Modelling Excess Mortality in Covid-19-like Epidemics

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We discuss a stochastic model to assess cumulative excess deaths during Covid-19-like epidemics for various non-pharmaceutical interventions. The model simulates three interrelated stochastic processes: epidemic spreading, availability of respiratory ventilators and changes in death statistics. Epidemic may spread either locally or globally. The local mode simulates virus transmission through contacts in the vicinity of the place of residence while the global mode simulates virus transmission through social mixing in public places, sport arenas, airports, etc, where many people meet, who live in remote geographic locations. Epidemic is modelled as a discrete time stochastic process on random geometric networks. In the simulations we assume that the basic reproduction number is $R_0 = 2.5$ and the infectious period lasts ca. ten days. We also assume that the virus leads to severe acute respiratory syndrome in about one percent of cases, which in turn almost surely lead to respiratory default and death, unless the patient receives an appropriate medical treatment supported by respiratory ventilation. For other parameters, like mortality rate or the number of respiratory ventilators per million of inhabitants, we take values typical for developed countries. We simulate populations of $10^5 - 10^6$ people. We compare different strategies: do-nothing, social distancing, reduction of social mixing and lockdown, assuming that there is no vaccine and no efficient medicine. The results of the simulations show that strategies that slow down the spread of epidemic too much are inefficient in reducing the cumulative excess of deaths. A hybrid strategy in which lockdown is in place for some time and is then completely released is inefficient as well.
I. INTRODUCTION

Mathematical and computer modelling have proved to be very useful tools for controlling existing infectious diseases [1–4] as well as for analysing and forecasting epidemics [5–7]. Modelling of infectious diseases and epidemics has a long history [8–11]. The foundations of the contemporary theoretical epidemiology were laid by W.O. Kermack and A.G. McKendrick [12]. Today, theoretical epidemiology is a matured field of research [1–4].

In the last decades the classical epidemic models have been reformulated in the framework of complex networks science [13]. Complex networks [14–17] are very well suited to encode heterogeneity of spatial distribution [18] and mobility of population [19–21]. New techniques, which go beyond the classical mean-field approach, have been developed and successfully applied to modelling of epidemic spreading in heterogeneous systems. Among these new techniques it is worth mentioning degree-based mean-field theory [22, 23], spatial and mobility networks [19–21] and meta-population approach [24] were one can superimpose hierarchical transportation network on the population distribution in communities, cities, regions and countries, to differentiate between disease transmission modes in the regional and global scales. The models became very realistic. They can be fed with real-world data and used to forecast real world epidemics [6, 7, 20, 25–30].

In this article, we are developing a model for Covid-19-like epidemics that lead in about one percent of cases to Severe Acute Respiratory Syndrome (SARS), causing respiratory failure and death [31]. In order to make the model fully realistic for a specific epidemic, say Covid-19, it would require taking into account all medical, demographic, social, economic, geographic and any many other factors with thousands of parameters. These factors are still under active investigation and need to be determined. That is why in this paper we talk about a class of similar epidemics rather than a specific one. We focus on key factors that are important to assess the impact of various epidemic containment measures on reducing excess deaths. They are the following:

- In the absence of a vaccine immunity can only be obtained through infection;
- People who get infected become infectious for some time;
- People who recover are immune to reinfection;
- About one percent of all infections lead to SARS;
- SARS occurrence is correlated with health conditions and the age of the infected person;
- SARS leads almost surely to respiratory failure and death unless the person receives an appropriate medical assistance supported by artificial ventilation;
- Respiratory ventilation decreases the death probability; The death probability is correlated with general health conditions of the patient;
- The health care system has a limited capacity - in particular, the number of intensive care beds and mechanical ventilators is limited;
- The mortality rate of other causes like cancer, cardiovascular diseases, diabetes and other chronic diseases increases during epidemic because of epidemic restrictions on medical procedures;

The model simulates the spread of epidemics, the availability of respiratory ventilators for patients with SARS during epidemic, as well as a stochastic process that shapes population reference statistics. These three interrelated stochastic processes are important for assessing the number of excess deaths during Covid-19-like epidemics and for selecting the optimal strategy to reduce deaths.

II. RESULTS

A. Modes of infection transmission

Epidemic spreading is simulated on geometric random networks [32, 33], see Section IV A for details. There are two different disease transmission modes: a local and a global one. Local transmission corresponds to geographic epidemic spreading through human-to-human contacts near the place of residence. This is modelled as transmissions between neighbouring vertices of the network. The global mode corresponds to the disease transmission through contacts at places, like hospitals, cinemas, sport arenas, schools, universities, churches, airports, communications means, workplaces and many others, where people, who live in different geographic locations, meet. This effect is
where we compare dynamics of the epidemics for four different scenarios which differ by the basic reproduction number. We show phase portraits for epidemics with different values of $\alpha$ and they are compared to a theoretical mean-field result (8) going through the symbols. The value of the basic reproduction number in the mean-field result is $R_0 = 2.53$. (Right) Two different simulations for $\alpha = 1.0$ (symbols) compared to the mean-field result (solid line), and three different simulations for $\alpha = 0.0$.

implemented in the model as transmissions between randomly selected nodes, independently of their positions on the network. We shall call this effect long-range social mixing. Long-range social mixing leads to outbreaks in remote locations and it accelerates epidemic spreading.

In the model there is a control parameter $\alpha$ which interpolates between these two modes of transmission. The epidemic spreads by long range social mixing with a probability $\alpha$ or it spreads locally with a probability $1 - \alpha$. For $\alpha = 1$ the epidemic is described by the classical SIR mean-field dynamics [3, 12] which depends only on the node degree distribution, while for $\alpha = 0$ - by a quasi-diffusive dynamics reflecting the geographic population distribution. In Fig. 1 we show phase portraits for epidemics with different values of $\alpha$ on random geometric networks with $N = 10^5$ nodes. See Section IV B for details. As one can see from Fig. 1 the results of simulations for $\alpha = 1.0$ are very well described the by the phase-portrait (8) of the classical SIR compartmental model [3, 12]. The number of infective agents is maximal at $S_{\text{max}}/N \approx 1/R_0 \approx 0.4$ and the herd immunity is achieved for $S_{\text{hi}}/N \approx 0.1 - 0.11$, which is seen in the chart as the place were the curve hits zero. This value is close to the mean-field prediction (8). The value of the basic reproduction number, $R_0 = 2.53$, of the best fit to the theoretical curve given by the mean-field solution (8) differs by one percent from the value $R_0 = 2.5$ used in the Monte-Carlo simulations. The difference can be attributed to the fact that the classical mean-field dynamics is deterministic [3, 12] and $R_0$ is a number, while in the simulations the dynamics is stochastic and $R_0$ is the mean value of a random variable. The variance of this random variable introduces some corrections to the effective value of $R_0$.

The phase portrait starts to deviate from the mean-field solution when $\alpha$ decreases (see Fig. 1) that is when long-range social mixing gets smaller. As shown in the right panel in Fig. 1 the phase portraits for different simulations for $\alpha = 1$ lie on top of each other and are consistent with the classical SIR solution. For $\alpha = 0$ the curves differ from each other and change in a stochastic way.

The herd immunity value $S_{\text{hi}}$ weakly depends on $\alpha$ (see Fig. 1). The values of $S_{\text{hi}}/N \approx 0.10 - 0.11$ are almost identical for $\alpha = 1$, and $\alpha = 0$. What depends on $\alpha$ is the height of the curve which is a few times larger for $\alpha = 1$ than for $\alpha = 0$. This means that long-range social mixing significantly speeds up epidemic spreading. The effect is illustrated in Fig. 2 where we compare dynamics of the epidemics for four different scenarios which differ by the basic reproduction number $R_0$ and the long-range social mixing parameter $\alpha$. One can see that the spread of epidemic depends on the basic reproduction number $R_0$ as well as the long range social mixing parameter $\alpha$. The parameter $\alpha$ can be reduced by closing airports, schooles, churches, sport arenas, etc. The reproduction number can be lowered to a value $R'_0 < R_0$ through social distancing that is maintaining physical distance between people, reducing the frequency of personal contacts, wearing masks as well as disinfection, quarantine, isolation, etc.

In the next section we shall assess effects of these actions on the total mortality during epidemic using Monte-Carlo simulations of Covid-19-like epidemics.
The health care system capacity is modelled by a single. The model is implemented by superimposing SIR dynamics on a background stochastic dynamics, see Section IV B. The population size is \( N = 10^7 \), the mean node degree is \( \langle k \rangle = 100 \), the expected duration of the infectious period is \( \tau = 10 \) in all four cases presented in the figures. (Left) The number of infectious agents \( I(t) \) as a function of time \( t \) expressed in days from the beginning of the epidemic. (Right) The number of immune agents: \( I(t) + R(t) = N - S(t) \), where \( I(t), R(t) \) and \( S(t) \) are the numbers of infectious, recovered and susceptible agents, respectively. For scenario 1, the herd immunity level 90\% is reached in \( t = 199 \) days. In scenarios 2,3,4, the herd immunity levels: 89\%, 59\%, 50\% are reached in: \( t = 398, 444, 1678 \) days, respectively.

### B. Assessing mortality rate for different scenarios

We make the following assumptions in the model to incorporate these factors:

- We assume that in the the studied period there is no vaccine and no medicine available so one can only apply non-pharmaceutic interventions to mitigate epidemic spreading. To be specific let us assume that this period is 1000 days. The non-pharmaceutic interventions are divided into two classes: social distancing and reducing long-range social mixing. The first strategy is implemented in the model by reducing the reproduction number \( R_0 \) to some effective value \( R'_0 \) which is smaller than the basic reproduction number \( R_0 \), while the second one - by lowering the value of the long-range social mixing parameter \( \alpha \);

- The basic reproduction number is \( R_0 = 2.5 \). The mean infectious period is \( \tau = 10 \) days.

- The correlations between the occurrence of SARS and health conditions are modelled by dividing the population into a class of healthy people and a class of people who have some health issues, chronic diseases, or other problems requiring treatment and regular contacts with a doctor. The two classes are labeled in the model by \( H \) and \( C \), respectively. We assume that the percentage of people in the \( H \) class is \( p_H = 75\% \) and in the \( C \) class \( p_C = 25\% \). We assume also that SARS occurs with a probability \( p_{H,sars} = 1/300 \) in the \( H \) class and with a probability \( p_{C,sars} = 3/100 \) in the \( C \) class. This gives one percent on average for the whole population.

- The probability of dying of SARS is about 90\% in the \( H \) compartment and 100\% in the \( C \) compartment. The probability drops to about 10\% in the \( H \) class and to 30\% in the \( C \) class, if the person receives intensive medical care supported by mechanical ventilation.

- The number of available mechanical ventilators is 270 per million inhabitants. This sets the upper limit on the number of people who can be simultaneously ventilated;

- The mortality rate of other diseases than those caused by the virus is 27 per million per day and it increases to 30 per million per day during the epidemic. In other words, the background mortality increases by about 10\% during the epidemic.

The model is implemented by superimposing SIR dynamics on a background stochastic dynamics, see Section IV B. The background dynamics models the basic health and mortality statistics in the population. In our model it is implemented as a Markov chain which preserves the total size of the population by replacing deaths by newborns. The stationary state of the Markov chain reproduces the statistics of the \( H \) and \( C \) classes and the mortality rate of causes not related to the virus, see Section IV C for details. The health care system capacity is modelled by a single...
FIG. 3. The cumulative number of excess deaths for different scenarios in Monte-Carlo simulations of epidemic for population of $N = 10^5$ agents on random geometric network. The number is calculated as a difference between the cumulative number of deaths and $2.7t$, where $2.7$ corresponds to daily mortality rate in the population of hundred thousand people if there is no epidemic. (Left) The top curve corresponds to the worst case scenario, that is none of SARS patients receives medical assistance during the epidemic. The four curves below correspond to the scenarios 1-4 given in the main text. They also correspond to the four scenarios shown in Fig. 2. The curve in the bottom is an example of the background mortality data generated by Monte-Carlo in the absence of epidemic. In this case the number of excess deaths fluctuates around zero. (Right) The cumulative number of excess deaths for scenarios 1, 5, 6. One can see that the strategy of maintaining lockdown for some time and then replacing it by the do-nothing strategy, does not significantly reduce the total number of deaths as compared to the situation when the do-nothing strategy is introduced at the beginning of the epidemic.

number, which accounts for the number of available respiratory ventilators. The ventilators are used to ventilate SARS patients. The problem occurs when the number of SARS cases exceeds the number of available ventilators, because some people will not receive a required treatment and thus will have a lesser chance to stay alive. A ventilator is occupied by the SARS patient until he or she recovers or dies and only then it can be used for another SARS patient. It may take ten days on average, so the number of occupied ventilators saturates quickly if there are more than $27$ new SARS cases per million inhabitants per day.

We are now going to compare the total death toll of the simulated epidemic for six scenarios:

1. $R'_0 = R_0 = 2.5$ and $\alpha = 1.0$. This simulates do-nothing scenario. Epidemic spreads without any restrictions.
2. $R'_0 = R_0 = 2.5$ and $\alpha = 0.0$. This simulates a suppression of virus transmission through reducing long-range social mixing.
3. $R'_0 = 1.5 < R_0$ and $\alpha = 1.0$. This simulates social distancing and lowering the transmission rate.
4. $R'_0 = 1.5 < R_0$ and $\alpha = 0.0$. This simulates lockdown. Both, the local and global transmission modes are restricted.
5. Lockdown for 300 days, as in item 4, and then do-nothing scenario, as in item 1.
6. Lockdown for 600 days, as in item 4, and then do-nothing scenario, as in item 1.

Fig. 3 shows results of Monte-Carlo simulations of the accumulated excess death toll in the epidemic as a function of time for a population of $10^5$ people for 1000 days, for the six different scenarios. The first four scenarios are shown in the left panel in Fig. 3 and the remaining two in the right panel. In the left panel we additionally draw two reference curves representing the worse case scenario when no SARS patients receive required medical assistance during epidemic, and the best case when there is no epidemic. The charts in the right figure show what happens when lockdown is introduced right at the beginning of the epidemic, maintained for 300 or 600 days, and lifted afterwards. As a reference we also show the curve for scenario 1 which is identical as in the left figure.

Comparing the scenarios we see that after 200 days there are 703 excess deaths in scenario 1, 249 in scenario 2, 275 in scenario 3 and 70 in scenario 4, so the lockdown strategy might seem to be optimal. However when one checks the total number of surplus deaths after 1000 days, which is 709, 467, 431 and 397 in scenarios 1-4, respectively, one can see that lockdown is much less advantageous than strategies 2, 3. The total death toll for scenario 4 is comparable as for scenarios 2, 3 but in this case the epidemic is not yet dying out in contrast to the cases 2, 3 where the epidemic is over (compare Fig. 2). For scenario 4 the accumulated excess deaths curve is still rising after 1000 days. One can
FIG. 4. (Left) The number of SARS patients and the number of occupied intensive care beds equipped with ventilators. In the simulations presented in the figure, the number of ventilators is limited to 27. The difference between the two curves corresponds to the number of patients who will not receive adequate medical assistance. The figure shows results of Monte-Carlo simulations for scenarios 1, 2, 4. (Right) The number of deaths per day in scenario 1 and 2. For scenario 1 one can see a clear peak during the fast spread of epidemic. For scenario 2 one can also see a peak but it is wider and lower, and it only slightly exceeds fluctuations of the background mortality. We do not plot curves for other scenarios because the figures would be less transparent.

So far we have presented results of simulations for \( N = 10^5 \). We end this section by noting that one obtains qualitatively the same results for larger populations. As an example we compare simulations for \( N = 10^5 \) and \( N = 10^6 \) on geometric random networks with the mean degree \( \langle k \rangle = 100 \). The initial condition for SIR dynamics is \( I_0 = 5 \) infectious agents placed on randomly selected vertices of the network of size \( N = 10^5 \). All others agents (vertices) are initially susceptible. The corresponding initial condition is \( I_0 = 50 \) for \( N = 10^6 \). Also other parameters are correspondingly rescaled, for example the number of available respiratory ventilators is ten times larger, the expected number of deaths per day is ten times larger etc, to keep these numbers proportional to the population size.

The results of the comparison are shown in Fig. 5. In the left panel we compare SIR dynamics. The figure shows the fraction of infected people as a function of time. We see that the curves obtained in Monte-Carlo simulations for \( \alpha = 1 \) are almost identical for \( N = 10^5 \) and \( N = 10^6 \), as expected. This is the mean-field regime. For \( \alpha = 0 \) epidemic spreading depends on geometric details of the network. Epidemic spreading has a quasi-diffusive character in this case. For the initial conditions that we described, the average distance between locations where epidemic initially outbreaks is the same in the two cases, so one can expect that also the duration of epidemic is comparable. And indeed, this is what one can see in Fig. 5 on the left hand side. The right panel shows the number of all SARS patients and ventilated ones, for \( N = 10^6 \). The curves display a similar pattern as the one for \( N = 10^5 \) shown in Fig. 4, except that the statistical fluctuations are now smaller due to the law of large numbers.

### III. DISCUSSION

We have conducted a Monte-Carlo study of epidemic spreading on random geometric networks to assess efficiency of non-pharmaceutic interventions in reducing the total number of surplus deaths during Covid-19-like epidemics. We have discussed strategies based on social distancing and restricting long-range social mixing. They have different effects on epidemic spreading. Social distancing reduces the basic reproduction number \( R_0 \) to some effective reproduction number \( R'_0 < R_0 \). Restrictions on long-range social mixing reduce virus transmission between remote places. When
The number of SARS deaths depends on the number of respiratory ventilators. If the daily number of new SARS cases exceeds $V/\tau$ where $V$ is the number of ventilators and $\tau$ is the number of days of using one ventilator for one SARS patient, some people with SARS will not be ventilated and will have less chance to survive. If one assumes that there are 270 ventilators per million, and $\tau = 10$, then $V/\tau = 27$. As long as the number of SARS patients is below 27 per million then the number of deaths caused by SARS is minimal. This effect can be achieved by slowing down epidemic. On the other hand the number of excess deaths of other causes will increase proportionally to the epidemic duration so it is not beneficial to slow it down too much. The optimal solution is to keep the number of SARS cases close to the capacity of the health care system, but not much below it. In regions, where the number of available ventilators is very small and $V/\tau$ is comparable to the excess mortality of other diseases the best solution is to do nothing. For example, in one assumes that there are 3 excess deaths of other causes during epidemic per day per million and there are less then $V = 30$ ventilators per million then the do-nothing approach is optimal. For the same reason it is not very beneficial to slow down the epidemic to the extent that there are only a few SARS cases per day. In this case the epidemic will last a long time and there will be surplus deaths of other causes. We have also shown that a strategy of maintaining lockdown for some time and then releasing it by removing all restrictions has a similar effect on the number of deaths as if one introduced the do-nothing strategy right at the beginning. The deaths differ only by the time when they occur: in the do-nothing scenario the mortality is large at the beginning of the epidemic while in the other case it is large when the lockdown is released. A strict lockdown makes sense only if one expects that an effective medicine or a vaccine are going to be approved in a short time, or if one needs some time to increase the capacity of the health care system by buying new respiratory ventilators and training people, etc. Otherwise, the optimal strategy is to keep the epidemic progress at the level that the number of SARS patients at any time is roughly equal to the number of respiratory ventilators. If the number of SARS cases is much larger than that, too many people will die of SARS; if it is much smaller than that, the epidemic will last too long and as a result too many people will die of other causes.

For the simulated epidemic the strategies based on either social distancing or reducing long range social mixing (scenarios 2,3) give results close to the optimal one. Indeed, as one can see in Fig. 4 the number of SARS deaths in the peak of epidemic in scenario 2 does not significantly exceed the background mortality. In reality, however, it is much more difficult to tune parameters that control the spread of the epidemic and to find a way to implement appropriate actions in society.

Social distancing reduces the effective reproduction number to a value $R'_0 < R_0$ below the basic reproduction number. The herd immunity level for $R'_0$ is smaller than the herd immunity level for $R_0$. This means that after lifting restrictions on social distance and restoring normal contacts between people, the percentage of immune people will be below the herd immunity level. The system will be unstable, in the sense that a new single infective person may trigger a new outbreak of epidemic. The situation is similar to superheated liquid, where boiling may occur spontaneously.
at any time. For example, the total number of deaths in the simulated epidemic is comparable for scenarios 2 and 3 (see Fig. 3), but the percentage of immune people at the end of epidemic is 89% in scenario 2 and 50% in scenario 3, as one can see in Fig. 2. The value 89% is close to the herd immunity level for $R_0 = 2.5$ while 50% is far below it. This means that the epidemic in scenario 3 can restart from the level 50% when the restrictions are lifted and a new infective person appears. This example shows that strategies reducing long range social mixing bring better effects than social distancing. They are however much more difficult to implement.

The model that we described in this paper can be developed in many directions. One can introduce a full MSEIR dynamics, one can expand the background dynamics by implementing age groups, each having own distribution of health conditions and death statistics, one can introduce social mixing matrices for different public places where long range social mixing takes place, one can use the meta-population approach to model in detail the long range social mixing mechanisms and one can also introduce some temporal aspects.

IV. METHODS

A. Random geometric networks

Random geometric networks are constructed by the proximity rule [32, 33]. Two nodes are connected by an edge if they lie within the given distance from each other. The simplest example is a network constructed by connecting randomly distributed points in a $d$-dimensional Euclidean space. One can apply this construction to imitate a geographical proximity network in two-dimensions to model a network of contacts between people. For sake of simplicity we assume that the points are uniformly distributed on a two-dimensional square with the periodic boundary conditions. This can be done by generating pairs of coordinates $(x_i, y_i)$, $i = 1, \ldots, N$ consisting of $2N$ independent random numbers uniformly distributed on the unit interval $[0, 1]$ and connecting any two points $i$ and $j$ by an edge of the network if the distance between them is smaller than $\epsilon$: $\Delta x_{ij}^2 + \Delta y_{ij}^2 \leq \epsilon^2$. For the periodic boundary conditions the coordinate differences are calculated as follows $\Delta x_{ij} = \min(|x_j - x_i|, 1 - |x_j - x_i|)$ and analogously for $\Delta y_{ij}$. The node degree distribution of the network obtained in this way follows the binomial law

$$P(k) = \binom{N-1}{k}a^k(1-a)^{N-1-k},$$

where $a = \pi \epsilon^2$ is the area of a circle of radius $\epsilon$. The mean degree distribution is $\langle k \rangle = (N-1)a$, and the variance $\sigma^2(k) = (N-1)a(1-a)$. When $a$ is of order $1/N$ the distribution becomes Poissonian in the large $N$ limit. The average clustering coefficient of the network is large $\langle C \rangle = 1 - \frac{6\pi^2}{25\epsilon^2} \approx 0.586503$. [32]. This means that neighbours of a node are directly connected with a very high probability. This feature of geometric random networks constitutes the main difference to Erdős-Rényi random graphs [34], for which the clustering coefficient is of order $1/N$. This has also consequences for epidemic spreading on such networks.

B. Agent-based implementation of SIR dynamics

We use a discrete time stochastic implementation of the standard SIR dynamics [3, 12]. The network is populated with agents residing on its nodes. The population is divided into three classes: susceptible (S), infectious (I) and recovered (R) which describe the state of each agent at time $t$. The states change in the course of evolution according to epidemic rules which are implemented in the model in the form of a discrete time stochastic process. Time is counted in days from the outbreak of the epidemic. Initially, that is for time $t = 0$, one agent, or a few ones are infectious, while all others are susceptible. An infectious agent remains infective for $\tau$ days on average, and then it recovers. This is simulated in the model by assuming that the probability of remaining infective till next day is $q$ and of recovering is $1 - q$. The life time distribution of infectious state is given by the following exponential law

$$P_i(t) = (1 - q)^{t-1}, \quad t = 1, 2, \ldots$$

The mean life time of infectious state is related to the probability $q$ as follows

$$\tau = \langle t \rangle = \sum_{t=1}^{\infty} tP_i(t) = \frac{1}{1 - q}$$

which means that for

$$q = \frac{\tau - 1}{\tau}$$
the expected infectious period is \( \tau \) days. Clearly, for \( \tau \gg 1 \) the probability distribution (2) can be approximated by \( P_i(t) \approx e^{-t/\tau}/\tau \). Once an infective person recovers he or she remains recovered for the rest of the evolution. If a susceptible agent has a contact with an infective one and this contact is sufficient for disease transmission the agent becomes infective. In the model there are two different transmission modes: a local one – between neighbouring nodes on the network, and a global one – between randomly selected nodes independently of their locations on the network. The probability of the global transmission is \( \alpha \) and of the local one \( 1 - \alpha \). For \( \alpha = 0 \) the spread of epidemic is entirely shaped by local properties of the network while for \( \alpha = 1 \) only on average properties of the whole network. The global mode is well captured by the classical mean-field dynamics [3, 12].

Let \( p \) be a probability that the disease is transmitted from an infectious agent to a susceptible agent within one day. The probability \( p_t \) of a transmission within \( t \) days is \( p_t = 1 - (1 - p)^t \). The life time of an infectious state is a random variable (2). The transmission probability for the whole life-time can be calculated as the expected value of \( \langle p_t \rangle = 1 - ((1 - p)^t) \) for the random variable \( t \) (2). This yields

\[
\langle p_t \rangle = \frac{\tau^p}{1 + p(\tau - 1)}
\]

for \( q \) given by (4). An agent has on average \( \langle k \rangle \) neighbours, so the number of infections generated by a single infective in a fully susceptible population is

\[
R_0 = \langle k \rangle \frac{\tau^p}{1 + p(\tau - 1)}.
\]

This equation relates the basic reproduction number \( R_0 \) to the parameters of the model.

The epidemic evolution is implemented in a synchronous way in the model which means that all states are updated simultaneously. States at time \( t + 1 \) are computed from states at time \( t \). The following rules are used to update the states. If a node is recovered at time \( t \) it remains recovered at time \( t + 1 \). If a node is infectious at time \( t \) it remains infectious at time \( t + 1 \) with a probability \( q \). Otherwise, it changes to recovered. If a node is susceptible at \( t \) it changes to infectious with a probability \( p_s \). Otherwise it remains susceptible. The probability \( p_s \) of becoming infectious is related to the transmission probability \( p \) by the following relation \( p_s = 1 - (1 - p)^\ast \) where \( i \ast \) is an effective number of infectious neighbours

\[
i = (1 - \alpha) i_n + \alpha \langle k \rangle I/N.
\]

In the local transmission mode, that is for \( \alpha = 0 \), the quantity \( i \ast \) is proportional to the number of direct infectious neighbours \( i_n \). In the global transmission mode, that is for \( \alpha = 1 \), the quantity \( i \ast \) is proportional to all infectious nodes on the network \( \langle k \rangle I/N \). When \( \alpha \) changes from zero to one, \( i \ast \) interpolates between the two limiting cases.

In the classic approach one usually uses the continuous time formalism [3, 12]. The epidemic evolution is described by a set of first order ordinary differential rate equations for the fractions of susceptible, infectious and recovered agents: \( s(t) = S(t)/N \), \( i(t) = I(t)/N \), \( r(t) = R(t)/N \). The epidemic outbreaks if \( s(0)R_0 > 1 \). The quantity

\[
\phi(t) = i(t) + s(t) - \frac{1}{R_0} \ln s(t) = \text{const}
\]

is conserved during the evolution [3, 12]. \( s(t) \) is a non-increasing function of time \( t \) and \( r(t) \) is a non-decreasing function. The infectious fraction, \( i(t) \) increases for \( t < t_{\text{max}} \) and reaches a maximum for \( t = t_{\text{max}} \) such that \( R_0 s(t_{\text{max}}) = 1 \). Indeed, as one can see from eq. (8), the derivative \( di/ds = -1 + \frac{1}{R_0 s} \) changes sign when this condition is fulfilled. For \( t > t_{\text{max}} \) the epidemic begins to die out and \( i(t) \) decreases from the maximum to zero: \( i(t) \rightarrow 0 \) when \( t \rightarrow \infty \). The fraction of susceptible population for \( t \rightarrow \infty \) gives the level of herd immunity \( s(t) \rightarrow s_{\text{hi}} \). The value \( s_{\text{hi}} \) can be found from Eq. (8). In particular, if \( i(0) \) is very close to zero and \( s(0) = 1 - i(0) \), then \( s_{\text{hi}} \) is a solution to the equation \( \ln s_{\text{hi}} = R_0 (s_{\text{hi}} - 1) \). This yields \( s_{\text{hi}} \approx 0.4172, 0.2032, 0.1074, 0.0595 \) for \( R_0 = 1.5, 2, 2.5, 3 \), respectively, to give some examples.

We use the following input parameters in the Monte-Carlo simulations of the epidemic on geometric random networks: the number of agents \( N \), the mean node degree \( \langle k \rangle \), the basic reproduction number \( R_0 \), the expected duration of the infectious period \( \tau \), the probability \( \alpha \) of long range transmission. As an initial configuration we choose a configuration which consists of \( I_0 \) randomly selected infectious nodes. The remaining nodes are susceptible. The probability to remain infectious till the next day is calculated from Eq. (4). The probability of virus transmission from an infectious agent to a susceptible one within one day is calculated from Eq. (6) which gives

\[
p = \frac{1}{\tau \left( \frac{k}{I_0} - 1 \right) + 1}.
\]

An example of input values used in the simulations is \( N = 10^5 \), \( \langle k \rangle = 100 \), \( R_0 = 2.5 \), \( \tau = 10 \), \( \alpha = 0 \), \( I_0 = 5 \).
C. Modelling background conditions

In order to assess the impact of epidemics on death statistics one has also to determine the death statistics and the background conditions in the absence of epidemic. This is an interesting and very complex problem since it involves demographic factors, efficiency of health care systems, statistics of diseases, and many other factors. This is beyond the scope of this paper. Here we restrict ourselves to model only primary factors which may help to understand how the death statistics changes during epidemic. In the model this is implemented as follows. The population is divided into classes according to health conditions. In the simplest version of the model we introduce only two classes which correspond to healthy people and people with chronic diseases. We label the classes by $H$ and $C$, respectively. The division is symbolic, but it allows including in the model statistical correlations between health conditions and mortality. The mortality rate in the $C$ class is significantly higher than in the $H$ class. The second important difference is that the death probability during epidemics increases faster in the $C$ class than in the $H$ class. The details are given in the next section.

We assume that the size of population is constant during the epidemic. The number of deaths is compensated by the number of newborns. Denote the fraction of healthy people at time $t$ by $h(t) = H(t)/N$, the fraction of chronically ill people by $c(t) = C(t)/N$ and the fraction of deaths by $d(t) = D(t)/N$. We have $h(t) + c(t) + d(t) = 1$.

We implement the population dynamics as a discrete time stochastic process (Markov chain) with the following evolution equation

$$
(h(t+1), c(t+1), d(t+1)) = (h(t), c(t), d(t)) \begin{pmatrix}
\beta & 0 & 0 \\
0 & 1 - \gamma & \gamma \\
1 & 0 & 0 
\end{pmatrix}.
$$

The matrix in this equation is a stochastic matrix. It describes the transition probabilities between the states $H, C, D$. The transition rates $p_{DH}$ and $p_{DC}$ add up to one $p_{DH} + p_{DC} = 1$, which means that the number of deaths is equal to the number of newborns. The parameter $p_{DH}$ is the probability that newborns will be healthy at the beginning. For sake of simplicity, but without loss of generality, we additionally assume $p_{HD} = p_{DC} = p_{CH} = 0$. The condition $p_{HD} = 0$ means that the mortality rate of healthy people is zero or much smaller than the mortality rate of chronically ill people. The condition $p_{DC} = 0$ means that a death is replaced by a healthy newborn. The condition $p_{CH} = 0$ means that a chronically ill person does not become healthy again. Under these assumptions the last equation can be simplified to

$$
(h(t+1), c(t+1), d(t+1)) = (h(t), c(t), d(t)) \begin{pmatrix}
1 - \beta & \beta & 0 \\
0 & 1 - \gamma & \gamma \\
1 & 0 & 0 
\end{pmatrix}.
$$

The transfer matrix has only two free parameters: $\beta$ – the rate of becoming chronically ill and $\gamma$ – the rate of dying. This stochastic process has a stationary state

$$
h_* = \frac{\gamma}{\beta + \gamma + \beta \gamma},
\quad c_* = \frac{\beta}{\beta + \gamma + \beta \gamma},
\quad d_* = \frac{\beta \gamma}{\beta + \gamma + \beta \gamma}.
$$

We assume that $d_* = 27 \cdot 10^{-6}$. The value $d_*$ is adjusted to a typical mortality rate in European countries which is 27 per million per day. In most of our simulations we have $h_* \approx 0.75$ and $c_* \approx 0.25$, which means that the simulated population consists in 75% of healthy people and in 25% of people with chronic, cardiovascular, oncologic or other serious diseases. The corresponding parameters of the transfer matrix (11), which reproduce the stationary state, are $\beta \approx d_*/h_* = 36 \cdot 10^{-6}$ and $\gamma \approx d_*/(1 - h_*) = 108 \cdot 10^{-6}$.

We conclude this section with two remarks. We have assumed that there is no direct transfer from $H$ to $D$, from $D$ to $C$ and from $C$ to $H$ within one day, by setting $p_{HD} = 0, p_{DC} = 0$ and $p_{CH} = 0$. One should note that the effective transfers rates between these sectors are non-zero already for two days

$$
\begin{pmatrix}
1 - \beta & \beta & 0 \\
0 & 1 - \gamma & \gamma \\
1 & 0 & 0 
\end{pmatrix}^2 = \begin{pmatrix}
(1 - \beta)^2 & \beta(2 - \beta - \gamma) & \beta \gamma \\
\beta \gamma & (1 - \gamma)^2 & \gamma(1 - \gamma) \\
\gamma & \beta & (1 - \gamma)^2 
\end{pmatrix}.
$$
The second remark is that the square or a higher power of the transfer matrix (11) is also a stochastic matrix. In principle one can replace the original transfer matrix by any power of it, and interpret it as a daily transfer matrix. This will not change the stationary state. The stationary state is a left eigenvector of the transfer matrix associated with the eigenvalue 1 and it is identical for the transfer matrix (11) or any power of it. The transfer matrix (11) has three eigenvalues. The one which has the largest absolute value is $\lambda_1 = 1$ and the second largest is $\lambda_2 \approx 1 - \beta - \gamma$. The eigenvalue $\lambda_2$ tells us about correlations of states at different times $t, t'$. The correlation function decays exponentially as $\exp(-|t - t'|/T)$. The correlation time $T$ can be derived from $\lambda_2$: $T \approx -1/\log(\lambda_2) \approx 1/(\beta + \gamma)$. For the transfer matrix (11) $T$ is of order $10^4$. By raising this matrix to the $n$-th power and interpreting the resultant matrix as a daily transfers matrix one can reduce the autocorrelation time from $T$ to $T/n$.

D. Simulating death statistics during epidemic

We assume that the infection leads to SARS with a probability $p_{H,\text{sars}} = 1/300$ in the $H$ compartment and with a probability $p_{C,\text{sars}} = 3/100$ in the $C$ compartment. This yields on average

$$p_{\text{sars}} = h_s p_{H,\text{sars}} + c_s p_{C,\text{sars}} \approx 1/100 = 1\%$$

where $h_s \approx 3/4$ and $c_s = 1/4$, as described in the previous section. Because $p_{C,\text{sars}}$ is nine times larger than $p_{H,\text{sars}}$ this imitates a correlation between the occurrence of SARS and the health conditions of the person. In the algorithm this is implemented by moving new members of the $H$ and $C$ classes with the probabilities $p_{H,\text{sars}}$ and $p_{C,\text{sars}}$ to the SARS risk group. Members of this group will develop SARS after infection.

Another factor which plays an important role in the death statistics during epidemics is the fatality rate for SARS. The rate depends on the age and the general health conditions of the patient, and on whether the patient is ventilated or not when SARS develops. In the model we distinguish four situations, labeled by $C_0$, $C_1$, $H_0$, and $H_1$:

- $C_0$: Patients with SARS from the $C$ class who are not ventilated
- $C_1$: Patients with SARS from the $C$ class who are ventilated
- $H_0$: Patients with SARS from the $H$ class who are not ventilated
- $H_1$: Patients with SARS from the $H$ class who are ventilated

and we set probabilities of dying of SARS to 1.0, 0.3, 0.9, 0.1 for the $C_0, C_1, H_0$ and $H_1$ groups, respectively. In the simulations we use daily SARS mortality rates, which are related to the probabilities of dying in the whole period by an equation identical to Eq. (5) in which $p_{sars}$ is interpreted as the probability of dying of SARS during the whole period and $p$ is the probability of dying within one day. Assuming that $\tau = 10$, the corresponding daily rates are 1.0, 0.041, 0.474, 0.011 for the $C_0, C_1, H_0, H_1$ compartments, respectively. This choice of parameters corresponds to the assumption that respiratory ventilation increases the probability of staying alive from 0% to 70% for people with SARS in the $C$ class, and from 10% to 90% for people with SARS in the $H$ class. During epidemic the number of people with SARS may easily exceed the number of ventilators available. We assume that there are $V = 270$ ventilators (intensive care beds) per million inhabitants as in some European countries. A patient with SARS occupies a ventilator until he or she dies or recovers. This takes ten days on average. So one can expect that if there are more than 27 new SARS cases per day, the demand for ventilators will exceed the health care capacity.

The ventilator availability is simulated as follows. At any moment of time we keep track of the number of available ventilators. If this number is larger than zero, and there is a new SARS case, the number is decreased by one, and the SARS patient is moved from the $C_0$ to $C_1$ or $H_0$ to $H_1$ compartment, respectively. The ventilator is occupied until the patient recovers or dies, in which case the number of available ventilators is increased by one. Initially, the number of ventilators is set to 270 per million.

Another factor which has to be taken into account in assessing the epidemic total death toll is a lower efficiency of the healthcare system during epidemic. This has an impact on the increase of deaths of other causes which are not directly related to SARS. The effect is more pronounced in the group of people with chronic diseases who require continuous medical assistance, but it is also seen in the group of healthy people because regular medical procedures are slowed down due to epidemic precautions in hospitals. One would have to conduct systematic statistical surveys to estimate this effect. Here we just assume that the number of deaths of other causes than those directly related to SARS increases from 27 to 30 per million per day during epidemic - roughly by 10%. We implement this effect by changing values of the parameters $\beta$ and $\gamma$ in the transfer matrix (11) from $\beta = 36 \cdot 10^{-6}$ and $\gamma = 108 \cdot 10^{-6}$ to $\beta = 40 \cdot 10^{-6}$ and $\gamma = 120 \cdot 10^{-6}$, during epidemic.
ACKNOWLEDGMENTS

I thank Krzysztof Malarz for interesting discussions and assistance in preparing plots.

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