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Effectivity of various vaccination scenarious on scale-free networks

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Declaration of Authorship

I, Mykola KYRYCHENKO, declare that this thesis titled, "Effectivity of various vaccination scenarious on scale-free networks" and the work presented in it are my own. I confirm that:

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- Where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated.
- Where I have consulted the published work of others, this is always clearly attributed.
- Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work.
- I have acknowledged all main sources of help.
- Where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself.

Signed: _____

Date: _____

"The First Law: Performance drives success, but when performance can't be measured, networks drive success."

Albert-László Barabási

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by Mykola KYRYCHENKO

Abstract

Nowadays because of technological progress many fields of study get a second wind. This is because many of them need complex computations and a lot of features were analysed at the level of the idea. In my opinion networks are one of them. Alongside with technological progress many fields such as social networks, financial networks and computer networks are developing. Almost all the spheres of life touch those fields. Because of this connectivity many concepts could be considered under the perspective of networks. This perspective can explain many common concepts in the math language. Many common questions can be answered by numbers and those answers are more accurate. Moreover the fields considered as networks have a lot in common and the leading ideas of one sphere could be applied to the other sphere.

Acknowledgements

I would like to thank my supervisors Viktoria Blavatska and Yurij Holovatch for helping and supporting me during this work. I would like to thank my parents who were always interested in my personal growth and sacrificed a lot of mental and material resources for my future.

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Chapter 1

Introduction

In this work we analyse the effectiveness of various vaccination scenarios on scale-free networks. Epidemic spreading has been a highly discussed topic recently with millions of people dying every year due to epidemics. The importance of the topic has been further proven by COVID pandemic, with minor slowdowns of the pandemic saving numerous lives. Spreading of the epidemic was analysed with help of complex networks. We are simplifying complex networks by representing them as graphs with nodes being individuals and links representing individuals' relations. Each individual(node) is described by a specific state at a given time. Those nodes and edges were viewed under cellular automata topology. In other words we will consider the state of each individual (agent-based stochastic approach) and a spreading process via predefined rules. We have implemented many vaccination methods on the different networks and models. Spreading processes behave differently according to the generation method but have many features in common. For better understanding of those features we implemented different compartment models to enhance understanding of spreading processes. The goal of this work is to analyse the spreading processes using various network types. Consequently, to discover patterns and identify ways to slow down the spreading.

1.1 Spreading processes

One of the options to slow down the pandemic is to figure out the factors determining speed of spread and influence those factors. This could be done via mathematical modelling of the network. In Alun L.Lloyd and Robert M.May paperLloyd and May, 2001 authors analyse the spreading processes of computer viruses as well as biological viruses. Common features were found to unite both spreadings under one concept. The concept to study epidemic dynamics and spreading processes was called compartmental models. A compartment model provides mathematical tools to study transformation between different compartments of a system. Kermack and McKendrick layed a foundation for epidemics modelingKermack and McKendrick, 1927. The model is created using following steps. All individuals in a network are labeled according to the model parameters we chose. Speaking in terms of graphs, nodes of a graph are labeled with predefined values. At the same time, nodes might change their states and labels via predefined scenarios. Those scenarios and the state changing are described by simple differential equations with values β and γ.

$\beta > 0$ is the contact rate. This rate shows how many susceptible individuals could be infected by one infected individual during a given period of time with the whole population being susceptible except one individual. Basically it represents the probability of transmitting infection between infected and susceptible individuals.

$\gamma > 0$ is a recovery rate. Basically it is a probability for an infected individual to recover. It can also be viewed as the average duration of a disease (an average time an individual needs to recover). A basic reproduction number $R_0 = \beta / \gamma$ is a ratio that shows the expected proportion of infected individuals in a population where all individuals are susceptible except one Dietz, 1976.

For example, let's consider a kettle with water as a closed system. For a period of time we are boiling it. At the beginning, all water molecules are in a state W-water. During the boiling process some of the W entities with some probability turn into steam and change its state to S. Worth mentioning that S entities can not turn back into W because a steam in such environment can not transform back to water (the transport rule of a compartment model). At the end of the process all molecules are in state S. In this work we are considering SI, SIS and SIR models. Linda J. S. Allens workAllen, 1994 and A. Lajmanovich and J. A. YorkeLajmanovich and Yorke., 1976 proposed a deterministic model in a discrete-time case alongside with defining the different compartment models which are implemented in this paper.

1.1.1 SI model

The basic and the simplest model is named SI model according to the two possible states which individuals might have (S-susceptible and I-infected). This model is used to describe micro parasitic infections where individuals don't get immunity. It means that individuals never recover and have lifelong infections. The model is considered during some time T. Let's consider a case with a static predefined population of size N. Most individuals initially are in a state S, the predefined number of individuals starts at I. In any given time $0 \leq t \leq T$ the following rule takes place.

$$S(t) + I(t) = N$$

This means that a fraction of susceptible individuals is proportional to the fraction of the infected individuals. The example with kettle is actually a SI model.

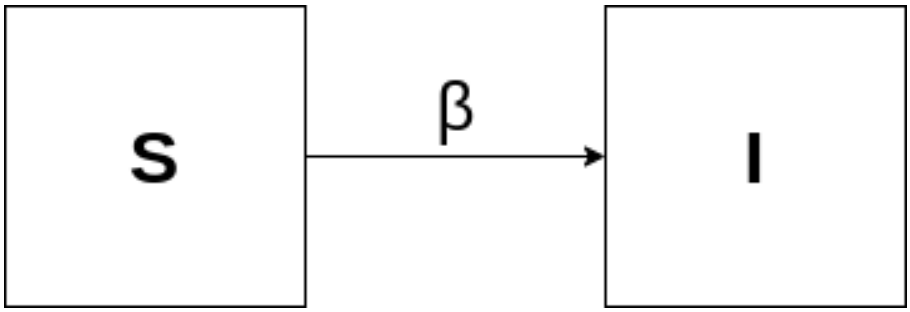

FIGURE 1.1: SI diagram

1.1.2 SIS model

SIS model is a compartment model similar to SI where individuals might be in two possible states S, I. In this model the population hardly leaves the infected state. The main difference compared to SI model is that after an individual becomes infected there is still a probability to change his state to S. The significant impact of studying and analysing spreading processes in SIS and SIR models was introduced in Boguñà and Pastor-Satorras, 2002Pastor-Satorras and Vespignani, 2001Pastor-Satorras and Vespignani, 2002Y.Moreno, Pastor-Satorras, and Vespignani, 2005Moreno and Vázquez, 2003Barthélemy et al., 2005. Population in this model does not develop immunity to the disease. Most sexually transmitted diseases could be described via SIS model. Even RNA viruses such as coronavirus or the simple cold due to fast mutation also fall into a category analysed via the SIS model. Let's consider an environment where each member of a population is born in S state. In that case the differential equations that describe spreading process look like:

$$\frac{\partial S}{\partial t} = -(\beta/N)SI + \gamma I$$

$$\frac{\partial I}{\partial t} = (\beta/N)SI - \gamma I$$

Where $N = S(t) + I(t)$ – the population size. Basic reproduction number $R_0 = \beta\gamma$.

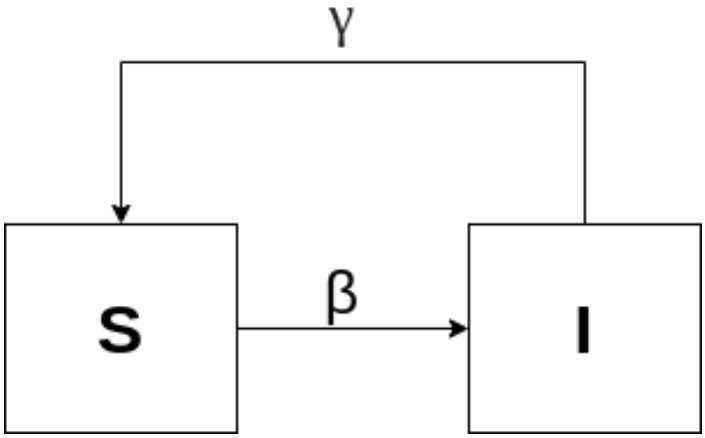

FIGURE 1.2: SIS diagram

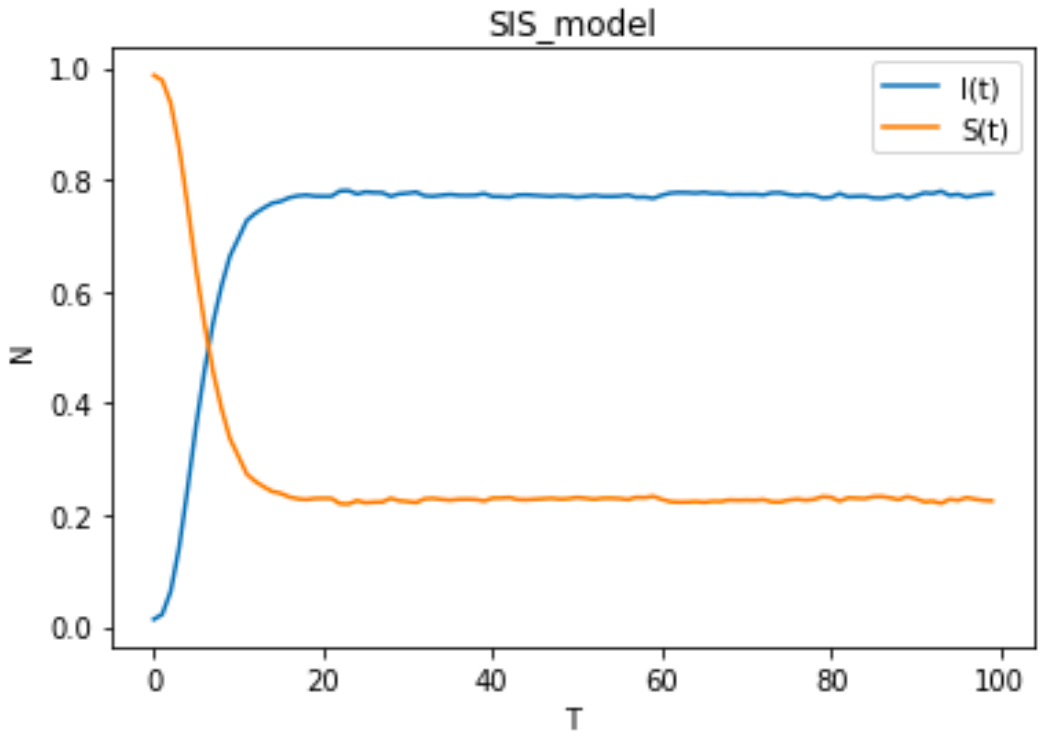

FIGURE 1.3: custom SIS graph

1.1.3 SIR model

One of the most widely used epidemic models is the SIR model. In a SIR model, all individuals in the population with size N have three possible states: S-susceptible, I-infected, R-recovered. In this model individuals are changing their state via the following rule, S goes only to I and I goes only to R. R state is considered as a recovered and is fully removed from the spreading process (math has no mercy while treating recovered and "dead" individuals in the same way). Compared with two other previous models, in the SIR model individuals develop immunity (state R). This model could be applied to childhood diseases (measles, chickenpox). Let's consider an environment where the whole population is susceptible to some disease (S state). We assume that birth rate is equal to death rate. The dynamics of a SIR model is described by the next equations:

$$\frac{\partial S}{\partial t} = -\beta SI/N$$

$$\frac{\partial I}{\partial t} = \beta SI/N - \gamma I$$

$$\frac{\partial R}{\partial t} = \gamma I$$

Where $b \geq 0$.
$N = S(t) + I(t) + R(t)$ - the population size.
As we assumed that birth rate is equal to death rate δ:
Basic reproduction number $R_0 = \beta\ /\gamma$.

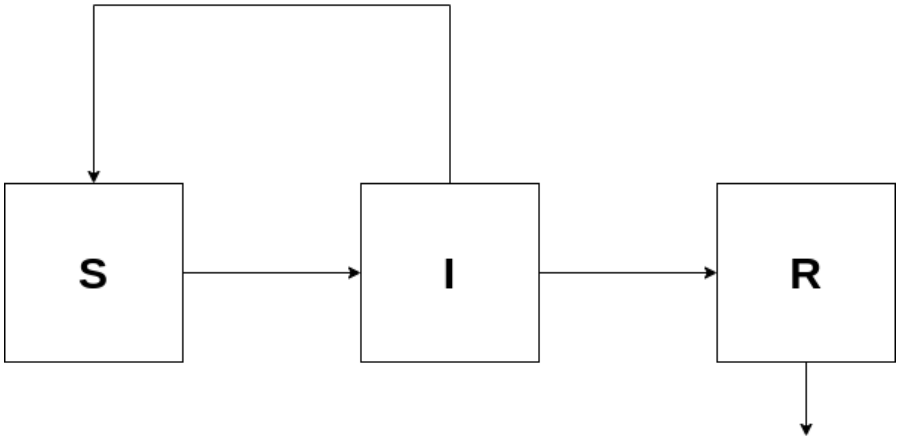

FIGURE 1.4: SIR diagram

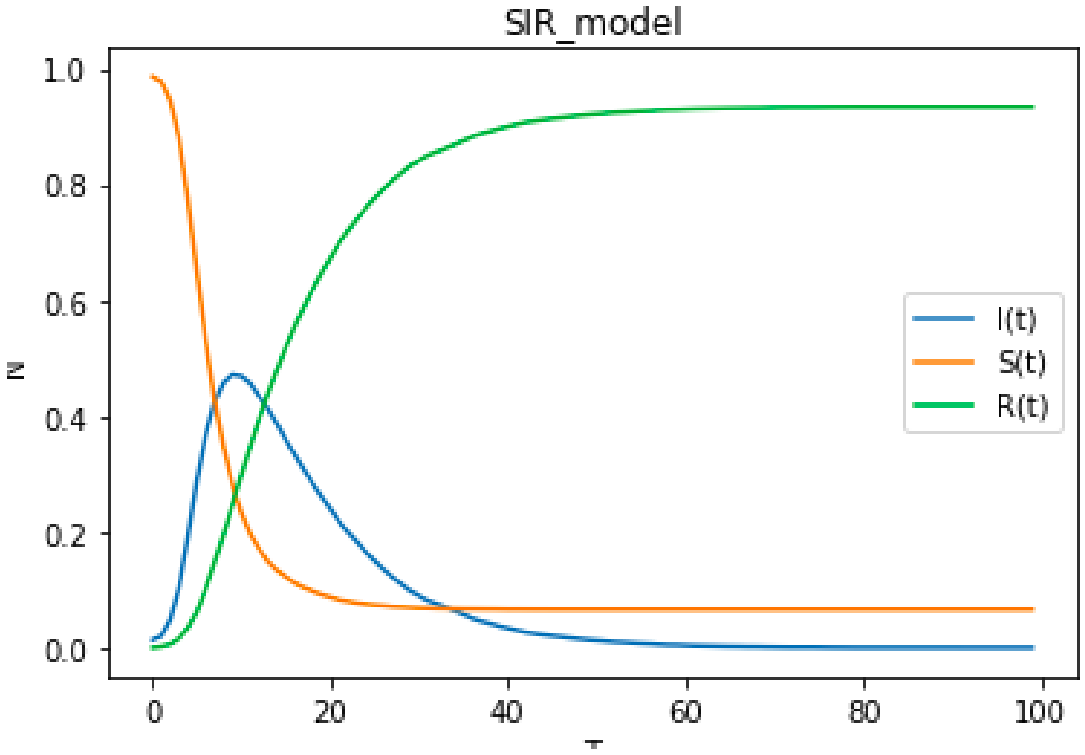

FIGURE 1.5: custom SIR graph

1.2 Complex networks

The idea of complex networks is applied in various fields including social networks, biological networks, financial networks,etc. Most interactions between entities or individuals happening within complex systems and associated relationships could be simplified and further represented by graphs [14]. Not long ago following the development of hardware scientists masivelly began to study complex networks. The first works are dated at the end of the previous century. Main advantage of this approach is an ability to analyze complex systems using advanced mathematical tools and consequently derive insights that are usually hidden from the observer. In a graph, individuals are represented by the graph's nodes and their interactions

by links between the nodes(edges). Social networks or WWW do not have any restrictive rules for individual relations (some of the individuals have more neighbors and respectively more relations) meaning that such types of networks have no scale. Complex networks, unlike tree graphs or mesh graphs, are classified by non-trivial features and complex structure. Complex networks are also called scale-free networks (SF) with node degree distribution p(k) obeying a power-law:

$$p(k) \sim k^{-\lambda}$$

ResearchCohen, D.ben-Avraham, and Havlin, 2002 conducted that analyzed networks including World Wide Web(WWW), Social Networks or even complex societal networks are described by

$$2 \leq \lambda \leq 3$$

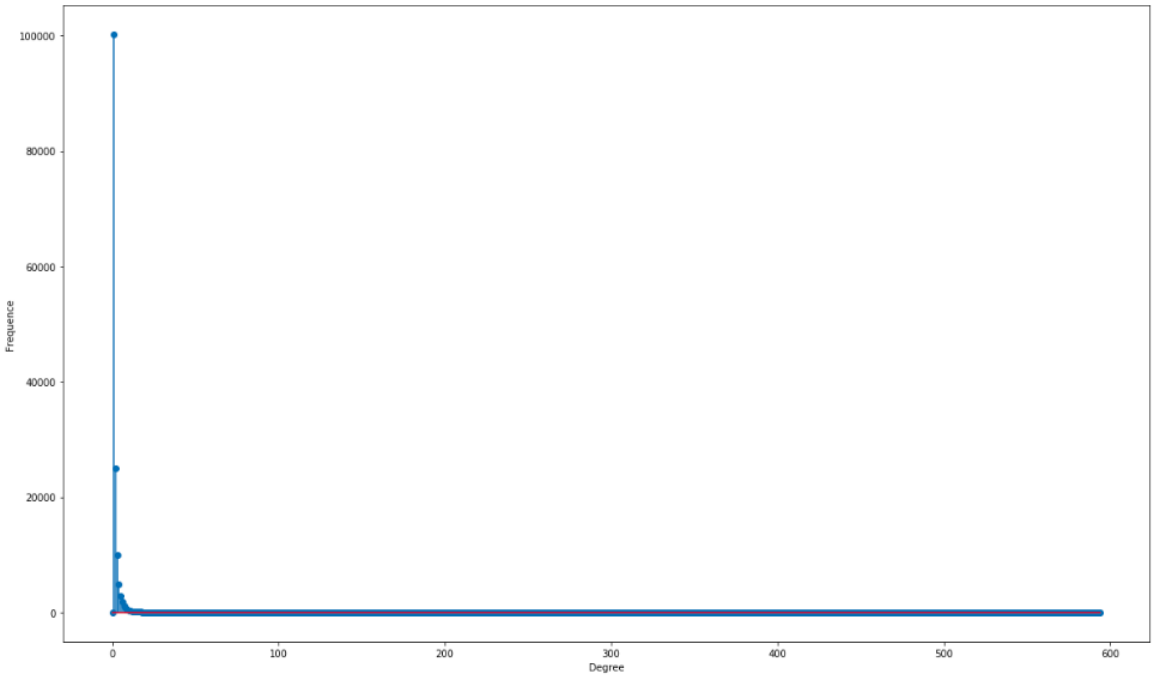

FIGURE 1.6: power law distribution of custom BA graph

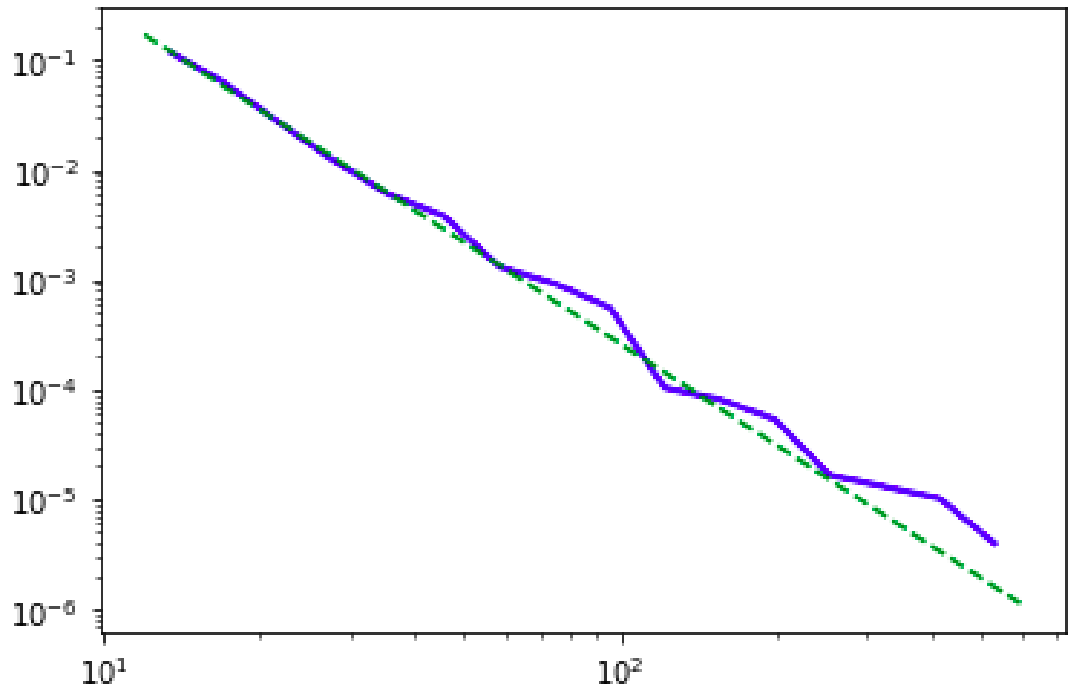

FIGURE 1.7: log form of power law distribution of custom BA graph

First graph shows a scale-free network depicted by Barabasi-Albert Graph (BA)Barabási and Albert, 1999. This model has λ parameter $\lambda = 3$.
BA networks also follow two rules:

- Growth
- Preferential attachment

Growth implies that a new node always could be added. It differs in comparison with random network models where the number of nodes is fixed. In BA networks the number of nodes always grows. For instance, following networks obey mentioned rules and can be described using BA graphs.

1) In WWW by creating a new web page that is connected to other web pages we add an additional node and expand the network.

2) In society by giving birth to a child who will communicate with other individuals we add an additional node and expand the network.

3) In the science world by starting a new thread of research the network built by papers and researches start to expand.

Preferential attachment means that a newly created node will be connected to popular nodes with higher probability than to the unpopular ones. Popularity is defined by the number of connections node has. Popular nodes are called hubs. This is different to random network models where node's neighbours are chosen randomly. For example:

1) A new individual more likely will create an account in Facebook (the most popular social network) than in other social networks. At least because the chance that he or she is familiar with Facebook is higher.

2) The more a football player plays in a regular season the higher chance that he or she will play in a finals just because of his or her confident performance.

3) The more close people of an individual are customers of a specific bank the higher chance that he will be a new customer of that bank. That bank will look more secure to put money in and he or she will tend to be a new customer of that specific bank rather than put his or her money to unknown random bank.

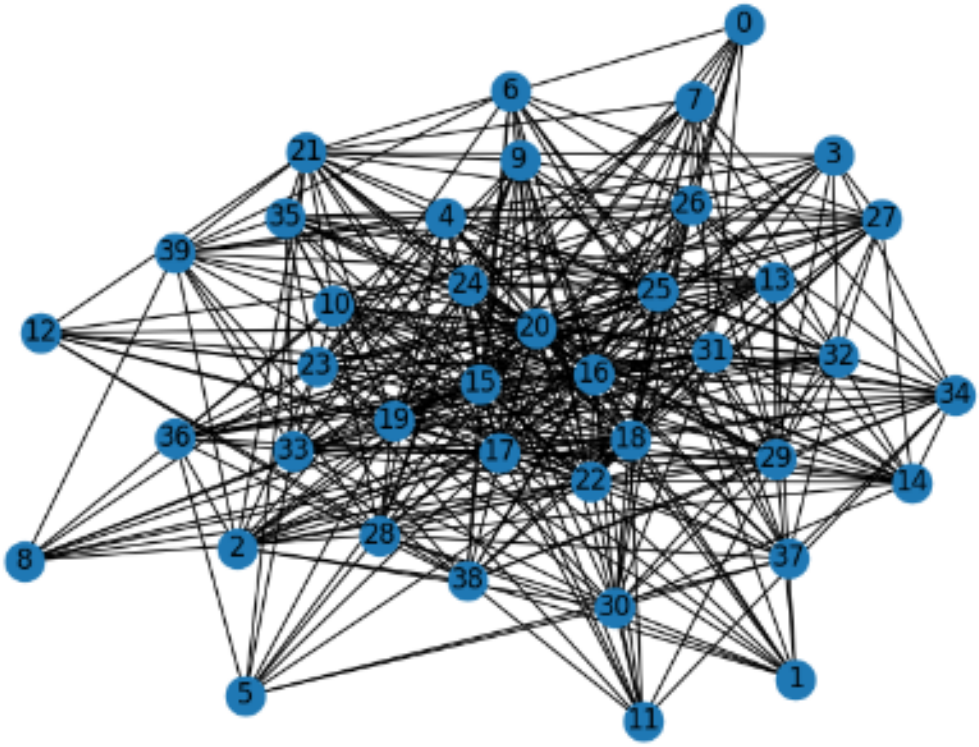

FIGURE 1.8: BA graph 40 nodes

1.3 Cellular automaton

A cellular automaton(CA) is a discrete mathematical model described by the cells grid and the relation rules (the rules of the cells state changes). Wolfram in his work Wolfram, 2002 lays the groundwork of a modern understanding of the cellular automata backbone. Basically it is a model which can explain a system with discrete time and space. The set of states of the cells is predefined. The states could be considered in a digital way or any other. Cells of that system could be placed in different ways. The number of cells (the population size) could be either finite or infinite. There are a lot of types of CA but in this work we are interested in a deterministic one. As mentioned previously, the time is considered to be discrete. In a specific point of time a cell could be in one specific state from the system's set of states. A state of a cell depends only on the states of its neighbors and its own previous state (a state in the previous period of time). The concept of a neighbor is a key concept in CA theory because it could be viewed as a main tool that sets the dynamics concepts. The states are changing only following the predefined rules of a system. Such a system is called memoreless because the cells "remembers" only the last state in the previous point of time. This is similar to DTMC(a discrete time Markov chain) where time and states are discrete. There are a lot of similarities with previously described compartment models. Let's consider how SI, SIS and SIR compartment models are implemented following the mentioned scenario. Let's say that we analyse models during some discrete time T. The population has a fixed size N. That means that for SI and SIS models the fractions are described in the following way:

$$S(t) + I(t) = N$$

For the SIR model:

$$S(t) + I(t) + R(t) = N$$

Where $0 \leq t \leq T$
The whole population changes the states every time stamp. Consider a δ to be a state of population.

$$\delta = \{S, I, R\}$$

Time is discrete and changes: r->$t+1$.According with time change, the i^{th} individual ($i = \{0 \dots N\}$) changes its state as $\delta_i -> \delta_{i+1}$ with respect to the rules of the system. Algorithm of the change for each model will looks like

SI model

- choose individual i
- if $\delta_i(t) = S$ then the state with probability β becomes infected $\delta_i(t+1) = I$
- if $\delta_i(t) = I$ do nothing

SIS model

- choose individual i
- if $\delta_i(t) = S$ then the state with probability β becomes infected $\delta_i(t+1) = I$
- if $\delta_i(t) = I$ then the state with probability γ becomes susceptible $\delta_i(t+1) = S$

SIR model

- choose individual i
- if $\delta_i(t) = I$ then the state with probability γ becomes susceptible $\delta_i(t+1) = R$ else with a probability β one of the susceptible neighbor(j) becomes infected ($\delta_i(t) = S$ -> $\delta_i(t+1) = I$)
- if $\delta_i(t) = S$ or $\delta_i(t) = R$ do nothing

Chapter 2

Literature review

In this section I would like to introduce works which were used fundamental for our research and identify gaps in existing researches that our work will be exploring. There are plenty of works where authors investigate spreading processes on complex networks. The first work I analysed was written by Viktoria Blavatska and Yurij HolovatchBlavatska and processes, 2020. In their research SI, SIS, SIR models were implemented via cellular automata algorithm. The dynamics of the spreading processes were shown by comparing different values of contact rate and using the critical value of basic reproduction number as a threshold (value that is a border of disease-free equilibrium and endemic equilibrium). Non-homogeneous environment was considered where individuals take part in a spreading process similar to a case where a part of a population has already gotten sick and gained some immunity to the disease. On the other hand it is analogical to a vaccination process. Effectiveness of each corresponding scenario was compared. In the models the probability for susceptible individuals to become infected is calculated dynamically depending on the number of its infected neighbors. Linda J. S. Allen in her workAllen, 2008 introduced three different methods for formulating stochastic epidemic models. She implemented SIS and SIR deterministic models in order to analyse those stochastic modeling processes: DTMC (discrete time Markov chain), CTMC (continuous time Markov chain) and SDE (stochastic differential equation). I was mostly interested in the DTMC model as its idea is similar to CA(cellular automata) but the other approaches gave me a better understanding of the basic concepts of spreading processes. Besides a common case where a population has fixed size, the author considers a case with a variable population size. She introduces the concepts of a birth rate and death rate which depend on the population size. For example the equation for the SIS model in this case will look like:

$$\frac{\partial S}{\partial t} = -(\beta/N)SI + (\beta+\gamma)I$$

$$\frac{\partial I}{\partial t} = (\beta/N)SI - (\beta+\gamma)I$$

Where $b > 0$ - the birth rate.
Besides analysing the final size of a population she underscored the next concepts: outbreak of a disease and a duration of epidemic. The different parameters of a model that cause the outbreak were introduced and illustrated via graphs. The expected duration of an epidemic was analysed in the SIS model. For the stochastic SIS model the probability of absorption is equal to 1 because there is no immunity in a system but depending on the parameters the time of an absorption is different.

The work that helped me the most in understanding the construction algorithms of the graphs obeying power-law distribution was introduced by Holovach et al[14].

Authors introduced the models of network construction (Erdős–Rényi model, Watts–Strogatz model, Barabasi-Albert model). The algorithms of constructing those models were explained in depth which helped me to build a complex network. Also as a part of Watts–Strogatz model concept of a small world model was introduced where ansamble graphs are traced.

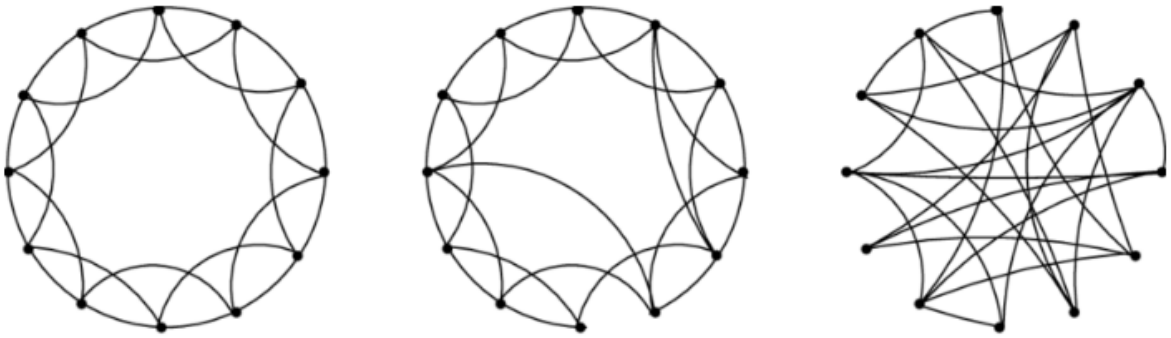

FIGURE 2.1: The Watts-Strogatz model for small-world graphs.

Authors introduced two mechanisms that are the base to construct Barabasi-Albert graph(growth and preferential attachment) and a step by step algorithm of building this graph. Also authors introduced a randomization method for building a complex network using which concepts I was building graphs for my simulations. In addition to the above, the authors introduced the main features of graph theory (clusters, graph ensembles, transitions in graphs, graph energy, betweenness centrality...) and their applications that helped me to better understand the "importance" of specific nodes in a network which helped me to implement different vaccination methods.

Chantan Nguyen and Jean M. Carlson in their workNguyen and Carlson, 2016 introduced real-time vaccination in a stochastic SIR model. They tried to simulate a real world case where vaccine deployment delay, limited vaccines and other features have been taken into account. They considered the epidemic spreading from city to city where those cities could be viewed from graph perspective as clusters (analysis of subpopulation). At the end they identify a tradeoff between those features and propose methods and protocols of vaccination. They introduced a concept of coupled populations. It was explained by the real world case of two cities where an individual from one city can contact individuals from the other city. The population of cities remains the same even if an individual from city A in a specific point of time is located in city B. Obviously they considered that the amount of those individuals is very small. They considered the outbreak as a threshold and took vaccination actions depending on the parameter values that caused the outbreak. Also one of the real world features that they introduced is herd immunity which occurs when a critical fraction of the population is vaccinated (basic reproductive number becomes such that the epidemic process begins to die out). Using that value of basic reproductive number as a lower threshold they analyse a minimum number of vaccinations needed in order to overcome the disease in a small period of time. The optimal method of vaccination is determined by minimizing the expected final size of infection with respect to the fraction of vaccine allocated in each city. That work clarified the non-static methods of vaccination that inspired me to implement

my own protocols on the SIS model. The results of their work are illustrated on the Figure below

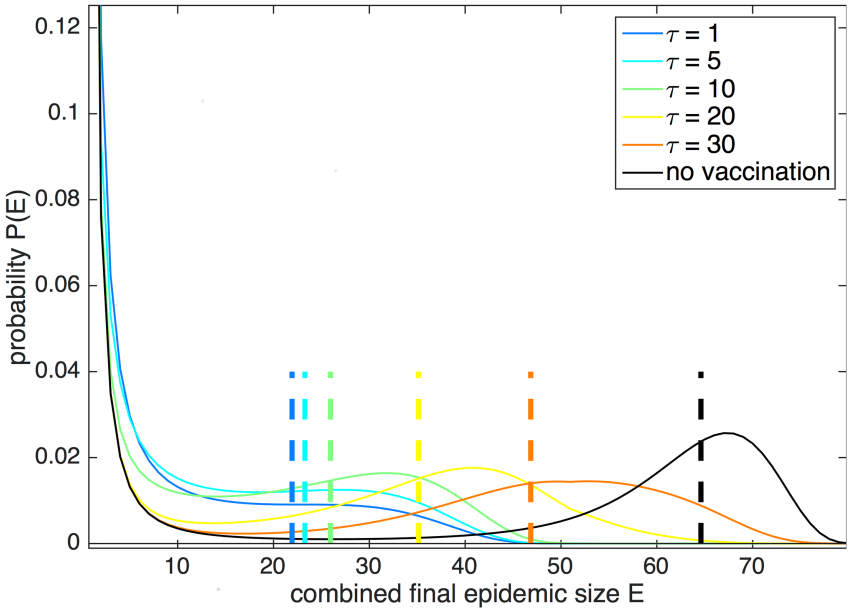

FIGURE 2.2: SIR vaccinated

Where the section of a graph where function grows could be considered as an outbreak.

Chapter 3

Implementation

In this section I would like to describe how the networks used were constructed and the vaccination protocols implemented based on the approaches described in the previous section. This section is divided into two parts. The first part is the algorithms of the complex network construction and the second one is the vaccination protocols for SIS and SIR models which were introduced in **1.1.2** and **1.1.3**.

3.1 Barabasi-Albert model

The first complex network I used was Barabasi-Albert graph. It's λ coefficient is equal to 3. The detailed algorithm of building BA model is introduced hereBarabási and Albert, 1999Barabási and Albert, 1999. As was mentioned in **1.2** that algorithm has two base rules.

- Growth is starting from the small population of individuals(nodes denoted as n_0). At every time step a new node $n \leq n_0$ is added to a network
- Preferential attachment.
  The probability of the new node to become a neighbor of an existing node i depends on the node degree k_i of a node i.

$$P(k_i) = k / \sum_{j}^{N} k_j$$

  Where N is a population size of an observed point of time.

3.2 Custom network

To generate a scale free network we used a method called configuration modelBender and Canfield, 1978Molloy and Reed, 1995. It is a method of generating random graphs with predefined degree sequence. At the start we have some population with size N and with no relations (all the nodes are disconnected). The degrees of the nodes are in range $k_{min} \leq k_i \leq k_{max}$. We assign the next values:

$$k_{min} = 2$$

$$k_{max} = N^{1/2}$$

We measure an upper threshold to decrease the degree correlationsCatanzaro, Boguñá, and Pastor-Satorras, 2005. For example in the BA model we do not have such value

that is why in Figure 1.2 we do not have a straight line. To each node i we assign its k_i randomly. The edges in the network are added randomly according to the corresponding k_{th} of a node (we avoid multiple connections and self-connections). At the end we do have an adjugate square matrix containing 0th and 1th (1th for neighbors).

3.3 Vaccination methods

In this section I would like to introduce the vaccination methods implemented by the SIS and SIR models (the corresponding algorithms are described in the **chapter1**). Before diving into the topic I would like to introduce the critical basic reproduction number concept R_c. For the SIS and SIR model it differsDorogovtsev and Mendes, 2001.
For SIS model:

$$R_c = \hat{k}/\hat{k^2}$$

For SIR model:

$$R_c = \hat{k}/\hat{k^2} - \hat{k}$$

If R_0 value is higher that R_c then the spreading leads to the endemic state. If R_0 is lower that R_c spreading dies during some time, which depends on the difference. That value takes an important role in our investigation as we used it as a threshold. The next vaccination methods were implemented by the networks introduced above.

- BA model $\lambda = 3$
- Custom network with $\lambda = 2.1$
- Custom network with $\lambda = 2.2$
- Custom network with $\lambda = 2.3$

Where λ was introduced in **section 1.2**
From the equation derived in **1.2** we can conclude that the higher λ corresponds to a sparser network. In our simulations we consider having a limited number of vaccines. At the beginning all individuals of a population of a size N are in the S state.

3.3.1 Vaccinated SIR

The system described by implementing the SIR model for the complex networks will have some restrictions. Let's consider a situation when the population is aware of the coming disease but can not be gradually vaccinated. The individuals can be vaccinated in a predefined way. Meaning that we choose individuals which we would vaccinate only once and only at the beginning of the simulation (before the disease came).

- The first vaccination method which is implemented in this paper is a random vaccination. At the beginning of a model simulation we took a predefined number of random susceptible individuals and changed their state to R. Because state R is considered as immune and doesn't spread infection it can be considered as vaccinated. In the scope of analysing the spreading processes those individuals could be completely "removed" from a network. This method we consider to be the least effective vaccination method as we do not

specify any features of a graph. We used it for comparison with other methods in order to clarify their effectiveness.

- The second vaccination method which is implemented is target hub vaccination (ranked by hubs vaccination). The intuition behind this method is the next one. As hubs are the individuals with the most neighbors (nodes with the highest degree value) we want them to be in a state R because that state does not spread an infection. At the beginning of the model simulation we took a predefined number of susceptible individuals with the most neighbors and changed their state to R. Comparing the results we can see that target hub vaccination shows a better performance than a random one. It was concluded that vaccination of hubs is more effective than a vaccination of random individuals. But what if for some reasons it is almost impossible to determine specific hubs in some situations. To solve this problem the following idea was used. Consider a case when we don't know features of the individuals and therefore consider all individuals of a complex network to be the same. In other words we would like to improve the random vaccination algorithm.

  The next vaccination method that handles that idea is random neighbor vaccination. The intuition behind this idea is the following. We would like to increase a chance to randomly target a hub of a network. When we vaccinated individuals at random we were considering the whole scope of a network. But it is possible to narrow that scope. As was mentioned above we have a fixed number of vaccines. We would like to consider a scope of neighbors of a randomly chosen individual i. The probability of finding a hub in a scope with a size N_i is way higher than in a scope N. At the beginning of a model simulation we randomly choose some predefined number of individuals. Then we randomly choose a random neighbor of each individual found previously and vaccinate it. Not significantly but such a method on a hundreds simulations showed better results compared to a random vaccination considered before.

- But what if we go further and vaccinate a random neighbor of a random neighbor of a random individual. The intuition is the same but we would like to see at which step this idea will stop giving better results. At the beginning of a model simulation we randomly choose some predefined number of individuals. Then we randomly choose a random neighbor of each individual found previously. For each of those random neighbors we randomly choose one neighbor and change its state to R.

- The next method is called target vaccination with respect to betweenness centrality. Before introducing an algorithm I would like to describe a concept of betweenness centrality and why we have chosen it to vaccinate a networkHolovatch et al., 2006.
  Betweenness centrality is a measure of a node importance in the network. Consider the nodes x, y, i. It shows the number of the shortest paths which go through i.

  $$\sigma(i) = \sum(B(x,i,y)/B(x,y))$$

  Where $B(x,y)$ is the total number of the shortest paths and $B(x,i,y)$ the total number of the paths which go through the node i. σ(i) is also called load. At the beginning of the model simulation we took a predefined number of susceptible individuals with the highest value σ and vaccinate them.

3.3.2 Vaccinated SIS

The system described by implementing the SIS model on the complex networks which we would like to vaccinate will also have some basic restrictions. Unlike the SIR case here a population can be vaccinated dynamically but the number of vaccines is fixed for each time iteration.

- The first vaccination method is a random one. At each time iteration we randomly vaccinate a predefined number of susceptible individuals. Those V individuals could be considered as removed. We continue a process until the vaccines run out. We need this method for a lower threshold as it is supposed to be the worst vaccination method.

- The second vaccination method is based on the hubs targeting. The idea is similar to the approach used in the SIR model. At each time iteration we vaccinate a fixed number of the most popular individuals(hubs). As they are considered to be removed we recalculate the sorted table of hubs and repeat the process until the vaccines run out.

- The last method is partly based on the previous one but with some addition. Let's consider a situation where the disease had a tendency to begin spreading in some specific part of the network. But what if the "biggest" hubs (the most popular individuals) are in the other "end" of the network. As the vaccine number is fixed there are more important nodes to vaccinate. Our model is based on the cellular automaton algorithm meaning that the more infected individuals are in the network the faster disease is spreading. It means that we would like to start the vaccination process from the first iteration of time. To deal with it we proposed the next algorithm. At each time iteration for all susceptible individuals we find the shortest path to the infected individuals. The next step is to clarify the importance of susceptible individuals. It is done by introducing the importance coefficient C For an individual s corresponding C_s will be looking like

  $$C_s = k_s * e^{-R}$$

  Where R is the shortest path to an infected individual. The higher R corresponds to lower C_s.

Chapter 4

Results

In this section I would like to show the results of the experiments described above. The results are represented via images and tables. At the end of the section I will provide some short conclusions. In our experiments some of the parameters are constant and some are changing. The population size of the complex networks $N = 15000$ is fixed for all simulations.

In this section we introduced the results of the proposed methods **3.3.1**
The parameters for the further experiments
Number of initial infected individuals $I = 200$
Number of vaccines $= 200$
$\gamma = 0.6$
Total observation time $T = 100$
β is changing in range $0 \leq \beta \leq 1$
SIR vaccinations
The values that we used as a measure of effectiveness for the SIR model is a number of recovered individuals at some huge period of time. It could be considered as a post pandemic value because the number of infected individuals in the SIR model directly depends on the number recovered. Basically in some period of time we will not be susceptible enough to become infected and at this period of time the R stables too (it could be viewed as a cumulative number).
SIR vaccinations
The value that we used as a measure of effectiveness for the SIS model is a number of infected individuals at some huge period of time. The intuition is the next one. As infected individuals directly depend on the susceptible in some period of time the fraction will also stabilize (due to the algorithms of SIS and SIR model written in **1.3**).

- $\lambda = 3$ (Barabasi-Albert graph)
  The minimal $k = 2$
  The random vaccination

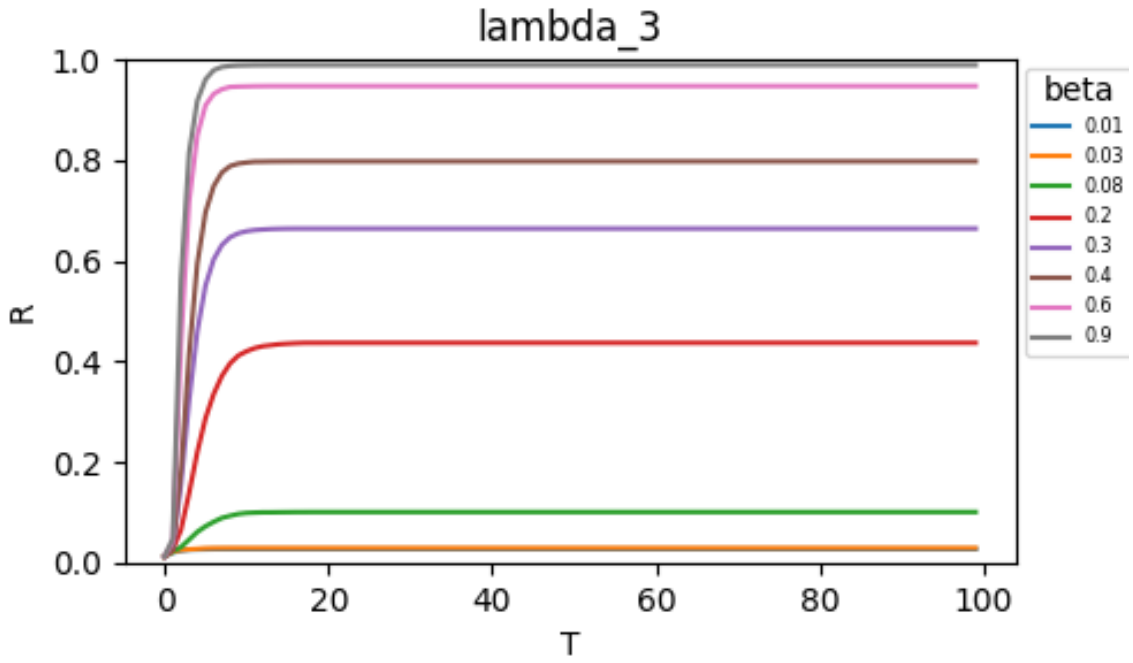

FIGURE 4.1: Random vaccination

The hubs vaccination

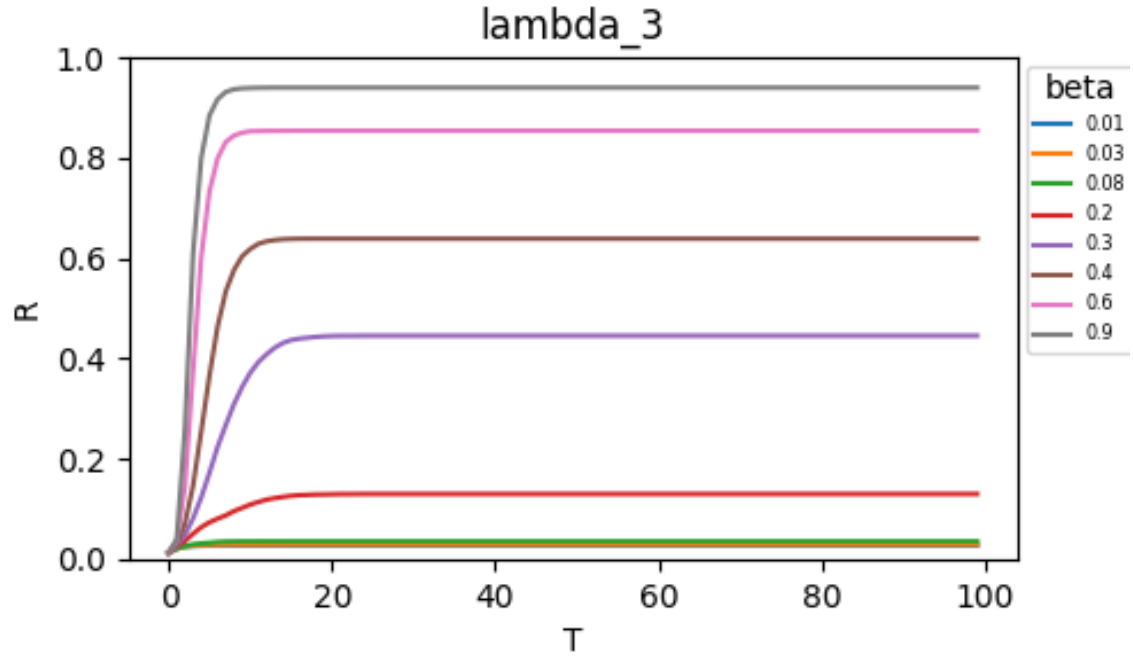

FIGURE 4.2: The target hubs vaccination

The random neighbor vaccination

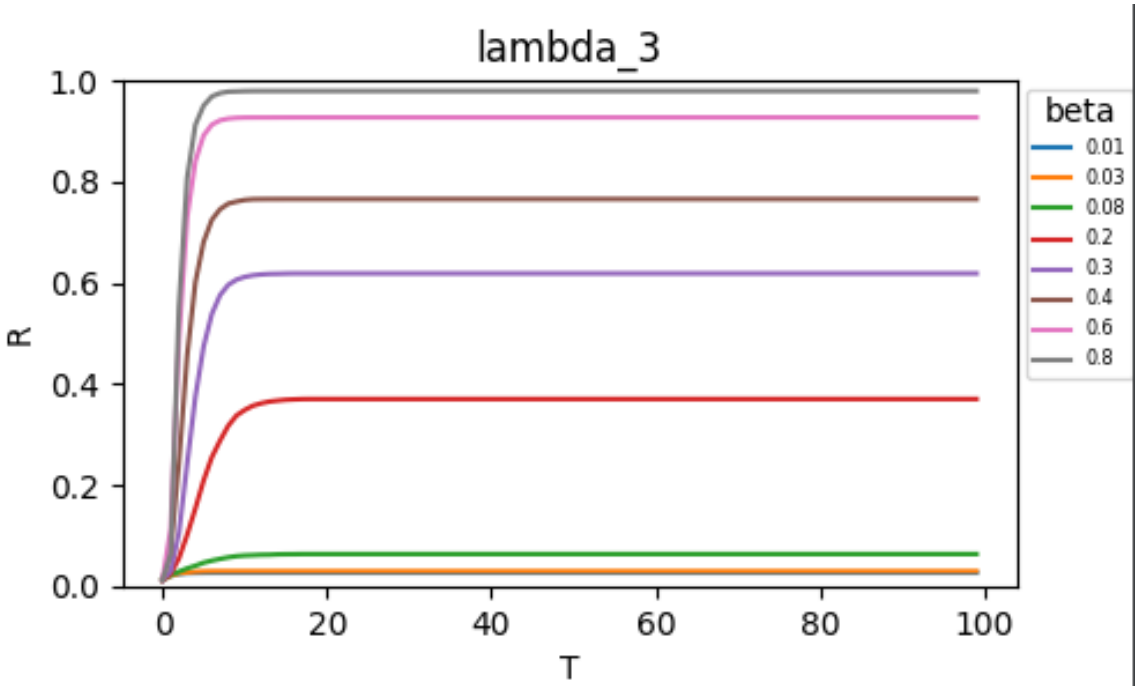

FIGURE 4.3: The random neighbor vaccination

The random neighbor of random neighbor vaccination

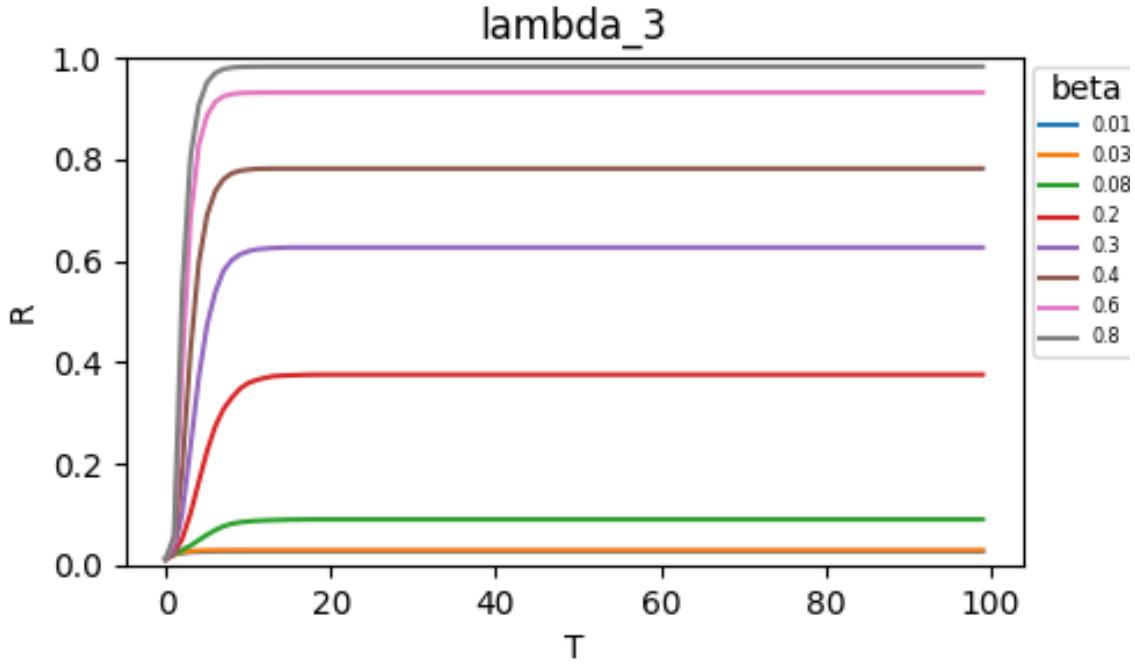

FIGURE 4.4: The random neighbor of random neighbor vaccination

- $\lambda = 2.1$
  The random vaccination

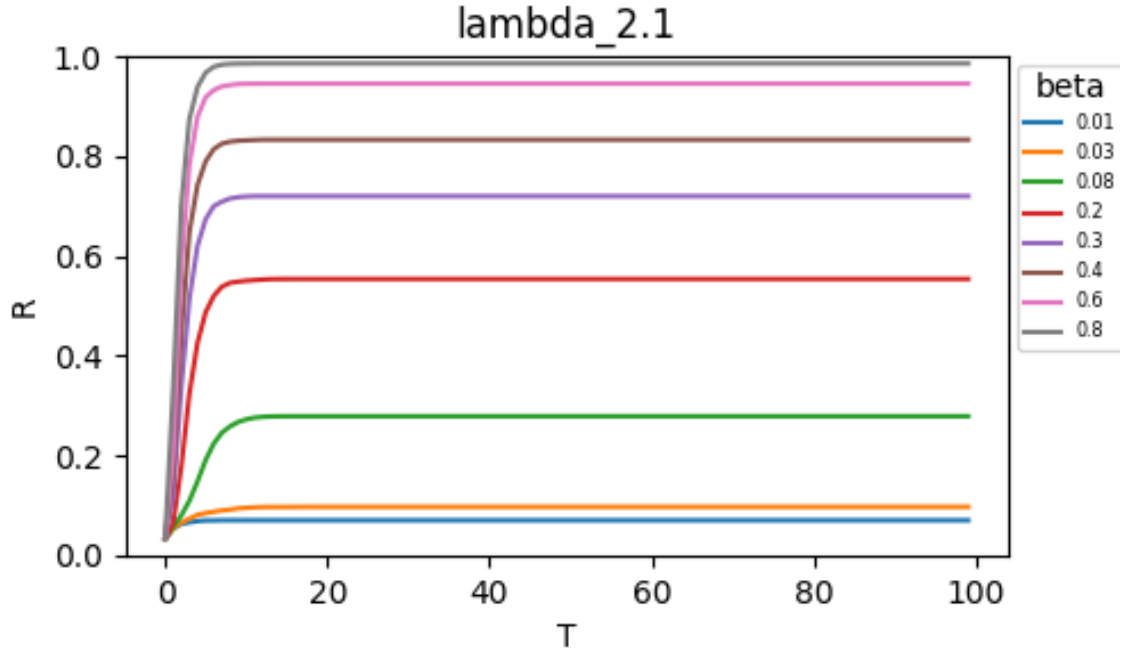

FIGURE 4.5: Random vaccination

The hubs vaccination

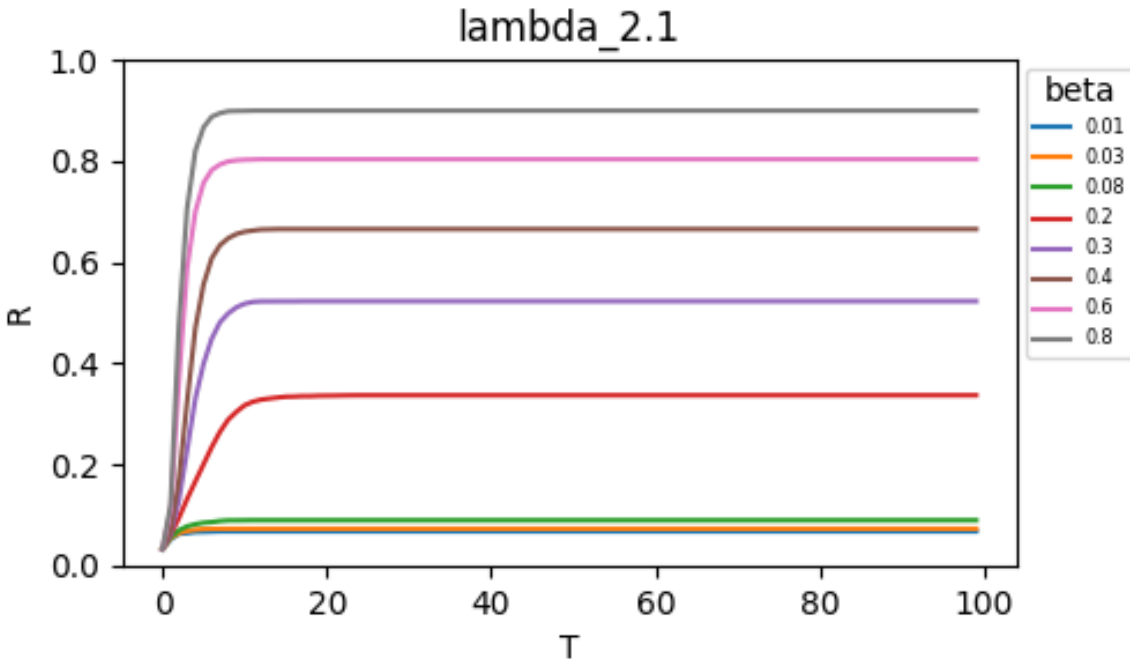

FIGURE 4.6: The target hubs vaccination

The random neighbor vaccination

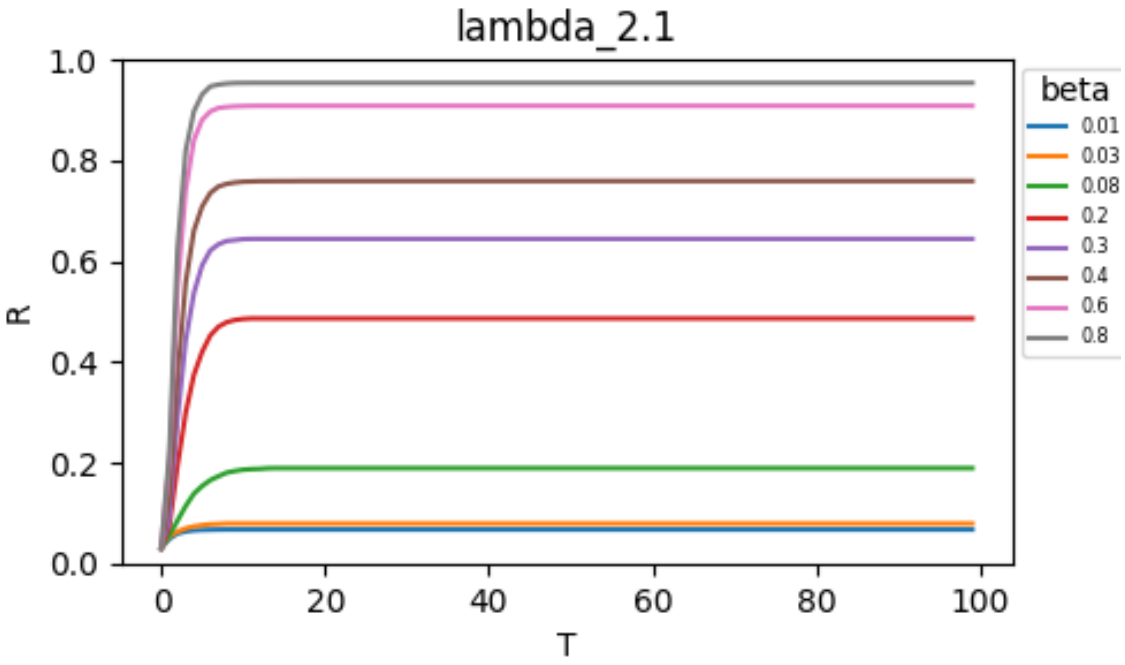

FIGURE 4.7: The random neighbor vaccination

The random neighbor of random neighbor vaccination

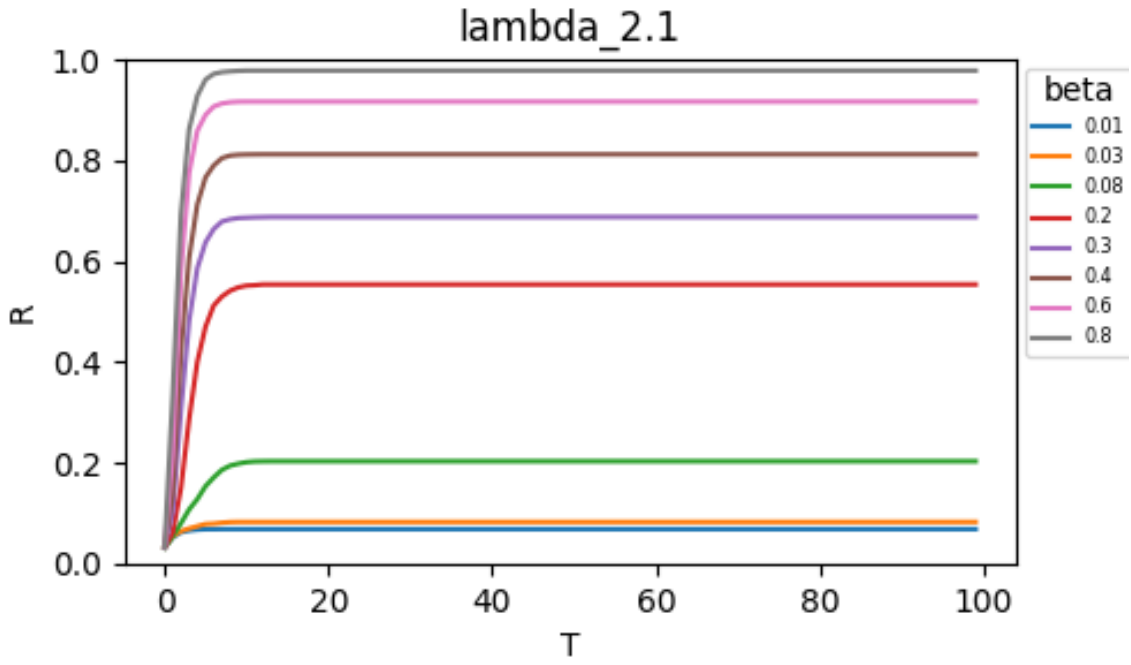

FIGURE 4.8: The random neighbor of random neighbor vaccination

- $\lambda = 2.2$
  The random vaccination

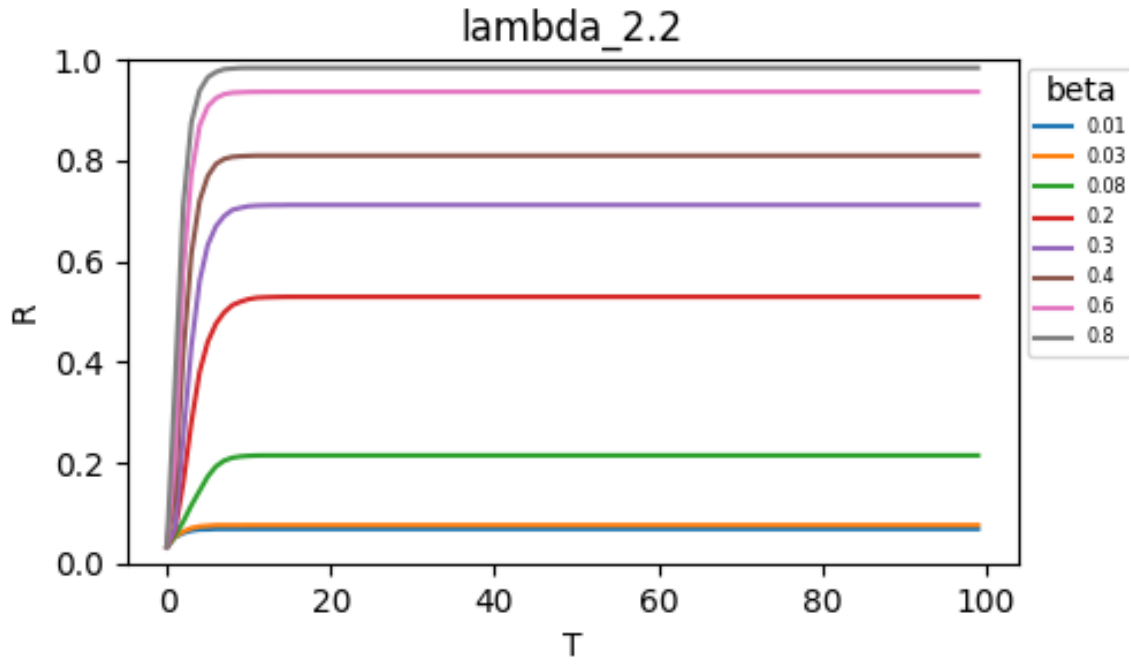

FIGURE 4.9: Random vaccination

The hubs vaccination

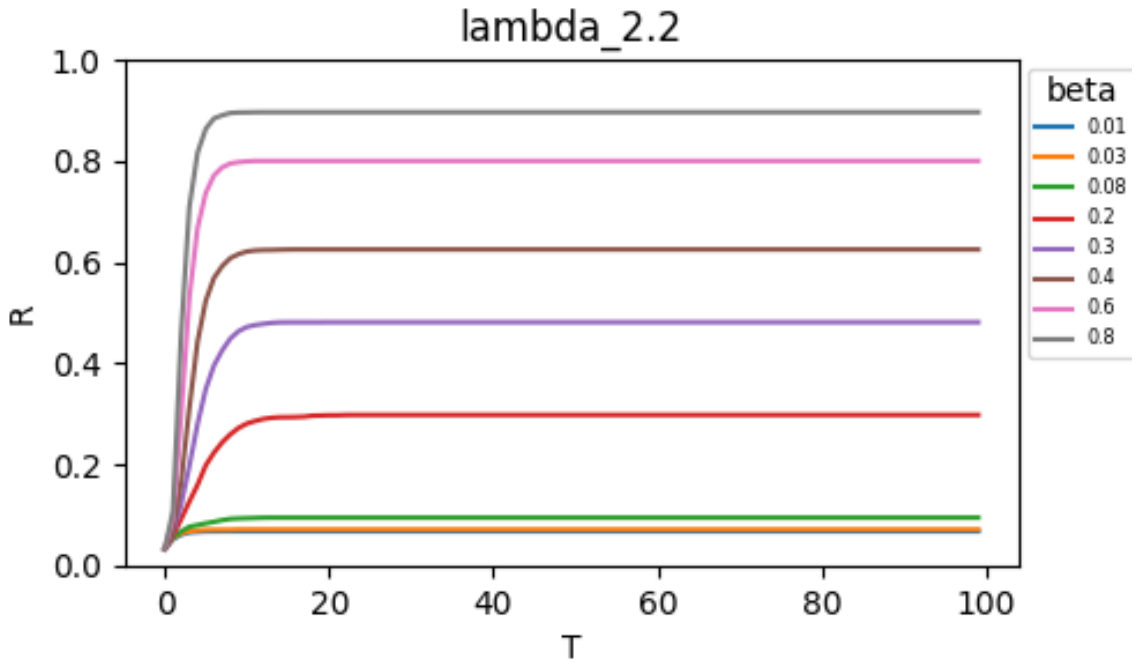

FIGURE 4.10: The target hubs vaccination

The random neighbor vaccination

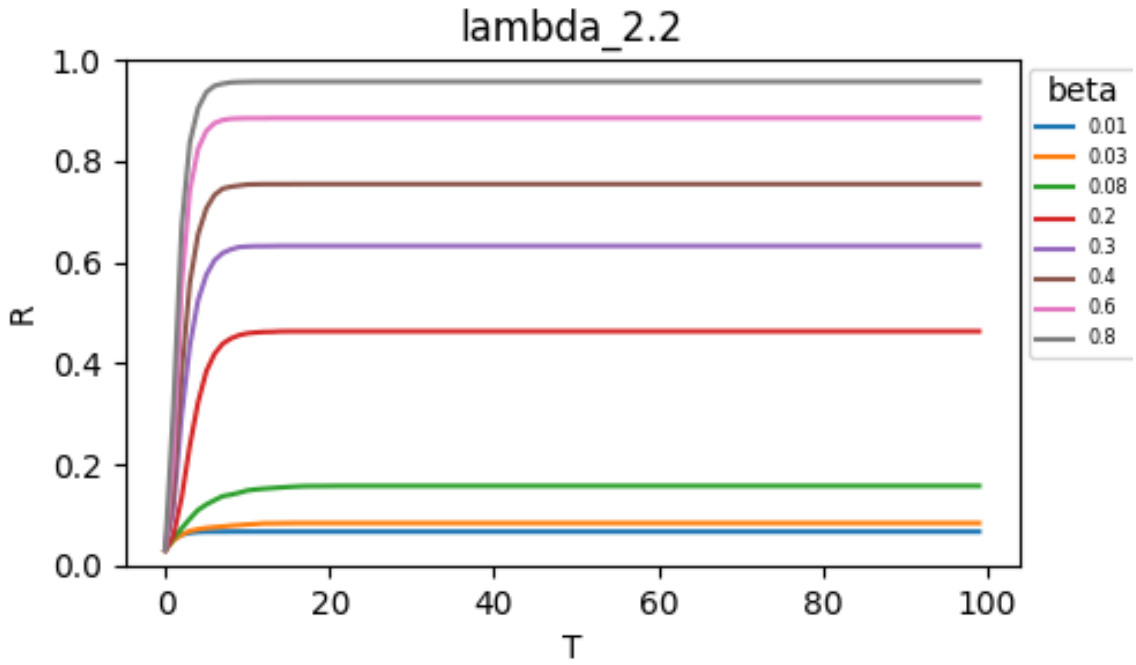

FIGURE 4.11: The random neighbor vaccination

The random neighbor of random neighbor vaccination

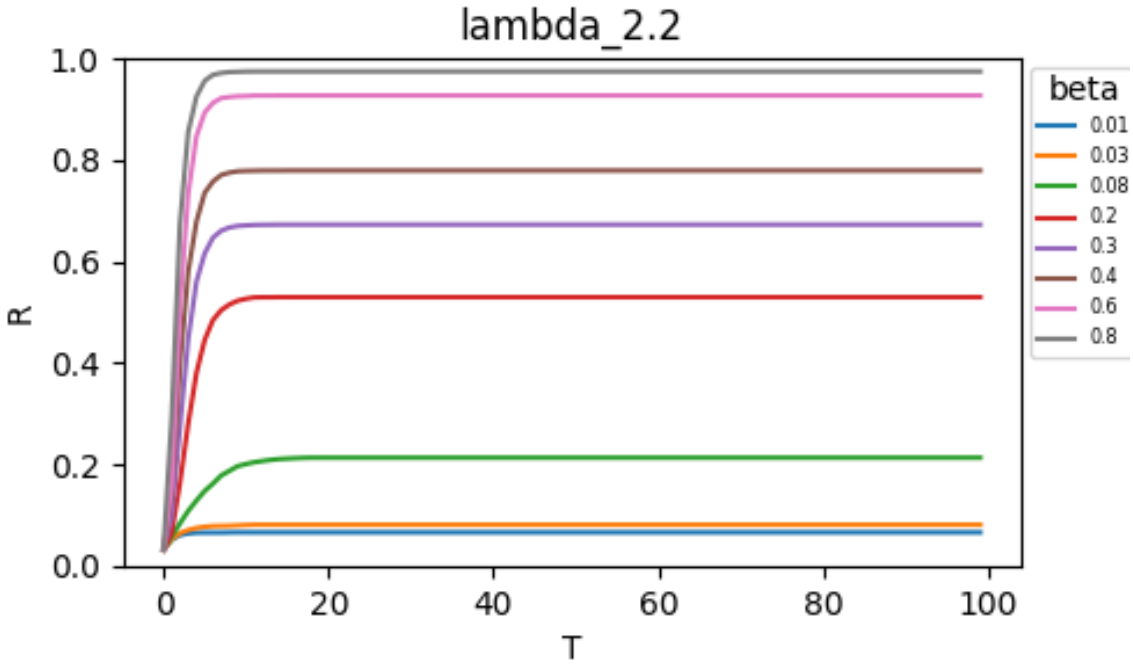

FIGURE 4.12: The random neighbor of random neighbor vaccination

- $\lambda = 2.3$
  The random vaccination

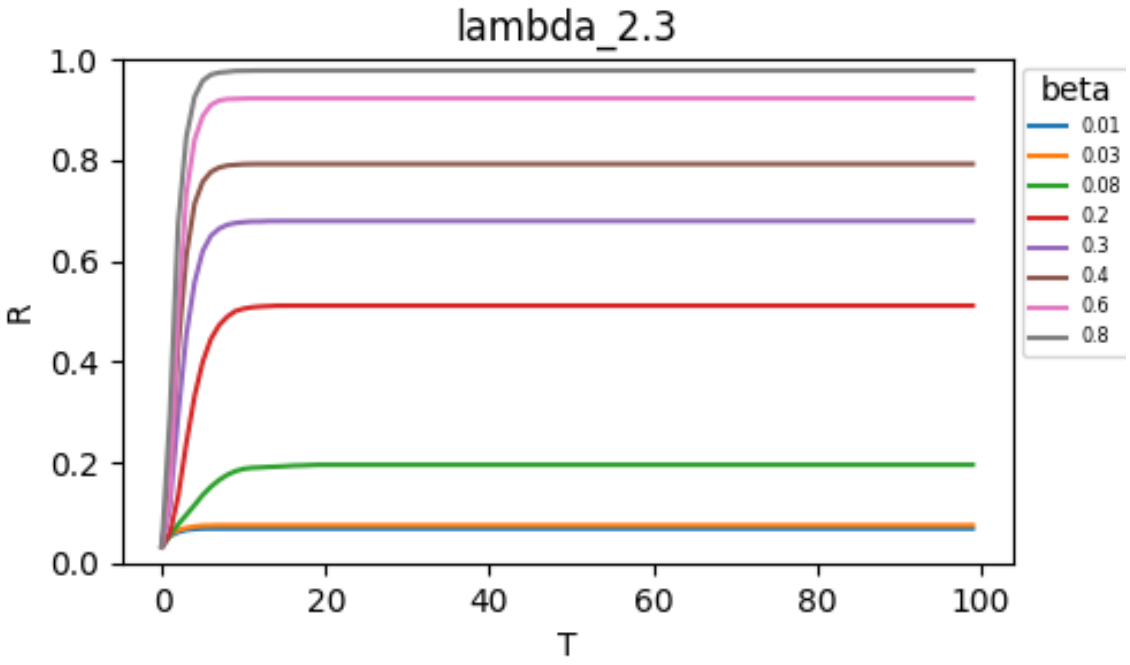

FIGURE 4.13: Random vaccination

The hubs vaccination

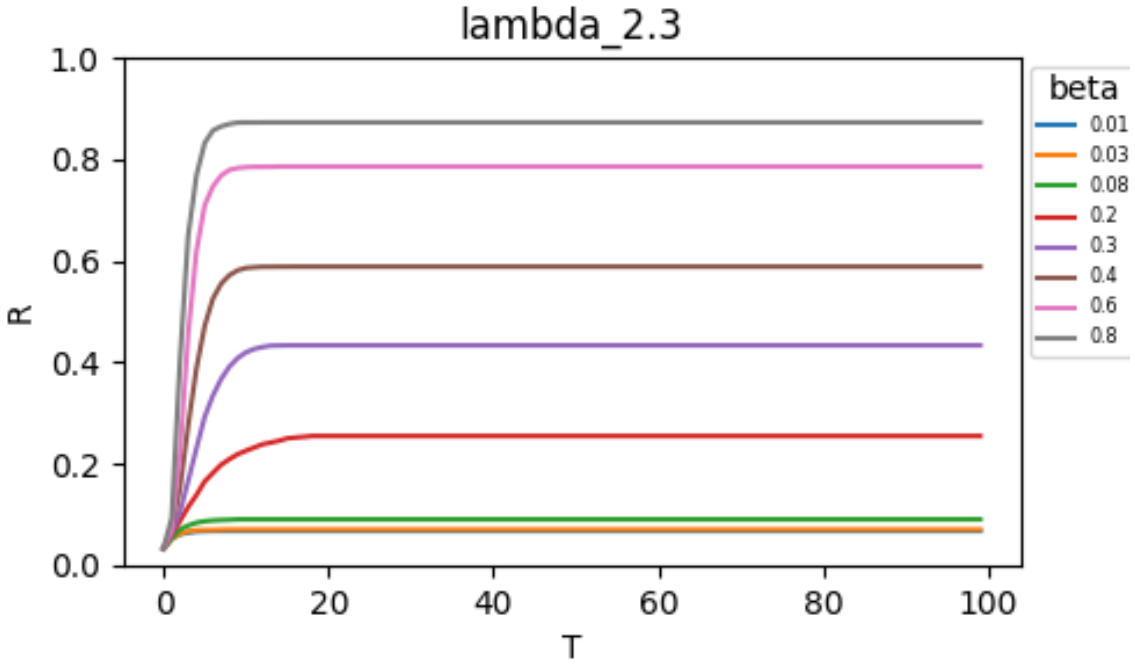

FIGURE 4.14: The target hubs vaccination

The random neighbor vaccination

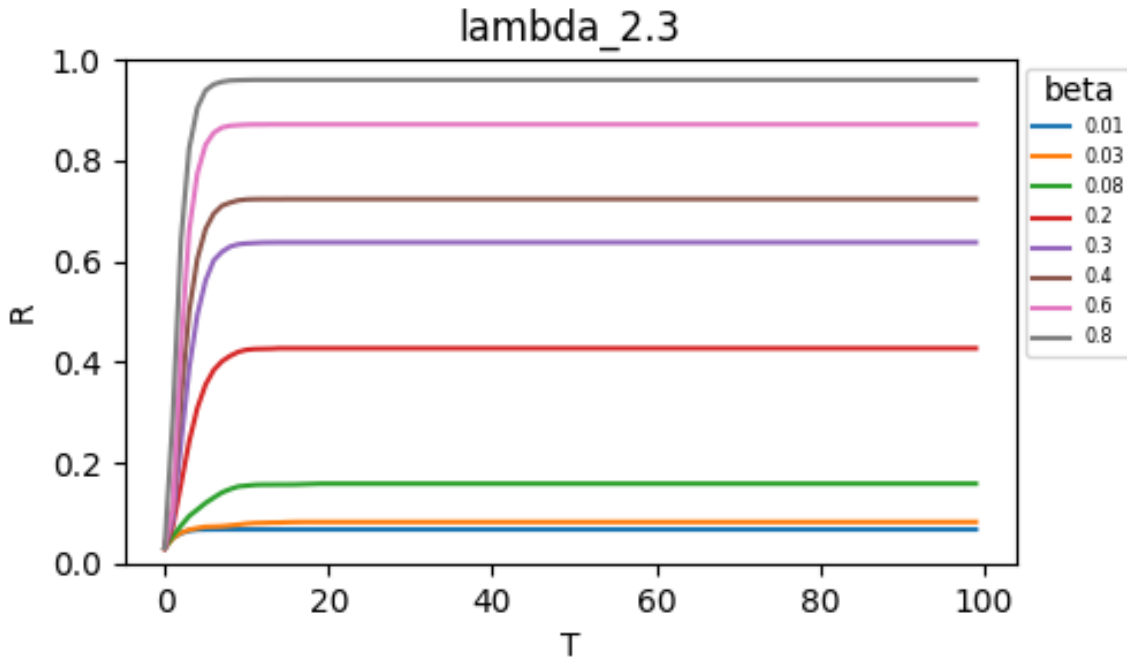

FIGURE 4.15: The random neighbor vaccination

The random neighbor of random neighbor vaccination

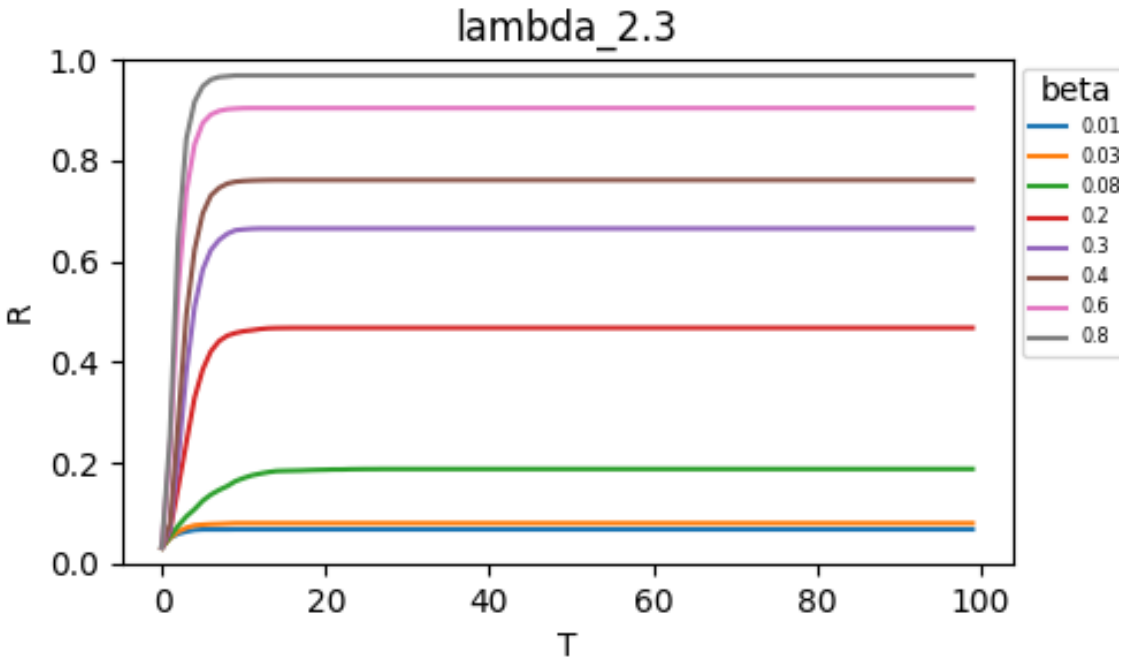

FIGURE 4.16: The random neighbor of random neighbor vaccination

	beta 0.01	beta 0.03	beta 0.08	beta 0.2	beta 0.3	beta 0.4	beta 0.6	beta 0.8
Random	0.02766666667	0.02986666667	0.1012666667	0.4370666667	0.6638666667	0.7972666667	0.9467333333	0.9885333333
Target hubs	0.02773333333	0.0296	0.03593333333	0.1303333333	0.4454	0.6386666667	0.8536	0.9394666667
Random neighbor	0.02733333333	0.03033333333	0.06426666667	0.3704666667	0.6185333333	0.7654	0.9266666667	0.9788
Random neighbor of Random neighbor	0.0282	0.03013333333	0.09086666667	0.3759333333	0.6262	0.7815333333	0.9311333333	0.9823333333
Betweenness centrality	0.0278	0.03046666667	0.08746666667	0.3674666667	0.6080666667	0.7326	0.9044666667	0.9796666667

TABLE 4.1: BA graph

	beta 0.01	beta 0.03	beta 0.08	beta 0.2	beta 0.3	beta 0.4	beta 0.6	beta 0.8
Random	0.07083333333	0.09766666667	0.279	0.5538333333	0.7196666667	0.8326666667	0.9448333333	0.9851666667
Target hubs	0.06816666667	0.07333333333	0.09016666667	0.337	0.5228333333	0.6651666667	0.8031666667	0.8991666667
Random neighbor	0.06819	0.08	0.1898333333	0.4865	0.6441666667	0.7588333333	0.9078333333	0.9533333333
Random neighbor of Random neighbor	0.069	0.08316666667	0.2035	0.5535	0.6875	0.8116666667	0.9161666667	0.9776666667

TABLE 4.2: $\lambda = 2.1$

	beta 0.01	beta 0.03	beta 0.08	beta 0.2	beta 0.3	beta 0.4	beta 0.6	beta 0.8
Random	0.06933333333	0.07683333333	0.215	0.5293333333	0.7113333333	0.8091666667	0.9356666667	0.9831666667
Target hubs	0.06833333333	0.072	0.09533333333	0.2981666667	0.4813333333	0.625	0.7998333333	0.8961666667
Random neighbor	0.06766666667	0.0845	0.158	0.4633333333	0.632	0.7541666667	0.8845	0.9565
Random neighbor of Random neighbor	0.06666666667	0.08166666667	0.2136666667	0.5296666667	0.6725	0.779	0.9266666667	0.974

TABLE 4.3: $\lambda = 2.2$

	beta 0.01	beta 0.03	beta 0.08	beta 0.2	beta 0.3	beta 0.4	beta 0.6	beta 0.8
Random	0.0695	0.076	0.1958333333	0.5111666667	0.679	0.792	0.922	0.9771666667
Target hubs	0.06816666667	0.071	0.255	0.2981666667	0.4335	0.5885	0.7855	0.8723333333
Random neighbor	0.06833333333	0.08316666667	0.159	0.4273333333	0.6371666667	0.7231666667	0.871	0.9593333333
Random neighbor of Random neighbor	0.0685	0.08066666667	0.1881666667	0.4678333333	0.6646666667	0.7608333333	0.9033333333	0.968

TABLE 4.4: $\lambda = 2.3$

Analysing the results above we can make some conclusion. First of all the importance of the hubs in a spreading process is enormous. The experiments implied on all complex networks proved it(hubs vaccinations are considered to be the best method). The most surprising results showed the neighbor vaccination method. Through many simulations it is constantly showing better results than the random individual vaccination. Actually it only underlines the importance of the hubs for the network. But if we fall deeper and implement the random neighbor of a random neighbor vaccination method it will not show the better results(meaning that the scope of finding hubs is already the most optimal in the random neighbor vaccination method). A little bit confusing are results of betweenness centrality vaccination method because it has high computational cost and needs more resources than other algorithms but its performance is on the same level as random neighbor method. The next “expensive” algorithm based on the shortest path to the infected individual does not show appropriate results either. It is because of the deterministic system. The hub vaccination shows better results because the coefficient which we introduced may be good in a long term perspective but a system is "memoryless". I assume the method will perform better but with another coefficient because the idea of the approach is promising. On the graphs could be easily seen the outbreak of a disease. Starting from the β coefficient 0.2. I have chosen appropriate β (0.01, 0.03, 0.08) to show the cases with disease-free equilibrium and endemic equilibrium (R_c is close to 0.03).

Chapter 5

Conclusions

In this work many algorithms of building complex networks were introduced alongside mathematical models to analyse spreading processes on the network. The models were based on the cellular automaton model. It was done to find out important graph features (represented by nodes) which we would like to vaccinate in an epidemic modelling. Many real world systems could be explained via networking abstraction analysing them by graph perspective could lead to new solutions of unsolved features. In this work we conclude that hub is the most important feature in the scope of the spreading process in the complex networks. Even if its relative location in the network is not "comfortable" for the spreading processes it tends to be a cluster and make all the other locations not "comfortable". Some of the algorithms are assumed to be able to improve because they are depending on the predefined coefficients not dependent on the system. Also the new improved randomisation vaccination method was successfully implemented and showing constantly better results than a simple one.

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