Simulation of SIS model over networks

Complex and Social Networks (CSN)

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1 Introduction

The study of infectious disease spread in complex networks has been a subject of considerable interest in the field of network science. Understanding how diseases propagate through populations is crucial for designing effective prevention and control strategies. In this context, the Susceptible-Infective-Susceptible (SIS) model provides a valuable framework for simulating the dynamics of disease transmission within a network.

The SIS model captures the essential features of an infectious disease by considering nodes in a network as either susceptible to the disease or infected. Susceptible nodes can become infected based on specified transmission probabilities, while infected nodes have the potential to become susceptible again. This model allows us to explore the temporal evolution of the disease within different network structures, providing insights into the factors influencing epidemic outcomes.

This laboratory project aims to investigate the spreading of a disease in the SIS model across various types of networks, namely Erdos-Renyi [3] random graphs, scale-free networks following Barabasi-Albert [1] preferential attachment, small-world networks based on the Watts-Strogatz [4] model, fully connected networks (complete graphs), and tree structures. Through systematic simulations, we aim to compare the dynamics of disease spread, emphasizing the impact of network topology on epidemic behavior.

The simulation parameters include the recovery rate (γ), the infection rate (β), and the initial fraction of infected nodes (p_0). By varying these parameters and conducting simulations on different network types, we seek to identify patterns in disease propagation and assess the resilience of each network to epidemic outbreaks.

Additionally, we plan to validate our simulations against theoretical predictions, particularly examining the epidemic threshold for each network type. The work by Chakrabarti et al. (2008) [2] has forecasted an epidemic threshold of $1/\lambda_1$, where λ_1 is the leading eigenvalue of the adjacency matrix of the network. Our simulations will explore whether the observed epidemic behavior aligns with this theoretical prediction.

The structure of this report is organized as follows: Section 2 provides a detailed overview of the results obtained, Section 3 includes a discussion and a summary conclusion on the findings, while Section 4 outlines the methodology for simulations.

2 Results

In this section, we present the results of simulating the spreading of a disease in the SIS model on various network types. As mentioned both in Sections 1 and 4, Erdős-Rényi random graphs, Barabási-Albert scale-free networks, Watts-Strogatz small-world networks, Fully Connected networks, and Trees were considered. Each network type was simulated under specific parameter values for the infection rate (β), recovery rate (γ), and initial fraction of infected nodes (p_0). Lastly, two distinct tasks were completed as part of the specific laboratory: disease spread analysis in different networks for fixed values of (β), (γ) and (p_0), and disease spread analysis based on different threshold values for each network. All the details about the performed methodology for obtaining the results presented in the following subsections are included in Section 4.

2.1 Disease Spread Analysis in Different Networks

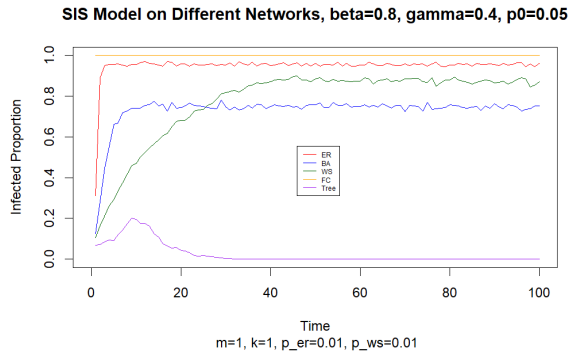
To begin with, in order to calculate the infected proportion of nodes over time for each network, the methodology explained in Section 4.2 was followed. The results for both parameter set 1 (Section 4.2) and 2 (Section 4.2) are included in Figure 1. Moreover, it is crucial to mention here some of the network's characteristics, calculated after the simulation of the disease spread. Basic information about each network is presented in Tables 1 and 2.

Name	λ_1	$Epid_{threshold}$	m	k_{ws}	p_{ws}	p_{er}
Erdős-Rényi (a)	11.18073	0.08943957	-	-	-	0.01
Barabási-Albert (a)	5.873073	0.1702686	1	-	-	-
Watts-Strogatz (a)	2.271662	0.4402063	-	1	0.01	-
Erdős-Rényi (b)	101.0141	0.009899607	-	-	-	0.1
Barabási-Albert (b)	31.60696	0.0316386	10	-	-	-
Watts-Strogatz (b)	20.21825	0.04946026	-	10	0.1	-
Erdős-Rényi (c)	200.8474	0.004978904	-	-	-	0.2
Barabási-Albert (c)	31.60696	0.0316386	100	-	-	-
Watts-Strogatz (c)	200.3708	0.004990747	-	100	0.2	-
Fully Connected	999	0.001001001	-	-	-	-
Tree	0	Inf	-	-	-	-

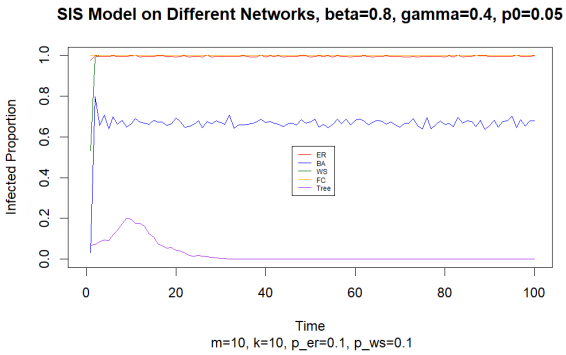
Table 1: Network characteristics for $\beta = 0.8$, $\gamma = 0.4$ and $p0 = 0.05$. Note: letters in the parentheses refer to the respective subfigure of Figure 1

Name	λ_1	$Epid_{threshold}$	m	k_{ws}	p_{ws}	p_{er}
Erdős-Rényi (d)	10.97519	0.09111461	-	-	-	0.01
Barabási-Albert (d)	5.618193	0.1779932	1	-	-	-
Watts-Strogatz (d)	2.388955	0.418593	-	1	0.01	-
Erdős-Rényi (e)	101.1271	0.009888548	-	-	-	0.1
Barabási-Albert (e)	31.60696	0.0316386	10	-	-	-
Watts-Strogatz (e)	20.2058	0.04949075	-	10	0.1	-
Erdős-Rényi (f)	200.8907	0.004977832	-	-	-	0.2
Barabási-Albert (f)	31.60696	0.0316386	100	-	-	-
Watts-Strogatz (f)	200.3412	0.004991485	-	100	0.2	-
Fully Connected	999	0.001001001	-	-	-	-
Tree	0	Inf	-	-	-	-

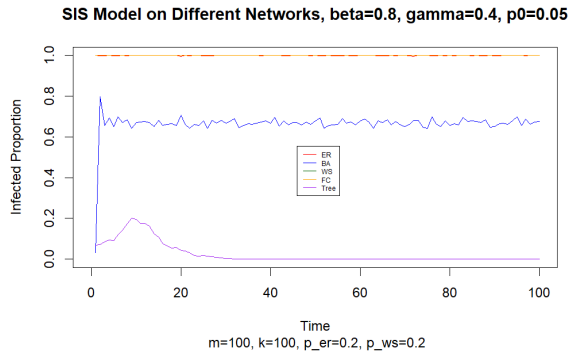
Table 2: Network characteristics for $\beta = 0.4$, $\gamma = 0.8$ and $p0 = 0.05$. Note: letters in the parentheses refer to the respective subfigure of Figure 1



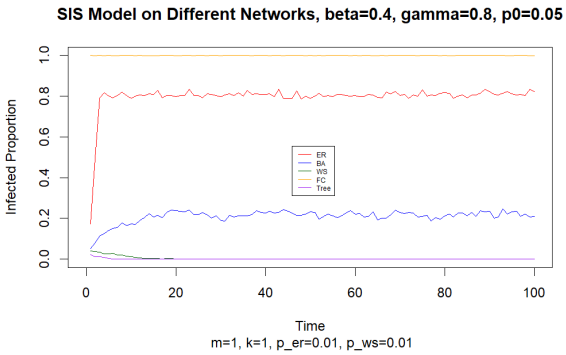
(a) Network comparison for $R_0 > 1$ (Eq. 2)



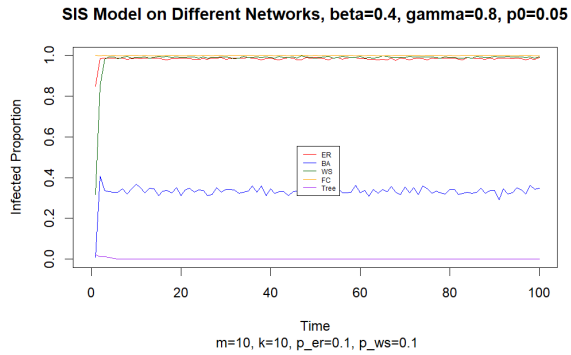
(b) Network comparison for $R_0 > 1$ (Eq. 2)



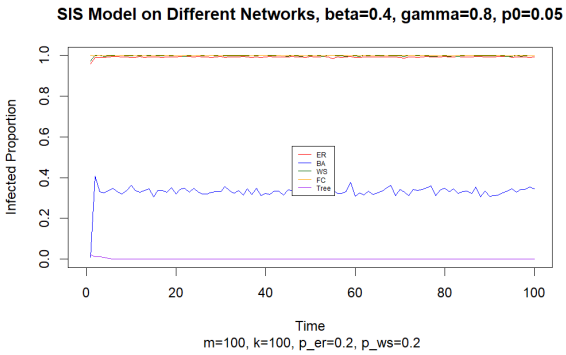
(c) Network comparison for $R_0 > 1$ (Eq. 2)



(d) Network comparison for $R_0 < 1$ (Eq. 2)



(e) Network comparison for $R_0 < 1$ (Eq. 2)



(f) Network comparison for $R_0 < 1$ (Eq. 2)

Figure 1: Network comparison for different scenarios of R_0 (Eq. 2) and initial network parameters

Although two distinct parameter sets were tested, one following values that would lead to an epidemic outrage and one to an epidemic shrinkage, the results obtained are very similar. More specifically, as it can be clearly seen, some specific types of networks are more prone to epidemics than others.

To be precise, it can be seen that in both cases (subfigures 1a-1c, and subfigures 1d-1f), the Tree network (G_{Tree}) experiences a very low proportion of infected individuals leading to the shrinkage of the epidemic. This result makes sense since, in the specific configuration, each individual node is only connected to 3 nodes (the root node and its children, except for the root). For that reason, the expansion of the epidemic is impossible to occur, even when β is way greater than γ. At the same time, a tree structure does not represent properly the nature of a social network structure.

On the other hand, in both cases, the Fully Connected network (G_{FC}) leads to the outrage of the epidemic with a constant infected proportion of 1, meaning that everyone is infected

as time develops since all nodes are connected with each other. Although, again, a social network structure will not follow a fully connected nature. The second ***worse*** results after G_{FC} are obtained for the Watts-Strogatz small-world network (G_{WS}) in both scenarios except subfigures 1a and 1d. In the first case, where $R_0 > 1$, the proportion of infected individuals is more or less constant around 1. In the second case, where $R_0 < 1$, the epidemic occurs again, but the proportion of infected individuals is slightly less. The only case, where the epidemic is vanished is 1d, where the initial parameters of k_{ws} and p_{ws} have very small values (1 and 0.01 respectively, see Table 2). The results are as expected, taking into account the nature of G_{WS}, where the clustering coefficient of this specific network type is high, meaning that a disease can be shared among nodes easily.

To continue with, concerning the Erdős-Rényi random graph (G_{ER}) and the Barabási-Albert scale-free network (G_{BA}) results for the two experiments fluctuate. In the first case (subfigures 1a-1c) G_{ER} behaves similar to G_{WS} and G_{FC} retaining a constant proportion of infected individuals around 0.95, where in the second one (subfigures 1d-1f) the proportion is slightly less, but again around 0.8, although when the parameter values of the network increase, the proportion tends to be equal to 1. In the first case, since $R_0 > 1$, the expansion of the epidemic is logical to occur even taking into account the randomness of G_{ER}, however for the second case, the expected result should be having the proportion of infected individuals in a lower level, or leading to a shrinkage of the disease. This result depends on the network's eigenvalues and the ratio of variables γ and β.

Lastly, the results for G_{BA} are interesting, achieving a infected proportion of 0.7 approx. in subfigures 1a-1c while it drops to 0.2-0.3 in subfigures 1d-1f. The intuition here is that the Barabási-Albert network simulates the scale-free nature of a large network meaning that it is resilient to the disease. Only in case that the hubs are the initial infected individuals, we should obtain that the whole network will get infected really quick, however this is considered as future work.

To conclude, in both cases, the comparison of each model's threshold with the basic reproduction number (Eq. 2) is accomplished, in order to validate the results along with the theory. More details on this comparison are presented as part of the submitted software (`compare_numbers` function).

2.2 Threshold Check per Network

In this subsection, the threshold for disease spread in each network type is investigated. To do so, the parameters γ and β are manipulated (as discribed in Section 4.3). By selecting parameter values slightly above and below the calculated threshold, the spread of the disease is simulated and analyzed, as well as, compared to theoretical predictions. The results of this experimental approach, which are presented below, provide insights into the resilience of different types of networks, shedding light on potential vulnerabilities and effective mitigation strategies.

2.2.1 Erdős-Rényi Network

Concerning the Erdős-Rényi random graph (G_{ER}), it is obvious that in all cases where γ and β are manipulated to have values that lead to a result above the threshold ($R_0 > 1/\lambda_1$), the epidemic is growing and expanding. It is interesting to mention here, for this case, that as the initial parameters of the network get higher values, the proportion of infected individuals is increasing as well. On the other hand, in all cases of $R_0 < 1/\lambda_1$, the epidemic vanishes, showcasing the validity of the theory. Although some minor fluctuations are present for the cases where the initial parameters of the network generate a highly connected structure, they still lead to the dispersion of the disease. The respective analysis is presented in Figure 2.

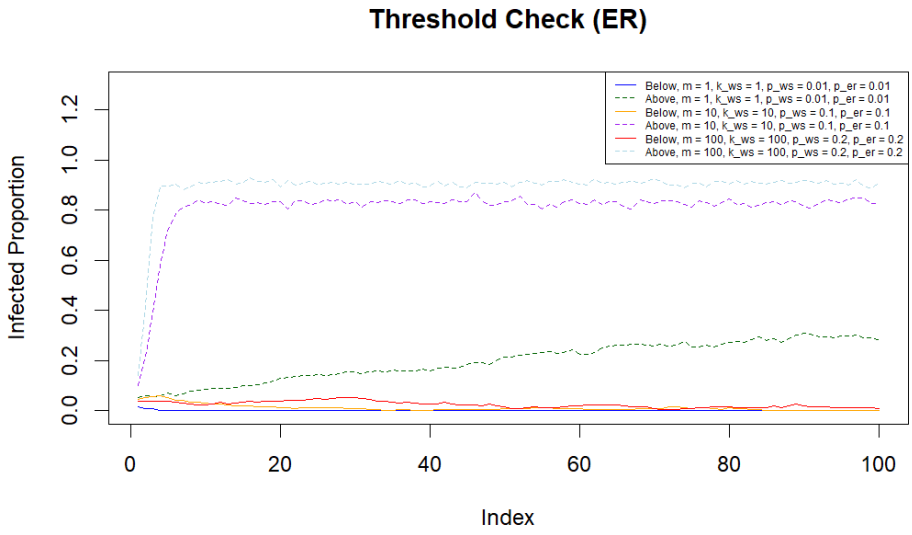


Figure 2: Threshold check of G_{ER}

2.2.2 Barabási-Albert Network

To continue with, the analysis for the Barabási-Albert scale-free network (G_{BA}) also coincides with the expected results. As mentioned before, as the Barabási-Albert network simulates the scale-free nature of a large network the expected result should present that it is resilient to the disease. Only in case that the hubs are the initial infected individuals, we should obtain that the whole network will get infected really quick. Based on Figure 3, this intuition is presented in the results, showing that the proportion of infected individuals do not surpass a maximum value of 10% for the cases where $R_0 > 1/\lambda_1$. On the other hand, in case where $R_0 < 1/\lambda_1$, for all examples, the infected proportion of the nodes is gradually decreasing. The fluctuations due to the different initial parameters of the network is as well presented here, although it seems that the results are robust to the changes of the initial parameters.

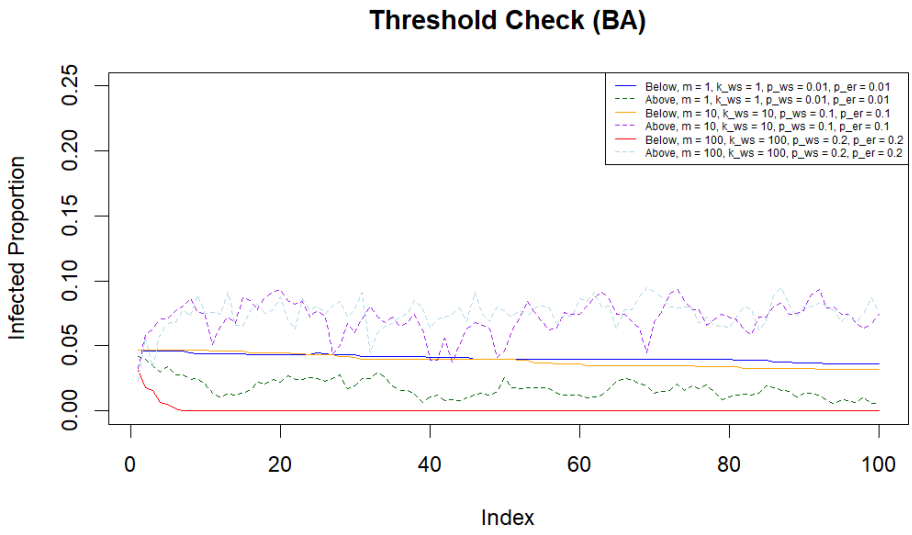


Figure 3: Threshold check of G_{BA}

2.2.3 Fully Connected Network

For the Fully Connected network (G_{FC}), the results presented in Figure 4 again follow the theory. This example clearly showcases that the forecast of Chakrabarti et al. 2008 [2], which states that the epidemic threshold is equal to $1/\lambda_1$, is absolutely true. In a fully connected structure, when $R_0 > 1/\lambda_1$, the infected proportion becomes rapidly equal to 100%, while when $R_0 < 1/\lambda_1$, the infected individuals immediately become 0%.

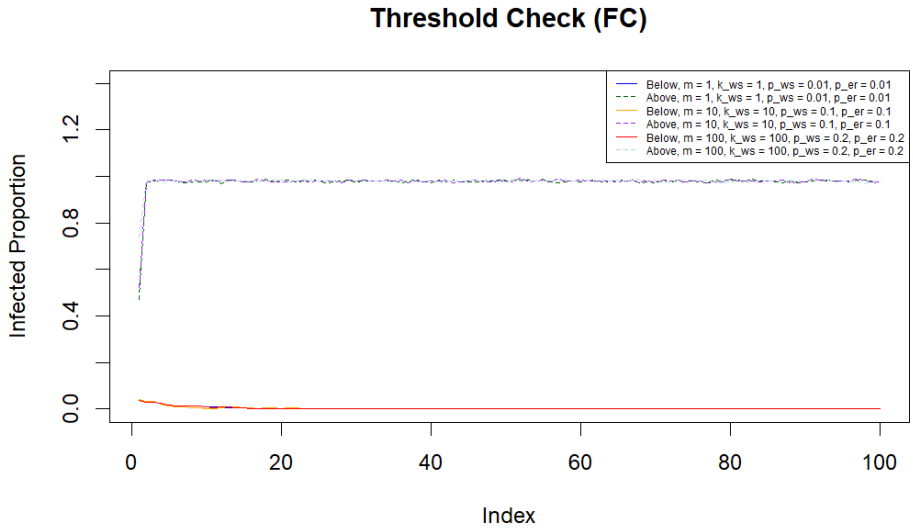


Figure 4: Threshold check of G_{FC}

2.2.4 Tree Network

Moreover, the analysis of the Tree network (G_{Tree}) is presented in Figure 5. The epidemic

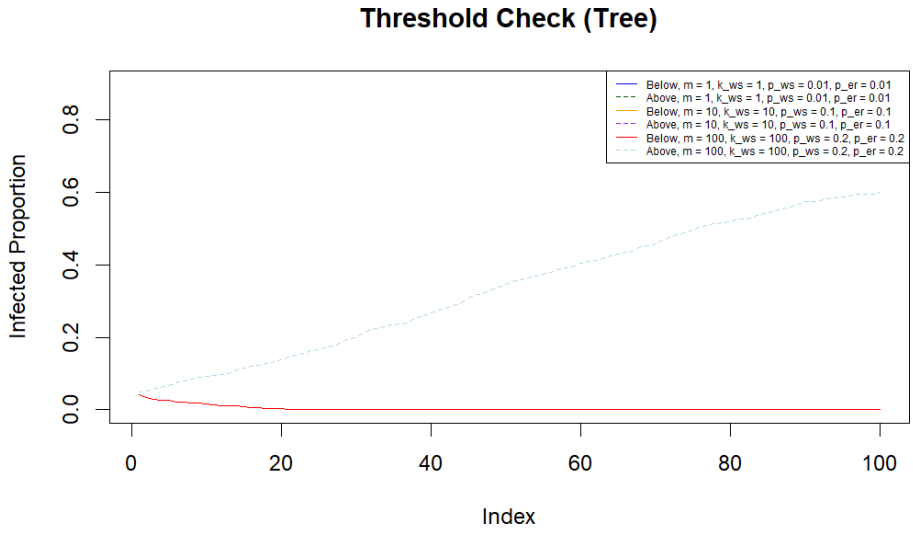


Figure 5: Threshold check of G_{Tree}

spread for the Tree network, is quite interesting. As mentioned before, since we have a tree structure, each individual has maximum 3 neighbors (out of 1000 total nodes). Although this is

true, meaning that we have a very sparse connected structure, still the theorem of Chakrabarti et al. holds still. It can be seen that when $R_0 > 1/\lambda_1$ the epidemic occur, while when $R_0 < 1/\lambda_1$ it vanishes immediately.

2.2.5 Watts-Strogatz Network

Finally, as for the Watts-Strogatz network (G_{WS}) Figure 6 includes the results. By observing in detail the simulation, the final conclusions are similar to the Erdős-Rényi random graph (G_{ER}). In every scenario where γ and β are adjusted to values resulting in a condition surpassing the threshold ($R_0 > 1/\lambda_1$), the epidemic exhibits growth and expansion. It is noteworthy to mention, in this context, that higher initial values of network parameters correspond to an increased proportion of infected individuals. Conversely, in instances where $R_0 < 1/\lambda_1$, the epidemic ceases, providing evidence for the theory's validity. Although there are slight variations in scenarios where the initial network parameters yield a highly interconnected structure, they still contribute to the dissemination of the disease.

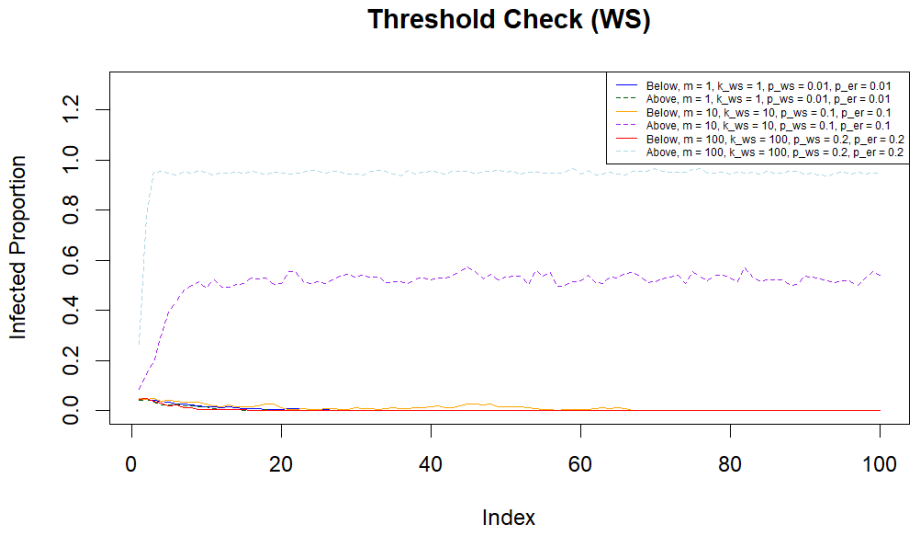


Figure 6: Threshold check of G_{WS}

3 Discussion

This study has explored the dynamics of disease spread across various network models, employing the SIS model to simulate scenarios in different network structures. Each network model exhibited distinct characteristics in terms of disease propagation, highlighting the profound impact of network topology on epidemiological dynamics.

3.1 Network-Specific Dynamics

In the Erdős-Rényi network, the disease spread was notably influenced by the probability of edge formation, leading to varied and somewhat unpredictable patterns of transmission. The Barabási-Albert network, characterized by a few highly connected nodes, demonstrated its resilience to the disease spread, aligning with real-world situations where only if certain individuals or nodes who can act as super-spreaders will lead to the rapid expansion of an epidemic. The Fully Connected Network, with its complete interconnectivity, showed an expectedly swift and widespread transmission of the disease, underscoring the risks associated with densely connected networks.

Conversely, the Tree Network, due to its hierarchical and sparsely connected nature, exhibited a slower and more controlled disease spread. This is indicative of how limiting connections in a network can effectively slow down the transmission of diseases. The Watts-Strogatz model, embodying characteristics of both regular and random networks, presented an interesting case with its small-world properties, demonstrating moderately quick disease transmission with notable local clustering effects.

3.2 Implications and Conclusions

The findings of this study emphasize the significance of network topology in understanding and predicting disease spread. Networks with higher degrees of connectivity are more vulnerable to rapid disease outbreaks. In contrast, networks with sparse connections (such as Tree networks) can inhibit the spread, serving as a model for effective disease control strategies. Moreover, part of the findings was the validation of the Chakrabarti et al. (2008) [2] forecast on the epidemic threshold of $1/\lambda_1$.

4 Methodology

4.1 General Methodology

In this section, we outline the general methodology employed in our study to investigate the dynamics of disease spread, emphasizing the simulation approach and parameter variations across distinct network structures.

4.1.1 Simulation of SIS Model

The simulation of the Susceptible-Infective-Susceptible (SIS) model is conducted on various network structures, namely Erdős-Rényi random graphs (G_{ER}), Barabási-Albert scale-free networks (G_{BA}), Watts-Strogatz small-world networks (G_{WS}), and finally, Fully Connected networks (G_{FC}), and Tree networks (G_{Tree}). Although, it is clear that for the first three network types (G_{ER}, G_{BA}, G_{WS}) their initial parameters affect the final results. For this reason, several tries were conducted considering the initial parameters, in order to analyse their contribution to the results. More precisely, for Erdős-Rényi (G_{ER}) the initial parameter is the probability of edge creation p_{er}, for Barabási-Albert (G_{BA}) the initial parameter is the number of edges attached from a new node and finally for Watts-Strogatz (G_{WS}) the parameters are: k_{ws}, which is the number of nearest neighbors each node is connected to and a rewiring probability p_{ws}. The values tried together with their results are presented in Tables 1 and 2.

Moreover, the SIS model is governed by the following rules at each time step:

1. An infected node recovers with probability γ.
2. An infected node attempts to infect each neighbor with probability β.
3. Initially, only a random fraction p_0 of nodes are infected.

The simulation is performed for a fixed set of parameters: infection rate (β), recovery rate (γ), initial fraction of infected nodes (p_0), and the duration of the simulation (T). The status of each node is updated in parallel at each time step, and the proportion of infected nodes over time is recorded. Moreover, the dynamics of disease spread are visualized by plotting the proportion of infected nodes against time for each network type. Finally, the leading eigenvalue (λ_1) of the adjacency matrix for each network is calculated as it plays a crucial role in determining the epidemic threshold ($Epid_{threshold}$ (Eq. 1)).

4.1.2 Epidemic Threshold

To analyze the epidemic threshold for each network type, the leading eigenvalue of the adjacency matrix is computed using the `leading_eigenvalue` function. The epidemic threshold ($Epid_{threshold}$) is then determined as the inverse of the leading eigenvalue:

$$Epid_{threshold} = \frac{1}{\lambda_1} \tag{1}$$

Moreover, R_0, also called ***basic reproduction number*** in the SIS model, is a key parameter in epidemiology, representing the average number of secondary infections produced by one infected individual in a completely susceptible population. R_0 serves as a threshold for the onset of an epidemic. When $R_0 > 1$, the disease is expected to spread in the population, indicating an epidemic. On the other hand, when $R_0 < 1$ it suggests that the disease is not expected to exhibit epidemic behavior.

$$R_0 = \frac{\beta}{\gamma} \tag{2}$$

4.2 Disease Spread Analysis in Different Networks

In this task (Section 2.1), we investigate the dynamics of disease spread in the SIS model by considering two sets of parameter values and all the different networks. The idea of this experiment, is to test if any network type is more prone to an epidemic spread compared to the rest of the networks, for both cases of R_0 being greater or smaller than 1. Specifically, two sets of fixed parameter values are considered:

Parameter Set 1

- $n = 1000$ (Number of nodes)
- $\beta = 0.8$ (Infection rate)
- $\gamma = 0.4$ (Recovery rate)
- $p_0 = 0.05$ (Initial infection rate)
- $T = 100$ (Duration of the simulation)

The basic reproduction number (R_0) for this parameter set is calculated as $R_0 = \frac{\beta}{\gamma} = \frac{0.8}{0.4} = 2$. As mentioned above, R_0 serves as a threshold value and when $R_0 > 1$, the disease is expected to spread in the population, indicating a potential epidemic.

Parameter Set 2

- $n = 1000$ (Number of nodes)
- $\beta = 0.4$ (Infection rate)
- $\gamma = 0.8$ (Recovery rate)
- $p_0 = 0.05$ (Initial infection rate)
- $T = 100$ (Duration of the simulation)

For this parameter set, the corresponding R_0 is calculated as $R_0 = \frac{\beta}{\gamma} = \frac{0.4}{0.8} = 0.5$. In this case, $R_0 < 1$, suggesting that the disease is not expected to exhibit epidemic behavior.

For the two parameter sets, simulations are conducted on the network types mentioned before, to observe and compare the dynamics of disease spread. However, during the simulations performed with the two mentioned parameter sets, different values for the networks' initial values were tried (see Tables 1 and 2), in order to evaluate how they affect the results as well. The results will provide insights into the influence of different parameter values on the resilience of each network type to epidemic outbreaks, along with an examination of whether each network type is conducive to the spread of epidemics or not.

4.3 Threshold Check per Network

For the second task (Section 2.2), two new sets of parameter values are selected for performing the simulations: one slightly above and one slightly below the $Epid_{threshold}$ of each network. Specifically, once ϵ, a small positive constant, is both added and subtracted individually to/from the $Epid_{threshold}$, the new values of R_0 are generated (let's call them $R_{0_{below}}$ and $R_{0_{above}}$, respectively). Then by using the formula of Eq. 2, two distinct sets of β, γ are generated (by using the function `generate_random_values`), one for the case of $R_{0_{below}}$ and one for $R_{0_{above}}$, respectively. It is important to mention here that this procedure is completed for all network configurations

mentioned in Tables 1 and 2. Finally, it is obvious that results also depend on the value of ϵ. However, in the current implementation only the value of 0.05 was tested and further analyses are considered as future work.

The simulation results for both parameter sets are compared to evaluate the consistency with the theoretical prediction. The proportion of infected nodes over time is visualized for each case, providing insights into the forecast of the work by Chakrabarti et al. 2008 [2] which states that the epidemic threshold is equal to $1/\lambda_1$.

4.4 Comparison and Analysis

To assess the relative susceptibility of different networks to epidemic outbreaks, the proportion of infected nodes is compared across the different types of networks mentioned before. This comparative analysis is essential for understanding how network topology influences the dynamics of disease spread.

The comparison includes visualizations of the simulated epidemic curves for each network type, highlighting potential differences in their resilience to infectious diseases. Moreover, the relationship between the calculated β/γ ratio and the inverse of the leading eigenvalue is examined to validate the theoretical predictions regarding epidemic thresholds.

The results are presented graphically, and conclusions are drawn based on the observed patterns and comparisons across different network structures.

References

[1] Albert-László Barabási and Réka Albert. "Emergence of scaling in random networks". In: *science* 286.5439 (1999), pp. 509–512.

[2] Deepayan Chakrabarti et al. "Epidemic thresholds in real networks". In: *ACM Transactions on Information and System Security (TISSEC)* 10.4 (2008), pp. 1–26.

[3] P ERDdS and A R&wi. "On random graphs I". In: *Publ. math. debrecen* 6.290-297 (1959), p. 18.

[4] Duncan J Watts and Steven H Strogatz. "Collective dynamics of 'small-world'networks". In: *nature* 393.6684 (1998), pp. 440–442.