Extending the SIR model through Branching Processes

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1 Introduction

Compartmental models, like the SIR model, are widely researched and used for predicting the spread of infections. This model can be expanded upon in many ways. In this paper, the population is considered to consist of two categories; those with a large amount of social contacts, and those with a small number of social contacts. This is a distinction that can be clearly observed during many past events, e.g. in lock downs during Covid-19 pandemic. Here, one can separate the isolating and non-isolating individuals.

Because the focus is laid upon the start of an outbreak, branching processes are utilized. Although most of the conclusions stem from the analysis of the moment generation functions of the branching process, this process is also simulated in discrete time. For this reason, mainly the chance of a small-scale outbreak, compared to a large-scale outbreak is examined. When the infection reaches a certain threshold, the chance of it infecting the whole population becomes virtually 1, as seen in Figure 4.

The presented model is used to find critical points in the chance of a large scale outbreak occurring, considering different parameters. The time it takes for low-contact individuals to be shielded from an outbreak, when the infection only occurs in high-contact individuals, is also investigated. This will be applied to different relevant situations where the distinction between high-contact and low-contact individuals is warranted. Among these are the aforementioned lock downs, and nursing homes. This is useful to find the proportion of a population that needs to reduce the amount of contacts to prevent a large scale outbreak of an infection, or to protect a vulnerable subpopulation. The first infectious individual can be a low-contact or high-contact individual. This can have an impact on the overall behaviour of the infection, and is also studied in this paper.

2 Methods

Suppose we have a population with a fixed number of individuals N. We consider two categories of individuals: the first category, formed by individuals who have a small amount of social contacts, and the second category, with individuals who have many social contacts. We assume that a fraction p of all individuals is in the first category (low-contact individuals), with $0 \leq p \leq 1$. Therefore, a fraction $1-p$ of all individuals is in the second category (high-contact individuals).

We will model the spread of an infection in this population as a Markov chain, and we will approximate it as a two-type Branching Process. This approach will result useful for gaining insight into the initial spread of the infection.

We consider $S(t)$ susceptible, $I(t)$ infected and $R(t)$ recovered individuals at time t. To simplify the notation, we will denote $S(t), I(t), R(t)$ as S, I, R. We separate susceptible and infected individuals to account for the two categories of the population: S_1, I_1 account for individuals of type 1, and S_2, I_2 account for individuals of type 2. We do not account for the different categories among the recovered individuals, since doing so does not provide further information regarding the dynamics of the rest of the four variables, so we will still consider R to be all of the recovered individuals. Thus, we have that $S_1 + S_2 + I_1 + I_2 + R = N$.

We consider an equal rate of recovery across both types of individuals. To get dimensionless parameters, we take the time step $\tilde{\Delta}t$ to be the average time it takes for one individual to recover.

We assume that the behaviour of each infectious individual is independent from other infectious individuals. This means we can define a 2×2 matrix B of infection rates B_{ij}, such that $B_{ij}\frac{S_j}{N}$ is the amount of susceptible individuals of type j met and infected by an individual of type i in one time step, with $i, j \in \{1, 2\}$.

In the Markov chain, each state is specified by $\{S_1, S_2, I_1, I_2\}$. As the values of the transition matrix should be in the range $[0, 1]$, the time step should be made smaller to accommodate this. This is why we define a new time step, $\Delta t = \frac{\tilde{\Delta}t}{N}$. The matrix W of all transition probabilities from any state $\{S_1', S_2', I_1', I_2'\}$ to another

state $\{S_1, S_2, I_1, I_2\}$ in one time step is given by

$$W_{\{S_1',S_2',I_1',I_2'\}\to\{S_1,S_2,I_1,I_2\}} = \begin{cases} (B_{11}\, I_1' \;+\; B_{21}\, I_2')\, \frac{S_1'}{N}\, \Delta t & \text{if } S_1 = S_1' - 1,\ I_1 = I_1' + 1, \\ & \quad S_2 = S_2',\ I_2 = I_2' \\ (B_{22}\, I_2' \;+\; B_{12}\, I_1')\, \frac{S_2'}{N}\, \Delta t & \text{if } S_2 = S_2' - 1,\ I_2 = I_2' + 1, \\ & \quad S_1 = S_1',\ I_1 = I_1' \\ I_1'\, \Delta t & \text{if } S_1 = S_1',\ I_1 = I_1' - 1, \\ & \quad S_2 = S_2',\ I_2 = I_2' \\ I_2' \Delta t & \text{if } S_2 = S_2',\ I_2 = I_2' - 1, \\ & \quad S_1 = S_1',\ I_1 = I_1' \\ 1 - ((B_{11}\, I_1' \;+\; B_{21}\, I_2')\, \frac{S_1'}{N} & \\ \quad +(B_{22}\, I_2' \;+\; B_{12}\, I_1')\, \frac{S_2'}{N} & \\ \quad +I_1' + I_2'))\Delta t & \text{if } S_1 = S_1',\ I_1 = I_1', \\ & \quad S_2 = S_2', I_2 = I_2' \\ 0 & \text{otherwise} \end{cases}$$

The first two cases in the definition of W represent an infection of an individual of type 1 and an infection of an individual of type 2, respectively. The third and fourth case represent recoveries of type 1 and of type 2, respectively. The fifth case represents the case of no change, that is, where there is no infection and no recovery.[1]

The initial sizes of S_i and I_i, $i = 1, 2$ are defined as follows:

$$\begin{aligned} S_1 &= pN - I_1^0 \\ S_2 &= (1-p)N - I_2^0 \\ I_1 &= I_1^0 \\ I_2 &= I_2^0 \end{aligned}$$

where I_1^0 and I_2^0 denote the number of initial infectious individuals of type 1 and of type 2, respectively, and are predetermined parameters.

From this Markov process, we define a two-type branching process by linearising the transition matrix as follows. We have

$$I_1 \cdot \frac{S_1}{N} = I_1 \cdot \frac{pN - I_1 - R_1}{N} = pI_1 - \frac{I_1^2}{N} - \frac{I_1 \cdot R_1}{N}.$$

The terms $\frac{I_1^2}{N}$ and $\frac{I_1 \cdot R_1}{N}$ are quadratic terms, and can be ignored in the linearisation, as in the beginning of an infectious outbreak, $\frac{I_1^2}{N}$ and $\frac{I_1 \cdot R_1}{N}$ will be negligible for sufficiently large N. This results in

$$I_1 \cdot \frac{S_1}{N} \approx p\, I_1.$$

Note that this, in fact, can also be derived from $S_1 \approx pN$, i.e. the assumption that the susceptible population does not diminish significantly in the initial stages of an outbreak. As we are only interested in these starting stages of an outbreak, this is an appropriate assumption to make. Completely analogously, $I_2 \cdot \frac{S_2}{N}$ is linearised to become $(1-p)\, I_2$.

This linearisation can be represented in a new transition matrix, given by

$$W'_{\{I_1', I_2'\} \to \{I_1, I_2\}} = \begin{cases} (B_{11}\, I_1' \;+\; B_{21}\, I_2')\, p\, \Delta t & \text{if } I_1 = I_1' + 1, \\ & \quad I_2 = I_2' \\ (B_{22}\, I_2' \;+\; B_{12}\, I_1')\, (1-p)\, \Delta t & \text{if } I_2 = I_2' + 1, \\ & \quad I_1 = I_1' \\ I_1'\, \Delta t & \text{if } I_1 = I_1' - 1, \\ & \quad I_2 = I_2' \\ I_2' \Delta t & \text{if } I_2 = I_2' - 1, \\ & \quad I_1 = I_1' \\ 1 - ((B_{11}\, I_1' \;+\; B_{21}\, I_2')\, p & \\ \quad +(B_{22}\, I_2' \;+\; B_{12}\, I_1')\, (1-p) & \\ \quad +I_1' + I_2')\Delta t & \text{if } I_1 = I_1', \\ & \quad I_2 = I_2' \\ 0 & \text{otherwise} \end{cases}$$

Analysis via the probability generating functions

To study the probability of extinction (that is, the probability of achieving $I_1 = I_2 = 0$) via the two-type branching process, we will use the probability generating functions (pgf) known as the offspring pgf [2, 3]. With this approach, we will obtain the probability that one or a few infected individuals initiate a large-scale

epidemic (in contrast with the probability of the outbreak being small-scale, that is, extinguishing relatively quickly).

The offspring pgf of an infectious individual of type i takes the form

$$F_i(z_1, z_2) = \sum_{j,\,k\,=\,0}^{\infty} P_{j,k}\, z_1^j\, z_2^k$$

where $z_1, z_2 \in [0, 1]$ and $P_{j,k}$ is the probability that one individual of type i generates j infectious individuals of type 1 and k infectious individuals of type 2 [1, 2].

From the probabilities considered in the definition of W', the offspring pgf for I_1 takes the form

$$F_1(z_1, z_2) = \frac{1}{N} + (1 - (1 + pB_{11} + (1-p)B_{12})\frac{1}{N})z_1 + (1-p)B_{12}\frac{1}{N}z_1 z_2 + pB_{11}\frac{1}{N}z_1^2.$$

The first term $\frac{1}{N}$ corresponds to the probability that one infections individual of type 1 produces no infections in the next time step (that is, the individual recovers). Similarly, the term of z_1 corresponds to the probability of no change occurring: the individual produces one infection of type 1 (themselves). Last, the terms of $z_1 z_2$ and z_1^2 are the probabilities that the type 1 infectious individual infects an individual of type 2 and of type 1, respectively.

In a similar way we obtain the offspring pgf of I_2:

$$F_2(z_1, z_2) = \frac{1}{N} + (1 - (1 + pB_{21} - (1-p)B_{22})\frac{1}{N})z_2 + pB_{21}\frac{1}{N}z_2 z_1 + (1-p)B_{22}\frac{1}{N}z_2^2.$$

The mean number of offspring of type I_j from a parent I_i is [1, 2, 4]

$$m_{ij} = \frac{\partial F_i}{\partial z_j}(1, 1).$$

The matrix of expected values is given by

$$M = \begin{pmatrix} m_{11} & m_{12} \\ m_{21} & m_{22} \end{pmatrix} = \begin{pmatrix} 1 - \frac{1}{N} + pB_{11} & (1-p)B_{12}\frac{1}{N} \\ pB_{21}\frac{1}{N} & 1 - \frac{1}{N} + (1-p)B_{22}\frac{1}{N} \end{pmatrix}.$$

Its greatest eigenvalue is

$$\lambda = \frac{1}{2N}\left(\sqrt{p^2B_{11}^2 + 2p(1-p)(2B_{12}B_{21} - B_{11}B_{22}) + (1-p)^2B_{22}^2} + pB_{11} + (1-p)B_{22}\right) + 1 - \frac{1}{N},$$

and denotes the mean growth rate of the infected population [1].

By Theorems found in [1, 2],

(i) if $\lambda \leq 1$, the probability of extinction is 1.

(ii) if $\lambda > 1$, the probability of extinction of the whole infection is $u_1^{I_1^0}\, u_2^{I_2^0}$, where u_i, $i = 1, 2$, is the unique fixed point $F_i(u_1, u_2) = u_i$ such that $0 < u_i < 1$, and I_1^0 and I_2^0 are the number of initial infectious individuals of type 1 and of type 2, respectively.

Equations $F_1(u_1, u_2) = u_1$, $F_2(u_1, u_2) = u_2$ will be solved numerically for each particular set of parameters. As an example, considering $p = 0.4$, $B_{11} = 4.375$, $B_{12} = B_{21} = 7.5$, $B_{22} = 10$, we obtain $\lambda \approx 1.0057 > 1$ and the following numerical solutions for the fixed points:

$$(u_1, u_2) \in \{(-1.4867, 2.3387), (0.1545, 0.1129), (1.0, 1.0), (4.9750, -0.3683)\}.$$

From these solutions, there is exactly one that satisfies $0 < u_i < 1$, which is $u_1 = 0.1545$, $u_2 = 0.1129$. Thus the (numerical) probability of extinction for $I_1^0 = I_2^0 = 1$ is $u_1 u_2 = 0.1545 \cdot 0.1129 = 0.0174$. This quantity approximates the probability of a minor-outbreak being initiated [3]. The approximate probability of a large-scale epidemic developing is $1 - u_1 u_2 = 0.9826$.

In summary, we obtain that the probability of extinction of the whole infectious class, which serves as an approximation of the probability that an outbreak initiated by one infectious individual remains minor [3], is given by

$$\mathbf{P}_{small-scale} = \begin{cases} 1 & \text{if } \lambda \leq 1 \\ u_1^{I_1^0}\, u_2^{I_2^0} & \text{if } \lambda > 1 \end{cases} \tag{2.1}$$

where u_1, u_2 are the unique solutions of equations $F_1(u_1, u_2) = u_1$, $F_2(u_1, u_2) = u_2$ such that $0 < u_1, u_2 < 1$, and I_1^0 and I_2^0 are the number of initial infectious individuals of type 1 and of type 2, respectively.

On the other hand, the probability that a large-scale outbreak is initiate by I_1^0, I_2^0 infectious individuals is

$$\mathbf{P}_{large-scale} = \begin{cases} 0 & \text{if } \lambda \leq 1 \\ 1 - u_1^{I_1^0}\, u_2^{I_2^0} & \text{if } \lambda > 1 \end{cases} \tag{2.2}$$

Stochastic simulations

In addition to the analytical derivations, some methods have been implemented to derive results based on stochastic simulations.

First and foremost, we have implemented the simulation of outbreaks, with the primary objective of studying the initial spread of the infection and the probability of extinction in the initial phases. The implementation is in discrete time, and based on the transition matrix W for the simulations based on the Markov chain (the initial model), and the transition matrix W' for for the simulations based on the Branching Process.

It is worth noticing that, since the Branching Process offers a linearised approximation of the initial model based on a Markov chain, it is computationally less expensive, while still offering reliable results regarding the initial spread of the outbreak. Therefore, the Branching Process model will be used to run processes for which the initial approach based on W is too computationally expensive.

The chosen transition matrix is used to choose a random outcome (infection, recovery or no change in the state) in each time step, with the corresponding probability. For simplicity, the time step is assumed to be one (i.e. we simulate a discrete-time process). The evolution of the simulated outbreaks will be captured by the variables I_1, I_2 in each time step. These variables will then be analyzed to yield different results.

A counter of extinguished simulations at a given generation t_0 (or equivalently, at time t_0) is implemented. This counter is used to study the proportion of extinguished simulations at specific time points, and the general evolution of this proportion over time. We will be especially interested in how the proportion of extinguished simulations approximates the analytical probability of extinction, and how different parameters affect this proportion and its evolution over time. To this goal, we have implemented various numerical and plotting tools.

As meeting a person is a symmetrical relation, we always assume $B_{21} = B_{12}$. We are mostly interested in the case where the first category represents vulnerable individuals. Often, this will imply $p < 0.5$ and $B_{11} < B_{22}$, although we diverge from this assumption when modelling care facilities.

3 Results

We use the methods developed in the previous section to study the initial spread of an infection initiated among a population with low-contact and high-contact individuals.

We first explore the probabilities that the epidemic remains small-scale or becomes large-scale, given different initial conditions. Later, we explore how the outbreak evolves in the initial phases if the initial infectious patients have few contacts or many contacts. A question we look to answer is how long can low-contact individuals remain unaffected by the infection after an outbreak begins among high contact individuals. We will see how these different results and observations depend on the parameters considered.

Probability that an epidemic remains small-scale or becomes large-scale

From expression (2.1) we find out that the probability of an epidemic remaining small-scale (*versus* becoming large-scale, see expression (2.2)) depends on p and B. Moreover, this probability of extinction, i.e. the infection stays small-scale, increases with the proportion of low-contact population p, and decreases and with the infection rates B_{ij} (Fig. 1). For high enough p and low enough B_{ij}, the probability of extinction approaches and becomes 1 (Fig. 1).

Analogously, the probability of the outbreak becoming large-scale decreases with p, and increases with B_{ij}.

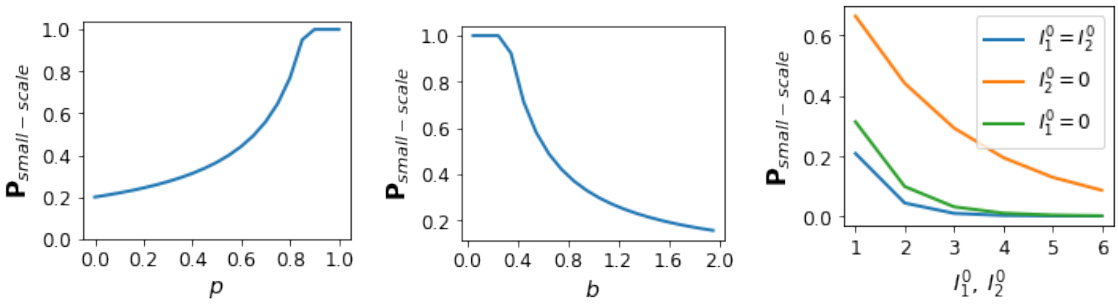

Figure 1: At the right and center, plots of $\mathbf{P}_{small-scale}$ for changing values of p and b (a parameter such that $b \cdot B$ is used as infection rates). At the right, plot of $\mathbf{P}_{small-scale}$ for changing values of I_i^0, for three cases: $I_1^0 = I_2^0$, $I_2^0 = 0$ (initial infected population of type 1 only) and $I_1^0 = 0$ (initial infected population of type 2 only). The parameters considered (when fixed) are $p = 0.1, B = \begin{pmatrix} 0.7 & \frac{1}{5} \\ 1 & \end{pmatrix}, b = 1, I_1^0 = I_2^0 = 1$.

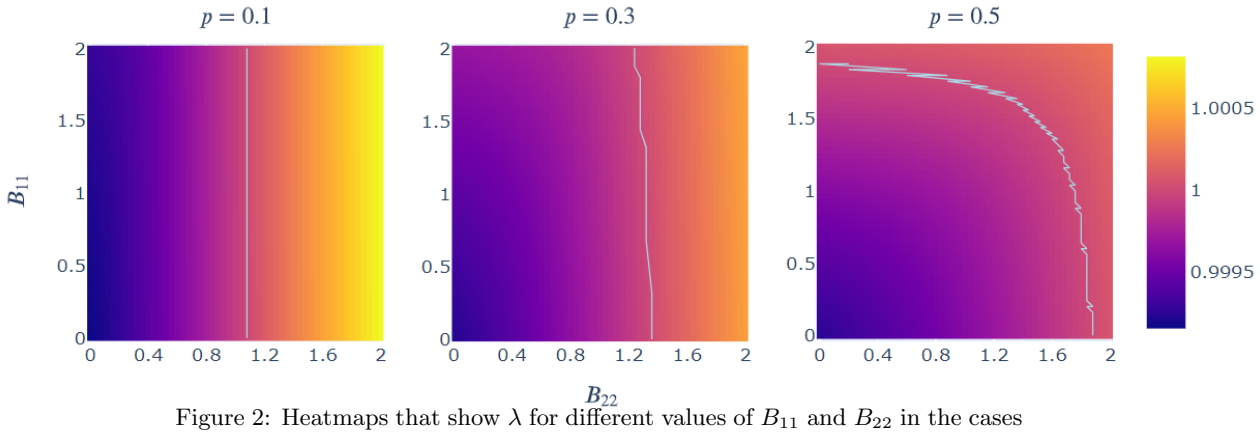

Figure 2: Heatmaps that show λ for different values of B_{11} and B_{22} in the cases of $p = 0.1$, $p = 0.3$ and $p = 0.5$. The light blue lines correspond to points where $\lambda \approx 1$ (with an accuracy of the order of 10^{-5}). The rest of the parameters considered are $B_{12} = B_{21} = 0.5, N = 1000$.

It is also worth noticing that, as the initial infectious population increases, the probability of a large-scale epidemic increases exponentially (equivalently, the probability of a minor outbreak decreases exponentially, see Fig. 1)).

In addition, it is of our interest to study how the probability of extinction changes for different values of B_{11} and B_{22}. Since a lot of numerical calculations would have to be performed to calculate $\mathbf{P}_{small-scale}$ for different values of B_{11} and B_{22}, we study the evolution of λ instead. λ represents the mean growth rate of the infected population. Therefore, the larger λ is, the lower the probability of the outbreak being contained and thus being small-scale (or, equivalently, the larger the probability of the outbreak being large-scale). See Fig. 2.

From Figure 2, we can infer that as p increases, the effect of B_{22} on the probability of an infection being small-scale, becomes smaller, and the effect of B_{11} increases. When $p = 0.1$, the influence of B_{11} is negligible.

Modeling the spread of an outbreak in the case of a lockdown

With this approach, results can be obtained for specific situations. For example, one situation that can be modeled is the case of a lockdown (Fig. 3). For a given infection, one can see how λ varies depending on the proportion of the population isolated (given by the parameter p) and the strictness of the lockdown (given by the parameters B_{11} and $B_{12} = B_{21}$). We assume an isolating person does not differentiate between the contacts they have. Therefore, in this case, we considered $B_{11} = B_{12} = B_{21}$. We are especially interested in which parameters lead to $\lambda \leq 1$, and thus to a certainty of the outbreak being small-scale, for a given typical infection.

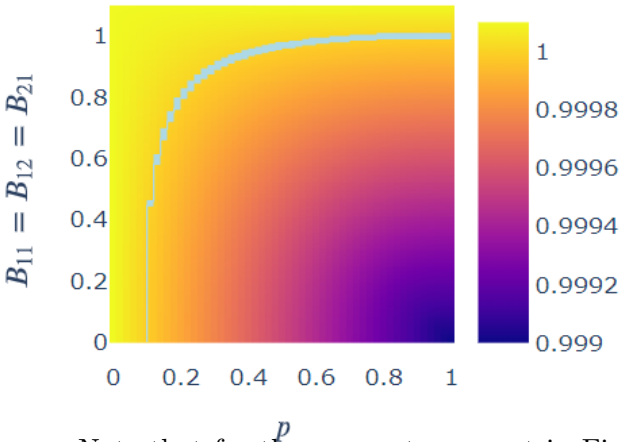

Figure 3: Heat map that shows λ for values of p between 0 and 1, and $B_{11} = B_{12} = B_{21}$ between 0 and B_{22}, corresponding to different proportions of population isolated and different grades of strictness in the case of a lockdown. The light blue line correspond to points where $\lambda \approx 1$ (with an accuracy of the order of 10^{-5}). The rest of the parameters considered are $B_{22} = 1.1$ and $N = 1000$.

Note that for the parameters as set in Fig. 3, if the lockdown consists of total isolation ($B_{11} = B_{12} = B_{21} = 0$), around 0.1 of the population should isolate to reach $\lambda \leq 1$. As a lockdown gets less strict, this proportion increases rapidly, until there is no proportion of the population that can isolate to ensure $\lambda \leq 1$. This happens at approximately $B_{11} = B_{12} = B_{21} = 1$.

Low-contact *versus* high-contact *patient zero*

We now look to answer how differently the outbreak evolves in the initial phases if the initial infectious patient has few contacts or many contacts. To do so, we will simulate both situations and plot the number of extinguished outbreaks against time.

As a first approach, to yield initial observations, we plot $I = I_1 + I_2$ over the initial time steps for a few simulations in each situation (Fig. 4). With this first approach, we observe that outbreaks initiated by a high-contact individual tend to grow faster.

Next, we simulate a higher number of outbreaks in each situation (to obtain more reliable results) and plot the proportion of extinguished simulations over time (Fig. 5). We observe that a larger proportion of simulated outbreaks extinguish in the first phases of the spread if they are started by an individual with fewer contacts. In the simulations with patient zero of type 1, 45.8% of the outbreaks are extinguished by the generation 1000. In contrast, only 21.7% of the outbreaks initiated by the patient zero of type 2 are extinguished by the same generation 1000.

The result observed in the simulations is supported by the fact that the probability of a minor-outbreak derived from the pgf is higher for the simulation initiated by a low-contact individual (0.5774) than by the high-contact patient (0.2196). The time-independent probability of extinction resulting of analytical methods can also be compared to the time-dependent one estimated through the simulations in Fig.

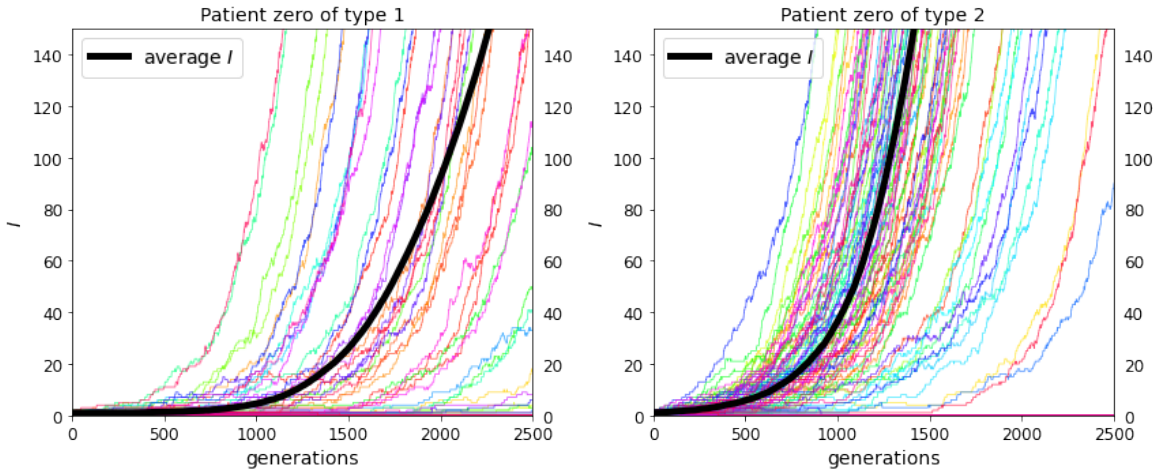

Figure 4: Results of 100 simulations of outbreaks initiated by one low-contact individual (left) and outbreaks initiated by one high-contact individual (right). Coloured lines represent $I = I_1 + I_2$, and the thick black line represents the average I over all the simulations in each generation. The parameters considered are $p = 0.1$, $B = \begin{pmatrix} 0.7 & \frac{1}{5} \\ 1 & \end{pmatrix}$ and $N = 1000$.

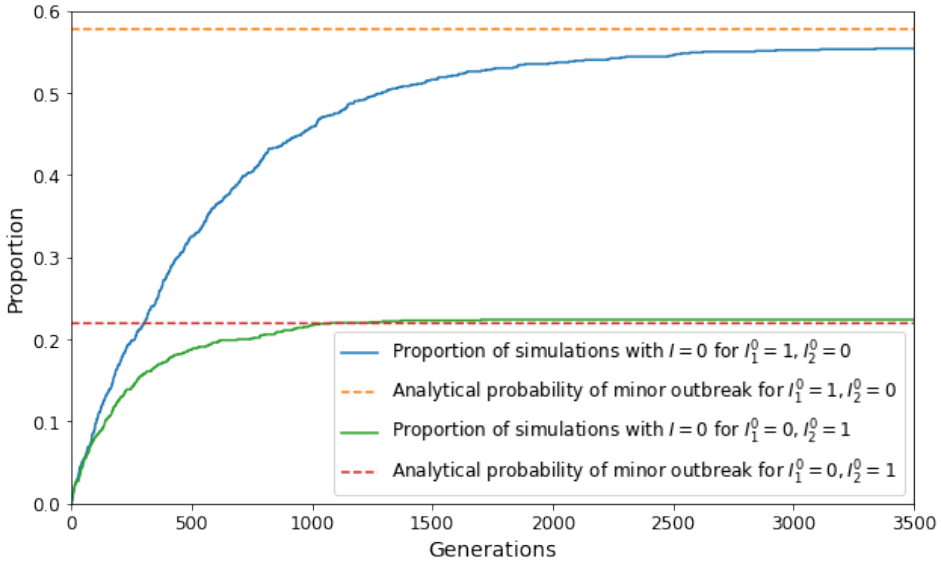

Figure 5: Results of 1000 simulations of outbreaks initiated by one low-contact individual (blue line) and outbreaks initiated by one high-contact individual (green line). Lines represent the proportion of simulations such that $I = I_1 + I_2 = 0$ for each moment in time. Dashed lines correspond to the analytical probability of extinction for the case of a low-contact patient zero (orange dashed line), and the case of a high contact patient zero (red dashed line). The parameters considered are $p = 0.1$, $B = \begin{pmatrix} 0.7 & \frac{1}{5} \\ 1 & \end{pmatrix}$ and $N = 1000$.

5. Important to note is that in the case of a high contact individual being the first infected person, the plateau phase (where almost no simulations die out) is reached remarkably faster than in the other case.

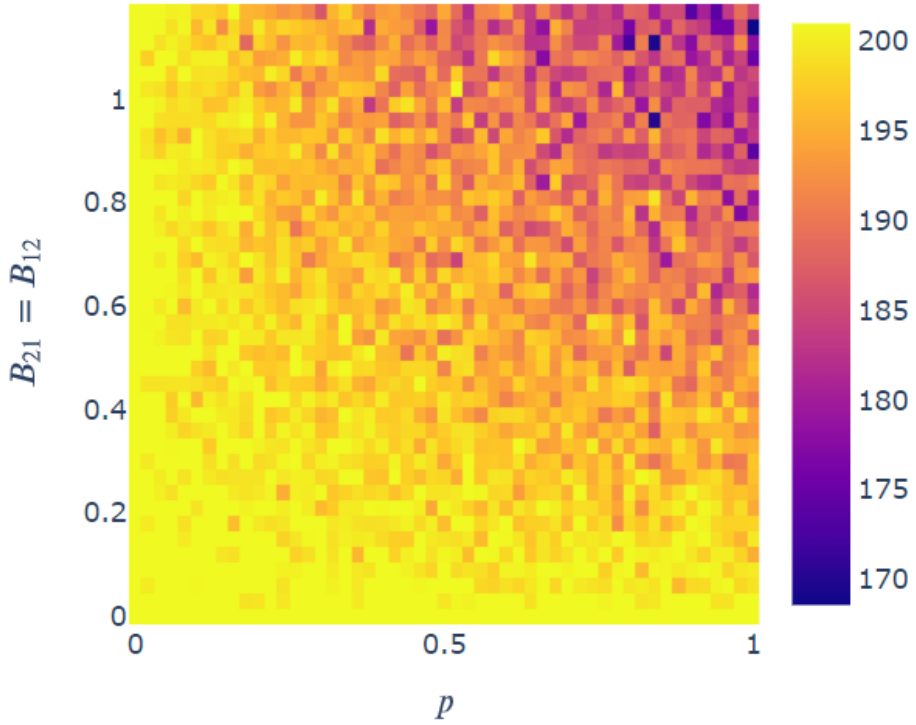

Figure 6: Mean generation up to which the type 1 population remains shielded from an outbreak initiated by type 2, over different values of p and $B_{12} = B_{21}$ in $[0, 1]$, and $[0, 1.2]$ respectively. The fixed parameters considered are $p = 0.1$, $B_{11} = 0.3$, $B_{22} = 1.1$ and $N = 1000$.The results have been obtained over 100 simulations running up to generation 200.

How long can low-contact individuals remain shielded after an outbreak begins among high-contact individuals?

One particular question we search to answer is: how long can low-contact individuals (type 1) remain unaffected by the infection after an outbreak begins among high-contact individuals (type 2)? We will also see how this quantity depends on different parameters.

We can see from Figure 6 that the low-contact population remains shielded for longer when p is smaller. Although this would seem obvious from the observation that this implies there are less low-contact individuals, it is important to keep in mind that a larger p implies a higher chance of a large-scale infection, and thus a generally faster spread to the population. This figure implies that, for the given parameters, a large amount of isolating people does not result in a longer buffer before the first infection of an isolating individual.

Note that these generations are in fact influenced by the infection's recovery rate, because the time step (or generation length) is defined as the average time it takes

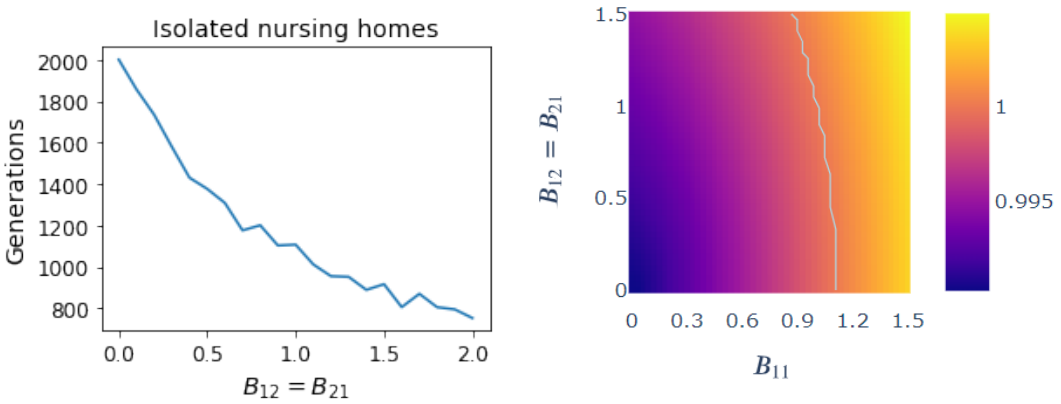

Figure 7: Left: mean number of generations up to which low-contact individuals are shielded, for different values of $B_{12} = B_{21}$ with $p = 0.9$, $B_{11} = 1.1$, $B_{22} = 0.3$, and $N = 100$. These results were gained over 2000 generations and 1000 simulations.
Right: values of λ when B_{11} and $B_{12} = B_{21}$ vary. The blue line corresponds with $\lambda = 1$. The set parameters have values $p = 0.9$, $B_{22} = 0.3$ and $N = 100$.

for an infected individual to recover from the infection.

Modeling the spread of an outbreak in a care facility

With this approach, we can also study how long can low-contact individuals remain unaffected by an outbreak initiated by high contact individuals in specific situations, such as the case of care facilities (Fig. 7).

We seek to answer how long can residents (modeled here as low-contact individuals) remain shielded after an outbreak begins among workers at a nursing home, depending on how strict the measures that prevent workers from infecting residents are.

In this case, we consider nursing homes as *isolated* from the exterior. The population exists only of residents and carers. Here, residents form the type 1 individuals, so p, the proportion of type 1 individuals, is be significantly high. Residents interact highly among themselves, more than they do with nurses, and there is very little interaction between workers among themselves. This implies a high B_{11} and low B_{22}, therefore $B_{11} > B_{22}$. The variation in $B_{12} = B_{21}$ correspond to preventive measures nurses apply to prevent an outbreak from happening among residents, by restricting carer-resident interactions. A change in B_{11} models restriction in resident-resident interactions.

4 Discussion

In general, the presented model is qualitatively different from the standard SIR model, and can be used in specific scenarios to answer questions the standard SIR model cannot answer. The use of a branching process implies that the model is most useful when the infection is not yet widespread, as seen in the assumption that $I << N$ in the linearisation from a Markov chain model to a branching process[3].

In the case of a lockdown, one can infer the effect of the strength of the lockdown, as well as the proportion of isolating individuals, on the reproductive value of the infection. This can also be used to calculate the chance of a large-scale infection occurring. As seen in Figure 2 and Figure 6, the extent to which one isolates does not have a big influence on the course of an infection, or on the protection of isolating individuals, if the proportion of isolating individuals is small. One can conclude that, in order to protect a subpopulation of vulnerable people, a big proportion of the total population has to isolate, and not only the vulnerable individuals.

When considering a care facility, the model can be used to gauge the effect of two types of measures one can take to lower infection rates. On one hand, the influence of change in interaction between carers and residents on λ and average time it takes for a resident to get infected, can be examined. From Figure 7, one can see that, with the considered parameters, the average shielded time of the residents can be doubled by restricting this interaction five-fold. However, to guarantee a small-scale infection, the interaction between the residents should also be considered.

As the time step is taken to be $\frac{1}{N} \cdot$ *average recovery time*, the parameters B_{ij} can be interpreted as $\frac{B_{ij}}{\textit{recovery rate}}$, and thus have no units in this interpretation.

One can see from the formula

$$\lambda = 1+\frac{1}{N}\left(\frac{1}{2}\left(\sqrt{p^2B_{11}^2 + 2p(1-p)(2B_{12}B_{21} - B_{11}B_{22}) + (1-p)^2B_{22}^2} + pB_{11} + (1-p)B_{22}\right) - 1\right),$$

that N acts to scale λ to 1 as N increases. This implies that $1-\lambda$ can get greater values when N is smaller. As this paper handles only cases where $N = 100$ or $N = 1000$, this explains why λ remains close to 1.

When considering the probability of an infection being small-scale, we use the formula $F_1(u_1, u_2) = u_1, F_2(u_1, u_2) = u_2$, where F_i is the probability generating function of I_i. This implies that the values u_1 and u_2 do *not* depend on N, as one can multiply both sides of both equations by N.

Possible extensions

As with any compartmental model, this model can be extended upon by adding more compartments. Typical categories to include are the exposed (but not yet infectious), deceased or vaccinated categories. One can also model a rate of transfer between recovered and susceptible individuals, where one loses the immunity to an infection. However, as the model is tailored to look into the initial stages of an infection, the time scale is likely not large enough to get qualitative differences.

The occurrence of N in the expression of λ is due to the scaling of the time step, to ensure the elements of the transition matrix W' can act as probabilities. The model can be extended by substituting a lower value for N in this scaling of the time step. This could be done by defining a cut-off point $\tilde{I}$, where if $I > \tilde{I}$, the infection is assumed to be large-scale. This would produce a bound on the values of the transition matrix, and thus give more desirable scaling value that brings about a higher variance in λ.

The model can be extended upon by increasing the amount of categories in which the population is divided. This can be used to model a wider array of situations, at the cost of complexity and interpretability, as the amount of parameters will increase.

All the parameters B_{ij} and the recovery rate are assumed to be constant through time. This is however, not accurate in many situations. The model can be extended by substituting these parameters with time dependent parameters. This would be able to represent change in policy and preventive measures based on the amount of infected individuals and time.

Instead of probability generations functions $F_i(u_1, u_2)$, one could investigate the time dependent moment generating functions. These can replace simulations, and be used to gain analytical insight in the stochasticity of the model.

Another way of obtain understanding of the stochasticity of the branching process, the variance of the chance of becoming small-scale can be calculated. This can be compared to the deterministic model, to find parameter ranges where the presented model differs significantly from a deterministic model.

5 Conclusion

A noticeable difference in the behaviour of a subpopulation brings about the relevance of representing this in an epidemiological model. The presented model can

be used to gain insight into these cases, more specifically the initial spread of an infection in such a population.

It is shown that the model can be used in specific situations to model this difference between two subpopulations, as in the case of a lockdown or a care facility. One substantial deduction is the fact that in a lockdown, the total population has to take measures to protect vulnerable individuals. Restricting interactions with or between vulnerable individuals will not guarantee them remaining shielded, and does not have a significant influence on the general spread of an infection.

With further research, extensions, and considerable review, the presented model can be used to help guide policy and public practice where applicable.

References

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Notes

1 Note that a dimensional analysis of this model does not deliver dimensionless probabilities. This stems from the fact that the value of the recovery rate is 1, and left out of the equation. This parameter has as dimensions the inverse of time and people, and would thus make the probabilities dimensionless.