Abstract

Studying how infectious disease spread and constructing simulation models can provide insights into how epidemics spread through populations helping us to develop effective strategies for controlling outbreaks, and reducing the number of people infected by diseases. Therefore, the goal of this project is studying the disease spread system under different simulated settings, and exploring the simulation model applications in the real-world diseases or other social behaviors that have the similar disease-spreading process.

Contents

1 Project Description

Understanding how infectious diseases spread can help us to develop effective strategies for controlling outbreaks, and reducing the number of people infected by diseases. Simulations can provide insights into how epidemics spread through populations and can incorporate various environmental factors. With simulation models, it is easier for researchers to evaluate the impact of different interventions and strategies.

In addition, the spread of information, cultural norms and other social behaviors can be conceptually modeled as a disease-spreading process. The similarity between the mathematical descriptions of disease-spreading and other phenomena let the epidemic modeling be widely researched and become a field crossing different disciplines.

2 Literature Review

Centers for Disease Control and Prevention (CDC) separates transmssions modes into two categories: (1) direct, such as direct contact and droplet spread and (2) indirect, such as airborne, vehicleborne and vectorborne. In Hiram, Petrizzelli, and Mazza's paper [6], they classified infectious diseases mainly into three categories in terms of the ways of infection. The first kind is airborne diseases, such as flu, Middle East Respiratory Syndrome, and COVID-19, for which the transmission is usually due to close physical interactions among people; the second kind is sexual-based diseases, such as AIDS, for which the transmission is due to a sexual interaction or blood-based infection; the third kind's transmission is vechicleborne and vectorborne diseases, which can be carried by contaminated surfaces of subjects in some cases, such as rhinovirus and SARS-COV-2, or more commonly, by insects such as mosquitoes. It should be noted that the three kinds of diseases are not mutually exclusive and modeling should be adjusted accordingly. These three classifications are reflected in the model as the difference in contact frequency and the number of edges in a network.

2.1 SIR and SEIR

Many models for the spread of infectious diseases in populations have been studied and applied to specific diseases. The susceptible/infected/recovered (SIR) model is one of the typical models that are commonly discussed when describing the behavior of epidemics. This model is appropriate for a rapidly spreading disease that confers immunity to its survivors. The paper from Side, Syafruddin and Salmi Md Noorani [13] applies the classical SIR model for the dengue fever disease, and finds it suitable for predicting the spread with data collected in South East Asia.

SEIR model is developed based on the SIR model with an extra parameter E indicating the fraction of exposed individuals who have been infected but are not yet infectious. Annas, Suwardi, et al's paper [1] ensures the numerical stability of the model and can be a reference model for the spread of COVID-19 with data collected in Indonesia.

2.2 Cellular Automata

Another discrete SIR-based model to simulate epidemic spreading is Cellular Automata, which is proposed by Stanislaw Ulam and John von Neumann in 1940s. Cellular Automata describes population as a grid of cells, with each cell being in one of the three states: susceptible, infected, or recovered. The state of a cell can affect its neighbourhood's states usually according to a fixed set of rules, otherwise with the updating rules following certain distributions, the model is extended to Stochastic Cellular Automata.

For sexualborne disease, Dos Santos, Maria and Courinho in their paper [2] found that cells infected by HIV organize themselves into spatial structures, and as a result the concentration of uninfected cells is decreased, which leads to AIDS. In Mo et al. work [10], they proposed a 3-dimensional Cellular Automata model representing a 3D single lymph node to model the acute phase, asymptomatic period and the AIDS phase of HIV infection. The 3D Cellular Automata performs more robust on parameters than the 2D model.

For insectborne disease, which is a subsidiary of vectorborne disease, Slimi et al. in their paper [14] modeled Chagas spreading by bugs in Middle and South America using Cellular Automata to develop a spatio-temporal description of the insects population dynamics at the scale of a village. By dividing each transition step to demography process and dispersal process that alternate over time, they proposed the concept of periodic spreadability to investigate temporal transmission risk of Chagas disease.

Gabric et al. [4] compared the efficiency of two vaccinations for using Stochastic Cellular Automata. They introduced two new compartments that are associated with the individuals vaccinated with the first and second doses, and hence a dynamic behaviour depends on the time between the doses. With this, they analysed three different scenarios of vaccination: unlimited doses, limited doses into susceptible individuals, and limited doses randomly distributed overall individuals.

2.3 Network Models

The original and simplest SIR model considers the population is fully mixed, which might be unrealistic in the real life. Thus, Newman [11] replaced the "fully mixed" aspect with a network of connections between individuals and provided exact solutions. Random networks with specified degree distributions have been widely studied. For example, Meyers et al. [9] extended the mathematics of contact network epidemiology, used random network with a Poisson degree distribution and a (truncated) power-law degree distribution, to provide new insight into the observed epidemiology of SARS. Newman [11] also extended his study to networks that represent structured populations, like a sexually transmitted disease. Similarly, Pastor et al. [12] also discuss a generalization of epidemic processes in complex, multi species reaction-diffusion processes, which is an area relevant in the analysis of epidemics in structured populations. Eames and Keeling [3]developed an intuitive mathematical framework to deal with the heterogeneities implicit within contact networks comparing with full stochastic simulations.

In the paper [6] by Hiram et al., they reviewed several epidemic models of spreading of infectious diseases regarding the spread of COVID-19, including compartmental models (SIR, SIRD, and SIRV) that assume contact structures being homogeneous, and contact-based model, a network model, that integrates a contact pattern based on temporal networks enabled by the data of cellular phones to the previous diffusion models. In the researches they reviewed, the contact-based model usually performs better. They also summarized several open-source or data-preloaded models including *Covasim* which is an open-source model developed to simulate coronavirus spreading, agent-based *Epigraph* and *COVID Spreading and Vaccination Model* by Hartnett et al..

Hartnett et al.[5] carried out the contact-based model mentioned above using mobile phone data to study the outbreak of COVID-19 in Oregon, Portland. They compared this model with a compartmental SEIR model and found that predictions based on the contact networks are more accurate and reliable. Their result also suggested that targeted vaccination of hub nodes in the network can accelerate epidemic control.

3 Conceptual Models

This section describes the conceptual simulation models we studied of the disease spread system. We created 3 types of models: SIR, Cellular Automata, and Network Model.

3.1 SIR and SEIR

3.1.1 SIR

SIR model uses $S * I$ to describe the encountering of susceptible population and infected population, which has a rate of β that result in transmission of the disease. Assume that recovering rate is γ and recovered population are immune to further infection. Therefore, β and its contribution in formula are our handles to fulfil our purpose of comparing models of infectious diseases with different transmission ways. We use *NHSN Standardized Infection Ratio* and https://en.wikipedia.org/wiki/Infection_rateas references.

3.1.2 SEIR

SEIR model assumes susceptible population encountered with infected population will not directly become infectious, but will first transform to the state of "Exposed" with the rate β and then to infectious state with a rate α, which describes the "incubation period" or "latent period" as it is usually called. This process α is the result of multiple factors including immune response and host susceptibility. We use https://en.wikipedia.org/wiki/Incubation_period as a reference for choosing α for diseases with different transmission agents.

3.1.3 SEIRS and Other Extensions

SEIRS model takes in consideration that for many diseases, the immunity of recovered population wanes over time, and allow a person in recovered state to return to susceptible state with a rate ξ.

1. SEIRS with Mitigation and Policy Parameter

   In this model, we use u to represent policy effects. An effective policy should be able to lower the expose or infection rate. Therefore, we replace β with $(1-u) * \beta$.

$$\begin{cases} \frac{dS}{dt} = -(1-u)\beta\frac{SI}{N} + \xi R \\ \frac{dE}{dt} = (1-u)\beta\frac{SI}{N} - \alpha E \\ \frac{dI}{dt} = \alpha E - \gamma I \\ \frac{dR}{dt} = \gamma I - \xi R \end{cases}$$

2. SEIRS with Dynamic Parameters In this part, we are considering the representing the processes of exposion, infection, and recovery with time-variant dynamic parameters $\beta, \alpha, \gamma, \xi$ and u instead of constant ones used in previous models. This will enable the model to capture environmental changes caused by the outbreak of an infectious disease that in turn affect transmission process. For example, since policy and mitigation requires time to act, the infection rate usually surge up given the current infected population and decrease after policy is taken; the recovery rate will drop if the infection is overwhelming and strain on the medical resources. The formula of dynamic parameters are decided based on historical cases and can be further improved with professional advices.

Some basic assumption we have that are going to be used in dynamic SEIRS models:

$$\begin{cases} \beta \propto S \\ \alpha \propto S, E \\ \gamma \propto \frac{1}{S} \\ \xi \propto t \\ u \propto I, E, \frac{1}{t} \end{cases}$$

3.2 Cellular Automata

Cellular Automata is a discrete model of a grid of cells, where each cell can be in a particular state. To simulate the epidemic, each cell represents a person and the state of each cell might be "susceptible", "infected", or "recovered" according to the classical SIR model.
In the Cellular Automata, at every step, the state of each cell is updated based on its neighbors' states and a set of rules. In the epidemic simulation, these rules can be based on factors such as the infection rate of the disease, the susceptibility of individuals to infection, and the parameters measuring interventions such as social distance, quarantine, wearing a mask, and vaccination.
In this project, we first implement a simple cellular model for disease spread assuming all people are fixed, and being infected or not is decided by their neighbors in a 2-D grid. According to the SIR model, the spread of disease from one cell to another is calculated based on contact with neighbors. Here for each pair, the probability of being infected by a neighbor is fixed. And we will consider the probability to be infected based on all eight neighbors for each cell. And during the given infectious period, the infected will spread to its neighbors with the same probability. Also, a recovered cell is no longer susceptible.
After finishing the simple implementation, we will add more details to our simulation. First, potential prevention measures such as practicing social distancing and wearing masks will be considered to lower the infection probability. Also, we will implement a model where individuals are likely to be infected again after recovery, which is possible for COVID-19. Overall, cellular automata will provide a flexible and powerful tool for simulating the spread of epidemics and understanding the factors that contribute to their spread.

3.3 Network Model

When using network dynamics for disease spread simulation, this project extends the SIR model. The Network SIR model is a powerful tool for studying the spread of infectious diseases in populations with complex social networks. Each person in a community is represented as a *vertex* in the network and each contact between two people is represented as an *edge* connecting their vertices. The number of edges emanating from a vertex, that is, the number of contacts a person has, is called the *degree* of the vertex. The simulation model assumes that each individual in the network can be in one of three states: susceptible (S), infected (I), or recovered (R).
The network SIR simulation model initials with a small number of infected individuals and simulates the spread of the disease over time. The model assumes that infected individuals transmit the disease to their susceptible (connected) neighbors with a certain probability. The probability of transmission depends on the number of infected neighbors of the infected individual, which is influenced by the network structure. The probability of recovery also depends on the parameters of the model, such as the duration of the infectious period.
For the simplified network SIR model, the mathematical expression of SIR part is the same as the original SIR that described above. The only different place is that, in the Network SIR model, the

transmission rate β is influenced by the network structure and the number of infected neighbors of each individual. The transmission rate can be calculated as:

$$\beta = \beta_0 \times \frac{k}{\bar{k}}$$

where β_0 is the base transmission rate, k is the number of infected neighbors of an individual, and $\bar{k}$ is the average number of neighbors in the network. This equation captures the idea that individuals with more infected neighbors are more likely to become infected, and that the transmission rate is higher in networks with higher connectivity.

4 Simulation Model

4.1 SIR and SEIR

4.1.1 Model Settings

To study the simulation of infectious disease given different transmission ways, we use SIR, SEIR, SEIRS, SEIRS with policy factors and Dynamic SEIRS models in this part. We specifically investigate three transmission ways: Airborne, Sextually-transmitted and Mosquito-borne.
For each transmission ways, to better simulate transmission routes, we choose three representative diseases that are known being dominated by each transmission way: Covid-19, HIV and Zika virus. We first apply SIR, SEIR, SEIRS, SEIRS with policy factors models to each diseases with SIR model as the performance baseline.
Each model has the same population size $N = 1000$, infected population $I_0 = 1$ and simulated time step $t = 1$ day. For Covid-19 and Zika virus, the simulated time frame is $T = 720$ days and for HIV, being more long-term disease, the time frame is $T = 3600$ days.

4.1.2 Parameters

The simulation of three infectious disease starts with constant parameters we described before. For dynamic SEIRS model applied to Covid-19, we have dynamic parameters:
$\beta(t)$: susceptible-to-exposed rate per person varied by t and $\frac{I}{N}$, for if the infected fraction is too large, the possibility of contact is increased.

$$\begin{cases} \beta = \min(0.9, \beta + 0.01), \text{ if } \frac{I}{N} > 0.5 \\ \beta = \beta, \text{ otherwise} \end{cases}$$

$\gamma(t)$: infectious-to-recovery rate per person varied by t and $\frac{I}{N}$, for effective treatment requires time to be developed, thus old methods should not be modeled as effective as customized ones, and if the infected fraction is too large, medical resources will be strained, which also lower the recovery rate.

$$\begin{cases} \gamma = 0.7\gamma, \text{ if } t \leq 90 \\ \gamma = 0.5\gamma, \text{ if } \frac{I}{N} > 0.1 \\ \gamma = \gamma, \text{ otherwise} \end{cases}$$

$u(t)$: policy effectiveness. Effective policy requires time to be designed and take place, also, as a epidemic going to its end, the policy will be treated with carelessness.

$$\begin{cases} u = 0, \text{ if } t \leq 60 \\ u = u, \text{ if } t > 60 \text{ or } \frac{I}{N} > 0.3 \\ u = 0.5u, \text{ if } t > 0.9T \end{cases}$$

4.2 Cellular Automata

4.2.1 Model Settings

The model will be implemented on a $N \times N$ grid, and for this paper, we choose N to be 40, which means there are 1,600 cells in total to represent individuals. Based on the SIR model, each cell will have 3 states, susceptible, infected, and recovered. Initially, we assumed that each cell has 99% to be susceptible and 1% to be infected, which is consistent with the other two models.
The model will be simulated daily. Each day, for a susceptible cell, the probability of being infected is based on its neighbors' states. If there are a total of k infected neighbors and the probability of being infected (PI) by one neighbor is p, then the overall probability of the susceptible cell being infected is:

$$P_{infected} = 1 - (1 - p)^k \tag{1}$$

For a recovered cell, the probability of being reinfected can be expressed similarly. Here we do not use p directly but use a reinfected probability p_r which should be much lower than p. And the overall probability to be reinfected given k neighbors are infected is:

$$P_{reinfected} = 1 - (1 - p_r)^k \tag{2}$$

And an infected cell will recover after a given recovery period, and it is infectious to its neighbors during this period.

4.2.2 Parameters

To simulate the spread of the epidemic and to observe the effect of potential prevention measures, the following parameters are used in our model.
N: this determines the size of the grid.
PI: the probability of infection for a suspicious individual, if the neighbor is an infected individual.
$recover\_day$: days an infected individual needed to recover.
PRI: the probability of reinfection for a recovered individual, if the neighbor is an infected individual.
sim_day: total simulation days.
$init\_PI$: the probability of an individual being infected on Day 1, used to initialize the simulation.

4.2.3 Model Output

For this simulation model, we will output the number of individuals in each state on each simulation day, as well as the state of the whole grid on the last day. And in the following part, we will show some simulation results with several sets of parameters.

4.3 Network Models

The network models attempt to characterize every interpersonal contact that can potentially lead to disease transmission in a community. The project extends the SIR model on different simulated networks: binomial network, the network with a Poisson degree distribution, network with a power-law degree distribution, Watts–Strogatz small-world network, and Barabási–Albert network with preferential attachment. Each network has the same population size ($N = 1000$) and similar average vertex degree k (around 10). And each network is applied on the same SIR model, with the transmission rate ($\beta = 0.3$), recovery rate ($\gamma = 0.1$) and 10 initial infected people.

4.3.1 Binomial Network

For each vertex under Binomial graph model (Figure 1), the network chooses each of the possible edges with probability $p = \frac{k}{N} = 0.01$. The binomial network assume each person could connect with every other people with the same probability, so the clustering coefficient of the network is relatively low, that is, there are rarely clusters in the community. We use this network model as a null model to compare against more complex, real-world networks.

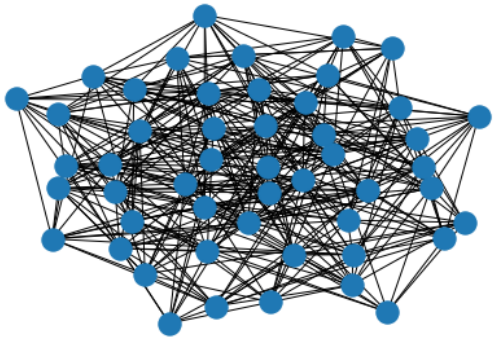

Figure 1: Binomial network example

4.3.2 Network with A Poisson Degree Distribution

The network with a Poisson degree distribution (Figure 2) is also used as a null model for comparison in this project. The network with the degree sequence that is generated from a Poisson distribution with parameter $\lambda = k$ (average degree is 10).

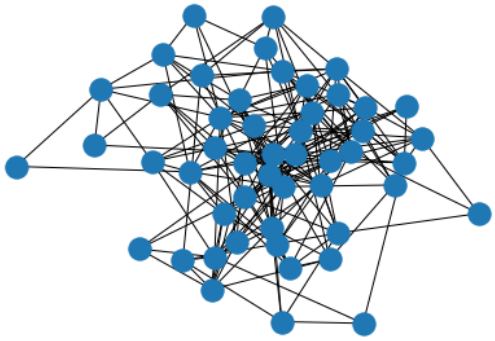

Figure 2: Network with A Poisson degree distribution example

4.3.3 Network with A Power-law Degree Distribution

This project also study the network with a truncated power-law degree distribution (Figure 3). This network has a heavy tail of superspreaders, which means a few vertexes having very high degree and many vertexes having low degree, as it shown in Figure 4. The superspreaders usually have a profound effect on outbreak patterns despite being few in number.

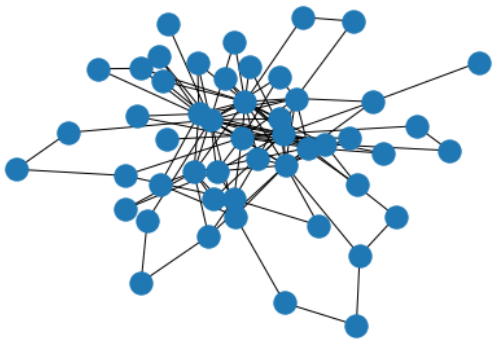

Figure 3: Network with A Power-law degree distribution example

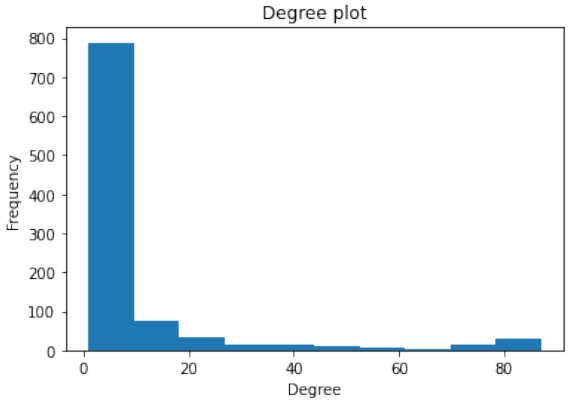

Figure 4: Degree histogram of the network with a Power-law degree distribution

4.3.4 Watts–Strogatz small-world network

A small-world network (Figure 5) is defined to be a network where the typical distance L between two randomly chosen vertexes (the number of steps required) grows proportionally to the logarithm of the number of nodes N in the network ($L \propto \log N$). In the network like this, the small world phenomenon of strangers being linked by a short chain of acquaintances. Small-world networks are defined with high-clustering coefficient, tending to contain cliques, meaning sub-networks which have connections between almost any two nodes within them. Also, in small-world network, most pairs

of nodes will be connected by at least one short path, which following from the defining property that the mean-shortest path length be small.

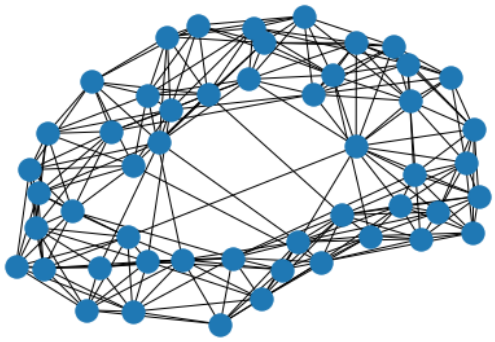

Figure 5: Watts–Strogatz small-world network example

4.3.5 Barabási–Albert network with preferential attachment

The Barabási–Albert network (Figure 6) is generated by using a preferential attachment mechanism. It incorporates two important general concepts: growth and preferential attachment. Growth means that the number of nodes in the network increases over time. Preferential attachment means that the more connected a node is, the more likely it is to receive new links. The degree distribution of Barabási–Albert network also follows a power-law (Figure 7).

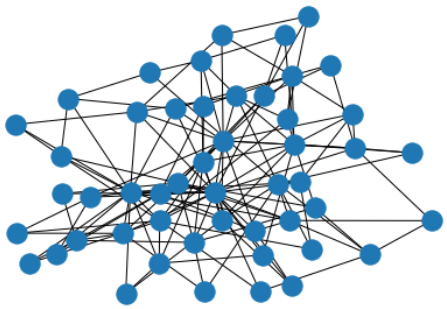

Figure 6: Barabási–Albert network example

5 Experimental Results and Validation

In this section, we construct simulators with each model in programming and simulate under different scenarios, then discuss the experimental results and validations.

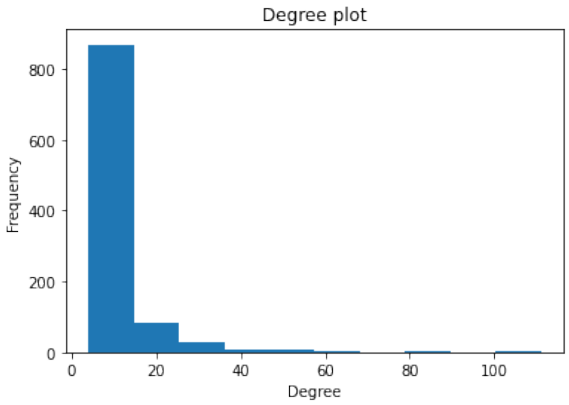

Figure 7: Degree histogram of the Barabási–Albert network

5.1 SIR and SEIR

5.1.1 Airborne: Covid-19

We refer to Kraus and Feuerriegel's paper [7] for deciding Covid-19 parameters and we have $\beta = 0.15, \gamma = 0.05, \alpha = 0.05, \xi = 1/180,$ and $u = 0.4$. Simulated results are shown in Fig.1.

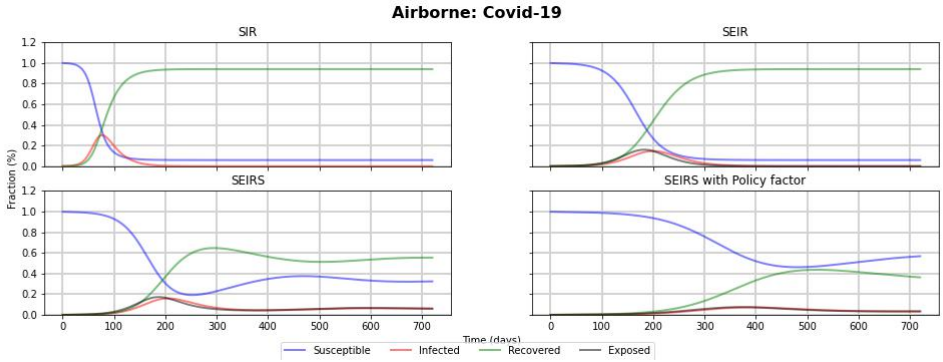

Figure 8: Airborne Disease

From the comparison plot of Figure 8, we can see that in SIR model, 80% of the population will be affected and ended up recovered, but the highest fraction of infected population at a given time stamp is less than 30%. In SEIR model and SEIRS model, the distribution of exposed population is left shifted and having higher peak comparing to infected population, which corresponds with the definition. Covid-19 has different strains and being recovered from one strain cannot protect one from catching other strains, and the vaccines have an effective protection for 6 months, therefore, SEIRS model is more logical to use. We can observe that in SEIRS model, susceptible and recovered population will not be stable after some time, but will start oscillation by first raising and dropping; exposed and infected population have thicker right tails comparing to SEIR model. And from SEIRS model with policy factor, we can see that if half of the population follows the policy and the policy is effective stopping people from getting exposed, the infected population will be greatly reduced

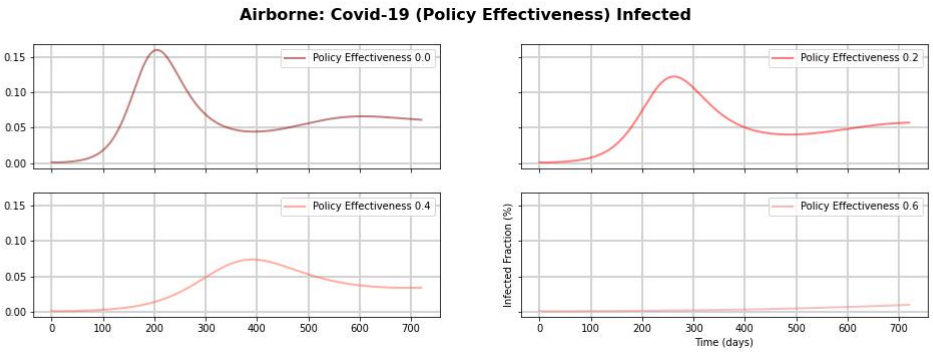

Figure 9: Airborne: Covid-19 Infection (Policy Effectiveness)

and the there will not be an outbreak.
Figure 9 shows when we vary the policy effectiveness u, the change of infected population over time. The higher the policy effectiveness, which is a product of policy quality and residents' obedience, the smaller the infectious fraction will be and the smoother the infectious line.
However, parameters are not reasonable to be constant in the real world. Considering the time policy and targeting treatment needs to happen and the possiblity of medical resources being strained out. We proposed a Dynamic SEIRS model for Covid-19. The result of Dynamic SEIR is shown in Figure 10, which has a more complex behavior and can capture the sharp increase of the outbreak.

5.1.2 Sexually Transmitted: HIV

We refer to https://www.hiv.gov/hiv-basics/overview/data-and-trends/statistics/ We expand the monitor time to 3600 days and set contact rate β as 12.6/100000 which is th estimated number of HIV infections in the U.S. was 34,800 divided by the whole population. Since there's no effective method to fully cure, we set $\gamma = 0$. But since ART treatment can prevent majority cases of HIV developing to AIDS, we set $\alpha = 0.2$. And $\xi = 0, u = 0.1$.
From Figure 11, we can see that SIR and SEIRs models are not the best way to simulate sexually transmitted diseases. For sexually transmitted diseases in general have extremely low contact rate β and the infection population is not outstanding given the whole population. But if we assume the infectious happens within a small group of people, the status of all being infected is unavoidable. Such unstable models cannot provide information and instruction on how to improve medication and policy.
Figure 12 shows when we vary the Contact Rate β to capture a wider range of contact distribution, the change of each fraction in spreading models over time. We can observe that even a little increase in contact rate will leads to sharp increase in overall infected population.

5.1.3 Mosquito-borne : Zika

We refer to https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5333268 and work from Lee et al.[8] for Zika features and we set $\beta = 0.071, \gamma = 1/7, \alpha = 1/10, \xi = 0,$ and $u = 0$.

From Figure 13, we can observe that for insects transmitted diseases, general SIR and SEIR models are also ineffective, for the population of agents should also be simulated along with the human(host) population. Also, for human population, pregnant population should be treated differently, for Zika virus cause serious birth defects. Both problems can be solved with a specialized

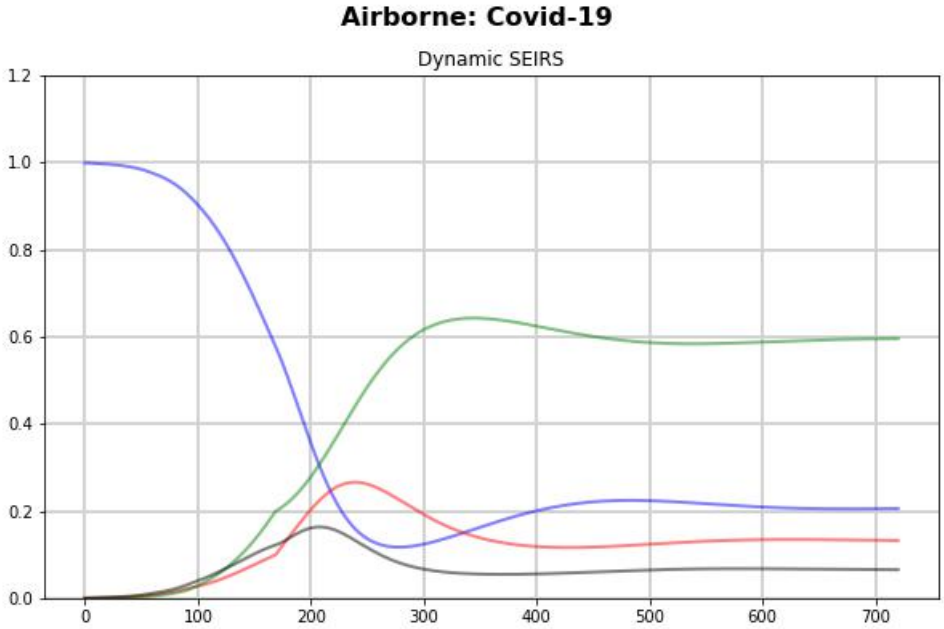

Figure 10: Airborne: Covid-19 Dynamic SEIRS

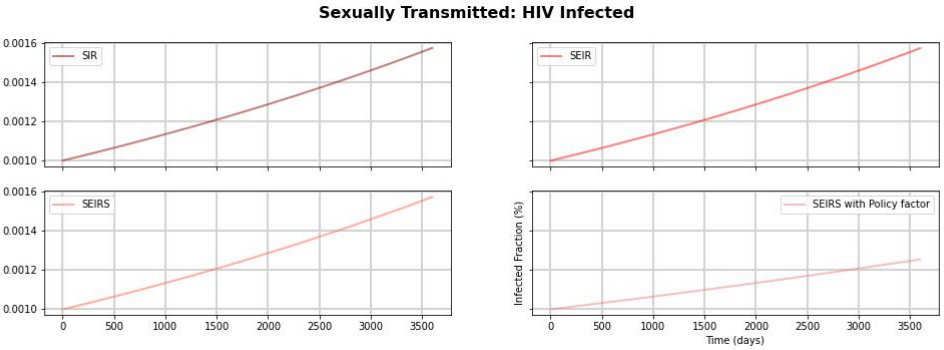

Figure 11: Sexually Transmitted Disease Infected

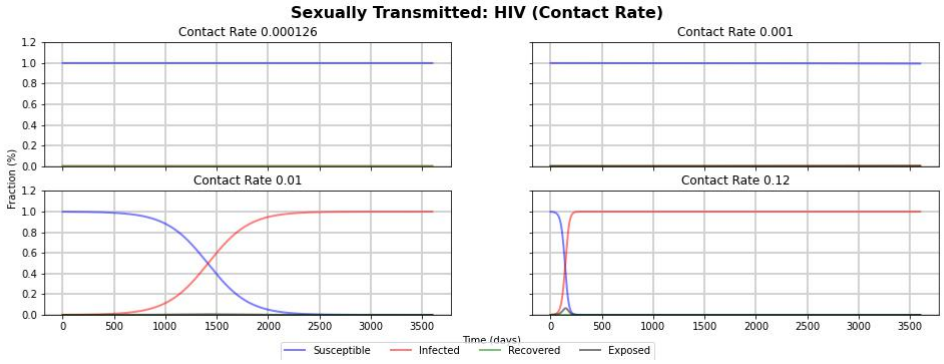

Figure 12: Sexually Transmitted Disease (Contact Rate)

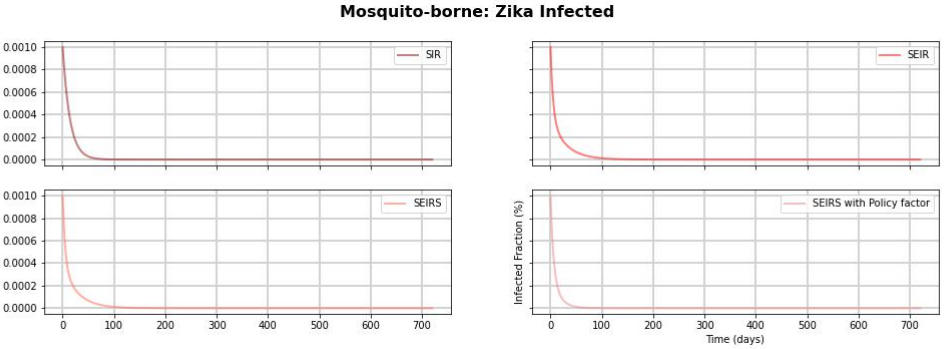

Figure 13: Mosquito-borne Disease Infected Fraction

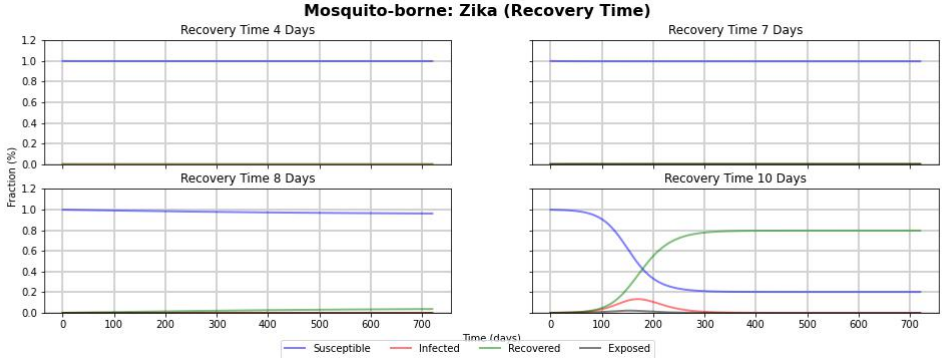

Figure 14: Mosquito-borne Disease (Recovery Time)

SEIR model.
Figure 15 shows when we vary the Recovery Rate γ based on the medical level of the local area, the change of each fraction in spreading models over time. We can observe that even a little increase in the required days to cure will leads to sharp increase in overall infected population.

5.2 Cellular Automata

For the simulations we conducted, some parameters are fixed. The grid is 40×40, the total simulation days are 100, and on the first day, each cell will have 1% to be infected.

5.2.1 Base Setting

In this setting, the probability of being infected by one neighbor is 0.3, and individuals will not be reinfected if recovered. And the recovery day is set to be 7. ($PI = 0.3$, $PRI = 0$, $recover\_day = 7$)

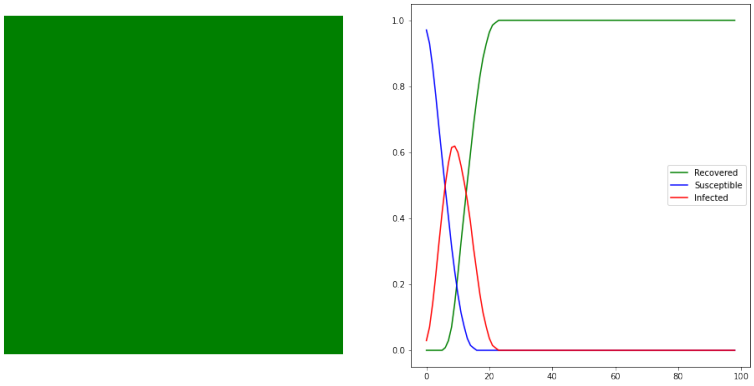

Figure 15: Base Model of CA

Here, green represents recovered individuals, red represents infected, and blue represents susceptible. We can see that after 100 days, all individuals are healthy and all of them are infected during the simulation.

5.2.2 Model considering Prevention Measures

In this setting, prevention measures such as social distancing, testing, and wearing masks are considered. And the probability of being infected by one neighbor is reduced to 0.1, and individuals will still not be reinfected if recovered. And the recovery day is set to be 7. ($PI = 0.1$, $PRI = 0$, $recover\_day = 7$)

This time, 99% individuals are infected during the process, but the peak of the infected curve is significantly reduced, which means the prevention measures can alleviate the pressure on the healthcare system.

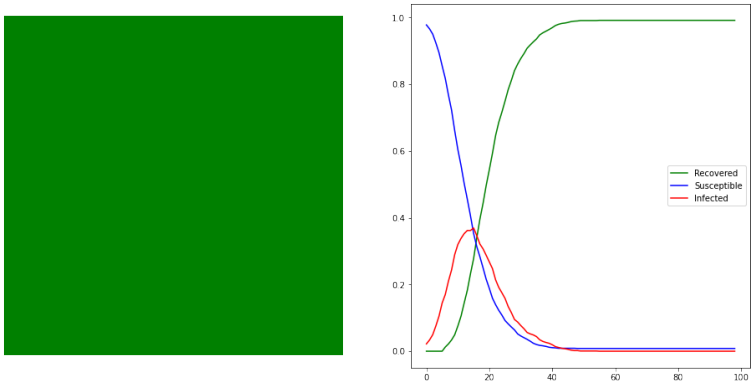

Figure 16: CA with Prevention Measures

5.2.3 Model with re-infection

We consider the re-infection this time based on the previous prevention measures. As being infected will boost immunity, we assume that the reinfection probability is 0.03. ($PI = 0.1$, $PRI = 0.03$, $recover\_day = 7$)

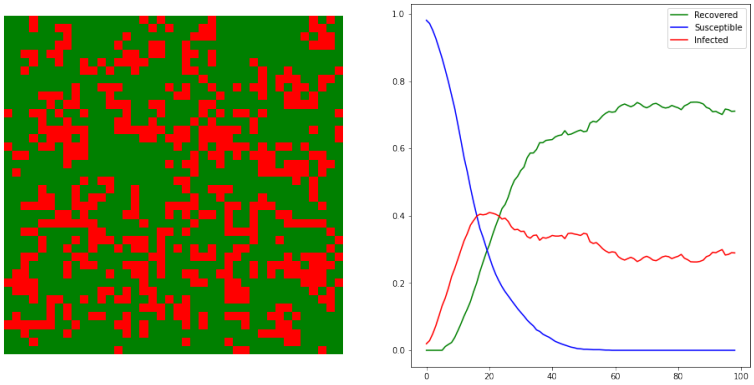

Figure 17: CA with Prevention Measures and Reinfection

Still, all individuals are infected during the simulation. And we can see that when considering

reinfection, the disease will continue spreading and is hard to be eliminated after 100 days.

5.2.4 Model with vaccination

Suppose vaccination can reduce the probability of being infected and reinfected, and we assume that it can reduce 40% based on the previous probability, which is reasonable based on the data for COVID-19. ($PI = 0.06$, $PRI = 0.018$, $recover\_day = 7$)

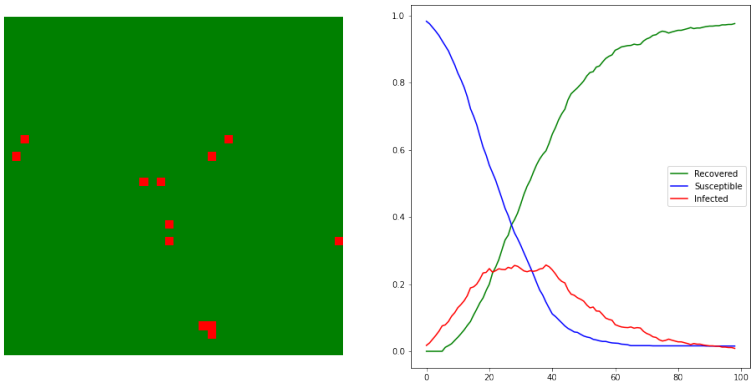

Figure 18: CA with Vaccination

This time, 98% individuals are infected during the process, and the infected curve goes down much

faster than the previous one, and it behaves more similarly to the one without reinfection, but with a smoother curve and can further reduce the pressure on the healthcare system.

5.2.5 Model with treatment

Suppose suitable treatment is discovered and the day needed for recovery is cut to 5. ($PI = 0.06$, $PRI = 0.018$, $recover\_day = 5$)

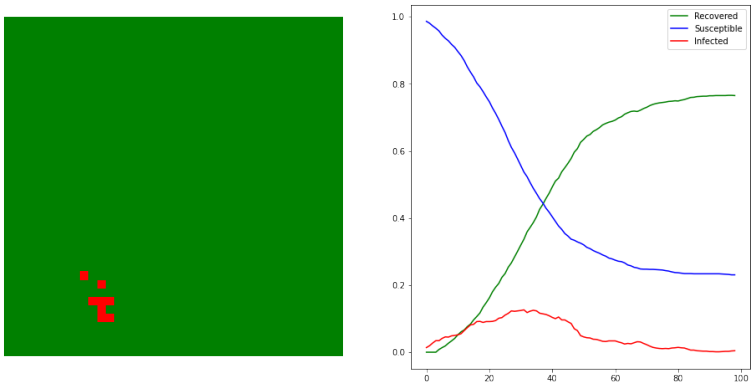

Figure 19: CA with Treatment

This time, 77% individuals are infected, which is much lower than the previous setting. And the

peak of infection is further reduced.

5.3 Network Model Comparison

Because of the experimental settings, the networks have the similar edge density and average degrees. Comparing network properties (Table 1) under this control, the Watts–Strogatz small-world network shows higher clustering coefficient than others. Comparing all random networks, the Watts–Strogatz small-world network model's behavioral is more closely resemble to the spread of infectious disease contact in the real-world. Typically, the clustering coefficient of around 0.2-0.4 and an average path length around 2-4 may be a good approximation of real-world social networks. However, whether the simulation model imitates the real-world depends on whether it capture the disease characteristics in spreading. For example, if it is more likely to have superspreaders for a disease, it is more appropriate to use a simulation network with power-law degree distribution.

Network	Edge density	Average short path length	Clustering	Average degree
Binomial	0.01	3.24	0.0095	10.07
Poisson	0.01	3.27	0.0088	9.86
Power-law	0.01	3.01	0.1819	10.05
Watts–Strogatz	0.01	3.85	0.3421	10.00
Barabási–Albert	0.01	2.98	0.0406	9.95

Table 1: Network properties

Looking at the Watts–Strogatz small-world network with different average shortest path length, when the length is short, a higher peak of infected population would be reached quicker. This result gives us the insight that isolation and increase the connect path length in the community is also a good prevention for infectious diseases.

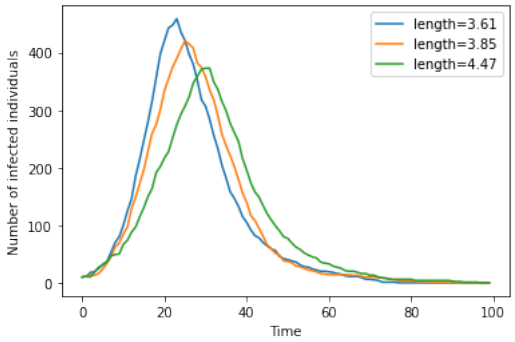

Figure 20: Infected individuals of Watts–Strogatz small-world model with different path length

Compare their SIR model simulation results with fully connected network which equivalent to the simple SIR model without network, finding that its infecting speeds is larger than all other network SIR models and it has higher peak value. The network models are closer to the reality, since it usually takes some time for the infected population getting to peak, and recovering as well.

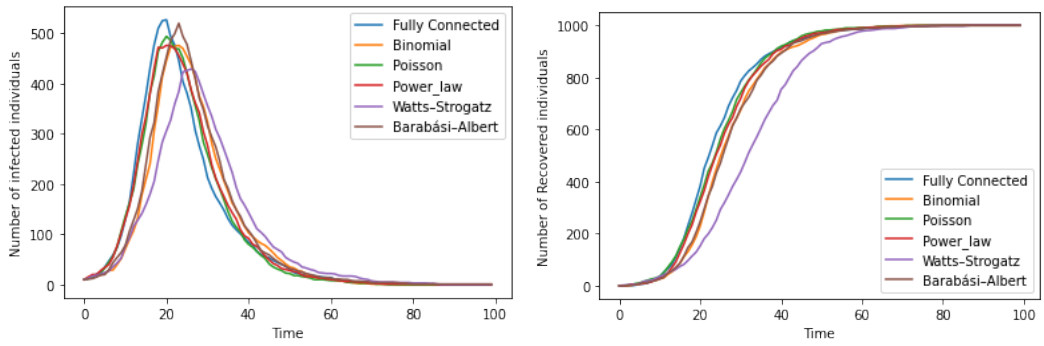

Figure 21: Infected (left) and recovered (right) individuals with different network models

6 Discussion and Conclusions

The difference in transmission ways affects the performance of traditional SIR and SEIR models. The "contact" happens during the Sextualy or insect-based transmission may not be fully captured by constant "contact rate". Airborne diseases are the most suitable to be simulated by SIR related models. The dynamic behavior of parameters should be further researched on besides current step functions. Deceased (natural and due to disease) and new-birth should also be taken into consideration if the disease is vital or the simulation time frame is long. There are a lot more details in each specific disease for the SIR related models to expand to cover.

The CA-based simulation involves infection and reinfection based on the state of individuals' neighbors, which is suitable for airborne diseases like COVID-19. And parameters about the infection and reinfection probability, as well as the treatment period length are included in our model, which offers a strong tool to investigate the effect of different disease control measures. Also, the concept of cellular automata allows more details to be considered for more complicated simulations, like more parameters or dynamic parameters. A potential improvement to the current simulation may be dynamic parameters, like the changing infection and reinfection rate, decreasing treatment time, or increasing vaccination rate. We can also construct a sparse grid where blank cells are allowed and individuals may move defensively to avoid contact with infected ones.

The network SIR model results exhibit that the network models are closer to the reality. It is more suitable than a fully connected network when simulating the disease spread and information spread. Especially, the Watts–Strogatz small-world model with the property of small average-shortest path length and high clustering coefficient is more closely resemble to the spread of infectious disease contact in the real-world. Also, the network model gives us the insight that isolation and increase the connect path length in the community is a good prevention for infectious diseases. We expect to extend our investigation of network-based model to the disease-spreading with more complex contact structure, like sexually transmitted disease.

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