Simulation of SIS Model over Weighted Networks

Complex and Social Networks (CSN)
Final Project

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Contents

List of Figures

List of Tables

1 Introduction

The landscape of social network analysis has predominantly been shaped by the study of unweighted networks. However, recent advances have begun exploring the more nuanced realm of weighted networks [1]. Weighted networks offer a more realistic representation of social interactions, which are inherently heterogeneous [2]. This heterogeneity is derived not only from the degree distribution of the networks but also from the distribution of weights assigned to the edges [2], encompassing normal, power-law, and exponential distributions, to mention a few.

In this paper, we delve into the Susceptible-Infected-Susceptible (SIS) model within the framework of graph theory, focusing solely on weighted networks. The SIS model serves as a pivotal tool for analyzing the impact of network structures on the dynamics of disease spread, enabling the assessment of interventions and understanding the persistence of infectious diseases in communities. In the current study the weights of the edges of the network represent contacts among individuals. In the context of sex transmitted diseases for example, this accounts for the number of sex acts among individuals, which certainly has an impact on the likelihood of being infected. Our focus is on the application of this model to complex networks, particularly those characterized by random, scale-free and exponential connectivity distributions, such as the random graph model of Erdős–Rényi (ER) [3], the the Barabási–Albert model [4], and finally the Watts and Strogatz model (WS) [5].

The study of epidemics in complex networks, as highlighted in the works [6, 7], reveals intriguing differences in disease dynamics based on the underlying network topology. At the same time, it is proven that the weight distribution of a weighted network largely impacts the epidemic spreading process [2]. Exponential networks, in which the nodes' connectivity distribution (the probability P(k) that a node is connected to other k nodes) is exponentially bounded [8], typically exhibit a distinct epidemic threshold, a critical point beyond which an infection becomes persistent [9]. Conversely, scale-free networks demonstrate a remarkable resilience to the spread of infections, often lacking a clear epidemic threshold [9]. This distinction underlines the significance of network topology in understanding and predicting epidemic patterns.

Through this research, we aim to contribute to the growing body of knowledge in network science, particularly in the context of weighted networks and their role in epidemic spread. By exploring various weight distributions—normal, power-law, and exponential—we seek to capture the diverse nature of real-world social interactions and their implications for disease transmission and control.

2 Related Work

In addition to the pivotal works referenced throughout this paper, the study conducted by Ye Sun et al. [1] served as an initial inspiration due to its detailed exploration of outbreak thresholds and epidemic prevalence within weighted SIS models. This research emphasizes the complex interdependencies inherent to the network's weighted structure. While our work drew from these valuable insights, the implementation strategy we adopted ultimately charted a distinct path, tailored to the unique objectives and parameters of our investigation.

Outbreak Threshold The outbreak threshold of an epidemic signifies the critical point at which the disease begins to spread significantly through the population. For uncorrelated networks, this threshold can be expressed in terms of the network's degree distribution and the average degree [1]. Specifically, it is related to the largest eigenvalue of the adjacency matrix of the network. In the context of the SIS model, when accounting for the weights of connections, the threshold condition is adjusted to accommodate the weighted transmission probabilities [1].

For the case of uncorrelated networks, the probability that an edge links to a node with degree k is $\frac{kp(k)}{\langle k \rangle}$, where $p(k)$ is the degree distribution of the observed network, and $\langle k \rangle$ is the average degree [1]. In addition, it is assumed that as long as the epidemic has not spread out yet, the infected node with degree k has only one ingoing link and $k-1$ outgoing links [10]. And the average number of susceptible nodes infected by an already infected node i is:

$$\langle n_i \rangle = \beta \sum_k \frac{p(k)k(k-1)}{\langle k \rangle} + \pi, \tag{1}$$

where π is the contribution of the probability to reinfect the ancestor (the node that infected node i, corresponding to i's ingoing link) [11].

For the SIS model where the reinfection is allowed, things get more complicated. Ye Sun et. al. [1] define π_t as the probability that j infects i if i has infected j yet. In this model, they set the recovery probability $\mu = 1$, which means that the infected node remains the infected state in just one step. Therefore, the interval that j remains infected and i remains susceptible is only 1, which leads to $\pi_t = \beta$. Incorporating the effect of competition between j and the other descendants of i, the reinfected probability π for the system can be calculated as following:

$$\pi = \pi_t \sum_{k'=0}^{\kappa-1} \frac{(\kappa-1)}{k'} (\beta\pi_t)^{k'} (1-\beta\pi_t)^{\kappa-1-k'} \frac{1}{k'+1}, \tag{2}$$

where $\kappa - 1 = \sum_k p(k) \frac{k(k-1)}{\langle k \rangle}$ is the branching factor that represents the average number of nodes influenced by node i and k' represents the infected neighbors of node i.

Neglecting the high-order term, it is obtained that $\pi \approx \beta$. According to Eq. 1, the epidemic threshold of SIS model is:

$$\lambda_c(\text{SIS}) = \frac{\langle k \rangle}{\alpha \langle k^2 \rangle}. \tag{3}$$

Epidemic Prevalence Epidemic prevalence, on the other hand, quantifies the steady-state fraction of the infected population. In the SIS model, and the work of Ye Sun et. al. [1], this is derived using a heterogeneous mean-field theory approach that assumes nodes with the same degree exhibit similar dynamics. The prevalence can be influenced by the network's average edge weight, reflecting the intensity of interactions across the network.

In the SIS model, the density of the infected nodes is used in the final state to illustrate the epidemic prevalence. In heterogeneous mean-field theory, it is supposed that all nodes of the same degree share similar dynamic behavior. Based on the microscopic Markov-chain approach [12], we can obtain the infected density of node degree k, I_k, for $t \to \infty$ as follows:

$$I_k = (1 - I_k)(1 - \psi_k) + (1 - \mu) I_k, \tag{4}$$

where ψ_k is the probability that the node with degree k is not being infected by any neighbors, which can be obtained by:

$$\psi_k = \prod_{k'} (1 - \beta I_{k'})^{C_{kk'}}, \tag{5}$$

where $C_{kk'} = kp(k'|k) = \frac{k'p(k')}{\langle k \rangle}$, represents the expected number of links from a node of degree k to nodes of degree k'.

Then the global epidemic prevalence can be obtained as follows:

$$\rho_I = \sum_k p(k) I_k. \tag{6}$$

Simulation Results Moreover, the simulations of this work were implemented considering the intricacies of weighted networks. Two scenarios were compared: one where link weights follow a uniform distribution and another with a Poisson distribution. Monte Carlo simulations were performed to verify theoretical predictions. Finally, The impact of the average edge weight on the outbreak threshold and epidemic prevalence is depicted in the following figures.

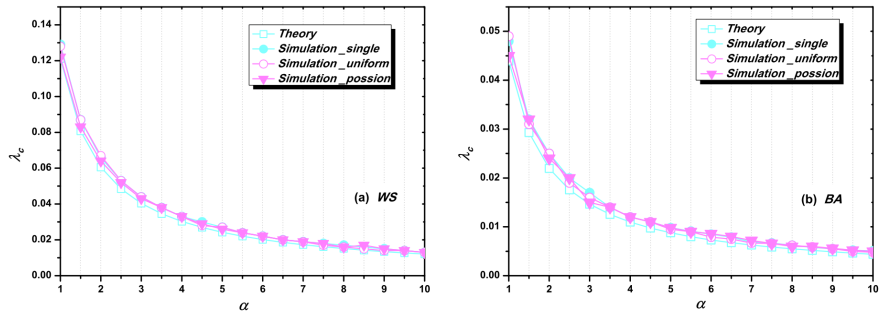

Figure 1: Epidemic threshold λ_c as a function of the average edge weight parameter α on the SIS model for (a) WS network and (b) BA network [1].

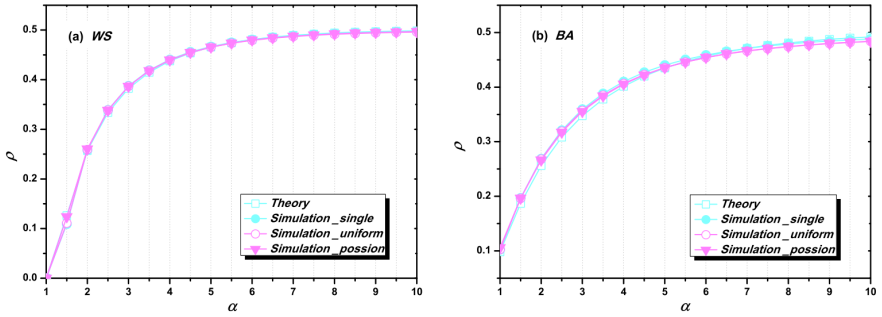

Figure 2: Epidemic prevalence ρ as a function of the average edge weight parameter α on the SIS model for (a) WS network and (b) BA network [1].

3 Results

The results of the simulations for the SIS model across the various network types and weight distributions that investigated are presented in the current section. Each plot represents the dynamics of epidemic spread over 100 time steps, showing the infected proportion of the network in 3 separate scenarios. One scenario includes values of γ and β that result exactly in the epidemic threshold **as calculated by Eq. 3** (stated as *On-threshold*), another scenario includes values of γ and β that lead to epidemic outrage (stated as *Outbrake*) and a final scenario with values that lead to the stability of an epidemic (stated as *Stability*). Moreover, Table 1 presents in a total view the values of the parameters in each different simulation scenario, for all models and distributions.

Table 1: Transmission rate (β), recovery rate (γ), weighted and generalized (Gen.) epidemic thresholds as well as weighted R_0 of simulations.

	β	γ	$1/\lambda_w$	R_0	Gen. Threshold
ER, Normal, On-Thresh	0.05	0.56	0.27	0.27	90.85
ER, Normal, Outbreak	0.05	0.16	0.27	0.92	170.60
ER, Normal, Stabilized	0.05	0.96	0.27	0.16	11.09
ER, Power-law, On-Thresh	0.45	0.60	2.22	2.22	80.36
ER, Power-law, Outbreak	0.45	0.20	2.22	6.72	160.12
ER, Power-law, Stabilized	0.45	1.00	2.22	1.33	0.61
ER, Exponential, On-Thresh	0.19	0.56	0.32	0.32	91.58
ER, Exponential, Outbreak	0.19	0.16	0.32	1.17	171.34
ER, Exponential, Stabilized	0.19	0.96	0.32	0.19	11.82
BA, Normal, On-Thresh	0.24	0.59	1.21	1.21	3.37
BA, Normal, Outbreak	0.24	0.19	1.21	3.79	5.87
BA, Normal, Stabilized	0.24	0.99	1.21	0.72	0.90
BA, Power-law, On-Thresh	0.44	0.59	1.52	1.52	3.13
BA, Power-law, Outbreak	0.44	0.19	1.52	4.72	5.63
BA, Power-law, Stabilized	0.44	0.99	1.52	0.91	0.71
BA, Exponential, On-Thresh	0.75	0.58	1.22	1.22	3.19
BA, Exponential, Outbreak	0.75	0.18	1.22	3.88	5.65
BA, Exponential, Stabilized	0.75	0.98	1.22	0.73	0.88
WS, Normal, On-Thresh	0.36	0.60	1.80	1.80	1.49
WS, Normal, Outbreak	0.36	0.20	1.80	5.48	2.48
WS, Normal, Stabilized	0.36	1.00	1.80	1.08	0.56
WS, Power-law, On-Thresh	0.49	0.60	2.04	2.04	1.23
WS, Power-law, Outbreak	0.49	0.20	2.04	6.21	2.11
WS, Power-law, Stabilized	0.49	1.00	2.04	1.22	0.50
WS, Exponential, On-Thresh	0.78	0.59	1.22	1.22	1.44
WS, Exponential, Outbreak	0.78	0.19	1.22	3.79	2.33
WS, Exponential, Stabilized	0.78	0.59	1.22	0.73	0.83

3.1 Erdös-Renyi Simulations

The Erdős-Rényi model, characterized by its random connectivity, was subjected to the SIS model simulations to examine the epidemic spread under varying weight distributions. Below, the infected proportions over 100 time steps for exponential, gaussian, and power-law weight distributions are presented.

In the gaussian distribution (Fig. 3) it is shown that while the on-threshold scenario experiences initial fluctuations, it stabilizes at an infection rate of approximately 24%. The outbreak scenario presents a more sustained infection level, with a significant proportion of the network remaining infected throughout the simulation (slightly higher than 50%). Again, the stability scenario results in a rapid and sustained decline to a very small portion of infections.

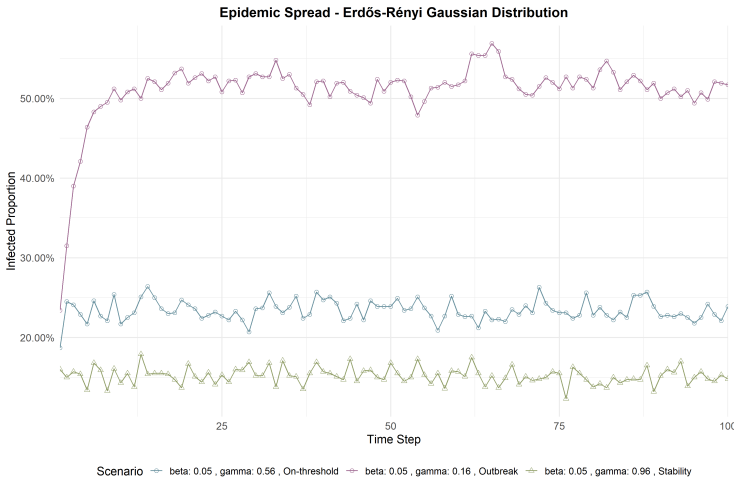

Figure 3: Epidemic spread in Erdős-Rényi network with gaussian weight distribution.

As for the power-law distribution (Fig. 4), all scenarios quickly diminishes to a low endemic state of 0%, with the only difference being in the number of time steps required for this even to occur.

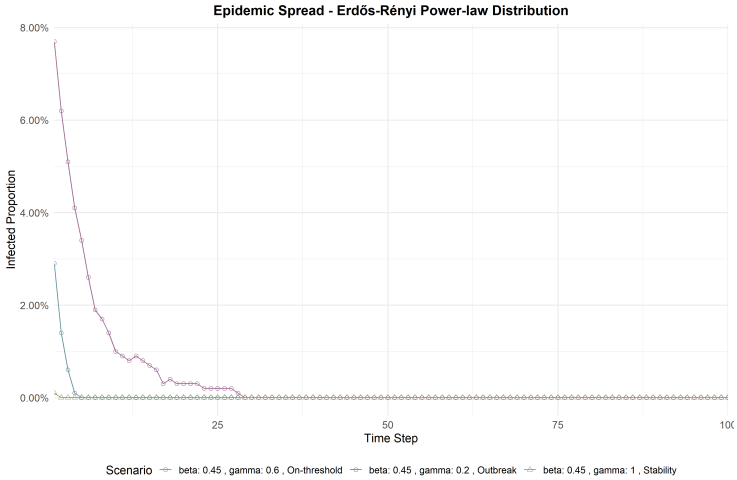

Figure 4: Epidemic spread in Erdős-Rényi network with power-law weight distribution.

In the exponential distribution (Fig. 5), the on-threshold scenario show fluctuations around a mean prevalence, which tends to be equal to initial infected proportion of the nodes, indicating a dynamic balance between infection and recovery. For the outbreak scenario, the infection peaks at a constant level of approximately 30% and it remains stable at this point for the remaining of the simulation. The stability scenario effectively suppresses the epidemic, leading to a very small proportion of infections, although it does not reach 0%.

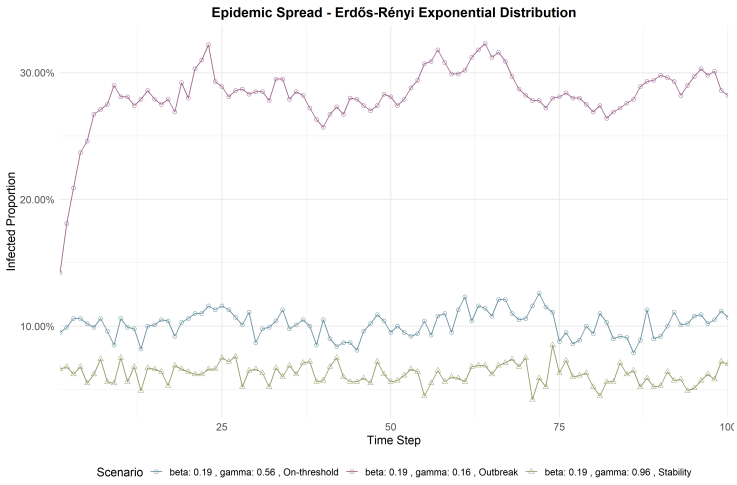

Figure 5: Epidemic spread in Erdős-Rényi network with exponential weight distribution.

The Erdős-Rényi simulations reveal that random network structures can either sustain an epidemic at varying levels or allow for quick control, depending on the balance between transmission and recovery rates. The impact of weight distributions on epidemic outcomes is apparent, with each distribution contributing to the overall dynamic in distinct ways.

3.2 Barabasi-Albert Simulations

In Barabási-Albert networks, the epidemic spread was simulated under three different scenarios of transmission (β) and recovery (γ) rates. The scenarios were chosen to reflect critical points in disease dynamics: reaching the epidemic threshold, causing an outbreak, and achieving stability in the spread of the disease.

In the gaussian distribution case (Fig. 6), the on-threshold scenario shows again a rapid decline in the proportion of the infected nodes, reaching close to a level of 0% after the first 25 steps of the simulation. The outbreak scenario, on the other hand, reaches a peak prevalence or around 30% that is retained throughout the simulation steps, whereas the stability scenario demonstrates a rapid decline to zero, suggesting successful epidemic control.

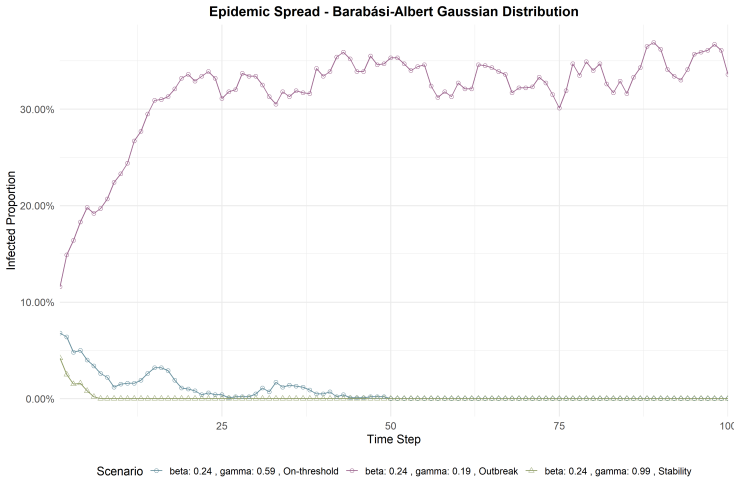

Figure 6: Epidemic spread in Barabási-Albert network with gaussian weight distribution.

Moreover, for the power-law distribution (Fig. 7), the epidemic threshold scenario presents a slow decline in prevalence, reaching values of 0% after the 26th step of the simulation. The outbreak scenario, on the other hand, starts with a moderate level of infected nodes (around 8%), but it also shows a remarkable tendency towards a stable state after initial fluctuations. The stability scenario quickly diminishes the spread of the epidemic, leading to its eventual disappearance.

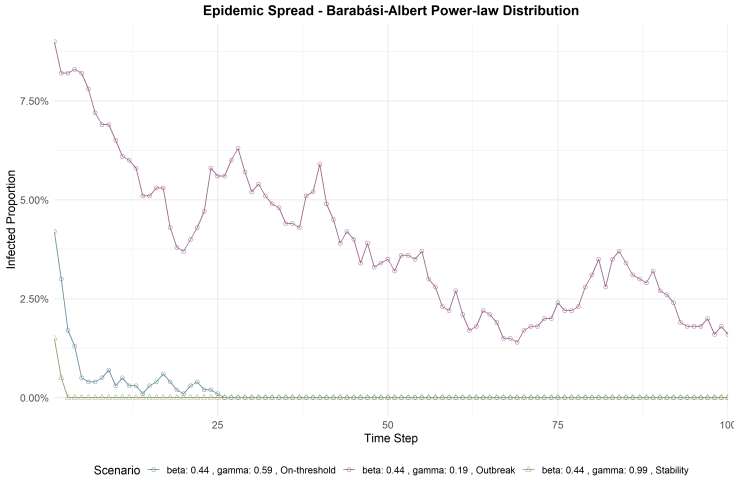

Figure 7: Epidemic spread in Barabási-Albert network with power-law weight distribution.

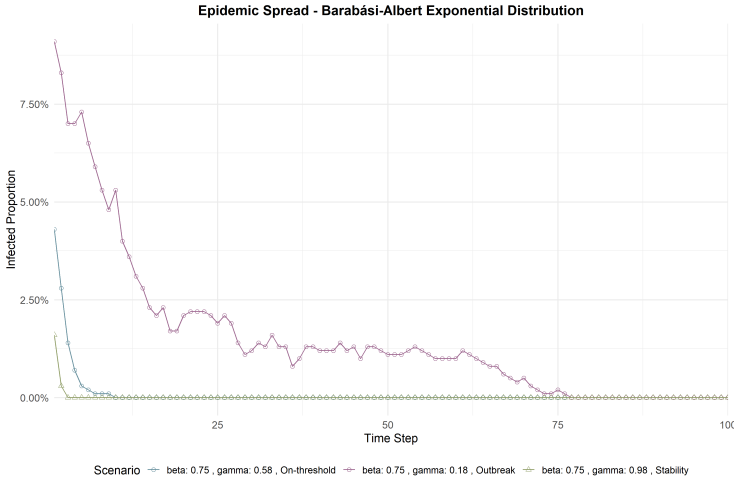

Figure 8: Epidemic spread in Barabási-Albert network with exponential weight distribution.

Lastly, the exponential weight distribution results (Fig. 8) show that for the on-threshold scenario, the epidemic quickly stabilizes at a low prevalence. The outbreak scenario leads to a higher prevalence, as expected, but eventually settles into a low steady state as well, suggesting that even with higher transmission rates, the epidemic can be contained over time. The stability scenario shows a rapid decline in infection, leading to the eradication of the epidemic.

These results underscore the sensitivity of epidemic dynamics to the underlying network structure and weight distribution. The Barabási-Albert network, known for its scale-free properties, demonstrates diverse behaviors under different epidemic parameters, reflecting the complex interplay between topology and disease spread.

3.3 Watts-Strogatz Simulations

The Watts-Strogatz model, renowned for its small-world characteristics, demonstrates interesting epidemic dynamics under the influence of various weight distributions. The simulations conducted across three scenarios — on-threshold, outbreak, and stability — provide insights into the interplay between network connectivity and disease spread.

For the gaussian distribution, as depicted in Fig. 9, the on-threshold simulation together with the stability scenario slightly fluctuates before settling at a negligible infection rate. Conversely, the outbreak scenario shows the persistence of the epidemic, maintaining a moderate proportion of infection throughout the simulation (approx. 14%).

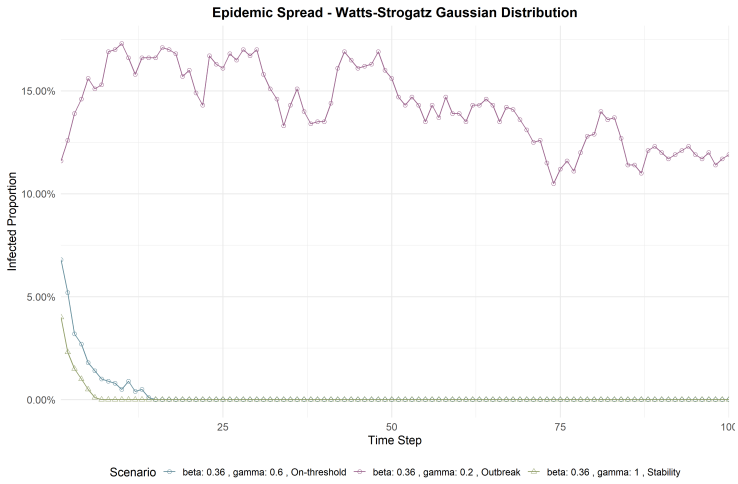

Figure 9: Epidemic spread in Watts-Strogatz network with gaussian weight distribution.

The power-law distribution results, shown in Fig. 10, reveal a quick containment of the epidemic, with the infected proportion decreasing sharply, for all scenarios, underscoring the non-persistent nature of the epidemic in this network configuration. The only slight difference is shown in the outbreak scenario where the infected proportion instead of directly decreasing to a percentage of 0%, it is linearly decreasing in the first 10 steps, before leading to infection evaporation.

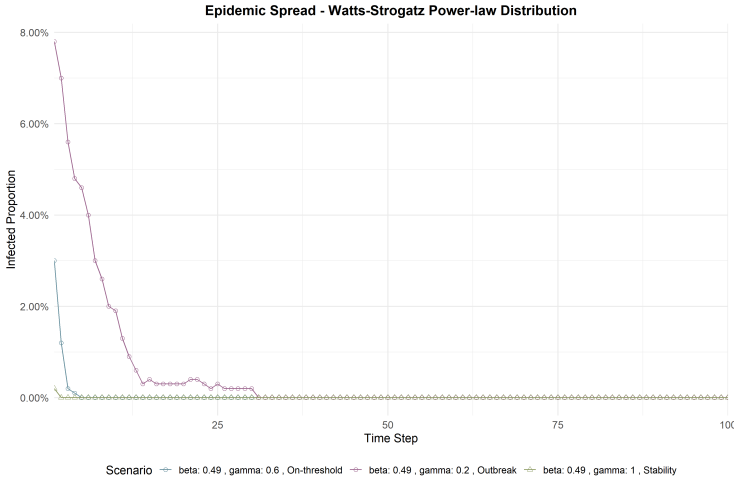

Figure 10: Epidemic spread in Watts-Strogatz network with power-law weight distribution.

Lastly, in Fig. 11, the on-threshold scenario for the exponential weight distribution shows a precipitous drop in infection, reaching zero prevalence rapidly, highlighting the network's resilience. The outbreak scenario peaks early but demonstrates the network's capacity to recover as the infection prevalence stabilizes at lower levels. Additionally, the stability scenario shows an effective disease eradication, indicating a high level of control over the epidemic spread.

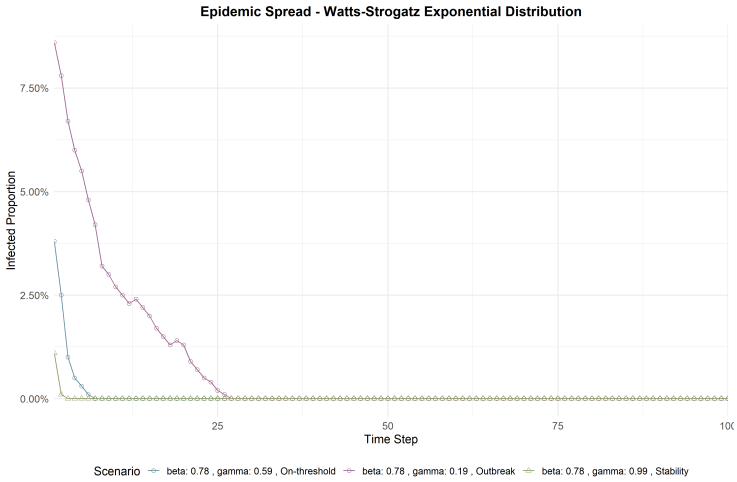

Figure 11: Epidemic spread in Watts-Strogatz network with exponential weight distribution.

These simulations across different weight distributions in the Watts-Strogatz network elucidate the crucial role of network topology in epidemic modeling. The small-world nature coupled with varying weight distributions significantly influences the course of the epidemic, which is critical for understanding and managing disease spread in real-world scenarios.

4 Discussion

The influence of degree distributions on the epidemic threshold for unweighted networks has already extensively been researched [7], [6], [13]. Some general findings for example are that networks with random (binomial) or exponential degree distributions do have an epidemic threshold, while scale-free networks, like the Barabasi-Albert model, seem to not have one. Though it should be mentioned that the threshold for exponential degree distributions [13] is different from the one that we are using in this paper.

Our results shown in Figures 6 to 11 confirm this assumption to a certain point, because both the Erdös-Renyi and the Barabsi-Albert model live up to their expectations except for one specific weight distribution each. Given the fact that the power-law distributed weights prevent the random network from having a threshold, just like the normal distributed weights prevent the scale-free network from having no threshold, leaves room for interpretation that the type of weight distribution might have a greater impact on the outcome of epidemic scenarios than the type of degree distribution has. It is also stated in [2] that the homogeneity of the weight distribution is an essential factor for the presence of high epidemic prevalence, which is not directly related to a threshold being present, but it confirms that the distribution of weights does have a relatively high influence on the behaviour of the network during epidemic disease spreading.

On an additional note, it seems that the influence of the exponential distribution of weights is somewhere between the normal and the power-law distribution. The influence is strong enough to depict a threshold oriented behavior of the Erdös-Renyi model, but it struggles to force that threshold oriented behavior on a scale-free network just like the normal distributed weights did. Since the shape of the exponential distribution pictured in figure 14 is more similar to the power-law distribution with much more low valued elements than those of high value, the lack of being able to keep a high spreading rate for scenarios where that clearly should be possible, might actually be just as expected.

Another relatively clear observation is that for cases where we are parameterized to be below the threshold, the networks based on the Erdös-Renyi model seem to be rather fluctuating at lower rates of infected population, while the other networks let the epidemic die out relatively instant. This also makes sense since the Erdös-Renyi network has much more connected nodes rather than clustered hubs of connections, meaning it is probably much easier to spread the disease. This is also confirmed by the diameter of the networks, which states how long the longest shortest path between two nodes in a network is, because even with consideration of the weights, the diameter for the Erdös-Renyi networks are at approximately 2, for Barabasi-Albert at ~ 50, and for Watts-Strogatz at ~ 229. This also underlines how the exponential distribution seems to be behaving more like a power-law distribution rather than a normal distribution. It also might help to explain the inability to comply with the threshold in all simulations that were based on the Watts-Strogatz network. Even though it was already mentioned that there is another threshold for this type of network,

not even the normal distributed weights were able to counter that absence of threshold behavior, though the network had a hard time to suppress the rate of infected nodes.

Lastly, we want to address the generalized threshold that was introduced in [7] for general networks and is also included in table 1. Since generalized networks are not specifically defined, we considered that this might be a general approach that can be taken for different types of networks. It is proposed that simulations should yield epidemic behavior for cases where the leading eigenvalue of the parameterized adjacency matrix is greater than 1. Said parameterized adjacency matrix can be calculated as follows:

$$M_{ij} = \beta_{ij} + (1 - \gamma) \times A_{ij} \tag{7}$$

where β_{ij} is similar to the weighted adjacency matrix only that it does not contain weights, but infection probabilities between nodes i and j, and A_{ij} is the classic adjacency matrix. However, as can be seen in table 1, this value is very often above 1 and sometimes even far beyond it even when there should not and also is not epidemic behavior. However, for the cases where an epidemic threshold was confirmed, the resulting epidemic behavior is proportional to the values of the generalized threshold, meaning that if no epidemic is present, setting parameters so that the leading eigenvalue of the parameterized matrix increases, will also result in more epidemic behavior and vice versa.

A further problem of this approach is that the calculation of parameters is not completely simple or exact. Since the calculation of the leading eigenvalue for the parameterized matrix is dependent on both, β & γ, neither of the parameters can be directly calculated based on the other. However, approximation for the parameter γ can be done by doing the following: let $le(X)$ be a function that calculates the leading eigenvalue of a matrix X. The parameter γ can then be calculated with the formula:

$$\gamma = -\left(\frac{1 - le(\beta_{ij})}{le(A_{ij})}\right) + 1 \tag{8}$$

5 Future Work and Limitations

In considering future enhancements and recognizing the limitations of the current study, several initiatives are proposed. Firstly, the development of a more robust hypothesis testing framework is critical. This involves formulating a structured experimental history that can systematically validate the accuracy of the epidemic threshold formula. Secondly, employing Monte Carlo simulations would be instrumental in solidifying the results, providing a more reliable statistical basis through repeated random sampling.

Further, there is a need for a deeper examination of the parameters involved in the initialization of network models. A nuanced understanding of these parameters will likely yield insights into the complex dynamics of network behavior during epidemics.

Lastly, optimizing the existing simulation software is essential to improve computational efficiency. Faster simulations would not only expedite the research process but also enable the exploration of larger and more complex networks, potentially leading to more generalizable findings.

Each of these areas presents an opportunity to refine and expand the current research, paving the way for more comprehensive studies in the field of network-based epidemic modeling.

6 Conclusion

This study has explored the intricate dynamics of infectious diseases on various complex network models under different weight distributions. Our simulations, based on the SIS model, have provided valuable insights into how the heterogeneity of interactions affects the spread and control of epidemics. We observed that the Barabási-Albert networks, characterized by their scale-free nature, exhibited significant resilience to epidemic outbreaks. On the other hand, Erdős-Rényi and Watts-Strogatz models displayed different patterns of disease spread, emphasizing the impact of network topology on epidemic dynamics.

One of the principal findings of this work is the nuanced understanding of how different weight distributions influence the epidemic threshold and prevalence. It was evident from the simulations that the distribution types have a non-trivial effect on the spread patterns, with the Gaussian distribution, in particular, showing a propensity to maintain a high prevalence rate in Barabási-Albert networks during outbreak scenarios.

Moreover, the conducted research highlighted the importance of precise parameter selection. The delicate balance between the transmission rate and the recovery rate can determine whether an epidemic will die out, stabilize, or spread uncontrollably. This reinforces the critical role of accurate parameter estimation in epidemic modeling and the potential for these simulations to aid in real-world disease control strategies.

The limitations encountered in this study pave the way for several avenues of future research. The need for more structured hypothesis testing to validate the threshold formula, the employment of Monte Carlo simulations for robustness, and the optimization of software for faster simulation runs are all aspects that can significantly enhance the reliability and applicability of our findings. Additionally, further work could explore the inclusion of more network initialization parameters and conduct an in-depth analysis of their effects on disease dynamics.

In conclusion, while our study has advanced the understanding of epidemic spread on weighted networks, it has also opened up new questions and highlighted the need for continued research.

7 Methodology

7.1 Approach

The methodology of this study revolves around a computational approach to simulate and analyze the dynamics of infectious diseases on various network models. The simulations are designed to mirror the complexities of real-world social interactions and disease transmission patterns. By employing network models such as Erdős–Rényi, Barabási–Albert, and Watts and Strogatz, our approach systematically varies parameters within these models to observe their impact on disease spread.

The integration of the SIS epidemiological model with the chosen weighted network structures is a key aspect of our approach. This model provides the framework for simulating the spread of an infectious disease, allowing us to track the infection status of individuals over time. The main goal of the current implementation is to investigate different ways of calculating the epidemic threshold, R_0, in an edge-weighted setup and analyze the conditions under which an infectious disease can persist or die out within a population based on this threshold.

In conducting these simulations, we aim to achieve a balance between computational efficiency and the accuracy of results. We utilize a combination of analytical techniques and numerical simulations to explore the parameter space of the models, ensuring that our findings are robust and can be generalized to a wide range of scenarios.

This section outlines our overall strategy for implementing the simulations and provides the rationale behind our chosen methodologies. Detailed algorithms, computational techniques, and the specifics of the simulations, including parameter selection and data analysis methods, will be elaborated in the following subsections. The approach also underscores the significance of network theory in understanding the complex interplay between network structure and disease transmission.

7.2 The SIS Model

The SIS model is a fundamental framework employed to study the dynamics of infectious diseases within structured populations. In this model, individuals are represented as nodes in a graph, and the edges between nodes denote potential transmission pathways. The acronym "SIS" stands for Susceptible-Infectious-Susceptible, reflecting the primary states an individual can occupy in the model. At each time step, susceptible individuals can contract the disease when connected to infectious individuals through edges, transitioning to the infectious state. Subsequently, infectious individuals have the opportunity to recover and revert to the susceptible state.

The model is governed by parameters such as the transmission rate β, recovery rate γ, and the portion of initially infected individuals denoted by p_0, influencing the spread and persistence of the disease within the population. In

unweighted networks, both β and γ are constant values, and all neighbors of a node i can be infected at time step t with the same probability. The basic reproduction number, denoted as R_0, is a fundamental concept in epidemiology [14]. It represents the expected number of secondary cases produced, in a completely susceptible population, by a typical infected individual during its entire period of infectiousness. It serves as a threshold parameter that predicts whether an infection will spread through the population (if $R_0 > 1$) or die out (if $R_0 < 1$). In the context of network theory, R_0 can be related to the largest eigenvalue of the adjacency matrix of the contact network, as shown by Chakrabarti et al. [15] .

However, this behavior is not expected in real social networks, where interactions between individuals are weighted, suggesting a higher or lower interaction rate, and thus the probability of getting infected is also influenced by the weight between two individuals. Additionally, Ye Sun et al. [9] propose that the actual infection rate would not be affected by the weight distribution on edges if the average weight of the whole network is fixed. There are also different ways to model weighted social interactions because not every disease spreads in the same way [2]. These complexities necessitate a nuanced approach to understanding disease spread in weighted networks, which this study aims to address.

7.3 Weight Distributions

The study of weight distributions in networks is crucial for understanding the complex dynamics of systems characterized by nodes and the interactions between them. The choice of weight distribution impacts the network's topology and, consequently, the dynamics of processes like epidemic spreading. As mentioned before, we explore three fundamental weight distributions: Normal (Gaussian), Power-law, and Exponential, each representing different aspects of connectivity in networks.

7.3.1 Normal (Gaussian) Distribution

The Normal, or Gaussian distribution, is defined by its mean and standard deviation, which shape its symmetric bell curve. The plots in Figure 12 illustrate the impact of varying the mean and standard deviation on the density of contacts. For our simulations, we have chosen a mean (μ) of 3 and a standard deviation (σ) of 1, representing a moderate average number of contacts with low variability.

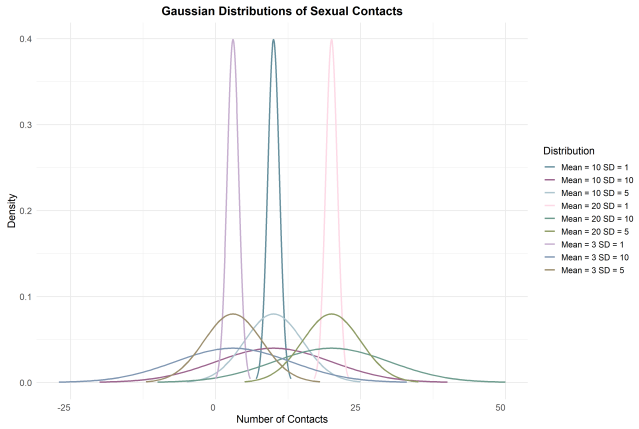

Figure 12: Gaussian Distributions of Sexual Contacts

7.3.2 Power-law Distribution

The Power-law distribution is characterized by the scale parameter x_{min} and the shape parameter α, which control the distribution's tail. Networks with this distribution are known for a small number of nodes with high connectivity, as shown in Figure 13. We use $x_{min} = 1$ and $\alpha = 2.5$ parameters to represent a network with a realistic degree of heterogeneity in node connections.

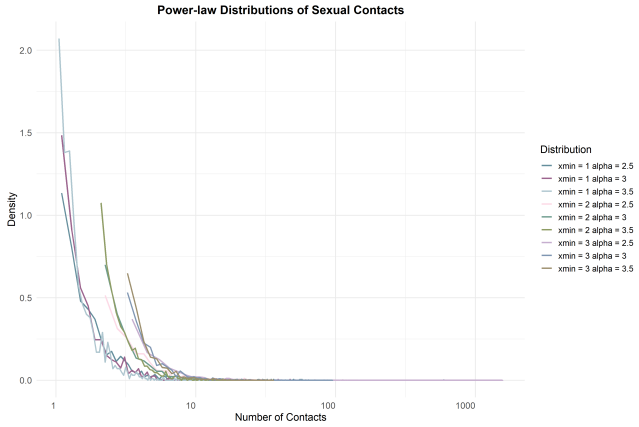

Figure 13: Power-law Distributions of Sexual Contacts

7.3.3 Exponential Distribution

Exponential distribution is defined by its rate parameter λ, which sets the decay of the distribution curve, depicting the probability of contacts between nodes. In Figure 14, we observe how different λ values affect the spread of contacts. For

our further simulations, we opted for $\lambda = 1$, indicating a relatively fast decay rate in the number of contacts.

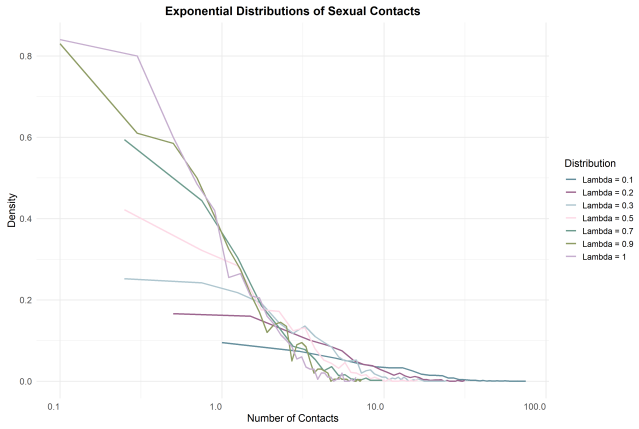

Figure 14: Exponential Distributions of Sexual Contacts

7.4 Network Models

Understanding the structure and dynamics of networks is essential for the study of complex systems. Here we focus on three foundational models that have shaped our understanding of network theory.

7.4.1 Erdős–Rényi Model

The Erdős–Rényi (ER) model is a cornerstone in the study of random graphs. It posits that networks form by connecting nodes with a fixed probability, resulting in graphs where the degree distribution follows a binomial or Poisson distribution in the limit of large network sizes. The ER model exhibits a phase transition at a critical connection probability, where a giant connected component emerges. This model has profound implications for understanding the robustness and fragility of networks, as it demonstrates how subtle changes in network density can lead to significant shifts in connectivity [3].

7.4.2 Barabási–Albert Model

The Barabási–Albert (BA) model captures the dynamics of network growth and preferential attachment. In this model, new nodes are added to the network one at a time and preferentially attach to existing nodes with a higher degree, leading to a "rich-get-richer" phenomenon. This mechanism results in a scale-free network with a power-law degree distribution, meaning that few nodes have a very high degree, while most nodes have a low degree. The BA model

has been instrumental in explaining the ubiquitous presence of hubs in real-world networks and has ramifications for network resilience and the spread of information or diseases [4].

7.4.3 Watts and Strogatz Model

The Watts and Strogatz (WS) model introduces the concept of network rewiring to interpolate between regular and random topologies. It begins with a regular ring lattice where each node is connected to its nearest neighbors and then randomly rewires connections with a probability p. This process can produce networks with high clustering coefficients and short average path lengths, a combination typical of small-world networks. The WS model has been pivotal in understanding the small-world phenomenon in social, biological, and technological networks, where most nodes are not neighbors but can be reached from every other by a small number of steps [5]. The presence of short path lengths facilitates rapid spreading phenomena, such as the spread of diseases or the propagation of information, highlighting the importance of network topology in dynamical processes.

7.5 Description of Implementation

7.5.1 General approach

In our simulations, we integrate the SIS model with weighted network structures to study the spread of infectious diseases. The implementation is carried out through a series of steps:

1. Network Creation: We construct weighted networks based on three different weight distributions: normal (Gaussian), power-law, and exponential. Each network is represented by an adjacency matrix with weights assigned according to the chosen distribution.

2. SIS Dynamics: For the SIS model, we iterate over each node in the network, calculating the probability of infection based on the weighted edges and the infection status of neighboring nodes. Recovery is also modeled probabilistically, with a fixed recovery rate.

3. Threshold Calculation: We compute the epidemic threshold by analyzing the inverse of the leading eigenvalue of the weighted adjacency matrix, which provides a critical value for the infection rate above which an epidemic would be expected.

4. Simulation Execution: We run simulations for a defined number of steps, keeping track of the number of infected individuals over time. This allows us to observe the epidemic's progression and steady-state behavior.

7.5.2 Details of the Weighted SIS Model

The Susceptible-Infected-Susceptible (SIS) model in a weighted network context is implemented as follows:

Network Creation Three types of weighted networks are created, as mentioned in previous subsections: normal (Gaussian), power-law, and exponential distributions of weights. The weights are then assigned to the edges of an unweighted base graph (g).

SIS Simulation The SIS model simulation over a given weighted graph (g) with parameters β (infection rate), γ (recovery rate), initial infection probability p_0, and number of steps is defined as follows:

1. The weighted adjacency matrix W and unweighted adjacency matrix A of the graph g are computed.
2. Each node is initialized as infected with probability p_0.
3. For each time step:
   (a) For each node i:
      i. Infection probability is calculated based on the weights of infected neighbors. If N_i is the set of neighbors of node i, then the infection probability, P_{inf}, is given by:
      $$P_{inf} = 1 - (1 - \beta_{avg})^{|N_i|} \tag{9}$$
      where β_{avg} is the average infection rate considering the weights of the infected neighbors, normalized by the maximum and minimum weights in the graph:
      $$\beta_{avg} = \left(\frac{\text{avg}(w_{infected})}{|N_i|} - w_{min} \right) / (w_{max} - w_{min}) \times \beta \tag{10}$$
      ii. Recovery probability for an infected node is equal to γ.

Threshold Calculations The threshold for the SIS model in weighted networks in our approach is calculated as follows:

- The weighted IBMF (Individual Based Mean Field) threshold is calculated as:
  $$\text{IBMF Threshold} = \frac{1}{\lambda_{max}} \tag{11}$$
  where λ_{max} is the leading eigenvalue of the weighted adjacency matrix.
- The generalized threshold, incorporating recovery rate γ, is given by the leading eigenvalue of the parameterized adjacency matrix:
  $$\text{Generalized Threshold} = W \times \beta + (1 - \gamma) \times A \tag{12}$$
  where W is the weighted matrix and A is the adjacency matrix.

Epidemic Spread Analysis Lastly, the epidemic spread is analyzed by plotting the proportion of infected individuals over time and calculating metrics such as R_0 based on the weighted network properties, meaning that essentially all β's in formulas are transformed considering the normalized average weight.

$$\frac{w_{avg} - w_{min}}{w_{max} - w_{min}} \times \beta$$

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