University of Amsterdam

Project: Stochastic and Spatial Models

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1 Introduction

The spread of infectious diseases through populations is influenced by a range of factors, including individual behaviours, spatial relationships, and network connectivity. Modeling these dynamics is essential for understanding disease transmission patterns and for developing effective intervention strategies. The COVID-19 pandemic illustrates the importance of such models: originating in Wuhan, the virus rapidly spread through travel routes, impacting connected cities before escalating into a global health crisis (Centers for Disease Control and Prevention, 2022). This pattern underscores the need for models that account for networked connections between populations—linked by transportation systems such as airports and highways—when predicting the spread of infectious diseases.

In this context, spatial network models play a critical role. By representing cities as nodes and transportation routes as edges in a network graph, spatial models simulate the pathways through which an infectious agent can travel across different regions. Such models provide insights into how network characteristics, such as the density and connectivity of nodes, influence disease spread. Thus, one of the central questions addressed in this paper is: **"How do the characteristics of a transportation network influence the spread of a disease through a population?"**

Stochastic modeling further enriches this approach by capturing the randomness inherent in disease transmission events. Unlike deterministic models, which use average rates and assume homogeneous populations, stochastic models consider the probabilistic nature of infection and recovery events. This is particularly valuable for simulating real-world conditions where random variations—due to factors like population size, contact rates, and individual movements—can lead to significant fluctuations in infection dynamics. Stochastic models can thus illustrate complex behaviours such as stochastic resonance, where random fluctuations amplify the effects of periodic changes, and increased transients, which describe significant temporary deviations from expected disease prevalence. This leads us to a second research question: **"How do stochastic variations in disease transmission influence the overall spread and peak infection levels within a population, and under what conditions do these variations lead to significant deviations from deterministic model predictions?"**

This paper combines both spatial and stochastic modeling approaches to study disease transmission across a network and to evaluate strategies for reducing infection rates. In particular, we examine how network characteristics, such as connectivity and clustering, impact the speed and extent of disease spread. Additionally, we propose a targeted vaccination strategy aimed at lowering peak infection prevalence within a given network, thereby offering insights into more effective disease control measures.

To address these research questions, this report begins with an overview of the theoretical foundations of both stochastic and spatial models. We then describe the methods used to construct and analyse these models, followed by the results and a discussion of key findings. Finally, we conclude with recommendations for intervention strategies based on our modeling outcomes.

2 Theory

2.1 Stochastic Model

In epidemiological modeling, stochastic models are essential for capturing the inherent randomness and unpredictability of disease transmission within a population. Unlike deterministic models, which use average rates and assume a homogeneous population, stochastic models consider the discrete nature of individuals and the probabilistic events of infection and recovery. This approach is particularly important when dealing with small population sizes or early stages of an

outbreak, where random fluctuations can significantly impact disease dynamics.

Stochastic models account for the fact that transmission events occur at random times and are influenced by chance interactions between individuals. These models can simulate scenarios where, for example, an infectious individual might not infect anyone else before recovering, leading to the extinction of the disease, or where a single infected person could cause a large outbreak due to random contacts.

One widely used stochastic framework is the continuous-time Markov chain model (Anderson 2012), which defines the system's state transitions based on probabilistic rules. In the context of the classic SIR (Susceptible-Infected-Recovered) model, the stochastic version incorporates randomness in the transmission and recovery processes. The model is characterized by the following transitions:

$$\begin{aligned} \text{Infection:} \quad & S + I \xrightarrow{\beta} 2I \\ \text{Recovery:} \quad & I \xrightarrow{\gamma} R \end{aligned}$$

Here, S, I, and R represent the numbers of susceptible, infected, and recovered individuals, respectively. The parameters β and γ denote the transmission and recovery rates. The probability of an infection event occurring in an infinitesimal time interval dt is proportional to $\beta SI/N$, where N is the total population size. Similarly, the probability of a recovery event is proportional to γI.

To simulate the stochastic SIR model, algorithms like the Gillespie algorithm (Gillespie 1977) are employed. The Gillespie algorithm generates statistically correct trajectories of the system by calculating the time to the next event and determining which event occurs (infection or recovery) based on their probabilities. This method accurately reflects the randomness in both the timing and sequence of events.

Stochastic models can exhibit phenomena that deterministic models cannot capture, such as:

- **Stochastic Resonance** : This occurs when random fluctuations (noise) enhance the system's response to a weak periodic input, like seasonal variations in the transmission rate β. The interplay between noise and periodic forcing can lead to amplified fluctuations in infection numbers, potentially triggering outbreaks that deterministic models would not predict.
- **Increased Transients** : Stochastic effects can cause significant temporary deviations from the expected disease prevalence. These transients are pronounced when the basic reproduction number $R_0 = \beta/\gamma$ is near the critical threshold of 1, or in small populations where random events have a larger impact.

An example of stochastic resonance is observed when small periodic changes in β are insufficient to cause oscillations in a deterministic model but, when combined with stochastic fluctuations, result in noticeable cycles of infection. Increased transients might manifest as sudden spikes or drops in infection numbers due to random variations, which could have critical implications for public health responses.

Understanding how stochastic models relate to parameters like population size N and transmission rate β is crucial:

- **Population Size** (N) : Smaller populations experience greater relative fluctuations, making stochastic effects more significant. In such populations, chance events can lead to the disease dying out or causing unexpectedly large outbreaks.

- **Transmission Rate** (β) : Higher transmission rates increase the likelihood of infection events, potentially amplifying stochastic resonance and leading to more substantial transients.

In conclusion, stochastic models provide a more nuanced and realistic representation of disease dynamics by accounting for randomness and individual-level interactions.

2.2 Spatial model

In a spatial model of the spread of a disease through a population, the relationships between people need to be included in the research. The way a people in a population relate to each other can be shown in a network in graph representation. An example of such a graph representation is shown in Figure 1. Shown are nodes which represent individuals of the population and edges between nodes which represents whether the individuals share a relationship such that they could infect each other. For reference, during the covid-19 pandemic this could be defined by being within a radius of 1.5 meters of each other and therefore, being able to infect the other person.

Note that indirect infections are disregarded in this context. So infections like malaria that happen because of mosquito bites are also possible. But this paper does not go into this any further.

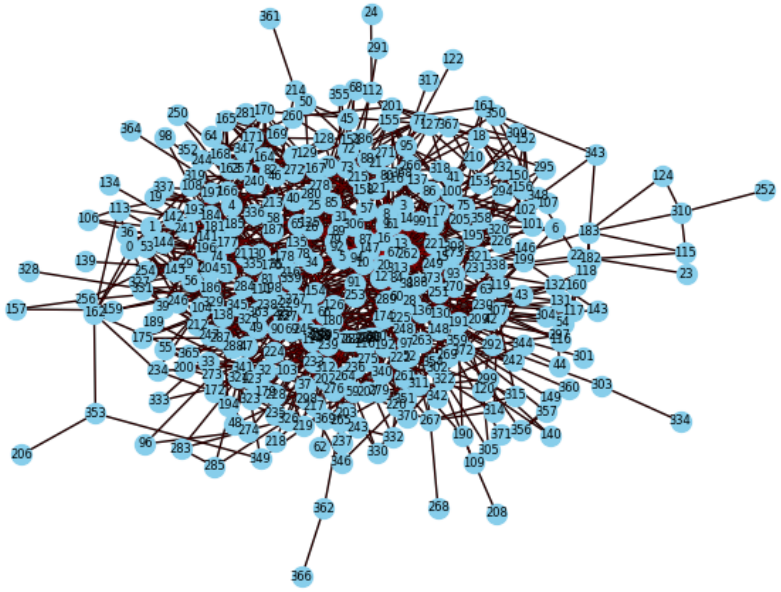


Figure 1: The sociopatterns data set as an example of a network with 374 nodes and 1265 edges

Nodes differ qualitatively based on some properties and of their position within the network. If a node have more edges, the probability of the node getting infected is higher than if it has less edges. If it does not have any edges (not shown in Figure 1, the probability of getting infected is even 0. Looking at the characteristics of the network itself some important characteristics are shown and explained in Table 2

These characteristics provide information about the infection risk within a network. Some have a positive relationship with infection, meaning that the higher the value of the character-

Characteristic	Explanation
Average degree	The average number of edges connected to a node.
Density	How dense the edges are in the graph.
Average clustering	Fraction of a node's neighbours that are connected.
Degree centrality	How many connections each node has.
Betweenness centrality	How many pairs of nodes communicate through a node.
Closeness centrality	How far a node is from the rest of the network.
Diameter	Longest shortest path between any two nodes.
Average shortest path length	Average steps along shortest paths between all node pairs.

Table 1: Relevant network characteristics influencing the spread of the disease.

istic, the higher risk of infection within the network. These are: 'average degree', 'density', 'average clustering', 'degree centrality' and 'betweenness centrality'. Others have a negative relationship with infection, meaning that the higher the value of the characteristic, the lower risk of infection within the network. These are: 'closeness centrality', 'diameter', 'average shortest path length'.

There have been different ways of constructing a network in a model. Three of these ways will be highlighted in this paper. Firstly, the Erdos-Reyni algorithm Erdios and Renyi 1959. This is an example of a random graph set by two parameters N and p. N represents the number of labelled nodes in the network and p represents the probability of any pair of nodes to be connected.

Secondly, the Watts-Strogatz algorithm Watts and Strogatz 1998. This is an example of a small world network set by three parameters N, k and p. N represents the number of nodes in the network. The N nodes are set in a topological ring structure. k is the number of closest nodes that each node is initially connected with. p is the probability of rewiring each edge. The reason that the Watts-Strogatz algorithm has been developed is because Watts and Strogatz noticed that networks were usually either completely random or completely ordered (Watts & Strogatz, 1998). They felt that there was a need for an algoirthm that could tune the degree of order or chaos. That is the function of the p-value since $p = 0$ is completely ordered and $p = 1$ is chaos.

Thirdly, the Barabasi-Albert algorithm Albert and Barabási 2002. This is an example of a scale free model set by two parameters N and m. N is the number of nodes which will be added to the network one by one. m is the number of edges that will be connected to a newly added node. In the Barabási-Albert algorithm, the newly added edges also connect to another node with a probability based on the degree of the nodes. As stated in the paper by Albert and Barabási: "the probability Π that a new node will be connected to node i depends on the degree k i of node i, such that

$$\Pi(K_i) = \frac{k_i}{\sum_j k_j}" \tag{1}$$

Albert and Barabási 2002

In terms of vaccination strategies, the proposition that the authors make is to vaccinate in order of the degrees of the nodes. Disregarding the ethical considerations of giving people with more friends better access to vaccinations, the authors believe that this strategy is better than a random vaccination strategy which will be referred to as the null strategy.

3 Methods

3.1 Stochastic model

3.1.1 SIR Baseline Model Description

The baseline SIR model divides the population into three compartments:

- **Susceptible (S)**: Individuals who can contract the disease.
- **Infected (I)**: Individuals who have the disease and can transmit it.
- **Recovered (R)**: Individuals who have recovered from the disease and are immune.

The model is governed by two key processes:

1. **Infection**: A susceptible individual becomes infected upon contact with an infected individual.
2. **Recovery**: An infected individual recovers and moves to the recovered compartment.

3.1.2 Gillespie's Algorithm Implementation

3.1.3 Defining Events and Rates

In Gillespie's Algorithm, we simulate the system as a series of discrete events occurring at random times. For the SIR model, we define two events:

1. **Infection Event** ($S \to I$)
   - **Rate** (a_1): The rate at which susceptible individuals become infected is proportional to the number of susceptible and infected individuals:

     $$a_1 = \beta \frac{S \cdot I}{N}$$

     where:
     - β is the transmission rate.
     - N is the total population size.
2. **Recovery Event** ($I \to R$)
   - **Rate** (a_2): The rate at which infected individuals recover:

     $$a_2 = \gamma I$$

     where:
     - γ is the recovery rate.

3.1.4 Algorithm Steps

The simulation proceeds as follows:

1. **Initialization**:
   - Set initial conditions: $S = N - 1$, $I = 1$, $R = 0$.
   - Set initial time $t = 0$.
2. **Event Selection and Time Advancement**:
   - Calculate the total rate: $a_0 = a_1 + a_2$.
   - Generate two random numbers r_1, r_2 uniformly distributed in $(0, 1)$.
   - Compute the time until the next event:

     $$\Delta t = -\frac{\ln(r_1)}{a_0}$$

     Update the time: $t = t + \Delta t$.
   - Determine which event occurs:
     - If $r_2 < \frac{a_1}{a_0}$, an infection event occurs.
     - Else, a recovery event occurs.
3. **State Update**:
   - Update S, I, R based on the event.
   - Record the new state and time.
4. **Termination**:
   - Repeat steps 2-3 until $t \geq t_{\max}$ or $I = 0$.

3.1.5 Controlling Noise Level (Bonus)

The noise level in the stochastic simulation can be controlled by adjusting the population size N. A larger N reduces the relative impact of stochastic fluctuations due to the law of large numbers, making the system behavior closer to the deterministic model. Conversely, a smaller N increases stochastic effects.

3.1.6 Comparison with Deterministic ODE Model

The deterministic SIR model is defined by the following ODEs:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta \frac{S \cdot I}{N} \\
\frac{dI}{dt} &= \beta \frac{S \cdot I}{N} - \gamma I \\
\frac{dR}{dt} &= \gamma I
\end{aligned}$$

We solve these equations numerically using the same initial conditions and parameters as the stochastic model to facilitate comparison.

3.2 Spatial model

In order to answer the research question, a model was made using Python version 3.12.3. In order to make a network, including the Barabasi-Albert, Watts-Strogatz and Erdos-Reyni networks, the python library networkx has been used. Ndlib is used for modeling the spread of the disease through the network. Scipy is used for statistic testing.

In an effort to be able to compare three different network algorithms equitably, the networks were made with an approximately equal number of nodes and edges as found in the sociopatterns data set, with 374 nodes and 1265 edges. For the Erdos-Reyni algorithm $N = 374$ and $p = 0.018$. For the Watts-Strogatz algorithm $N = 374, k = 7$ and $p = 0.55$ and for the Barabasi-Albert algorithm $N = 374$ and $m = 3$.

Concerning the vaccination strategy there are a few rules that have to be obeyed. Firstly, there is a finite x amount of vaccinations per iteration (the options are 1, 3, 5 or 10). Secondly, a maximum testing capacity of 200 was provided and thirdly, the test have a variable testing accuracy of 0.5, 0.75 or 1 respectively. The suggested strategy was for every iteration to rank-order the group of people who have not been vaccinated yet by degree from high to low. Now the people with the highest degrees are tested one by one until the tests had shown x amount of susceptible people, with x equal to the maximum allowed number of vaccinations per iteration. Then, the x amount of susceptible people would be vaccinated. The null strategy would be to randomly select x people from the group of people who have not been vaccinated yet. Statistics testing for the vaccination strategies will be done. If the infected curves for both the null strategy and the proposed strategy conform parametric testing, a paired samples t-test will be performed. If they don't conform, the Wilcoxon signed-rank test will be performed.

4 Results/Discussion

4.1 Stochastic model

4.1.1 Variability in Stochastic Simulations

We ran 100 stochastic simulations and plotted the number of infected individuals over time for each simulation, along with the deterministic solution and the mean of the stochastic simulations. The results are shown in Figure 2.

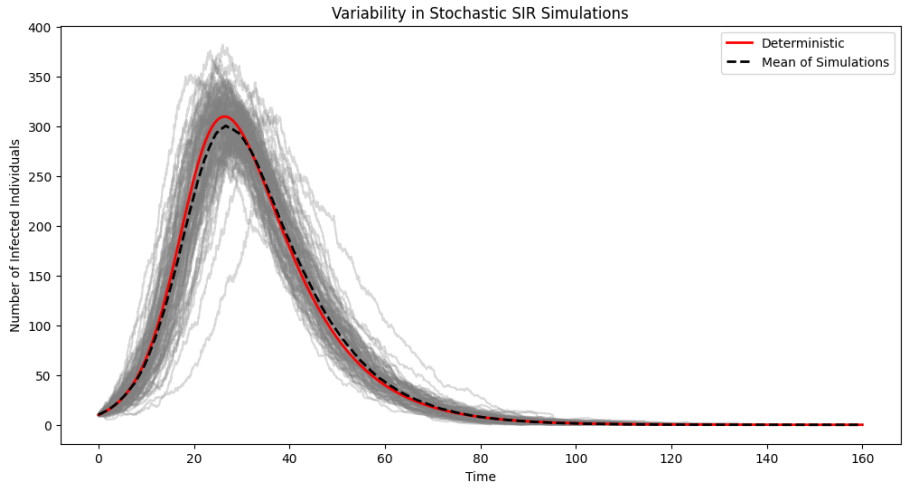


Figure 2: Variability in Stochastic Simulations

Observation: The stochastic simulations exhibit significant variability, especially around the peak of the epidemic. This variability arises due to the random nature of infection and recovery events in the stochastic model. The mean of the stochastic simulations closely follows the deterministic solution, highlighting that the deterministic model represents the average behaviour of the system.

4.1.2 Negative Covariance Between S and I

We computed the covariance between the number of susceptible and infected individuals over time across the simulations. The results are shown in Figure 3.

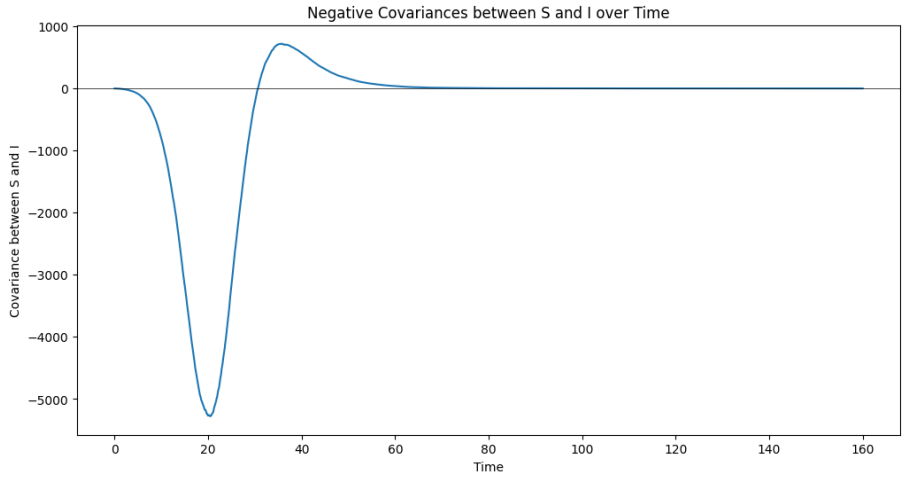


Figure 3: Negative Covariance between S and I

Observation: The covariance between S and I is negative during the course of the epidemic. This indicates that when the number of susceptible individuals is higher than average, the number of infected individuals tends to be lower, and vice versa. This negative covariance is a hallmark of the stochastic SIR model and is not captured in the deterministic model.

4.1.3 Increased Transients

We compared the time to reach peak infection in the stochastic simulations and the deterministic model. The results are shown in Figure 4

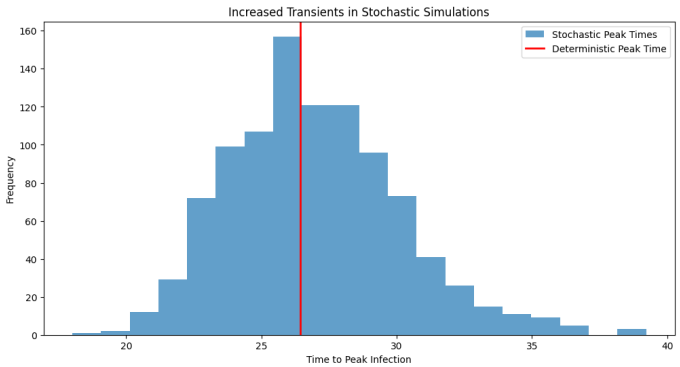


Figure 4: Increased Transients in Stochastic Simulations

Observation: The stochastic simulations show a spread in the time to reach peak infection,

indicating increased transients compared to the deterministic model. The deterministic model predicts a single peak time, whereas the stochastic model captures the variability in epidemic progression due to random events.

4.1.4 Stochastic Resonance

This graph below illustrates stochastic resonance, where initial conditions for the stochastic simulation were taken from a deterministic steady-state solution. Starting from this equilibrium, the stochastic model exhibits periodic fluctuations in infection levels due to the interplay between random noise and a weak periodic forcing applied to the transmission rate. Unlike the deterministic model, which remains stable at equilibrium, the stochastic simulation shows amplified oscillations, highlighting the resonance effect where random fluctuations synchronize with the periodic input, causing larger-than-expected variations around the steady state.

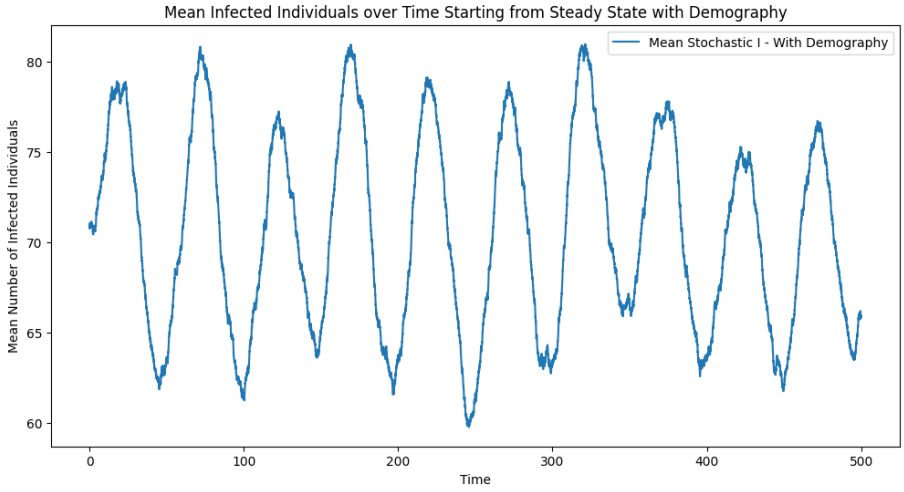


Figure 5: Fluctuations of a stochastic model, around an initial steady state equilibrium

Observation: The stochastic simulations display synchronization with the periodic changes in the transmission rate due to stochastic resonance. This effect is more pronounced in the stochastic mode

4.1.5 Extinction Simulations

Figure 6 above shows the extinction probability of an infectious disease as a function of the basic reproduction number, R_0, for different population sizes. Extinction probability here represents the likelihood that the disease dies out without reaching endemic levels within the population. The results highlight the critical role of population size in determining the extinction dynamics of the disease.

For smaller populations (e.g., $N = 500$ and $N = 1000$), the extinction probability remains close to 1 across a range of R_0 values, indicating a high likelihood that random events will lead to the disease's extinction, even when R_0 exceeds 1. This effect is primarily due to stochastic fluctuations, which have a stronger influence in smaller populations and increase the chances of disease extinction.

As population size increases, the extinction probability begins to show more sensitivity to changes in R_0. For instance, at $N = 5000$ and $N = 10000$, there is a noticeable dip in extinction probability around $R_0 = 1.2$. This dip suggests a transition point where the disease's persistence becomes more likely due to a higher effective transmission potential, making it less susceptible to

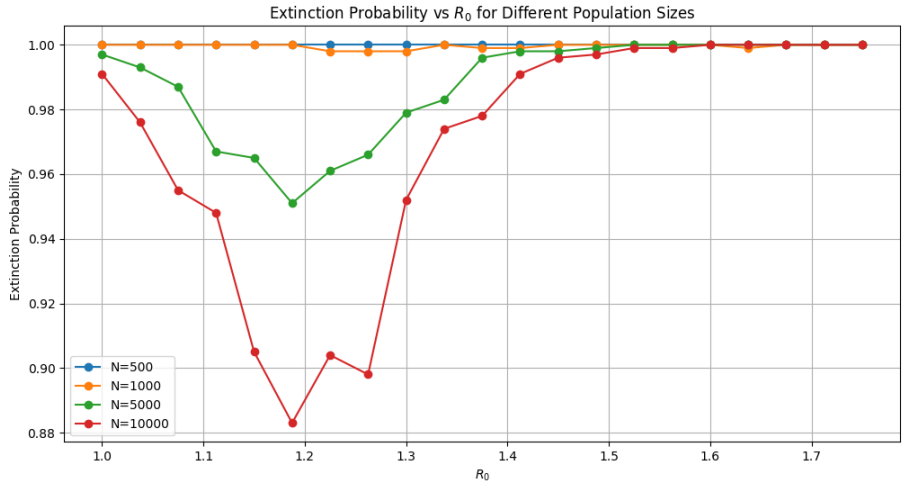


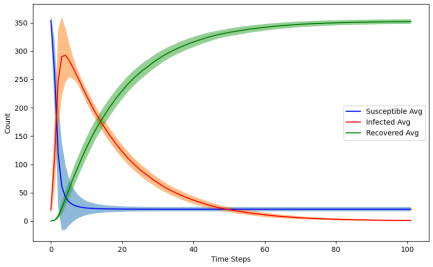
Figure 6: Extinction probability for differing values of R_0.

stochastic extinction. However, as R_0 increases further, extinction probability rises back to near 1, indicating that with sufficient control measures (reducing effective R_0), even large populations are prone to disease extinction.

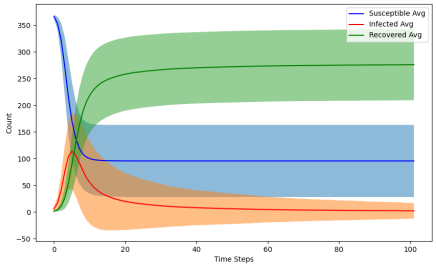
Overall, these results underscore the interplay between R_0 and population size in determining disease dynamics. Smaller populations are inherently more vulnerable to stochastic extinction events, while larger populations require a higher R_0 for the disease to overcome random fluctuations and persist. This finding has implications for public health strategies, as it suggests that interventions reducing R_0 could effectively induce extinction, especially in smaller or isolated populations.

4.2 Spatial model

Looking at the sociopatterns data set as the network and then being able to answer the research question, first a good combination of β and γ need to be used. If this combination of disease parameters varies by network type, the experiment would not be fair. In an effort to find appropriate values, the values of β and γ were varied. Then for some value of β, het mean and standard deviation was taken of the varying γ-values and plotted in Figure 7a. This was done vice versa in Figure 7b



(a) With $\gamma = 0.0595$ and values of β varying between 0.1 and 3.



(b) With $\beta = 0.245$ and values of γ varying between 0.01 and 1.

Figure 7: Mean and standard deviation of the sociopatterns data set network.

Looking at these figures it was decided for the answer to the research question to set the

value of β to 1.5 and the value of γ to 0.3.

In an effort to answer the research question, hundred networks each were made based on the algorithms of Barabasi-Albert, Watts-Strogatz and Erdos-Reyni. These networks had some characteristics where we show the mean and standard deviation from in Figure 8. Note that in an effort to be able to make an appropriate comparison, a goal in the setting of the parameters was to show an approximate equal number of edges is approximately acquired. In the results, significant differences between these characteristics will be made by comparing the means and standard deviations statistically. The authors felt that no statistics testing was needed because the differences between the values were either non-existent or notably different.

	Model	Erdos-Reyni	Watts-Strogatz	Barabasi-Albert
Nr_of_edges	mean	1259.280000	1.122000e+03	1.113000e+03
	std	35.652385	0.000000e+00	0.000000e+00
Average_degree	mean	96.617059	9.625000e+01	9.622594e+01
	std	0.095327	0.000000e+00	0.000000e+00
average_clustering_coefficient	mean	0.017291	4.475106e-01	6.380806e-02
	std	0.002891	1.210037e-02	1.060950e-02
density	mean	0.018054	1.608579e-02	1.595676e-02
	std	0.000511	0.000000e+00	0.000000e+00
average_degree_centrality	mean	0.018054	1.608579e-02	1.595676e-02
	std	0.000511	3.486925e-19	6.973851e-19
average_betweenness_centrality	mean	0.006211	1.105874e-02	5.705283e-03
	std	0.000119	3.379267e-04	9.754713e-05
average_closeness_centrality	mean	0.302506	1.968716e-01	3.234413e-01
	std	0.004083	4.677299e-03	3.920231e-03
diameter	mean	6.107692	9.540000e+00	5.030000e+00
	std	0.312404	6.422813e-01	1.714466e-01
average_shortest_path_length	mean	3.315291	5.113851e+00	3.122365e+00
	std	0.047306	1.257087e-01	3.628753e-02

Figure 8

Table 2: Network Characteristics for Different Algorithms

Algorithm	N	p	k/m
Erdos-Renyi	374	0.018	–
Watts-Strogatz	374	0.1	$k = 7$
Barabasi-Albert	374	–	$m = 3$

The spread of the disease through the populations made by the three different algorithms is shown in Figure 9. As can be seen in the figure, the Erdos-Reyni and Barabasi-Albert algorithms yield indistinguishable results. However, the Watts-Strogatz algorithm shows a delay (the peak of infected individuals occurs at a later moment in time than the other infected-curves) and a lower peak value.

Looking at the differences in characteristics in Table 2 between the Watts-Strogatz algorithm and the other two, the conclusion of this result is that the average clustering coefficient is significantly higher than in the other algorithms. This is not surprising when looking at how the algorithms work. Because the p-value is low, this means that most of the edges in the Watts-Strogatz network are connected with neighbours that are positioned close by. This means that the structure is quite ordered as can be seen in Figure 10. This orderedness can also be seen in

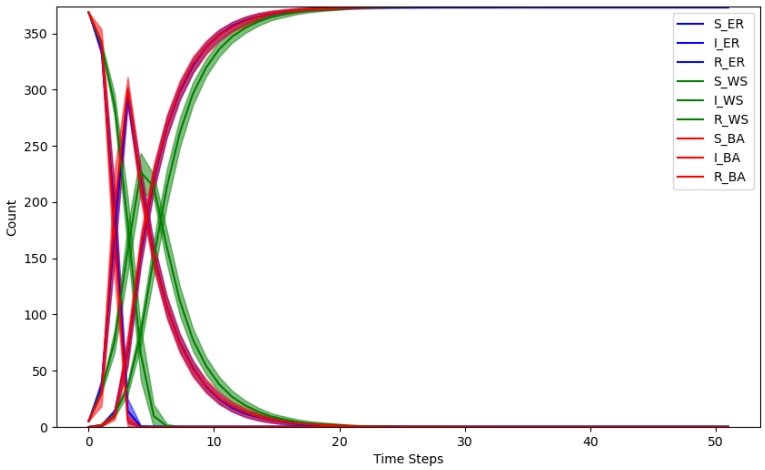


Figure 9: The spread of the disease through the population based on the three algorithms Erdos-Reyni (ER), Watts-Strogatz (WR) and Barabasi-Albert (BA). p-value of the rewiring of the WR algorithm was 0.1. For $p = 0.55$ or $p = 1$, the spread was indistinguisable from the other networks. With $\beta = 1.5$ and $\gamma = 0.3$

the 'average shortest path length', the 'average betweenness centrality' and the 'diameter' which are all significantly higher for the Watts-Strogatz network as compared to the other networks. The 'average closeness centrality' is significantly lower for the Watts-Strogatz network which is in alignment with the above. Given these characteristic differences, it is not surprising that the peak of infected people is delayed. Because the disease has to 'make more jumps' to go through the entire network. The fact that the peak is lower seems to be a direct effect of the delay in the network. It takes longer to go from one node to the other, so by the time the disease reached the end-node of a certain path, the begin-node of that path is already recovered.

Looking at the effects of the initial percentage infected it is seen that the WR network differs quite a lot from the other two networks when there are five individuals initially infected (Figure 10. In Figure 11 it can be seen that the difference between the WR network compared to the other networks decreases.

With regards to the vaccination strategy on the sociopattern data set network, a compar-

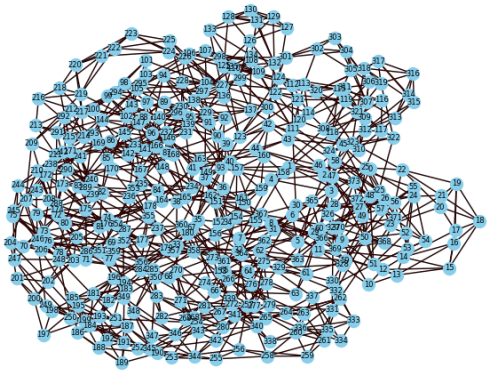
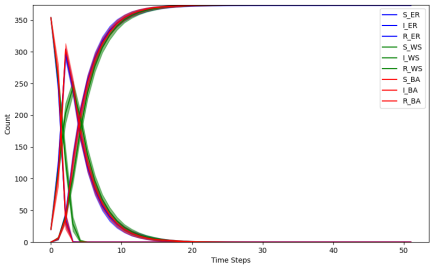
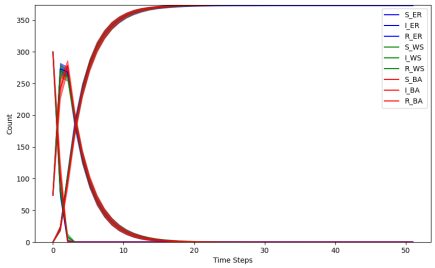


Figure 10: The graph representation of the Watts-Strogatz network with $N = 374, k = 7$ and $p = 0.1$ and 5 individuals initially infected.



(a) Initial number of infected individuals = 20



(b) Initial number of infected individuals $\approx$ 75 individuals (20%).

Figure 11: Mean and standard deviation of the three types of networks with $\beta = 1.5$ and $\gamma = 0.3$.

ison was made between a random vaccination strategy and a proposed strategy. The results of these strategies are shown in Figure 12. This figure shows a selection of the plots made. For the rest of the plot, the authors refer to the provided jupyter notebook. The figure shows that the curve does not show a lot of difference with 1 vaccination per timestep. Also, the testing accuracy does not impact the curve very much when there is only 1 vaccination per timestep, although the mean peak is lower and the standard deviations have less overlap. So this might point to a notable difference. The figure shows a more notable difference between the null strategy and the vaccination strategy with 5 vaccinations per timestep. As can be seen, this difference is notably more pronounced when the testing accuracy is 1 as compared to 0.5. This trend still holds when the vaccinations per timestep are 10: The mean peak infection is lower with more vaccinations per timestep and the mean peak infection is lower as the testing accuracy increases.

Statistics testing was used to find out whether the differences between the Null strategy and the proposed strategy are significant. It is concluded that in some instances, the paired samples t-test was acceptable and significant. For the reporting of the statistics testing, the authors again refer to the jupyter notebook provided with this paper. In all other cases, the Wilcoxon signed-rank test was performed and significant. For the reporting of the statistics testing, the authors again refer to the jupyter notebook provided with this paper.

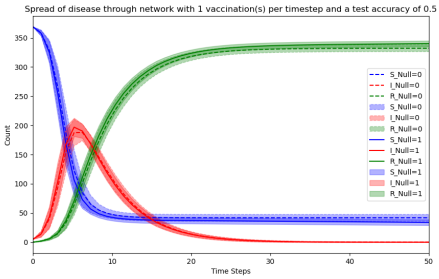
Considering these results, the authors conclude that their proposed vaccination strategy makes for a significantly lower peak infected proportion of the population than a random vaccination strategy.

5 Conclusion

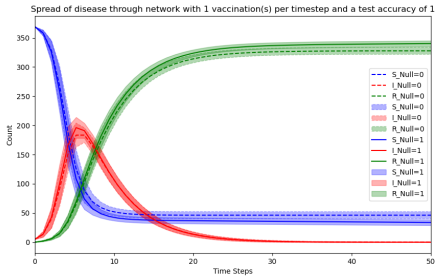
This paper attempted to answer the research question: "How do network characteristics influence the spread of a disease through a network?" It is concluded that the spread of a disease through a network is mostly affected by the network's degree of ordered-ness. This can be expressed by different network characteristics, such as an increased 'average shortest path length,' 'average betweenness centrality,' and 'diameter,' or a decreased 'average closeness centrality,' all of which contribute to a lower peak infection proportion during the epidemic.

Another goal for this paper was to design a vaccination strategy that outperforms a random vaccination approach. The proposed vaccination strategy effectively targeted key nodes based on network characteristics, leading to a significantly lower peak infection proportion than the random strategy.

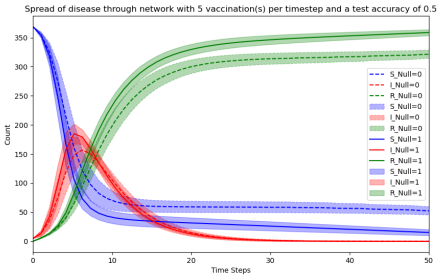
The analysis of the stochastic simulations provided further insights into the dynamics of dis-



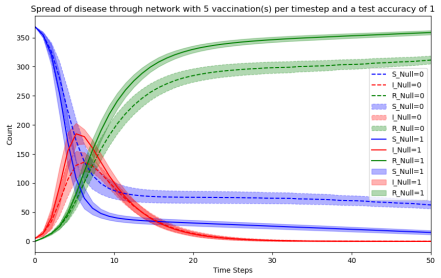
(a) Nr vacc = 1, test acc = 0.5



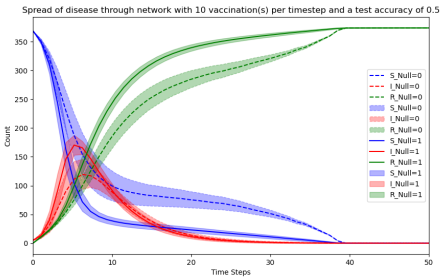
(b) Nr vacc = 1, test acc = 1



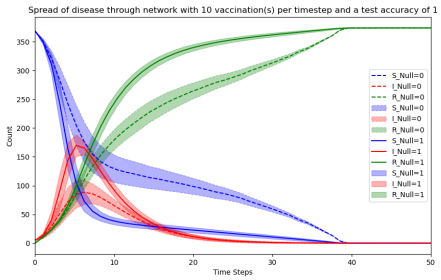
(c) Nr vacc = 5, test acc = 0.5



(d) Nr vacc = 5, test acc = 1



(e) Nr vacc = 10, test acc = 0.5



(f) Nr vacc = 10, test acc = 1

Figure 12: Mean and standard deviation of the three types of networks with $\beta = 0.3$ and $\gamma = 0.2$.

ease spread, emphasizing the impact of random variations in disease transmission on infection peaks and overall spread. Unlike deterministic models, which predict a smooth infection curve, the stochastic simulations demonstrated the phenomenon of stochastic resonance, where random fluctuations amplify periodic changes in transmission rates, causing larger-than-expected infection peaks. Additionally, increased transients were observed, particularly in smaller or highly connected populations, where random events could cause rapid and significant deviations from expected infection levels. This finding suggests that in real-world scenarios, stochastic effects can lead to unpredictable spikes in infection rates, reinforcing the need for adaptable and responsive intervention strategies. Together, these insights underscore the importance of incorporating stochastic modeling into network-based epidemic models to better anticipate and manage the dynamics of infectious disease spread.

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