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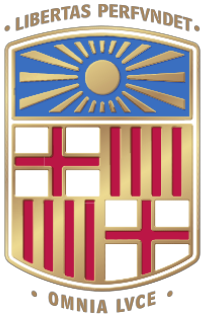
MASTER IN ARTIFICIAL INTELIGENCE



A4. Epidemic spreading on Complex Networks

Complex Networks

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0.1 Introduction

The aim of this work was to examine the nature of epidemic spreading through various types of networks using Monte Carlo simulations of the SIS model, and to understand how varying model parameters – such as spontaneous recovery probability and infection probability – and network parameters – such as number of nodes or probability of connection – impacts the spread.

We elected to use Python, as we are already familiar with the NetworkX library and many other Python libraries for analysis. Although this may have impacted the execution time of our code, we decided from the outset that this was a necessary tradeoff in order to benefit from the familiarity. In order to use NetworkX, it is important to have the version 1.8.0 of scipy, as it uses some functions that are not in the recent versions anymore. Other libraries we used are: numpy, pandas, matplotlib, and ndlib.

0.2 Algorithms, networks and parameters

Using the NetworkX package, it is important to convert the networks into a graph (using nx.Graph), since otherwise it could cause a problem later in the analysis due to the nature of NetworkX.

We also used the ndlib library from G. Rossetti [2017], which provides implementations of several spreading models. From this library, we used the SISmodel.py file as a foundation for our own SIS model implementation, and modified it to suit our own requirements.

We wrote our own implementation to model MMCA for use in comparison against the results from our SIS model. However, ultimately, we were not able to successfully run the code without crashing the session. Therefore, these comparisons will not be featured in the report, although the code can be examined in the included file.

0.2.1 SIS model and Monte Carlo

The Susceptible-Infected-Susceptible (SIS) Castillo-Chavez and Yakubu [2001] epidemic spreading model is characterized by two parameters:

- μ: spontaneous recovery probability.
- β: infection probability of a susceptible (S) individual when it is contacted by an infected (I) one.

The Monte Carlo simulation requires some extra parameters:

- N_{rep}: number of repetitions of the simulation. (100 and 50 repetitions)

- $\rho(0)$: initial fraction of infected nodes. (20% of initial probability to get infect)
- T_{max}: maximum number of time steps of each simulation. (900 time steps)
- T_{trans}: number of steps of the transitory. (100 steps)

For the Monte Carlo simulation we followed the suggested values for these parameters. In the beginning we tested some simulations with 100 repetitions, but reduced to 50 to take the final averages.

0.2.2 SIS with MMCA

The Microscopic Markov-Chain Approach Gómez et al. [2010] work with "microscopic" variables and uses the following equations for:

$$p_i(t+1) = (1-q_i(t))(1-p_i(t)) + (1-\mu)p_i(t) + \mu(1-q_i(t))p_i(t) \tag{1}$$

where $q_i(t)$ is the probability of node i not being infected by any neighbor

$$q_i(t) = \prod_{j}^{N}(1-\beta r_{ji} p_j(t)). \tag{2}$$

where:

- p_i: probability of node i being infected
- r_{ij}: probability of node i contacting node j

The macroscopic order parameter is given by the expected infection density ρ, computed as

$$\rho = \frac{1}{N}\sum p_i \tag{3}$$

To compare how the SIS model works in different environments, we selected to use a Barnabasi-Albert to represent a scale-free network, a Watts-Strogatz Network, and two real networks: dolphins and zachary_uw.

For the Barnabasi-Albert network we did three configurations of it, varying the size and the number of edges to attach from a new node to existing nodes constant. With graphs of size 500 nodes, we used both 2 and 5 for the latter value, and for the graph of size 750 nodes, we used only 2 edges.

For the Watts-Strogatz network we did four configurations, 500 and 750 nodes and the k value of 2 and 4. We kept the rewiring probability constant at 0.5.

We decided to pick dolphins and zachary_uw real network because of their size, and avoid the other real networks because even the next smallest one has more than 3000 nodes. Given the execution time of our networks with significantly smaller numbers of nodes, we found these larger networks to be infeasible.

0.3 ρ(β) plots

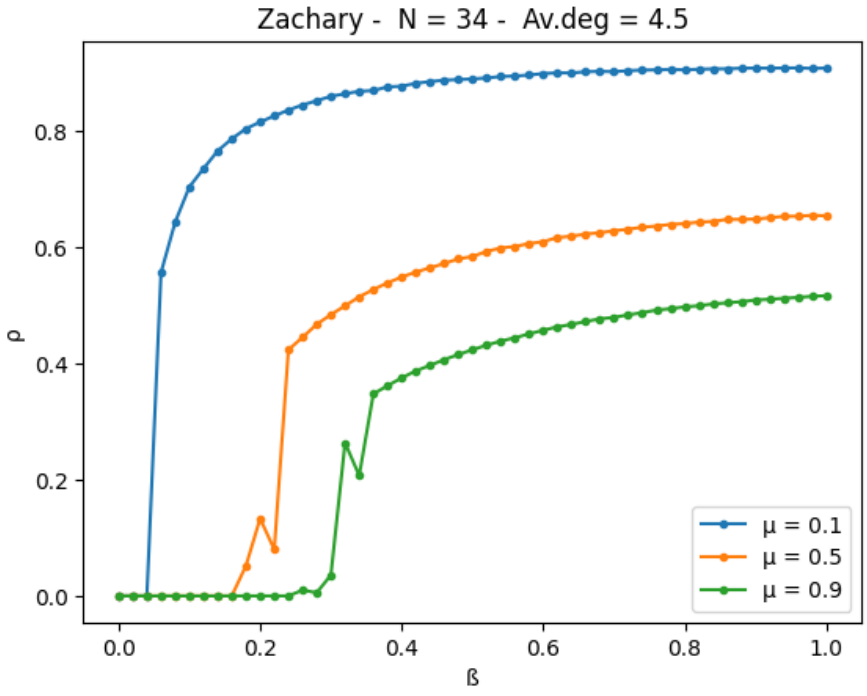

Figure 1: Comparison of the ρ vs β with the Zachary's real network

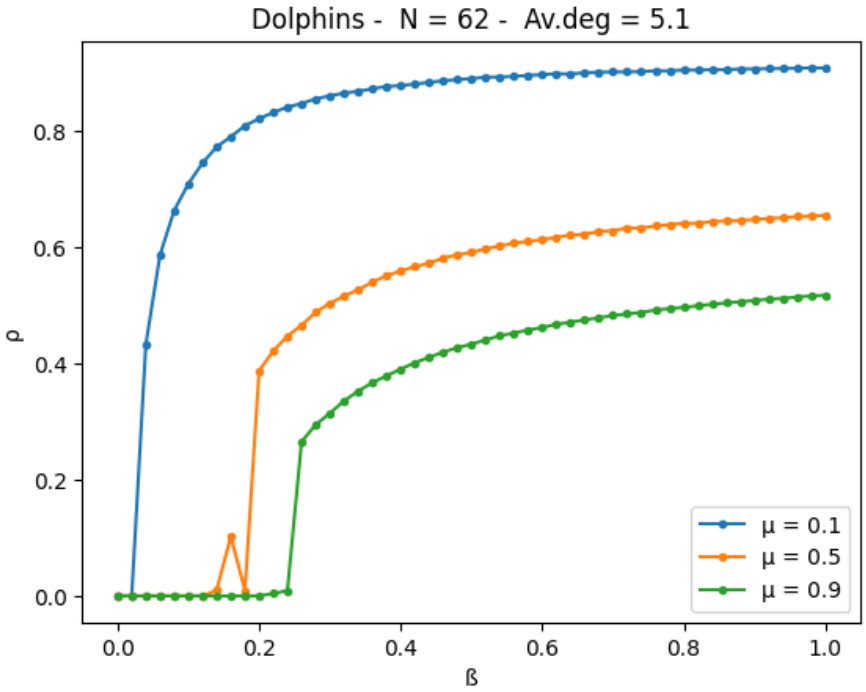

Figure 2: Comparison of the ρ vs β with the Dolphin's real network

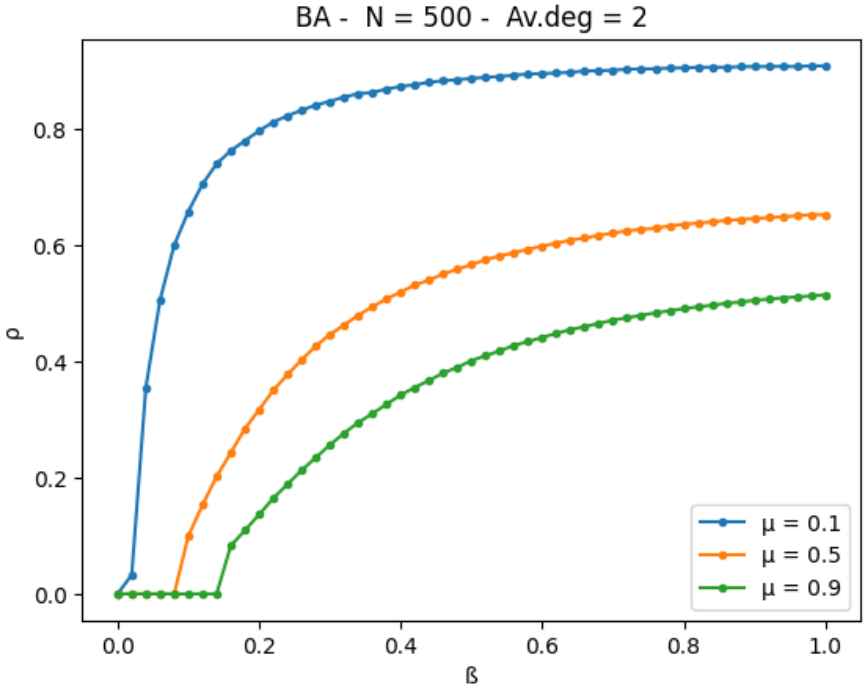

Figure 3: Comparison of the ρ vs β with the Barabasi-Albert network (500 nodes and 2 edges to attach)

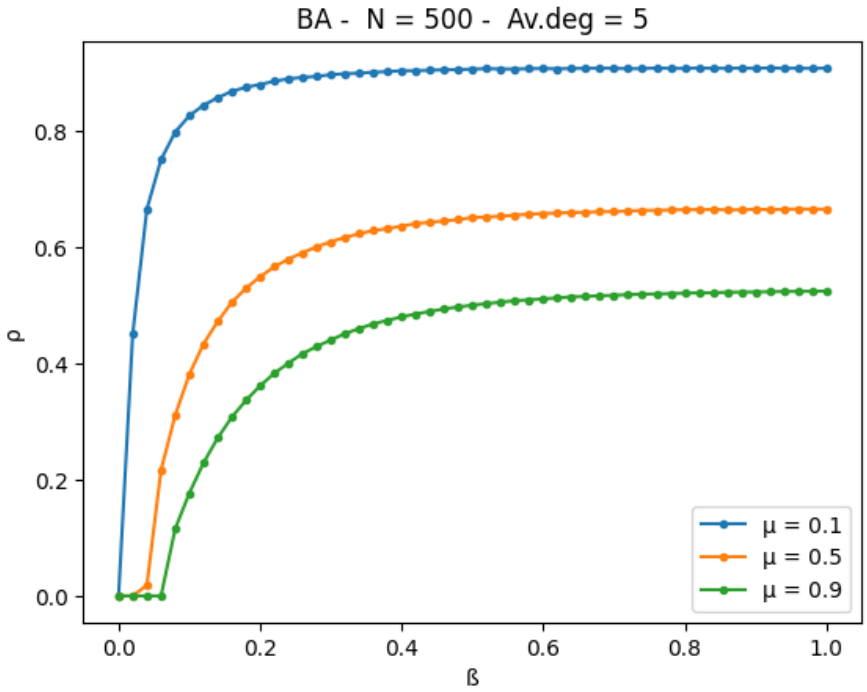

Figure 4: Comparison of the ρ vs β with the Barabasi-Albert network (500 nodes and 5 edges to attach)

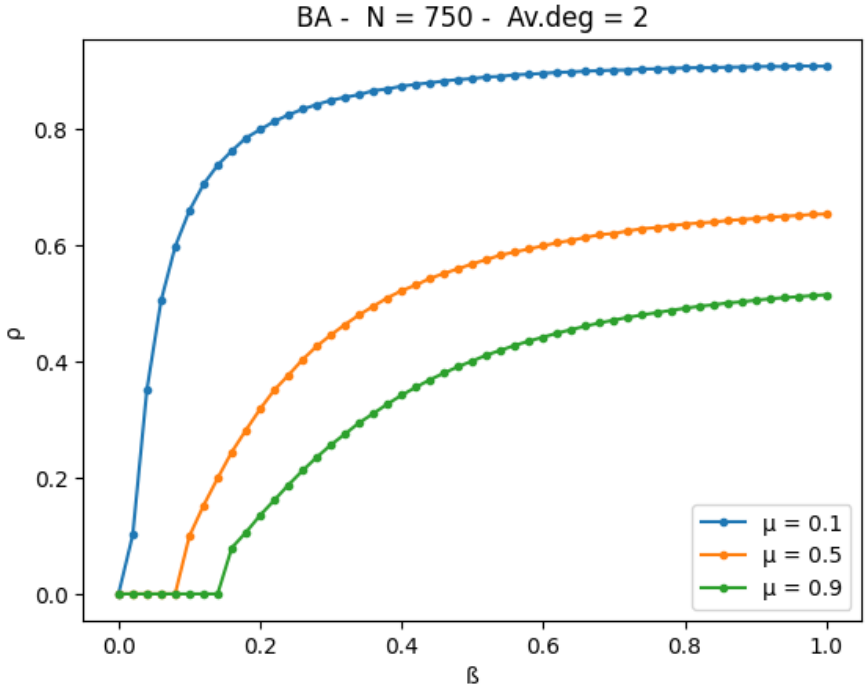

Figure 5: Comparison of the ρ vs β with the Barabasi-Albert network (750 nodes and 2 edges to attach)

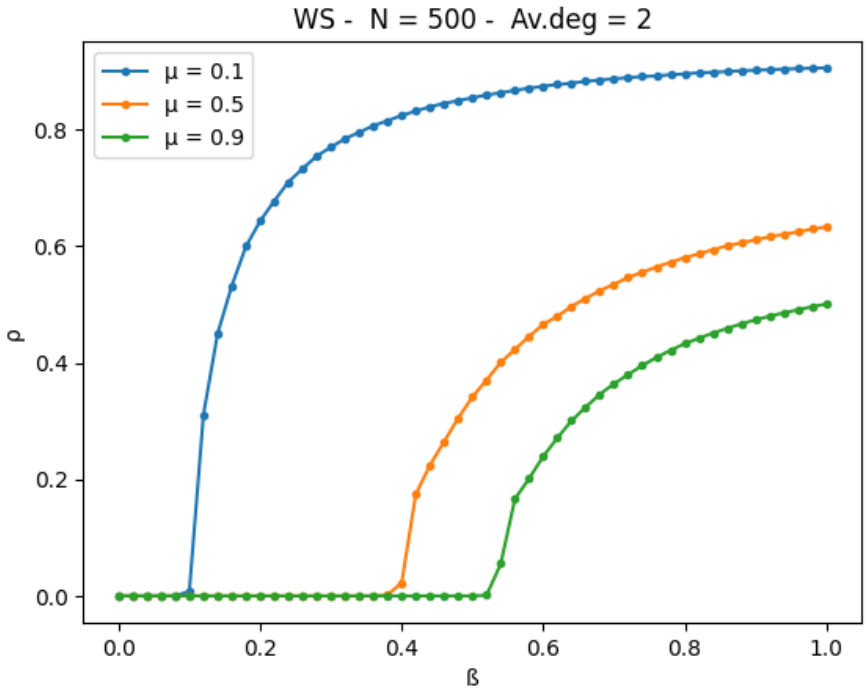

Figure 6: Comparison of the ρ vs β with the Watts-Strogatz network (500 nodes and 2 connections/node)

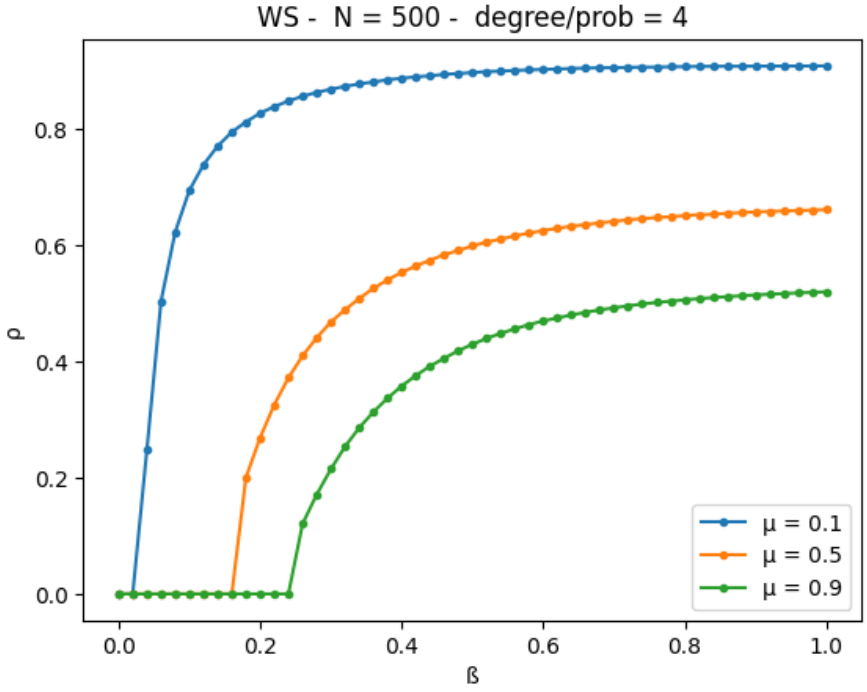

Figure 7: Comparison of the ρ vs β with the Watts-Strogatz network (500 nodes and 4 connections/node)

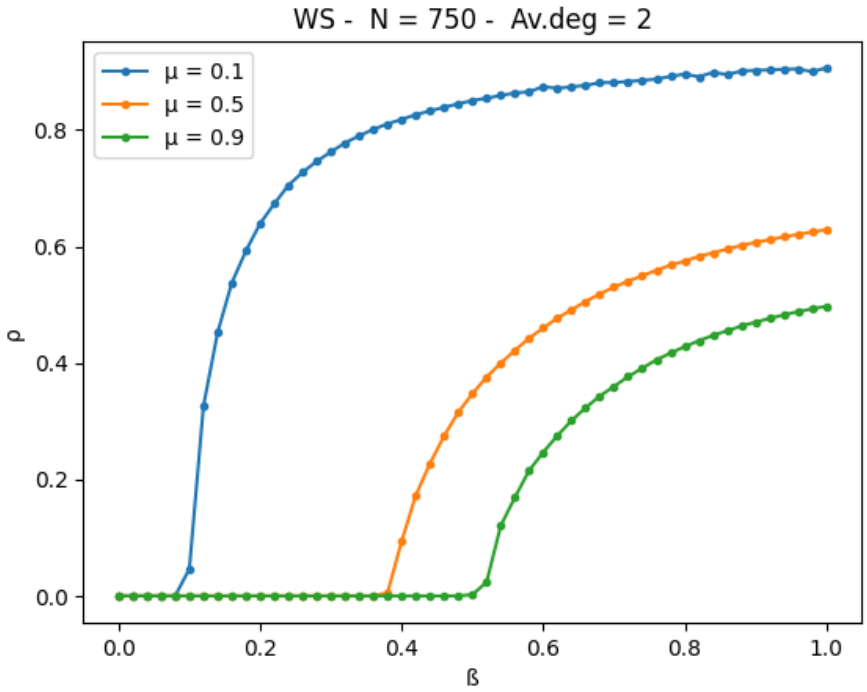

Figure 8: Comparison of the ρ vs β with the Watts-Strogatz network (750 nodes and 2 connections/node)

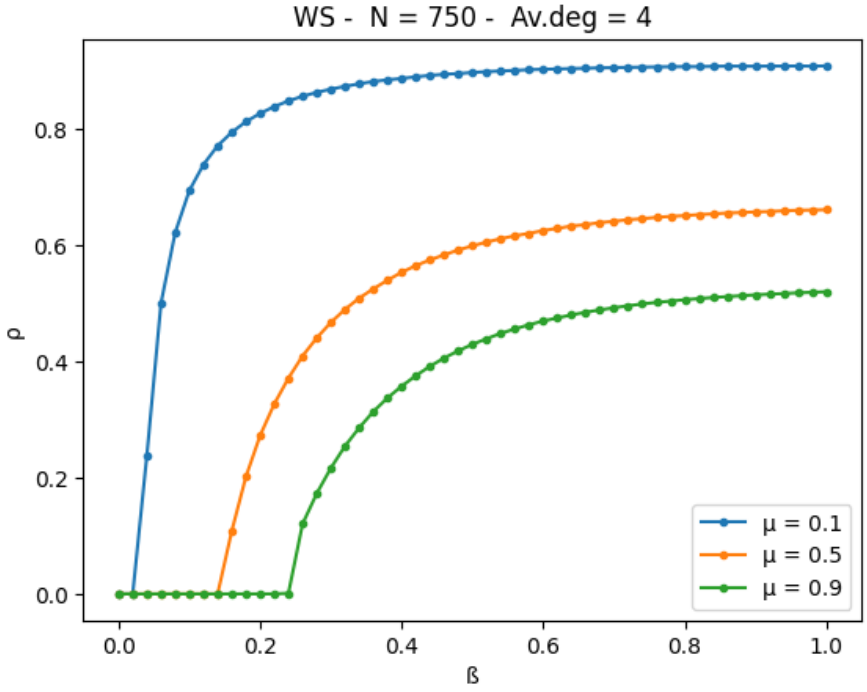

Figure 9: Comparison of the ρ vs β with the Watts-Strogatz network (750 nodes and 4 connections/node)

0.4 Discussion

In this work, we learned about the steps required for using the SIS model to simulate epidemic spreads, and how to implement MMCA.

Furthermore, we saw how changing the μ and β parameters changed the results. In general we could see a similar tendency for the same value of μ. For smaller values of μ, the value of ρ grows much more quickly, which is consistent with the idea that a low recovery rate will enable quicker spread of disease. Similarly, with higher values of μ, the high rate of recovery prevents disease from spreading quickly when the β values are low. Even as the β values increase, the increase in ρ is much less sharp than the increase when the recovery rate is low, stabilizing closer to around 0.5 depending on the network, as opposed to a ρ of almost 1.0 with the low values.

Another observation was that in our manually created networks, the curves were much smoother, with no overly sharp increases as seen in the real networks. This might be because of the more controlled structure in the manually created networks. Alternatively, it is interesting to note that, for example, between the Dolphins and the Barabasi-Albert network with 500 nodes and average degree of 2, the number of nodes and degree seem to have an effect on the shape of the curve. With a higher number of nodes and a lower degree, there are smoother curves. This is likely because the lower degree means there are fewer connections, meaning less likelihood of infection, and the larger number of nodes mean that each new infection contributes less to the overall ρ value. In a smaller network with a higher degree, the ρ value increases more sharply because the disease spreads more quickly between nodes even with low β values.

In the future, we would make some changes to improve our results and be able to draw more in-depth conclusions. For example, using a compiled language instead of an interpreted language like Python, would likely decrease our execution time and be more efficient overall. This would also likely have enabled the successful execution of our MMCA code, which would have contributed to further analysis. Finally, with more efficient execution, we could have explored more graph configurations with larger sizes and more connections to confirm whether the trends we identified are consistent.

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