A Spatial Markov Chain Cellular Automata Model for the Spread of Viruses

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Abstract We consider a Spatial Markov Chain model for the spread of viruses. The model is based on the principle to represent a graph connecting nodes, which represent humans. The vertices between the nodes represent relations between humans. In this way, a graph is connected in which the likelihood of infectious spread from person to person is determined by the intensity of interpersonal contact. Infectious transfer is determined by chance. The model is extended to incorporate various lockdown scenarios.

1 Introduction

At the time of writing, April 2020, the global human population is being hit by the Corona virus, in which some of the infected individuals can become seriously ill so that nursing at the intensive care unit is necessary, or that death occurs. The disease, COVID19, is often characterised by flu-like symptoms, which in some cases lead to excessive fever or even to lung inflammations. One of the serious problems regarding this disease is the high infection rate from person to person. In order to protect as many people as possible against the virus, many governments, including in Belgium, Britain and France, implement lockdown strategies. Other countries in Europe choose for herd immunity. Examples of such countries are Netherlands and Sweden. Both the Netherlands and Sweden show, despite their small population sizes, a relatively large mortality [1]. Despite their adopting of a firm lockdown strategy, Italy and Spain regret a high number of mortalities as a result of the Corona virus. Some critics claim that these high mortality rates are a consequence of having implemented the lockdown strategy only at a late stadium. Germany, on the other hand, has installed a lockdown strategy, and has registered many cases of infected people, however, fortunately Germany is only effected by a relatively low mortality. A reason for this low mortality rate could be the intensive testing policy that has been carried out by the Germans. Sweden and Netherland could possibly blame their high mortality numbers to the fact that testing is done on a small scale only. Only caregivers and very seriously ill people are tested in Sweden and Netherlands.

In order to predict the dynamics of the spread, death rate and recovery rate of the Corona virus, many different strategies are used. A very common model is the so-called SIR model, see [2] for the original paper. More modern elaborations on the SIR principle have been presented in [3] and [4]. In particular, the model in [4] bears some similarities with the approach that is presented in the current paper. This model simulates a homogeneous population that is exposed to a virus.
It contains a susceptible, resistant (recovered) and infected fraction of the population. Many more advanced models are variations on this strategy. One attempts to include spatial spread by the incorporation of diffusion terms, which are justified by random (unpredictable) migration and interaction of individuals. Other extensions are based on the incorporation of networks, which allows so-called jump processes so that airborne communication can be taken into account. The models described in [5] distinguish several challenges for network modelling in epidemics. The current model elaborates on the influence of the topology of the network on the evolution of the epidemic.

The current model has a stochastic nature. We consider a list of individuals, which can be related to each other. Each connection between two individuals contains a likelihood that a two individuals infect each other. Of course contact between individuals does not always lead to infection, and hence here a stochastic process is considered. The likelihood that each individual infects another is determined by the intensity of the contacts that the individuals have. Next to being infected, recovery is incorporated and once an individual has recovered, then it is assumed that the individual is immune to the disease. Since COVID19 can be a lethal disease in some cases, death has been incorporated as well. The model has been extended for modelling lockdown policies that certain governments have adopted. One of the advantages of the current approach is the small number of input parameters needed. A further innovation is the uncertainty quantification and the statistical assessment of the results.

Section 2 explains the mathematical model, which builds further on [6]. Section 3 contains the numerical implementation and the implementation of lockdown scenarios. Section 4 contains computer simulations and Section 5 ends up with conclusions.

2 The Mathematical Model

We consider a graph with nodes and vertices. The nodes represent individuals that can be in four states: susceptible, infected, resistant (or recovered), dead. If a person is susceptible, then this individual can be infected. Once the individual is infected, then, the person can either recover or die. If (s)he recovers then this person is assumed to be resistant. If a person is susceptible, dead or resistant, then (s)he will not spread the virus to other people (although this assumption may be subject to discussion because a non-infected could spread the virus by the hands or other objects, but this effect is neglected in the current modelling). The interpersonal relations are represented by vertices in the graph. Each connection between two nodes represent a connection. The connection is subject to an intensity, which represents the frequency that two individuals physically interact. This intensity determines the likelihood that, if one of the two individuals is infected, the disease is transferred from one to the other individual. Furthermore, infected individuals may recover or die.

Mathematically, we consider the following: We have $n$ individuals and a vector of length $n$, where each entry in this vector contains the state of individual. This vector is denoted by $v$, where the value of $v_i$ contains the integer states: $v_i \in \{1, 2, 3, 4\}$, where $v_i = 1$, $v_i = 2$, $v_i = 3$ and $v_i = 4$, respectively, correspond to the susceptible, infected, resistant and death states. All individuals are connected to other individuals by vertices between nodes (or individuals). The connection between person $i$ and $j$ is denoted by $a_{ij}$, where $a_{ij} = 0$ represents the case that individuals $i$ and $j$ have no physical contact. The entries $a_{ij}$ are assembled into the adjacency matrix $A$. Large values of $a_{ij}$ represent the intensity of the contacts. We will consider the dynamics of the spread of the disease in the coming subsections.

2.1 The transfer of the virus from individual to individual

First we consider the transfer from individual $i$ to individual $j$. Suppose that $a_{ij} \in [0, 1]$, then these two individuals are in physical contact. Suppose that person $i$ is infected and that person $j$ is susceptible. Then we assume that the infection of $j$ is a memoryless stochastic event and that
it follows an exponential distribution with infection probability rate $\lambda_{ij}$, given a time interval $\tau$, then the likelihood that person $i$ infects person $j$ is given by

$$P(v_j(t + \tau) = 2|v_j(t) = 1) = \int_t^{t + \tau} \lambda_{ij}(s) \exp(-\lambda_{ij}(s)(s - t))ds. \quad (1)$$

Furthermore, we have

$$P(v_j(t + \tau) = 1|v_j(t) = 1) = 1 - \int_t^{t + \tau} \lambda_{ij}(s) \exp(-\lambda_{ij}(s)(s - t))ds,$n

and hence

$$P(v_j(t + \tau) \in \{3, 4\}|v_j(t) = 1) = 0. \quad (3)$$

Death of individuals can also follow from other causes than Corona, but these causes are not incorporated in the current modelling. The transfer probability rate to node $j$ is determined by the infection state of the neighbour nodes of $j$, let the set of neighbours of node $j$ be given by

$$N_j = \{k \in \{1, \ldots, n\} : a_{kj} > 0\}. \quad (4)$$

For the transfer probability rate $\lambda_{ij}$, we have

$$\lambda_{ij}(t) = a_{ij}(t)\hat{\lambda}, \quad (5)$$

where we incorporated the time dependence of the intensity of contacts.

Next we consider all neighbours of node $i$ in the susceptible state ($v_i = 1$), that is the set $N_i$, consider the set of infected neighbours of node $i$, that is

$$N_i^I(t) := \{k \in N_i : v_k(t) = 2\}. \quad (6)$$

Then the likelihood that node $i$ will not transfer into the infected state is given by

$$P(v_i(t + \tau) = 1|v_i(t) = 1) = \prod_{j \in N_i^I(t)} (1 - \int_t^{t + \tau} \lambda_{ij}(s) \exp(-\lambda_{ij}(s)(s - t))ds). \quad (7)$$

This implies that

$$P(v_i(t + \tau) = 2|v_i(t) = 1) = 1 - \prod_{j \in N_i^I(t)} (1 - \int_t^{t + \tau} \lambda_{ij}(s) \exp(-\lambda_{ij}(s)(s - t))ds). \quad (8)$$

If we simplify the above expression such that $\lambda_{ij}(s) = \lambda_{ij}(t)$, then the above expression becomes

$$P(v_i(t + \tau) = 2|v_i(t) = 1) = 1 - \prod_{j \in N_i^I(t)} \exp(-\lambda_{ij}(t)\tau) = 1 - \exp(-\tau \sum_{j \in N_i^I(t)} \lambda_{ij}(t)). \quad (9)$$

Combined with the adjacency matrix, we obtain

$$P(v_i(t + \tau) = 2|v_i(t) = 1) = 1 - \exp(-\tau \sum_{j \in N_i^I(t)} a_{ij}(t)\hat{\lambda}). \quad (10)$$

This implies that the effective transfer probability rate for node $i$ is given by

$$\lambda_i^{eff} = \hat{\lambda} \sum_{j \in N_i^I(t)} a_{ij}(t). \quad (11)$$

This is summarised in Theorem 1, of which a similar version was proved in [6].

**Theorem 1:** Let node $i$ possess neighbours $N_i^I(t)$ that are infected. Then, assuming the adjacency matrix not to change during the time interval $(t, t + \tau)$, the effective probability rate in the exponential distribution for node $i$ to become infected is given by

$$\lambda_i^{eff} = \hat{\lambda} \sum_{j \in N_i^I(t)} a_{ij}(t).$$

Next, we consider the transition from being infected (possibly ill) to the recovered (resistent) or dead state.
2.2 Transition to recovery and death

People that are in the infected state await two different scenarios: recovery with being resistant or death. Some people recover very quickly after having had (very) mild or even no symptoms, whereas other people need a long time to recover or, worse, even pass away. In the current modelling, it is assumed that the recovery time follows an exponential distribution, that is

\[ P(v_i(t + \tau) \in \{3, 4\}|v_i(t) = 2) = \int_t^{t+\tau} \mu \exp(-\mu(s-t))ds, \]  

(12)

where \( \mu > 0 \) represents the probability rate for recovery. It has been assumed that \( \mu \) is constant. It is realised that \( \mu \) may be subject to temporal changes due to improvements of medical therapies against the disease. The mean recovery time from the moment that the patient was infected is determined by

\[ T_r = \frac{1}{\mu}. \]  

(13)

Let us assume that the mortality likelihood after infection is given by \( \alpha \), then, we assume that all people die who did not heal after a length of time interval \( T_d \) after infection. Hence suppose that individual \( i \) is infected at time \( t \), then \( T_d \) is determined by

\[ 1 - \int_t^{t+T_d} \mu \exp(-\mu(s-t))ds = \alpha \iff \mu \int_0^{T_d} \exp(-\mu s)ds = 1 - \alpha. \]  

(14)

This implies that

\[ 1 - \exp(-\mu T_d) = 1 - \alpha \iff T_d = -\frac{1}{\mu} \log(\alpha) = -\log(\alpha) T_r. \]  

(15)

Here we use the natural logarithm. Note that \( \alpha \) represents a probability, which does not exceed one, and in particular it is a small number in the order of at most \( 2\cdot3\% = 0.02 - 0.03 \) at most. For \( \alpha = 0.05 \), we have \( T_d \approx 3T_r = \frac{3}{\mu} \). We assume that patients that have been ill during more than a time-interval \( T_d \) have had so much damage to their vital organs (lungs and kidney) that they die. Mathematically, we can write if patient \( i \) got infected on time \( t_0 \), that is \( t_0 = \min_{t \geq 0} \{v_i(t) = 2\} \),

\[ v_i(t_0 + \theta) = \begin{cases} 3, & \text{if } \theta < T_d, \\ 4, & \text{if } \theta \geq T_d. \end{cases} \]  

(16)

3 Computational Implementation

The implementation has been done in Python. The current preliminary computations involve a simplified square topology, in which each each node has at most four connections. It is easy to revise this. We use a uniform transmissibility probability rate \( \hat{\lambda} \) to obtain the probability that the node changes from susceptible to infected during the time-step \( \tau \). To model transmission, the effective transmission probability rate is computed by the use of the adjacency matrix. Subsequently for each susceptible node a random number, \( \xi \), from the standard uniform distribution (between zero and one) is sampled, that is \( \xi \sim U(0,1) \). If the number is smaller than the probability of transmission from susceptible to infected then the state is changed from susceptible to infected, that is \( v_i \) is changed from 1 to 2, that is

\[ v_i(t + \tau) = \begin{cases} 2, & \xi < P(v_i(t + \tau) = 2|v_i(t) = 1), \\ 1, & \xi > P(v_i(t + \tau) = 1|v_i(t) = 1). \end{cases} \]

Otherwise, it stays in the susceptible state.
The fraction of individuals in state \( p \) entries \( ˆ A \) produce the standard Kronecker Delta Function:

\[
\delta_{p,k} : N \times N \rightarrow \{0, 1\} : \quad \delta_{p,k} = \begin{cases} 1, & \text{if } k = p, \\ 0, & \text{else}. \end{cases}
\]

This definition reproduces that:

\[
\beta = \beta(t) A,
\]

and in all expressions given earlier, \( A \) and its entries are replaced with \( ˆ A \) and its corresponding entries \( ˆ a_{ij} = \beta(t) a_{ij} \). Note that the lockdown policy depends on \( t \), and therefore \( \beta = \beta(t) \), where \( \beta : \mathbb{R}^+ \rightarrow [0, 1] \).

In order to compute the fractions of susceptible, dead, infected and resistant people, we introduce the standard Kronecker Delta Function:

\[
\delta_{p,k} : N \times N \rightarrow \{0, 1\} : \quad \delta_{p,k} = \begin{cases} 1, & \text{if } k = p, \\ 0, & \text{else}. \end{cases}
\]

The fraction of individuals in state \( p \in \{1, 2, 3, 4\} \) is given by

\[
f_{p}(t) = \frac{1}{n} \sum_{j=1}^{n} \delta_{p,v_{j}(t)}.
\]

This definition reproduces that \( \sum_{p \in \{1,2,3,4\}} f_{p} = 1 \). The computations are terminated as soon as the number of infected people equals zero.

In order to eradicate the virus, it is needed that the number of newly infected people is smaller than the number of people that recover from the virus. The likelihood that susceptible individual \( i \) gets infected during the time interval \((t, t + \tau)\) is given by

\[
P(v_{i}(t + \tau) = 2 | v_{i}(t) = 1) = \int_{t}^{t+\tau} \lambda_{\text{eff}} \exp(-\lambda_{\text{eff}}(s - t)) ds,
\]

which implies that the total number of people getting infected during the interval \((t, t + \tau)\) can be estimated by

\[
N_{\text{inf}}(t) = \sum_{j \in \{1, \ldots, n\}} \delta_{1,v_{j}(t)} P(v_{j}(t + \tau) = 2 | v_{i}(t) = 1) = \sum_{j \in \{1, \ldots, n\}} \delta_{1,v_{j}(t)} \int_{t}^{t+\tau} \lambda_{\text{eff}} \exp(-\lambda_{\text{eff}}(s - t)) ds.
\]

The number of people that either die or recover from the virus during the interval \((t, t + \tau)\) is given by

\[
N_{\text{noninf}}(t) = \sum_{j \in \{1, \ldots, n\}} \delta_{2,v_{j}(t)} \mu = n f_{2}(t) \mu.
\]

Since the virus decays if \( N_{\text{inf}}(t) < N_{\text{noninf}}(t) \), which implies

\[
\sum_{j \in \{1, \ldots, n\}} \delta_{1,v_{j}(t)} \int_{t}^{t+\tau} \lambda_{\text{eff}} \exp(-\lambda_{\text{eff}}(s - t)) ds < n f_{2}(t)(1 - \exp(-\mu \tau)).
\]
An upper bound for the number of individuals that get infected is determined by

\[ N_{\text{inf}}(t) < n f_1(t)(1 - \exp(-\nu \cdot \max_{(i,j)\in\{1,\ldots,n\}^2} |a_{ij}| \hat{\lambda} \tau)), \]

where \( \nu = \max_{j=1}^{|N_j|} \), which is the maximum number of neighbours of any individual. Hence a sufficient condition for a decay of the virus is obtained for

\[ f_1(t)(1 - \exp(-\nu \cdot \max_{(i,j)\in\{1,\ldots,n\}^2} |a_{ij}| \hat{\lambda} \tau)) < f_2(t)(1 - \exp(-\mu \tau)). \]

Taylor’s Theorem applied to both sides in the above equality implies

\[ \nu \cdot \max_{(i,j)\in\{1,\ldots,n\}^2} |a_{ij}| \leq f_2(t) \frac{\mu}{f_1(t) \hat{\lambda}}, \]

which is a sufficient condition to have the epidemic decay. This implies that the number of contacts between individuals should be decreased (decrease \( \nu \)) or the intensity of contacts should be decreased (decrease of \( \max_{(i,j)\in\{1,\ldots,n\}^2} |a_{ij}| \)) in order to have the epidemic vanish.

### 4 Computer Simulations

In the basic configuration, we consider a rectangular arrangement of 100 \( \times \) 100 nodes. Each node has five neighbours, except for the nodes on the boundary, which have three neighbours. Initially all nodes are in susceptible state, except for one nodal point in the centre of the domain. The numerical input values are given in Table 1.

| Parameter | Value | Unit |
|-----------|-------|------|
| \( n_x \times n_y \) | 100 \( \times \) 100 | - |
| \( \Delta t \) | 0.1 | day |
| \( \hat{\lambda} \) | 5 \( \cdot \) 10\(^{-3} \) | day\(^{-1} \) |
| \( \mu \) | 2.5 \( \cdot \) 10\(^{-1} \) | day\(^{-1} \) |
| \( T_d \) | 5 | day |

Table 1: Input values for the basic run

We stress that we have used hypothetic values in the current simulations and hence the simulations do not reflect reality. Calibration of the model is done in a future step. The current paper describes a feasibility study with preliminary simulation outcomes.

#### 4.1 A Basic Run

First we consider a run in which no lockdown policy is implemented. Figure 1 depicts the fractions of susceptible, infected, resistant and dead people. The results have been plotted in Figure 1. The first three parameter more or less follow the dynamics of the standard SIR models for the dynamics of an epidemic. The number of susceptible people decreases monotonically down to an end value, whereas the infected people increase during the early stages and decrease down to zero at the latest stages. This number has to decrease down to zero eventually since all infected people either recover and hence become resistant, or die. The curve for the infected people shows a slight non-monotonic behaviour due to the stochastic processes that sometimes allow more people to get infected than people transfer to death or recovery or vice versa. Furthermore the number of resistant also grows monotonically since resistant people cannot be infected again, and in the model they will not die of corona. In the first stages, the number of resistant and dead people follows more or less a logistic curve and so does the susceptible fraction. It can be seen that the epidemic has disappeared after a bit more than 100 days. In Figure 2 we plot the spatial distribution of the same run as in Figure 1. The red dots represent the infected individuals, the green dots are the resistant people, and the blue dots correspond to the dead people. The white dots are susceptible that have not
yet been in contact with the virus. It can be seen that there is an infection wave that spreads through the community over time. This wave is followed by dead and recovered people. Once a person is surrounded by recovered people and no longer interacts with infected people, then this person will never become infected. In this sense, this models protection of some people by resistant people. A weakness of the model is that in the current calculations, someone can be surrounded by dead people only. It is thought that these effects are 'second order effects' and therefore, we will disregard this issue for the time being. In future studies, this issue will be quantified.

4.2 Weak and strong lockdown scenarios

We consider a run in which we have a weak lockdown, that is we use

$$\beta(t) = \begin{cases} \tilde{\beta} = 0.5, & \text{for } t \in T_{ld} = (10, 50), \\ 1, & \text{else}. \end{cases}$$

This case corresponds to halving the amount of physical social interaction. It can be seen that such a lockdown does not change the number of casualties (dead) or resistant people in the end with respect to the case in which one does not have any policy. The only advantage is that the number of infected people is spread over a larger time-span so that the peak of patients at the hospitals is smoother. The results have been plotted in Figure 3. A more severe lockdown scenario, in which the amount of social interaction is reduced to 20% as follows

$$\beta(t) = \begin{cases} \tilde{\beta} = 0.2, & \text{for } t \in T_{ld} = (10, 50), \\ 1, & \text{else}. \end{cases}$$

During the time interval $t \in (10, 50)$, the maximum value of the adjacency matrix is equal to $\beta(t)$, and hence a smaller minimum makes it easier to satisfy condition (17).

The results have been plotted in Figure 4. It can be seen that the number of infected people remains small and here the number of people that is infected is smaller than the number of people that recover from the virus. Hence the number of resistant and dead people remains very small. This result indicates that lockdown scenarios (social distancing) can help reducing the number of infected people and ill people. This reduction could also correspond to having people wearing face masks.

![Fig. 1 The fractions of susceptible, infected, resistant and dead people as a function of time](image-url)
4.3 Uncertainty Quantification

Since the model is based on random principles, we present the results from twenty runs of the model to show the variability in the results using the basic input values from Table 1. For the sake of illustration, we do not incorporate lockdown scenarios. The results have been plotted in Figure 5. Despite the variation in the modelling results, all curves more or less reproduce the same trends. The epidemic is over after about a little more than a hundred days. The number of casualties is ranging between 25 and 30%, whereas the portion that becomes resistant is in the order of 65 – 70%. The number of people that is not infected at all lies in the order of 5%. Once again, we remark that we have just been modelling a hypothetic scenario. The calibration of the model to real outbreaks is beyond the scope of the current paper.

Using the Monte Carlo simulations with the model, we show how the model can be used to predict likelihoods of events such as that the length of the time interval that the epidemic exceeds a certain interval, the total fraction of people that have not been exposed exceeds a certain number, the total number of deaths exceeds a certain number or that the number of people that become resistant to the virus. In this simulation, 1000 Monte Carlo samples have been run using the input data set from Table 1. In order to obtain an indication of the Monte Carlo error, we estimate the relative Monte Carlo error of the stochastic variable $x$ by

$$MCE_N = \frac{s_{N}^{f}}{\bar{x}N\sqrt{N}} = \frac{\sum_{j=1}^{N} (x^{(j)} - \bar{x}^{N})^2}{(\bar{x}N)^2 N(N - 1)}.$$ 

![Fig. 2](image-url) Spatial plots of progression of the virus through the community. White, red, green and blue regions, respectively, represent susceptible, infected, resistant and dead individuals. Snapshots are after 1 day, 10 days, 50 days and 110 days.
Here \( N, s_N, x^{(j)}, \hat{x}^N \), respectively, represent the number of samples, sample standard deviation, value of \( x \) at sample \( j \), and the sample mean of \( x \) after taking \( N \) samples. In our simulations where we carried out 1000 samples, the relative Monte Carlo Error was about 0.02. In Figure 6, we show Monte Carlo estimates for the cumulative probability distribution for the fractions of died, resistant, and susceptible people. It can be seen that the most simulations indicate a fraction of about 0.275, 0.685, and 0.05 with some variation for the dead, resistant, and susceptible people. Furthermore, the duration times vary around 105 days. The cumulative probability densities that are presented in Figure 3 show the results once the epidemic has lasted for at least two weeks. Since the initial condition was taken as one infected node only, it happened in a few cases that the one node transferred from infected to resistant before contamination to other individuals could happen. A probabilistic model is able to predict such rare cases. These cases, and further cases in which the epidemic lasted shorter than two weeks have been ignored in Figure 6. For completeness, these cases were incorporated in Figure 7, where histograms for the duration time are presented for several (lockdown) scenarios.

Figure 7 shows histograms for various lockdown scenarios where the intensity of the lockdown policy has been varied. It can be seen that the number of occurrences that the epidemic is over within 10 days remains the same (note the scale of the horizontal axis), which is according to expectations since the lockdown strategy is applied after 10 days after viral outbreak. Further, it can be seen that a fierce reduction of interpersonal contact, which decreases the likelihood of contamination, significantly shortens the duration of the epidemic. All scenarios predicted that the epidemic was over within this lockdown period, except for one outlier, which has not be shown.

Fig. 3 The fractions of susceptible, infected, resistant and dead people as a function of time. A weak lockdown has been applied.

Fig. 4 The fractions of susceptible (left), infected, resistant and dead people (right) as a function of time. A firm lockdown has been applied. Note the change of scale.
here. A fierce reduction of inter-personal contact reduces the duration of the epidemic, and reduces the number of casualties and resistant (not shown here) dramatically. A weak lockdown, however, increases the duration of the epidemic.

Subsequently a batch of 1000 Monte Carlo simulations were run in which the input parameters have been sampled from statistical distributions:

\[ \tilde{\beta} \sim U(0.1, 1), \quad \min\{t \in T_{ld}\} \sim U(0, 20), \quad \max\{t \in T_{ld}\} \sim U(20, 40), \]

(18)

The results are shown in Figure 8. The duration of the epidemic is shown versus the severity of lockdown, as well as the fraction of people that are susceptible (who have not been infected) and the resistant (who recovered, but may have permanent health issues), and the dead versus the severity of the lockdown. All plots show a bifurcation, which results from the fact that in some runs the epidemic was over before it could spread significantly. This may happen since both the recovery rate and spreading rate are modelled on the basis of stochastic processes. In reality, this may also happen if the first receiver of the virus recovers before (s)he spreads the virus to other people. Regarding the bifurcating behaviour, in which there is a 'favourable' and 'non-favourable' branch (short duration versus long duration, large susceptible end fraction versus small susceptible end fraction, small resistant/dead end fraction versus large resistant/dead fraction). It can be seen that a more severe lockdown (smaller value of \( \tilde{\beta} \)) results into a larger number of occurrences in the favourable branch, which suggests a larger likelihood that a favourable scenario occurs in terms of smaller duration interval, smaller amount of dead and recovered people, and a larger number of people that did not develop any symptoms. It can also be seen that a very strict lockdown (reduction to 10% of the number contacts between people) no occurrences in the non-favourable branch were observed, which means that this will make epidemic die out rapidly with only few casualties.

This has been worked out in Figure 9, where the likelihood of a positive scenario in terms of number of casualties and duration of epidemic has been evaluated for different values of the severeness of the lockdown (\( \tilde{\beta} \)). The probabilities have been estimated using the Maximum Likelihood Principle. It is clear to see that a more severe lockdown (large reduction of inter-personal

![Fig. 5](image-url)

**Fig. 5** The fractions of susceptible, infected, resistant, and dead people as a function of time without any lockdown policies. Twenty runs are shown. The same input data as in Figures 1 and 2 from Table 1 have been used.
contacts) increases the likelihood that the epidemic will last during a relatively short time-interval. The same can be observed for the number of deaths.

The final simulations that are presented here involve a variation of the number of inter-personal contacts. Here we incorporate a likelihood $p_I$ to each inter-nodal connection. The random graph that we obtain is obtained by in principle connecting all nodes to one another. Subsequently, a random process is applied by sampling $\xi$ from a standard uniform distribution, that is $\xi \sim U(0, 1)$. Subsequently, we set for the initial adjacency matrix between node $i$ and $j$:

$$a_{ij} = \begin{cases} 
1, & \text{if } \xi < p_I, \\
0, & \text{if } \xi \geq p_I.
\end{cases} \quad (19)$$

This matrix determines the set of connected indices to any point $i \mathcal{N}_j$. This matrix is again updated by the random fluctuations in inter-personal contacts, by acting

$$a(i, j) \sim U(0, 1), \quad \text{if } j \in \mathcal{N}_i.$$ 

This option allows a flexible number of contacts between individuals and allows to have isolated subcommunities, in particular of $p_I$ is small. We show some preliminary results for $n = 10000$ with $p_I = 0.1$ and $p_I = 0.2$. It can be seen that the duration of the epidemic shortens significantly upon having a larger probability for connections, however, the number of casualties and recovered people increase as well for a larger probability. The number of susceptible people decreases of course with an increased likelihood for inter-personal connections. From this, it can be seen easily that a closed society is beneficial for preventing epidemic casualties. Some further computations were done for a lower likelihoods of 0.05 and lower, then the current parameter set gave no further proliferation of the virus. The results are shown in Figure 10.

Fig. 6 Cumulative probability distributions for the duration time of the epidemic, the number of susceptible people, resistant people and dead people after the epidemic has taken place. Cumulative probabilities on the vertical axis are interpreted as probabilities of the event that fewer than a certain fraction of individuals on the horizontal axis fall within the class of died, resistant, and susceptible people. The last diagram contains the duration time of the epidemic and its cumulative probability distribution that the epidemic last shorter than the time on the horizontal axis. The results were obtained by 1000 Monte Carlo samples of the probabilistic model using the input values from Table 1.
5 Conclusions

We implemented a spatial Markov Chain model using different network topologies for the progression of an epidemic using stochastic principles. We devised the model such that an uncertainty assessment, in the sense that the likelihood of different scenarios is computed, can be carried out. The model incorporates various lockdown scenarios and can be used to predict the time-evolution of epidemics under various lockdown strategies.

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Fig. 9 Probability that a given severeness of the lockdown strategy leads to a positive scenario. The horizontal axis gives $\hat{\beta}$, whereas the vertical axis gives the likelihood of a positive scenario in terms of duration of the epidemic and number of casualties.

Fig. 10 Evolution curve of the epidemic in terms of the fraction of susceptible, infected, resistant and dead people as a function of time for a random network model. Curves are shown for an inter-personal connection likelihood of $p_I = 0.1$ and $p_I = 0.2$. 