A Network approach on Epidemics: adapting the SIRVD Model to different types of Networks

Alma Mater Studiorum - University of Bologna
Department of Physics and Astronomy "Augusto Righi"
Master Degree in Theoretical Physics

Complex Networks Project

Katja Brunelli Erika Chinaia

Contents

1 Introduction

The aim of this project is to study the evolution of an epidemic and to do so, we employ two different approaches. The first one consists in just implementing the numerical equations typical of the specific models that we will simulate. However, this type of analysis is not completely realistic because it doesn't display how people among the community are really connected with each other. So, in order to take this feature into account, the next step is to try to implement the same epidemic dynamics using different types of network.
We will show the comparison between these two approaches for different cases. We will start with the standard SIRVD model with constant parameters and with networks that do not change the connections between the nodes with time. Then, to make the simulation even more realistic, we will make the links change with time by introducing such things as a lockdown or superspreader events.
Finally we will try to make a connection with real data from the COVID-19 pandemic in Italy.

2 Compartmental Models

Compartmental models are called in this way because they consider the population divided into different compartments (for example S, I and R) and it is possible to move from one compartment to another. Moreover, they consider individuals to be all connected to each other. The dynamics of such models is usually described by time dependent differential equations.

2.1 The SIR model

The SIR model is one of the most simple, yet accurate models one can use to simulate the spread of an epidemic among a population. As we already mentioned, the model is very basic since it takes into account only three states:

- **S** $\rightarrow$ number of susceptible individuals; these are all the individuals that have not yet contracted the disease, but are susceptible to it;
- **I** $\rightarrow$ number of infectious individuals; when an individual that has contracted the disease is nearby a susceptible one, the latter can become infectious;
- **R** $\rightarrow$ number of recovered individuals. In other models R could also indicate the removed i.e. deceased individuals, but we are going to consider a simpler model without births and deaths.

We need to specify that this model is predictive for a disease that is transmitted by human contact. We are also considering that a recovered individual cannot contract the disease again, becoming immune. The number of individuals in each state varies with time, but their sum is constant since we are considering a model without birth and death rates as already mentioned.

The dynamics of the epidemic (visible in figure 1) is governed by the following

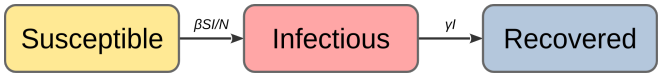


Figure 1: *Flow diagram of states in a SIR epidemic models and transition rates between them*

differential equations:

$$\begin{cases} \frac{dS}{dt} = -\frac{\beta}{N} IS, \\ \frac{dI}{dt} = \frac{\beta}{N} IS - \gamma I, \\ \frac{dR}{dt} = \gamma I, \end{cases} \tag{1}$$

where N is the total population and is given by $S + I + R$ at any time, β is the average number of contacts per person per time, multiplied by the probability of disease transmission in a contact between a susceptible and an infectious subject, γ is the recovery rate constant in the unit fraction of a person recovered per day per infected person, when time is in unit day such that $\gamma = 1/D$ with D average time period during which an individual is infectious.

2.2 The SIRVD model

The SIRVD model is an evolution of the SIR model that includes two more possible states. In particular the possible states are now susceptible-infected-recovered-vaccinated-deceased. In figure 2 we can see how the dynamics of the SIRVD model

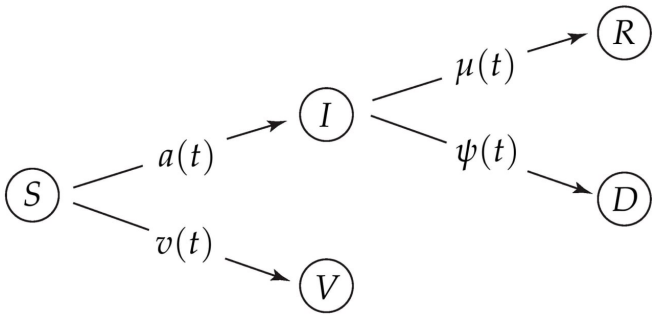


Figure 2: *Cartoon of the SIRVD model*

works. The differential equations in this case are:

$$\begin{cases} \frac{dS}{dt} = -a(t)SI - v(t)S, \\ \frac{dI}{dt} = a(t)SI - \mu(t)I - \psi(t)I, \\ \frac{dR}{dt} = \mu(t)I, \\ \frac{dV}{dt} = \nu(t)S, \\ \frac{dD}{dt} = \psi(t)I \end{cases} \tag{2}$$

where $a(t), \nu(t), \mu(t), \psi(t)$ are the infection, vaccination, recovery, and fatality rates, respectively.

This model is more realistic because it also takes into account the possibility of getting vaccinated and the possibility of death. However, it is still not totally realistic because it does not take into account the scenario where the individuals can be vaccinated after getting the disease, the scenario in which individuals can become infectious after being vaccinated and other possible cases. Among them we chose to analyze the possibility for recovered individuals to become susceptible again and we do it by introducing a new parameter $\sigma(t)$ called breakthrough parameter. Taking this into account, the equations that control the dynamics of the pandemic are:

$$\begin{cases} \dfrac{dS}{dt} = -a(t)SI - v(t)S + \sigma(t)R, \\ \dfrac{dI}{dt} = a(t)SI - \mu(t)I - \psi(t)I, \\ \dfrac{dR}{dt} = \mu(t)I - \sigma(t)R, \\ \dfrac{dV}{dt} = \nu(t)S, \\ \dfrac{dD}{dt} = \psi(t)I. \end{cases} \tag{3}$$

3 Network of Pandemics

In this section we are going to illustrate the different types of networks that we use in the simulation [1]. We will display their main features and how they are related to the pandemic dynamics.

3.1 Erdős-Rényi Network (ER)

The Erdős-Rényi model is the one typically used to work with random graphs and it can be divided in two different types:

- **G(N, L)** $\rightarrow$ in this model the graph is chosen uniformly at random from all the graphs that have n nodes and L links;
- **G(N, p)** $\rightarrow$ in this model there is a unique macroscopic parameter p which is the probability of connection between nodes, so unlike the previous model, there is not a fixed number of links.

For our analysis we will use the $G(N, p)$ model which is the simplest null model. For more detailed properties of this kind of network see the original paper from Paul Erdős and Alfrèd Rényi [2].

For the purpose of our analysis it is useful to consider this kind of graph because of its simplicity. However, it is not very realistic for simulating the connections between a real life community because of its random nature. In fact there are two properties observed in a real-world network that this model does not account for:

- **High clustering coefficient** $\rightarrow$ since the probability of generating a link is uniformly random there is no possibility of creating clusters;
- **Hubs** $\rightarrow$ formally, the degree distribution of Erdős-Rényi graphs converges to a Poisson distribution, rather than a power law observed in many real-world, scale-free networks.

In the next two sections we will see two models that solve these problems; in particular the Watts–Strogatz model addresses the first one and the Barabási–Albert model accounts for the second.

3.2 Watts–Strogatz Network (WS)

The Watts–Strogatz model is a minimal extension of an Erdős-Rényi network and its main features are a lattice structure and the rewiring parameter. The former produces a locally clustered network, while the randomly rewired links dramatically reduce the average path lengths. This results in the network having the following properties:

- **Small world property** → the *small world phenomenon* is a fascinating feature that has been observed. In particular, choosing two random individuals anywhere on earth, there is a path of at most six acquaintances between them. In fact this is also known as *six degrees of separation*. From the network point of view it means that the average distance between any two nodes (i.e. average path length) is unexpectedly small. In fact it scales as $\ln(N)$ and not as a polynomial which is the expected behaviour of regular lattices.
- **High clustering** → as previously said the average clustering coefficient of a real network is higher than the one of a random graph.

To build the network we need the total number of nodes N, the average degree $\langle k \rangle$ (assumed to be an even integer) and the rewiring parameter β such that $0 \leq \beta \leq 1$, $1 \ll \ln(N) \ll \langle k \rangle \ll N$. The model constructs an undirected graph with N nodes and $(N\langle k \rangle)/2$ edges in the following way:

- the first step is to create a regular ring lattice, a graph with N nodes each connected to $\langle k \rangle$ neighbors, $\langle k \rangle/2$ on each side;
- the second step is taking every edge of each node and rewire it with probability β.

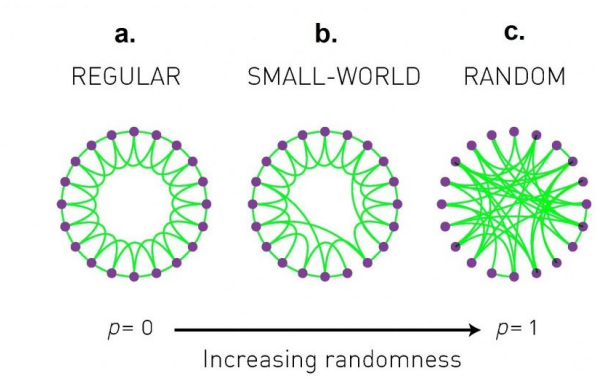


Figure 3: *Graphic representation of a Watts–Strogatz model where the rewiring parameter β is denoted as p*

The limit cases $\beta = 0$ and $\beta = 1$ correspond to a regular lattice and a random network respectively. All other values of β $(0 < \beta < 1)$ correspond to the Watts–Strogatz network also known as the small world model. In figure 3 there is a graphic representation of these cases.

We use this kind of model in our analysis because it efficiently simulates local communities with few long range contacts. However, the choice of the parameter β is crucial to have a more realistic simulation.

3.3 Barabási–Albert Network (BA)

The models we have presented so far, despite being valid for our purposes, do not consider how the network comes into being. To account also for this feature, we can use the Barabási–Albert model. In fact, the philosophy behind this model is simple yet crucial: "*To undertand the topology of a complex system, we need to describe how it came into being*" [1].
The two main characteristics of this model are:

- **Growth** → unlike for random networks where the number of node is fixed, the number of nodes in this model continuously increases at every step.
- **Preferential attachment** → when new nodes are introduced in the network, they are more likely to be connected to already existing nodes with a high number of links. This is in contrast with random graphs where the links are chosen randomly.

To create a Barabási–Albert network, we start with m_0 nodes with arbitrarily chosen links, as long as each node has at least one. At each step a new node is added with $m\,(\leq m_0)$ links that connect it to m already existing nodes. The new node can in principle be connected to any of the existing ones, however, due to the preferential attachment, it will have a much higher probability to be connected with nodes with a higher degree. In fact, the probability of the new node being connected to a node i of degree k_i is:

$$\Pi(k_i) = \frac{k_i}{\sum_j k_j}. \tag{4}$$

It is easy to see that a higher k_i corresponds to a higher $\Pi(k_i)$; for this reason the network will display particular structures such as hubs, that are nodes that have a very high degree. The creation of such hubs is the result of the *rich-gets-richer phenomenon.*
After t steps the model generates a network with:

$$\begin{cases} N = t + m_o & \text{nodes,} \\ m_0 + mt & \text{links.} \end{cases} \tag{5}$$

The obtained network has a power-law degree distribution with exponent $\alpha = 3$.
An important thing to notice is that the combination of growth and preferential attachment results in the network being scale-free, i.e. its properties do not depend on the number of links and nodes added at each step.

In the context of an epidemic simulation this kind of model can generate super-spreader events due to the presence of hubs. The main limitation of this network is that it is less realistic for small communities.

3.4 Network Properties and their Epidemiological Meaning

In this paragraph we present a table in which we relate some complex networks' parameters to their epidemiological meaning in all the models that we presented above.

Network Parameter	**Erdős–Rényi (ER)**	**Watts–Strogatz (WS)**	**Barabási–Albert (BA)**
Average Degree $\langle k \rangle$	Controls how easily disease can spread. Higher $\langle k \rangle \rightarrow$ higher chance of epidemic.	Same as ER. Key in determining outbreak size and speed.	Same effect, but influence is dominated by hubs.
Degree Distribution	Poisson $\rightarrow$ most nodes have similar degrees. The pandemic spreads uniformly.	Still narrow $\rightarrow$ few nodes differ greatly in degree.	Power-law $\rightarrow$ presence of super-spreaders. Large impact on spread and persistence of epidemics.
Clustering Coefficient	Low $\rightarrow$ interactions are random.	High $\rightarrow$ more local loops. Encourages localized outbreaks or repeated exposures within small groups.	Low $\rightarrow$ less redundancy in local connections, but long reach due to hubs.
Average Path Length	Short$\rightarrow$ disease can spread fairly efficiently.	Also short, even with high clustering $\rightarrow$ supports both local and global spread.	Very short$\rightarrow$ extremely fast disease transmission possible.
Hubs (high-degree nodes)	Absent $\rightarrow$ nodes are similar.	Rare $\rightarrow$ most nodes have similar degree.	Present $\rightarrow$ key drivers of transmission. Crucial targets for intervention.
Epidemic Threshold	Well-defined $\rightarrow$ if transmission probability exceeds a critical point, outbreak occurs.	Same $\rightarrow$ clustering may slightly alter threshold.	Threshold may vanish $\rightarrow$ epidemics can occur even with low transmission probability.

Table 1: *Epidemiological interpretation of network parameters across ER, WS, and BA networks in SIRVD models.*

To summarize, we can draw the following conclusions on how the three types of networks we presented behave in an epidemic simulation:

- **Erdős–Rényi** $\rightarrow$ due to its random nature the spread is moderate and predictable and a random immunization is effective;
- **Watts–Strogatz** $\rightarrow$ in this type of network the spread begins as local and then becomes global due to the high clustering coefficient that favours contained but repeated outbreaks;
- **Barabási–Albert** $\rightarrow$ the spread is fast and persistent. Since the dynamics is dominated by hubs a targeted immunization is essential.

4 Implementation

For this project we have implemented the classical SIRVD model and its variation that considers also the breakthrough parameter (see 2.2) in two ways: implementing numerically the compartmental model and using a network structure.
For the network simulation we also made additional variations that will be explained in the following paragraphs.

4.1 Which Network?

We implemented our simulation using the three types of networks presented in the previous section. As we saw, each of them has some parameters that characterize it and to create the network we just need to give them in input to our code. In particular, ER takes in input the connection probability p; WS takes in input the average degree $\langle k \rangle$ and the rewiring probability β (denoted with p from now on to avoid confusion with the infection rate); BA takes in input the parameter m (see 3.3). All of them take also in input the total number of nodes N.

4.2 Node's State Evolution

After the network is created, I nodes are randomly initialized as infectious and the remaining $N - I$ are susceptible S. After setting the initial state of the network in this way, the state of each node is the evolved in a probabilistic way. We can easily generalize the vaccination, recovery, fatality and breakthrough rates to probabilities multiplying them by the time interval one takes into account. This approximation is valid since we are considering a small time interval (in all the simulations we take a time interval of 1 day). However, for bigger time intervals one must consider a Poisson process taking into account an exponential distribution for probabilities. When dealing with the infection rate we need to be careful since the infection rate of the SIRVD model ($a(t) = \beta/N$) multiplied by the number of infectious individuals $I(t)$, must be adapted to the network. In particular, I/N represents the fraction of infectious an individual has contact with. We can not directly consider the infection rate $a(t)$ as a probability because in its definition all the individuals of the population are considered to be connected with each other. However, since we are not dealing with totally connected networks we have to adapt this parameter to our scopes. To explicit the fact that our network is not totally connected, I/N becomes I_N/k_i where I_N is the number of infected neighbours a node has and k_i is the degree of each node. In this way, the probability for an individual of being infected is given by

$$p = \frac{\beta}{k_i} I \Delta t. \tag{6}$$

Let's notice that if $k_i \to N - 1$ than the infection rate is again the one of the SIRVD model.
Once we have all the probabilities properly defined, the evolution of the states is done by confronting them with a number randomly extracted from a uniform distribution. Of course all the probabilities are properly normalized.

4.3 *Constant or Not Constant Parameters: this is the Question*

In our simulation of the compartmental model we assumed all the parameters to be constant with time. However, in principle, this does not have to be true.

From the site https://ourworldindata.org/coronavirus we were able to extract the real world data relative to the COVID-19 pandemic in Italy. In particular, we extracted the real data of vaccinated individuals per day, new infectious per day and the cumulative number of deaths. We also assumed an average recovery time of 7 days and an average breakthrough time of 30 days. With these information we can invert the discrete version of the SIRVD differential equations (obtained using the Euler's Method)

$$\frac{S(t+1)-S(t)}{\Delta t} = -a(t)S(t)I(t) - \nu(t)S(t), \tag{7}$$
$$\frac{I(t+1)-I(t)}{\Delta t} = a(t)S(t)I(t) - \mu(t)I(t) - \psi(t)I(t), \tag{8}$$
$$\frac{R(t+1)-R(t)}{\Delta t} = \mu(t)I(t), \tag{9}$$
$$\frac{V(t+1)-V(t)}{\Delta t} = \nu(t)S(t), \tag{10}$$
$$\frac{D(t+1)-D(t)}{\Delta t} = \psi(t)I(t), \tag{11}$$

to get the values of all the parameters day by day

$$a(t) = \frac{I_{new}}{S(t)I(t)}, \tag{12}$$
$$\sigma(t) = \frac{1}{\tau_B}, \tag{13}$$
$$\mu(t) = \frac{1}{\tau_R}, \tag{14}$$
$$\nu(t) = \frac{V(t+1)-V(t)}{S(t)}, \tag{15}$$
$$\psi(t) = \frac{D(t+1)-D(t)}{I(t)}, \tag{16}$$
$$\tag{17}$$

where I_{new} is the number of new infected per day, τ_B is the average breakthrough time and τ_R is the average recovery time, and the values of the remaining observables

$$S(t+1) = S(t) - I_{new} - (V(t+1) - V(t)) + \sigma(t)R(t), \tag{18}$$
$$I(t+1) = I(t) + I_{new} - \mu(t)I(t) - \psi(t)I(t), \tag{19}$$
$$R(t+1) = \mu(t)I(t) + R(t) - \sigma(t)R(t). \tag{20}$$

Let's notice that, as already mentioned, we have taken $\Delta t = 1$.
In our network simulations we implemented both the possibilities of having constant parameters and the extracted parameters. In this way we can see how the networks behave with real world data.

4.4 Dynamics

Another feature that we implemented is the possibility of considering a dynamical network. In particular, we added the possibility of modifying the already existing connections between individuals. To do so we again use a probabilistic approach: at every time step we randomly remove and add a certain number of edges. We consider

the probability of addition and removal to be equal so that the total number of links is statistically the same.
This is done to simulate the fact that people do not always interact with the same ones.

4.5 Events

To make the simulation even more realistic, we added the possibility of having special events such as a lockdown and a superspreader event (for example a rave party).
The lockdown is implemented by randomly removing a certain fraction of edges for a given period of time after which the same ones are inserted back into the network.
Similarly, the superspreader event is implemented by adding a certain fraction of new edges in the network for a given period of time, after which they are removed.
In our simulations we choose to remove the 90% of the connection during a lockdown and to add the 50% of the connections during a superspreader event.

4.6 Targeted Infection

Most of the features that we added work on a random basis. This is fine for the ER and WS networks since they have a random nature. However, the BA network has a more elaborate structure given by the presence of the hubs. For this reason we added the possibility of building the initial state in two different ways: setting the initial infectious in the nodes with highest degree or in the ones with lowest degree. In this way we are able to see how differently the epidemic spreads, highlighting the fact that the hubs are the key drivers of transmission.

5 Results

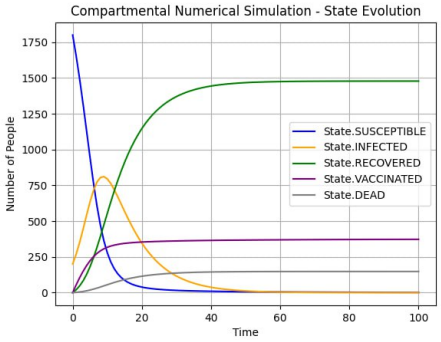
In this section we are going to present all the results obtained from the simulations. In particular we are going to show the behaviours of the three networks under different situations (described in the following subsections) and to compute some general observables: the day in which the infection has its peak, the maximum number of infectious individuals reached during the epidemic, the epidemic duration (whose end is estimated as the day in which the number of infectious goes below 1) and the case fatality rate which represents the virus lethality (estimated as the number of deaths at the end of the epidemic over the number of total infectious during all the epidemic).

5.1 Comparison between Numeric and Static simulations for ER, WS and BA Networks

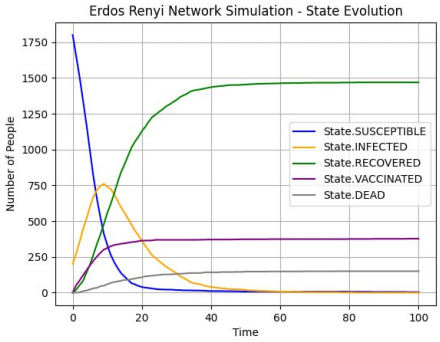
In this subsection we are going to compare the numeric simulations with the static version of all the three networks with constant parameters. Firstly, we are going to do it for the classical SIVRD model without the breakthrough and then we are going to do the same including it. Then we are going to study two particular cases, corresponding to the limits in which $\nu = 0$ and $\nu \to 1$. The former corresponds to the SIRD model, while in the latter we assume that the majority of the population has been vaccinated. The results are shown in the following pages.

5.1.1 SIRVD

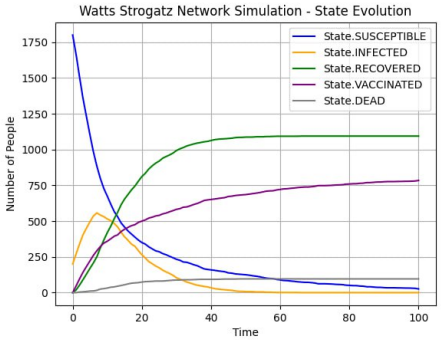
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $I_{init.} = 200$, simulation time = 100days. In Fig.4 and Tab.2 we show the results.



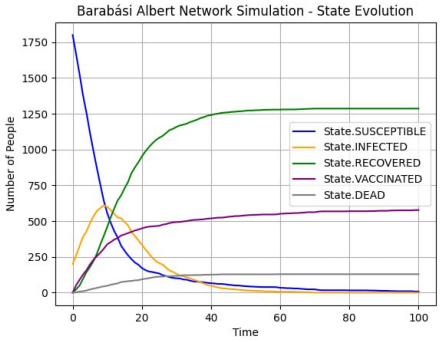
(a) Numerical Simulation



(b) Erdős–Rényi with $p = 0.03$



(c) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$



(d) Barabási–Albert with $m = 3$

Figure 4: *SIRVD results*

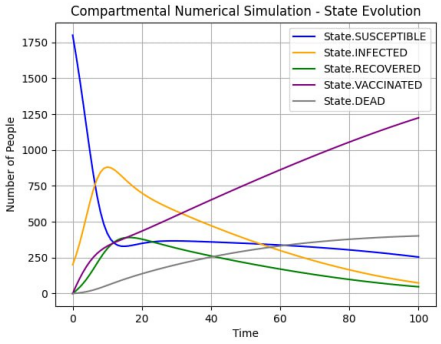
	Numerical	**ER**	**WS**	**BA**
Infection Peak Day	9	9	7	9
Infectious Peak	810.7	760	490	610
Epidemic Duration	72	82	78	70
Case Fatality Rate	0.091	0.093	0.098	0.091

Table 2: *Epidemic Parameters Across Different Network Models*

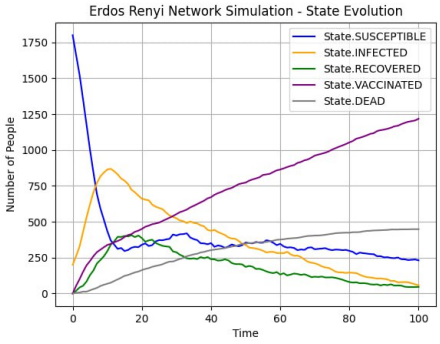
From these graphs we can see that all the types of the networks we considered are well suited to adapt the SIRVD model. Among them, the ER network is the one that approximates the numerical simulation best because it has the most similar structure to a totally connected network.

5.1.2 Breakthrough

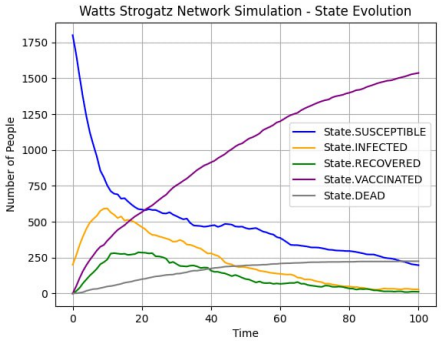
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 100 days. In Fig.5 and Tab.3 we show the results.



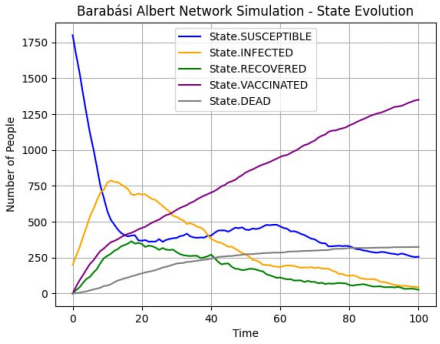
(a) Numerical Simulation



(b) Erdős–Rényi with $p = 0.03$



(c) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$



(d) Barabási–Albert with $m = 3$

Figure 5: *SIRVD with Breakthrough results*

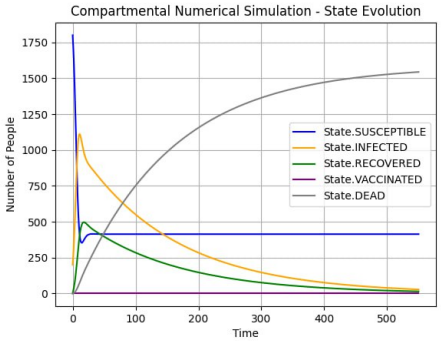
	Numerical	**ER**	**WS**	**BA**
Infection Peak Day	10	9	10	11
Infectious Peak	881.1	849	593	788
Epidemic Duration	160	155	101	101
Case Fatality Rate	0.091	0.089	0.084	0.088

Table 3: *Epidemic Parameters Across Different Network Models*

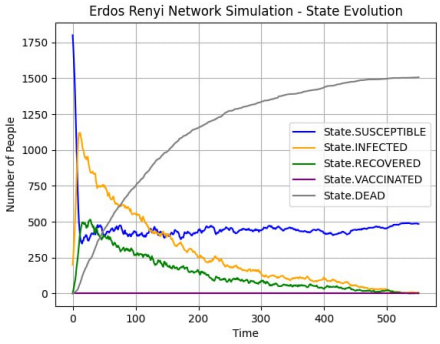
In the network simulations we see an oscillatory behaviour due to breakthrough parameter. We can see that in this case the recovered are much lower than in the previous one because they can go back to the susceptible state.

5.1.3 No-Vax

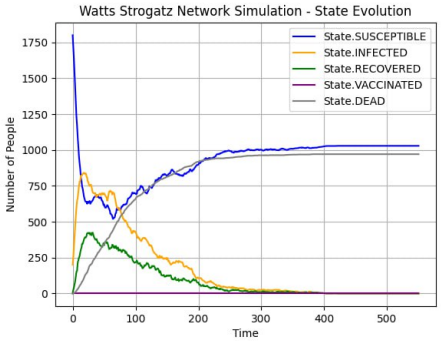
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 550 days. In Fig.6 and Tab.4 we show the results.



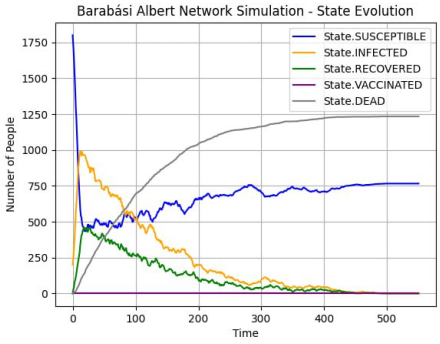
(a) Numerical Simulation



(b) Erdős–Rényi with $p = 0.03$



(c) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$



(d) Barabási–Albert with $m = 3$

Figure 6: *No-Vax results*

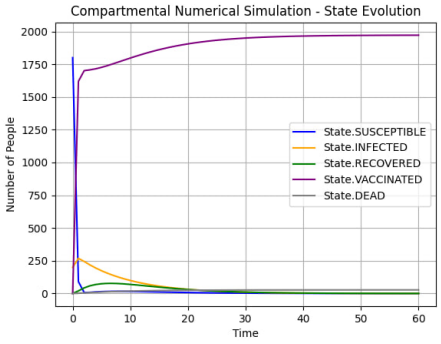
	Numerical	**ER**	**WS**	**BA**
Infection Peak Day	11	11	18	12
Infectious Peak	1111.64	1109	841	991
Epidemic Duration	501	501	415	494
Case Fatality Rate	0.091	0.092	0.093	0.090

Table 4: *Epidemic Parameters Across Different Network Models*

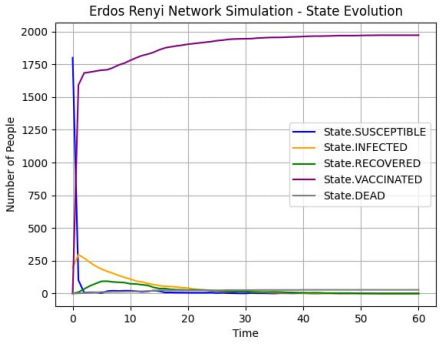
We wanted to simulate a situation where nobody in the population gets vaccinated. As expected there is a very high peak of infectious at the beginning of the epidemic and a very high number of deaths. So, we can conclude that it is important for people to get vaccinated.

5.1.4 Pro-Vax

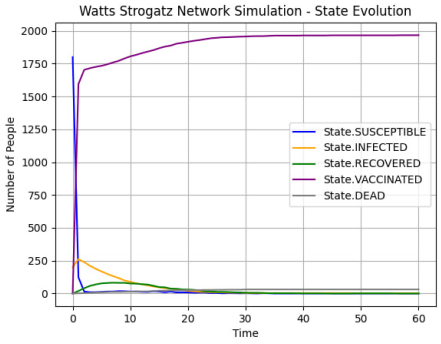
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.9$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 60 days. In Fig.7 and Tab.5 we show the results.



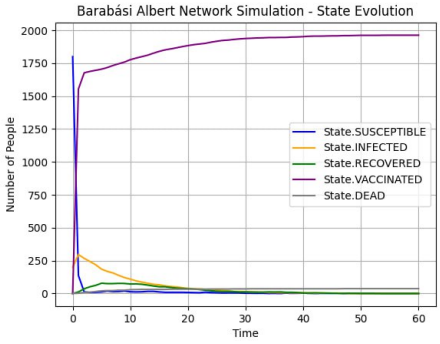
(a) Numerical Simulation



(b) Erdős–Rényi with $p = 0.03$



(c) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$



(d) Barabási–Albert with $m = 3$

Figure 7: *Pro-Vax results*

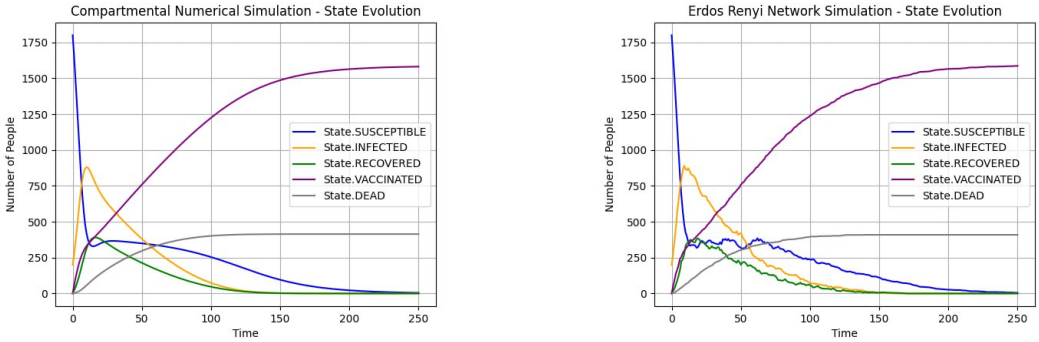
	Numerical	**ER**	**WS**	**BA**
Infection Peak Day	1	1	1	1
Infectious Peak	268	296	262	296
Epidemic Duration	50	45	61	48
Case Fatality Rate	0.091	0.088	0.110	0.120

Table 5: *Epidemic Parameters Across Different Network Models*

As opposed to the previous case we see that when the majority of the population gets vaccinated the infectious peak is much lower and also the epidemic duration is reduced by a factor of ten.

5.2 Comparison between Numerical and Static simulation of a ER Network with $p = 1$

In this simulation we show that a ER Network with $p = 1$ is equivalent to the numerical simulation because all the nodes are connected with each other. In fact, in this case the graph is completely connected.
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 250 days. In Fig.8 and Tab.6 we show the results.



(a) Numerical Simulation

(b) Erdős–Rényi with $p = 1$

Figure 8: *Results of the Comparison between Numerical and Static simulation of a ER Network with* $p = 1$

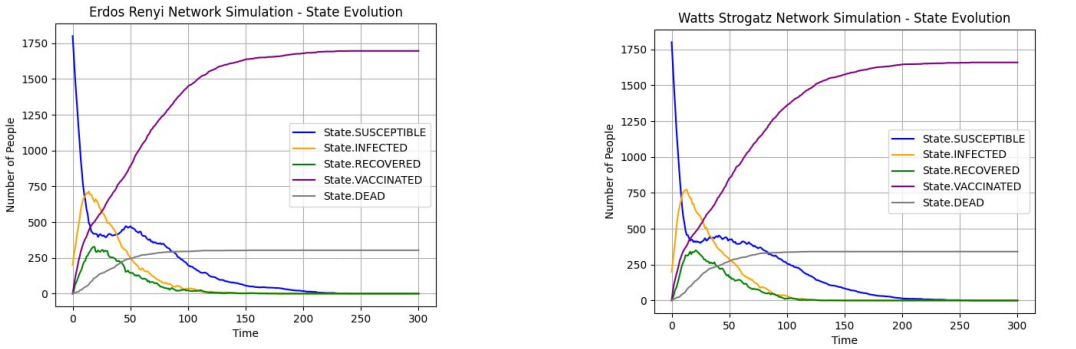
	Numerical	**ER**
Infection Peak Day	10	9
Infectious Peak	881.1	891
Epidemic Duration	160	162
Case Fatality Rate	0.091	0.088

Table 6: *Epidemic Parameters Across Different Network Models*

As we expected a totally connected ER network approximates extremely well the numeric SIRVD model.

5.3 Comparison between a ER and WS Network

In this simulation we show that a WS Network with $p = 1$ and $\langle k \rangle = 6$ is (almost) equivalent to a ER Network with $p = \frac{\langle k \rangle}{N-1}$.
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 300 days. In Fig.9 and Tab.7 we show the results.



(a) Erdős–Rényi with $p = \frac{\langle k \rangle}{N-1}$ (b) Watts–Strogatz with $\langle k \rangle = 6$, $p = 1$

Figure 9: *Results of the Comparison between a ER and WS Network*

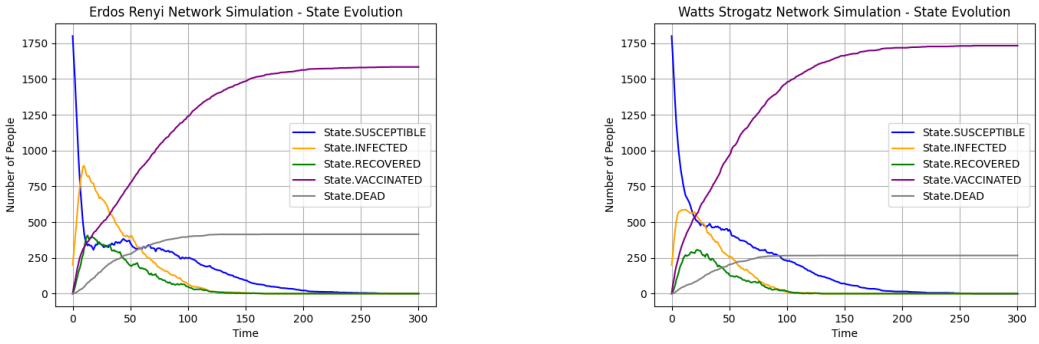
	ER	**WS**
Infection Peak Day	14	13
Infectious Peak	715	774
Epidemic Duration	171	144
Case Fatality Rate	0.091	0.095

Table 7: *Epidemic Parameters Across Different Network Models*

We can see that the two networks give very similar results, however, they are not completely equivalent due to differences in the degree distribution, clustering coefficient and edge formation of the two networks.

5.4 Comparison between a ER and a WS dynamical Network

In this section we are not considering the BA network, since randomly modifying the links would also modify the intrinsic structure of the BA network.
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 300 days. In Fig.10 and Tab.12 we show the results.



(a) Erdős–Rényi with $p = 0.03$

(b) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$

Figure 10: *Results of the Comparison between a ER and WS Dynamical Network*

	ER	**WS**
Infection Peak Day	10	12
Infectious Peak	893	588
Epidemic Duration	164	128
Case Fatality Rate	0.090	0.083

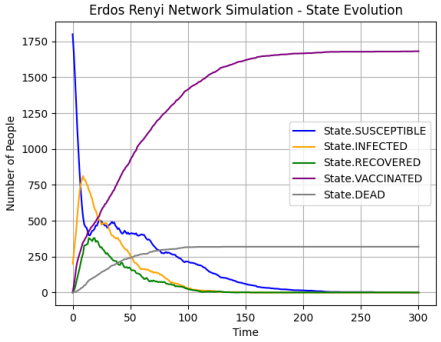
Table 8: *Epidemic Parameters Across Different Network Models*

We can see that the dynamics does not affect the general behaviour of the epidemic because both the dynamics and the ER and WS networks are implemented on a random base.

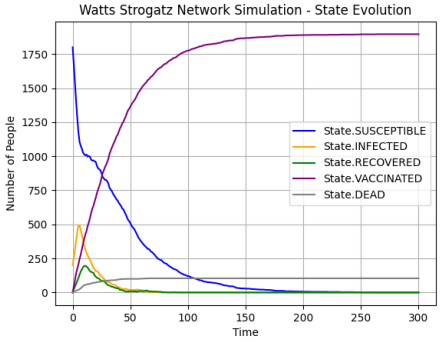
5.5 Comparison between a ER and a WS dynamical Network with Events

For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 300 days.

5.5.1 Lockdown

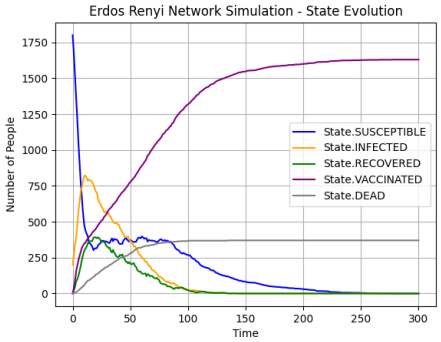


(a) Erdős–Rényi with $p = 0.03$

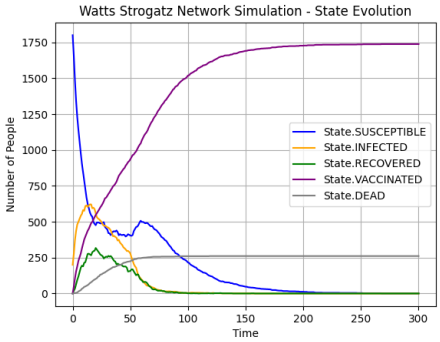


(b) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$

Figure 11: *Comparison between ER and WS with lockdown in days* 5 *to* 50 *(L1)*



(a) Erdős–Rényi with $p = 0.03$



(b) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$

Figure 12: *Comparison between ER and WS with lockdown in days* 50 *to* 100 *(L2)*

	ER	WS
Infection Peak Day	9	5
Infectious Peak	812	494
Epidemic Duration	152	89
Case Fatality Rate	0.090	0.107

(a) *Epidemic Parameters for L1*

	ER	WS
Infection Peak Day	11	15
Infectious Peak	822	621
Epidemic Duration	139	155
Case Fatality Rate	0.089	0.093

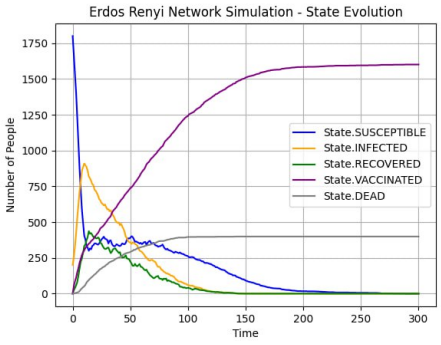
(b) *Epidemic Parameters for L2*

Table 9: *Comparison of Epidemic Parameters in Two Scenarios*

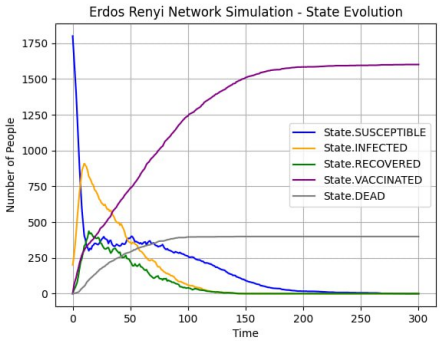
From the values of the infectious peak and the epidemic duration we see that the WS network is the one that can better approximate a situation with lockdowns. As

expected having a lockdown at the beginning of the epidemic reduces the infectious peak and the duration.

5.5.2 Superspreader

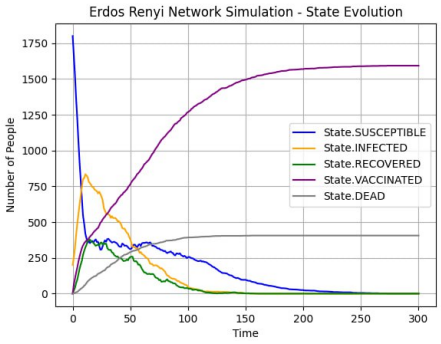


(a) Erdős–Rényi with $p = 0.03$

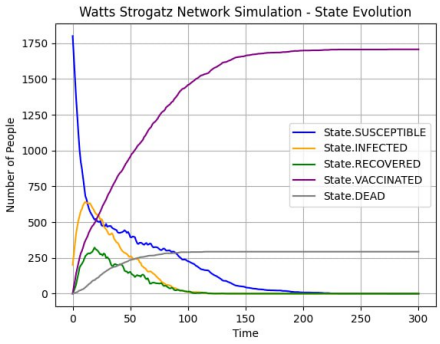


(b) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$

Figure 13: *Comparison between ER and WS with superspreader in days* 5 *to* 15 *(S1)*



(a) Erdős–Rényi with $p = 0.03$



(b) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$

Figure 14: *Comparison between ER and WS with superspreader in days* 50 *to* 60 *(S2)*

	ER	WS
Infection Peak Day	10	13
Infectious Peak	909	741
Epidemic Duration	146	126
Case Fatality Rate	0.087	0.094

(a) *Epidemic Parameters for S1*

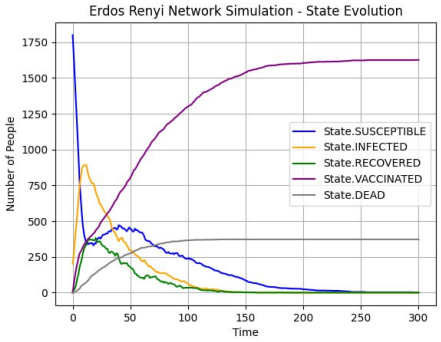
	ER	WS
Infection Peak Day	11	11
Infectious Peak	834	642
Epidemic Duration	155	128
Case Fatality Rate	0.093	0.092

(b) *Epidemic Parameters for S2*

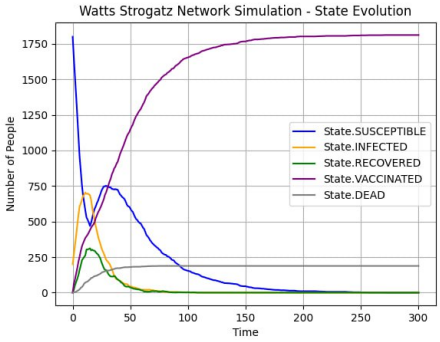
Table 10: *Comparison of Epidemic Parameters in Two Scenarios*

Also in this case the WS network is the one that better approximates the introduction of a superspreader event. The superspreader at the beginning of the epidemic produces more infectious than a later one because at the beginning there are more susceptible individuals that are neither vaccinated nor dead.

5.5.3 Lockdown and Superspreader



(a) Erdős–Rényi with $p = 0.03$



(b) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$

Figure 15: *Results of the Comparison between a ER and WS Dynamical Network with a superspreader in days* 1 *to* 10 *and a lockdown in days* 15 *to* 60

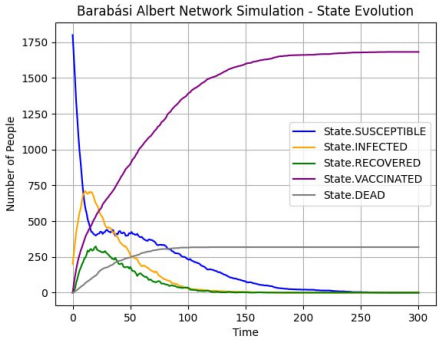
	ER	**WS**
Infection Peak Day	11	11
Infectious Peak	892	704
Epidemic Duration	178	104
Case Fatality Rate	0.092	0.099

Table 11: *Epidemic Parameters Across Different Network Models*

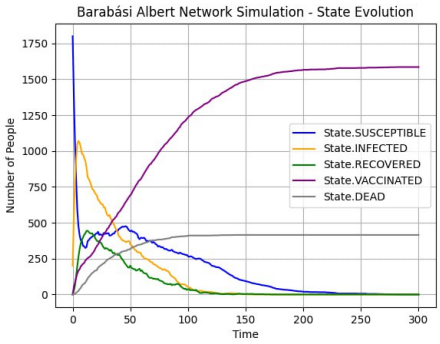
We see that having a lockdown right after a superspreader event reduces the epidemic duration. Again the WS network is the one that better simulates this case.

5.6 Comparison between Static BA Networks with different target options

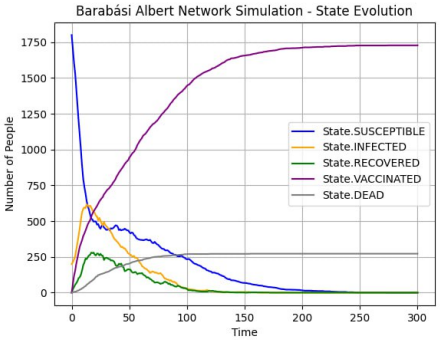
In this simulation we compare the BA network without any target (like the one we already got) with a BA in which the initial infectious are the most/least connected nodes.
For this simulation we use the following values for the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 300 days.



(a) BA with $m = 3$ and no target



(b) BA with $m = 3$ and target on most connected nodes



(c) BA with $m = 3$ and target on least connected nodes

Figure 16: *Results of Comparison between Static BA with different target options*

	BA no target	**BA target high**	**BA target low**
Infection Peak Day	11	5	13
Infectious Peak	710	1070	616
Epidemic Duration	178	154	163
Case Fatality Rate	0.092	0.089	0.084

Table 12: *Epidemic Parameters Across Different Network Models*

In this simulation we exploited the hub structure of the BA network. In fact we see that when the infection is targeted to the hubs, the infectious peak is much higher.

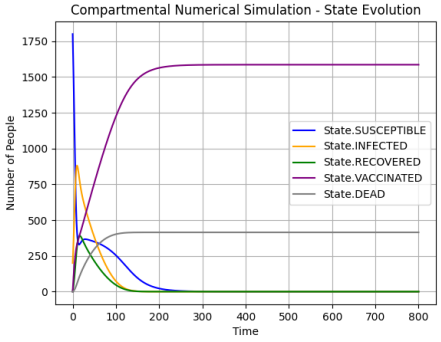
5.7 Comparison between Numeric Simulation with constant parameters and ER, WS and BA Networks parameters from real data

In this section we compare the numeric simulations with constant parameters and ER, WS and BA Networks parameters from real data. For the numeric simulation we use the parameters: $N = 2000$, $\beta = 0.5$, $\mu = 0.1$, $\nu = 0.03$, $\psi = 0.01$, $\sigma = 0.2$, $I_{init.} = 200$, simulation time = 300 days. For the simulations with parameters from real data we took as starting date first 23/02/2020, then 01/05/2020 and finally 01/10/2020. The ending date for all of the three simulations is 31/12/2022.

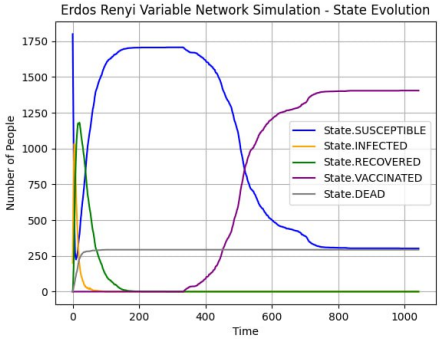
5.7.1 23/02/2020

	Numerical	ER	WS	BA
Infection Peak Day	10	5	4	5
Infectious Peak	881.1	1033	652	949
Epidemic Duration	160	99	55	62
Case Fatality Rate	0.091	0.13	0.13	0.12

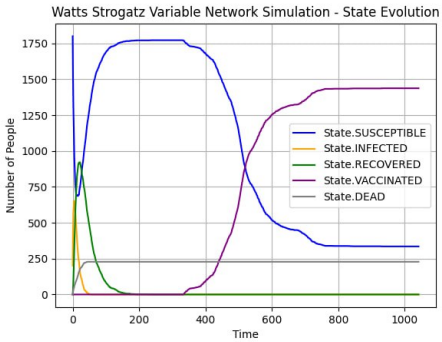
Table 13: *Start 23/02/2020 Epidemic Parameters Across Different Network Models*



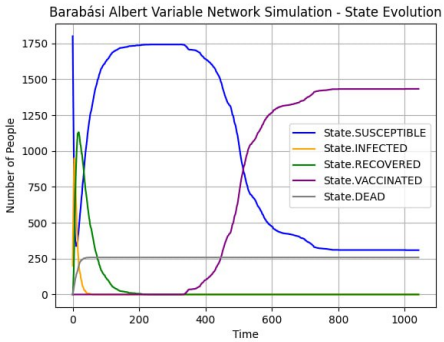
(a) Numerical Simulation



(b) Erdős–Rényi with $p = 0.03$



(c) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$



(d) Barabási–Albert with $m = 3$

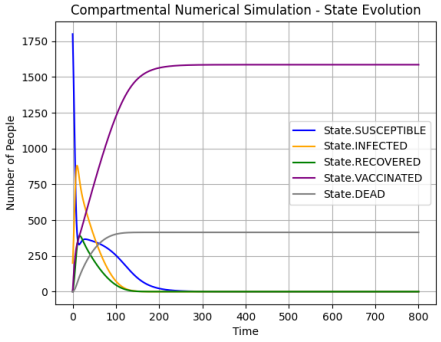
Figure 17: *Results of comparison between Numeric Simulation with constant parameters and ER, WS and BA Networks parameters from real data with starting date* 23/02/2020

We can see that starting on 23/02/2020 we have an infectious peak that is due to the fact that it was the beginning of the epidemic and we were not in lockdown yet.

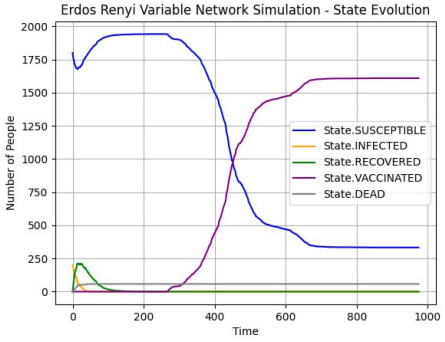
5.7.2 01/05/2020

	Numerical	ER	WS	BA
Infection Peak Day	10	0	0	0
Infectious Peak	881.1	200	200	200
Epidemic Duration	160	53	50	80
Case Fatality Rate	0.091	0.13	0.13	0.12

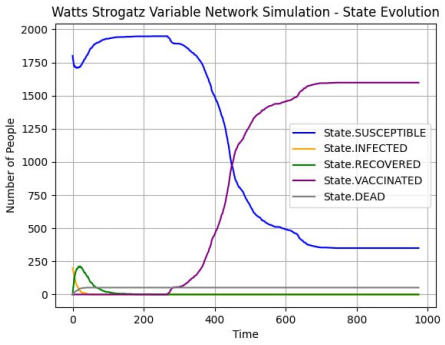
Table 14: *Start 01/05/2020 Epidemic Parameters Across Different Network Models*



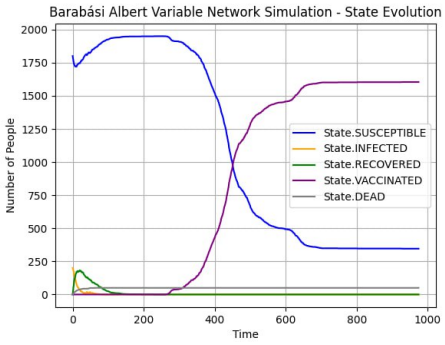
(a) Numerical Simulation



(b) Erdős–Rényi with $p = 0.03$



(c) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$



(d) Barabási–Albert with $m = 3$

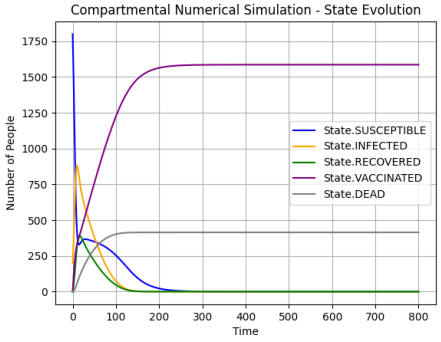
Figure 18: *Results of comparison between Numeric Simulation with constant parameters and ER, WS and BA Networks parameters from real data with starting date* 1/05/2020

Starting from 01/05/2020 we do not see an infectious peak because we were in full lockdown.

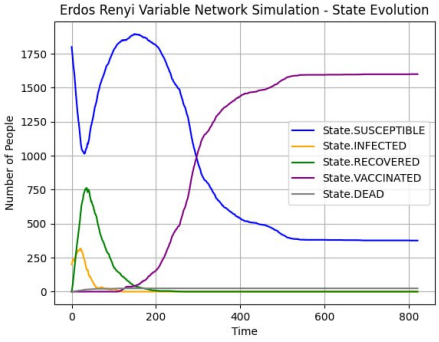
5.7.3 01/10/2020

	Numerical	ER	WS	BA
Infection Peak Day	10	20	5	1
Infectious Peak	881.1	316	213	202
Epidemic Duration	160	119	83	112
Case Fatality Rate	0.091	0.013	0.018	0.01

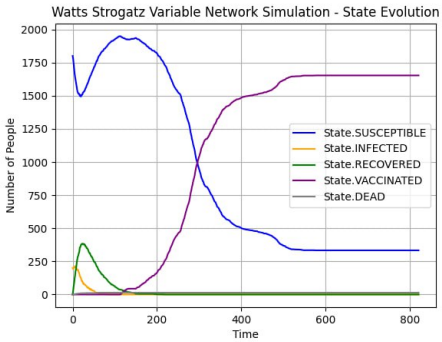
Table 15: *Start 1/10/2020 Epidemic Parameters Across Different Network Models*



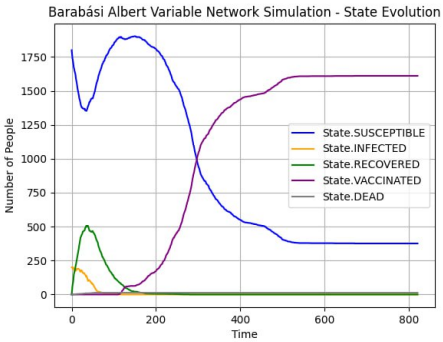
(a) Numerical Simulation



(b) Erdős–Rényi with $p = 0.03$



(c) Watts–Strogatz with $\langle k \rangle = 6$, $p = 0.03$



(d) Barabási–Albert with $m = 3$

Figure 19: *Results of comparison between Numeric Simulation with constant parameters and ER, WS and BA Networks parameters from real data with starting date* 1/10/2020

Starting from 01/10/2020 we see a small infectious peak because we were not in lockdown anymore, but we were still adopting the safety measures such as wearing masks and also the recovered individuals had some immunity.
However, in all the simulations we can see that the compartmental SIRVD model is not well fitted by the real world data.

6 Conclusions

We have reproduced successfully the behaviour of the SIRVD model using different types of networks. Also the modifications that we made are in line with what one would expect. The only simulation that does not reproduce well the compartmental SIRVD model is the one with real world data. This is not surprising since an actual epidemic has a more complex dynamics. For example, the total doses of the vaccine were three and not one, people could get infected even after getting vaccinated and also individuals who got infected could get the vaccine afterwards.

References

[1] Albert-László Barabási. *Network Science*. Cambridge University Press, 2016.

[2] A. Erdős P. Rényi. "On the evolution of random graphs". In: *Mathematical Institute of the Hungarian Academy of Sciences* 10 (1960), pp. 891–921. DOI: 10.1002/andp.19053221004.