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An analysis of stochastic epidemic modeling with a time-varying contact rate

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This thesis aims to analyze how interventions affect an epidemic. To do this, we will consider two stochastic general epidemic models, one with a constant rate of infection (i.e., no interventions) and one with a time-varying contact rate corresponding to the interventions implemented. More precisely, we studied numerical calculations and simulations for each model's reproduction number, final size and looked into the concept of flattening the curve. Then, we also studied the optimal timing for reducing the contact rate in the SIR epidemic model with interventions to minimize the outbreak and stretch the epidemic, which is known as the time of intervention.

Contents

1 Introduction

Infectious diseases are something we want to control and understand the behavior of. By constructing mathematical models that describe how a disease can be transmitted between individuals in a population, we gain insight into the dynamics of the disease. Such mathematical models have been used to describe a variety of infectious diseases. For example, Ross (1927) modeled the transmission of malaria [1] and the most recent coronavirus (2019). In general, there are two main types of epidemic models: stochastic and deterministic.

Early modeling contributions were often deterministic and addressed questions about the magnitude of an outbreak, the possibility of a large outbreak and the effect of vaccination prior to disease occurrence. In a stochastic epidemic model some additional questions can be observed, such as the probability of a large outbreak [1]. One advantage of a deterministic model is its simpler analysis since there is no element of randomness [2] while a stochastic model has the advantage of being a more realistic model since the element of randomness is taken into account. But hence, the stochastic model may be more difficult to analyze than the deterministic model.

In this thesis we will study the stochastic epidemic model, more specifically the stochastic *general* epidemic model, in a closed homogeneous mixing community, which is described in Section 2. For further information on the stochastic model as well as more reading on the deterministic model, see, for example, Andersson, H. and Britton, T. (2000) and Britton, T. (2009).

We will aim to analyze the impact of interventions on the epidemic. In order to achive this, we examine two distinct stochastic general epidemic models: one with a constant contact rate, i.e. without interventions, and one with a time-varying contact rate corresponding to the interventions implemented.

For a model where no interventions are performed during the time period, we will use numerical calculations and simulations to examine the basic reproduction number and the final size of the epidemic. For the model with interventions, we will use similar methods to those used for the prior model to examine the effective reproduction number and the final size of the epidemic. We will also look at the concept of flattening the curve, which means to slow down the outbreak dynamics and instead stretch out the outbreak over time [4], and we will do this for two different methods of introducing interventions which are described in Section 2. One method is directly influenced by the one of Michael Höhle in the article *"Flatten the COVID-19 curve"* (2020), to compare his method with the second one. We will also examine the optimal timing for reducing contact rates in epidemic models with interventions, known as the time of intervention.

In Section 4, we examine the results obtained from the numerical calculations and our simulations. We will also analyze the differences in the results between the models with and without interventions.

A brief overview of this thesis is as follows: In Section 2 we will go through some important model theory that is needed in this thesis. In Section 3 we will

analyze the results and present them. In Section 4 we discuss the results and clarify/justify some important notes or ambiguities. And lastly, in Section 5 we present the conclusions that can be drawn.

2 Model theory

2.1 Stochastic general epidemic model

The stochastic model consists of three sets: Susceptibles (S), Infectives (I), and Recovered (R), and it is assumed that at any time an individual is in one of these three sets [1]. The set of susceptibles is the set of individuals who have not yet been infected and are therefore susceptible to the disease, while the set of infectives is the set of individuals who are infectious and can therefore spread the disease further. An infected person will eventually recover from the disease and move to the last set of recovered individuals. These recovered individuals cannot be re-infected and are therefore immune. Thus, there are only two possible paths that an individual can take: from S to I and from I to R. For this reason, the model is said to be a SIR epidemic model [1].

We also assume that the epidemic spreads in a closed homogeneous uniformly mixing community. A closed community implies that there are no deaths, births, immigration, or emigration during the period. By a homogeneous uniformly mixing community, we mean that we do not take into consideration social groups or structures, and therefore all contacts of individuals are randomly distributed in the community [1].

Let $S(t)$, $I(t)$ and $R(t)$ represent the number of susceptible, infectious and recovered respectively at time t. Initially we assume that the number of these is given by $S(0) = n$, $I(0) = m$ and $R(0) = 0$, where $n + m$ corresponds to the size of the population. The value of m must be at least one for a disease to be able to spread in a society. If $m = 0$, there is no initial infective and thus no one can become infected. In this thesis we assume $m = 1$.

The sequence of events in this model can be described as follows. An infectious person has contact with other individuals randomly in time at a rate βn and each such contact is with an individual chosen uniformly at random from the population, all contacts of infectives are assumed to be mutually independent [1]. Hence, the contact rate with a specific individual is our β. If the contacted individual is susceptible, he/she will be immediately infected and instantly able to infect other individuals [2]. Otherwise, this contact has no effect. An infected person will then remain in that state for a random period $T_I \sim Exp(\gamma)$, with mean $\tau = 1/\gamma$. After T_I, the infective eventually recovers and is immediately immune. The infectious periods are assumed to be independent and identically distributed and also independent from the contact processes [1]. The epidemic starts at time $t = 0$ and ends when there are no infectious people.

This is called the stochastic general epidemic model, which is our main focus in this thesis. Since the infectious period T_I is exponentially distributed, which shows a lack-of-memory property, the stochastic general epidemic model can be

described as a continuous-time Markov process with two possible jumps. The rates for the two different transitions in such a process are described in Table 1 [1].

Event	Transition	Rate
Infection	$S \to I$	$\beta \cdot S(t) \cdot I(t)$
Recovery	$I \to R$	$\gamma \cdot I(t)$

Table 1: Transition rates for the two possible events, infection, and recovery, in a stochastic general epidemic model, described as a continuous-time Markov process

The transition rates can be derived immediately from the definition of the model since for the transition $S \to I$ to occur, susceptible individuals must have contact with infectives, and the transition $I \to R$ is the removal rate γ multiplied by the number of infected at time t.

2.2 Basic reproduction number

In this thesis, we will present and use two variations of reproduction numbers, the basic reproduction number, and the time-varying effective reproduction number. Both of these are measurements of how big an eruption can be, found by calculating the average number of secondary infections by a typical infective before recovering. The former is time constant and used when an infectious individual is surrounded by an entirely susceptible population. The latter is defined when interventions occur in some form and are the average number of secondary cases generated at time t. The effective reproduction number is presented in Section 2.6.

The basic reproduction number in a general stochastic epidemic model is given by [3]

$$R_0 = \frac{\beta}{\gamma} S(0).$$

The above expression of R_0 can be explained as follows. An infectious individual has an average infection period of $\tau = 1/\gamma$, and during this period, the infective infects susceptibles at a rate $\beta \cdot S(0)$.

The critical value that separates a major outbreak from a minor outbreak is $R_0 = 1$ [1]. For this reason, R_0 is also considered as the epidemic threshold. And since R_0 is defined as the average number of secondary infections, it is evident that if an infective, on average, spreads the disease to less than one person, then the infection will die out before a major outbreak can occur. Thus, it can be shown that there is a minor outbreak with probability 1, if and only if $R_0 < 1$. Otherwise, there is a positive probability of a major outbreak. A detailed proof of this can be found later in Section 2.3. Note that even if $R_0 > 1$, it is still possible that the epidemic never takes off since we assume some uncertainty [1].

2.3 Epidemic threshold

As mentioned in section 2.2, the reproduction number is also considered the epidemic threshold. In this section, we will look more into this, and to do so, we need to look closer into an early epidemic approximation. The early stage of an outbreak can be approximated by a continuous-time branching process as follows.

We consider a population where the life span of different individuals are independent and identically distributed according to a random variable T_I and where they independently give birth at time points according to a Poisson distribution with intensity βn. This applies to all generations [2]. The number of ancestors in the population is denoted by X_0, and their offsprings form the first generation X_1. Hence X_n denotes the size of the nth generation [5].

We will assume that each individual will produce j offspring with probability p_j during his/her life span, i.e., in the epidemic, each infected person infects j new individuals with probability p_j during his/hers infectious period [5]. Let D denote the number of immediate offspring from a single individual and therefore let [5]

$$E(D) = \sum_{j=0}^{\infty} j p_j.$$

Suppose now that we only have one initially infected person, $X_0 = 1$, we note that

$$X_n = \sum_{i=1}^{X_{n-1}} D_i,$$

letting D_i represent the number of offsprings of the ith individual in the $(n-1)$st generation, together with the fact that $E(D_i) = D$, we obtain [5]

$$E[X_n] = E[E[X_n|X_{n-1}]] = E[X_{n-1}E(D)] = E(D)E[X_{n-1}].$$

And since we only have one initially infected person, $X_0 = 1$, we get the following equations [5]

$$\begin{aligned} &E[X_1] = E[D], \\ &E[X_2] = E[D]E[X_1] = E[D]^2, \\ &... \\ &E[X_n] = E[D]E[X_{n-1}] = E[D]^n. \end{aligned}$$

We now let π_0 denote the extinction probability of the epidemic branching process, still assuming $X_0 = 1$. Since we have on average $X_0 E(D)^n = E(D)^n$ individuals in the nth generation, it is evident that $\pi_0 = 1$ if and only if $E(D) \leq$

1 [2]. If we instead turn to the case where $E[D] > 1$, we obtain that $\pi_0 < 1$. We can calculate π_0 via [5]

$$\pi_0 = \sum_{j=0}^{\infty} P\{\text{population dies out}|X_1 = j\}p_j.$$

However, since the population dies out if and only if all of the independent branches go extinct, π_0 satisfies $\pi_0 = \sum_{j=0}^{\infty} \pi_0^j p_j$. It can also be shown that π_0 is the smallest solution to the equation [5].

We now examine the branching approximation process for our epidemic. We consider a sequence of standard SIR epidemic processes, and we now want to show that it agrees with the epidemic process. The proof below is inspired by the article "Stochastic epidemic models and their statistical analysis" by Håkan Andersson and Tom Britton.

We first construct a branching process, as we did before, and suppose that the probability space holds the individual life histories $\eta_{X_0-1}, \eta_{X_0-2}, ..,$ where η_i is a list containing the ith individual's lifespan and time points at which this individual give birth [2]. Let $i \leq 0$ and $i \geq 1$ be the life histories of X_0 and the ith individual born, respectively.

Now we will use an independent sequence of i.i.d. distributed random variables defined on the probability space, each uniformly distributed on $(0, 1)$, to be able to create all of the epidemics processes with $S(0) \geq 1$. We fix n and label the initial susceptibles $1, 2, .., S(0)$ [2]. Now, X_0 in the branching process can be interpreted as the initial infectives in an epidemic, and births corresponds to contacts. If the contacted individual is susceptible, he/she becomes immediately infected. If not, he/she and all of his/her successors in the branching process are ignored in the epidemic process, and the individual is called a *ghost*. The death of an individual that´s not a ghost equals removal in the epidemic process, which leads to a process that is the same as standard SIR epidemic processes [2].

However, it is important to note that the two processes, the branching process, and the epidemic process, only agree until the time of the first ghost. Lastly, $E(D) = \beta n \tau$ and hence, it corresponds to the basic reproduction number R_0 in a stochastic SIR model. This result shows that R_0 can determine if a large outbreak occurs or not [2].

2.4 Flattening the epidemic curve

The concept of flattening the epidemic curve corresponds to slowing down the outbreak dynamics. There are various reasons why this approach is essential. But one main reason is that stretching out the outbreak over a longer period will ensure that a larger amount of individuals who need medical attention receive this [4]. More precisely, if the curve is flattened, fewer people will be infected at the same time t, and an obvious peak in the epidemic curve could be avoided. This is especially helpful in cases of limited health capacity. Other advantages

may be to save time to carry out more research and hence find better treatments and/or vaccines [4].

In order to flatten the epidemic curve, we want to reduce the reproduction numbers, i.e., the average number of secondary infections by a typical infective before recovering, and we can do this in two possible ways. The first one is to reduce the number of contacts individuals have with each other which, as mentioned before, corresponds to reducing β. And the second one is to reduce the effective infectious period. The latter can be done by regular tests for the virus and quarantine, as this will help minimize the period that an infectious person is among the other individuals in the community.

2.5 Epidemic model with interventions

For the SIR epidemic model with interventions, we instead have a time-varying contact rate. This means that during the period, we are putting in preventive measures, for example, social distancing, which will decrease the number of contacts one person has and hence make our $\beta(t)$ smaller. We will do this for two different methods of introducing interventions in order to compare these in the discussion in section 4.

The first method is directly influenced by the one given by Michael Höhle in the article *"Flatten the COVID-19 curve"* (2018) and can be described as follows. We will have three different $\beta(t)$ values that only depend on time [4]:

$$\beta(t) = \begin{cases} \beta_0, & \text{if } t \leq t_1, \\ \beta_1, & \text{if } t_1 < t \leq t_2, \\ \beta_2, & \text{if } t_2 < t, \end{cases}$$

where β_0 is the initial β value of the disease, i.e. $1 \cdot 10^{-4}$. During the time interval $[t_1, t_2]$, a large reduction of the contacts will take place, and after some time, the preventive measures are slightly relaxed. We thus have $\beta_1 < \beta_2 < \beta_0$. We will use $\beta_1 = c_1\beta_0$ and $\beta_2 = c_2\beta_0$ where $c_1 \leq c_2$ [4].

For the second method, we no longer will have a piecewise alteration. However, the values still only depend on time and can be described as follows: As previously, we assume that we have a value β_0 if $t \leq t_1$, where $\beta_0 = 1 \cdot 10^{-4}$. During the time interval $[t_1, t_2]$, minor control measurements are constantly made to reduce the number of contacts. Thus contacts in this interval decrease linearly and, hence, the value of $\beta(t)$. At the time $t = t_2$, all constraints are put in place, and after some time, the preventive measures are slightly relaxed again. And this will happen constantly over some time. This means that during the period $[t_3, t_4]$, the value of $\beta(t)$ increases linearly. And this is illustrated in Figure 1 below

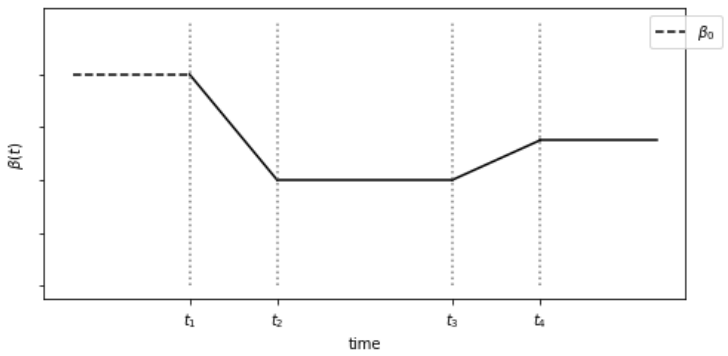

Figure 1: An illustration of how we will introduce the interventions in the second approach. Within the time intervals $[t_1, t_2]$ and $[t_3, t_4]$ interventions are introduced and removed respectively.

This can also be mathematically defined as follows:

$$\beta(t) = \begin{cases} \beta_0, & \text{if } t \leq t_1, \\ \beta_1, & \text{if } t_2 < t \leq t_3, \\ \beta_2, & \text{if } t_4 < t, \\ \mathrm{r}_1\beta(t-1), & \text{if } t_1 < t \leq t_2, \\ \mathrm{r}_2\beta(t-1), & \text{if } t_3 < t \leq t_4, \end{cases}$$

where β_1 and β_2 are defined in the same way as in the first method. The variables r_1 and r_2 correspond some small constant that depends on the percentage by which we want to decrease and increase $\beta(t)$, respectively. For example, if $\beta(t)$ decreases by one percent for each step in the interval $[t_1, t_2]$, $r_1 = 0.99$. We thus have $r_1 < r_2$.

It is important to note that the timing of the interventions matters and that they must be done properly in order to stretch the outbreak and reduce the final size of the epidemic [4], so the presented times t for the different methods may vary. For example, the time $t = t_1$ at which we initiate the first preventive measures may be different for the different methods. This is further analyzed in Sections 3 and 4.

In what follows, we will for clarity refer to these as piecewise intervention and linear intervention respectively, due to the fact that we in the second method use a linearly decreasing and increasing $\beta(t)$ (even though it does not change linearly all the time).

2.6 Effective reproduction number

When an epidemic eventually develops it is no longer appropriate to use the basic reproduction number. This is since the number of susceptible individuals is decreasing over time and interventions can take place in the community. As mentioned before, such interventions may be restrictions or guidelines which can

contribute to changed values of β and γ, respectively, over time. For instance, introducing restrictions that reduce the number of contacts individuals have with each other corresponds to reducing β, while imposing isolation on infected individuals corresponds to reducing γ. The former example is what we are doing in the epidemic model with interventions presented in Section 2.5. It is in these cases we use the time-dependent effective reproduction number which is given by [6]

$$R_e(t) = \frac{\beta(t)}{\gamma(t)} S(t).$$

Here we instead calculate the number of secondary infections by an infected person before recovering at time t and can thus vary over the period. The critical value is still 1. For a value $R_e(t) < 1$, the number of new infections will decrease. Otherwise, the number of new infections will increase [6]. The motivation used for the basic reproduction number formula can also be used to motivate the formula of the effective reproduction number.

3 Results

Throughout the simulations, we will, for simplicity, only consider models with one initially infected person and a population with $5,000$ susceptible individuals. We also only assume an infectious period of five days on average and hence $\gamma = 1/5$. The only variable that will vary during the thesis and the simulations is β.

For the SIR epidemic model without interventions we have a constant value of $\beta = 1 \cdot 10^{-4}$. This indicates, together with the values given above, that we have a basic reproduction number R_0 as high as 2.5 which implies that a major epidemic may occur. All simulations and code in this paper were calculated using the computer program R.

3.1 SIR without interventions

We will first look at the results of the general stochastic SIR model without interventions. We start by simulating and presenting the plot of the number of infected individuals during the course, i.e., $I(t)$.

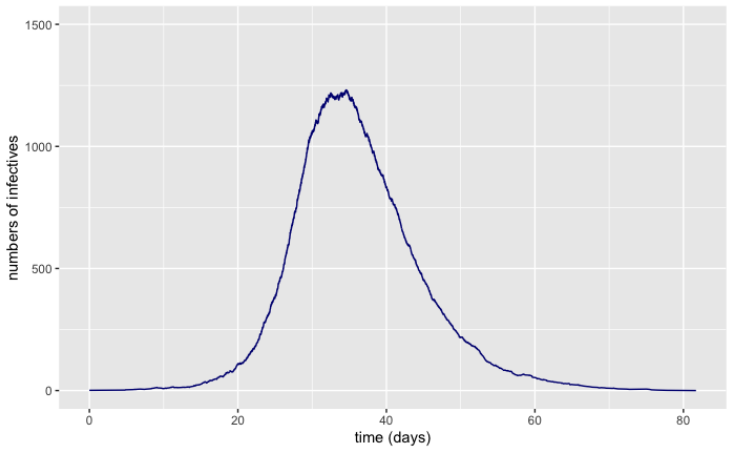

Figure 2: One simulation of the general stochastic model in a population with $5,000$ susceptible individual, one initially infected and $R_0 = 2.5$.

In Figure 2, we can see that the outbreak extends over 80 days and has, at most, roughly $1,250$ individuals infected at the same time, and this occurs approximately at time $t = 35$. We can write this as $I(35) = 1,250$.

We now find the final size Z, defined as the total number of infected individuals by the end of the epidemic. We find Z by subtracting the number of susceptible individuals at the end of the epidemic from the total population. We know that $S(0) = 5,000$, $I(0) = 1$, and we get the number of susceptible at the end of the epidemic from the simulation we just ran. We thus get $5,001 - 575 = 4,426$ individuals. To see the final size for several simulations of this specific model, we now simulate $10,000$ epidemics and present the final size for these in a so-called final size distribution plot.

Now we can from Figure 3 see that we will either get a minor or a major outbreak. This is due to the fact that the histogram unmistakably demonstrates that, in the $10,000$ simulations, the final magnitude of the pandemic in 40% of the cases is close to 0. In the other cases, the final size is between $4,000$ and $5,000$, which indicates a major outbreak. This is not surprising given our high R_0 value and, if an outbreak takes off, it is likely that it will not end until the majority of the population has been affected.

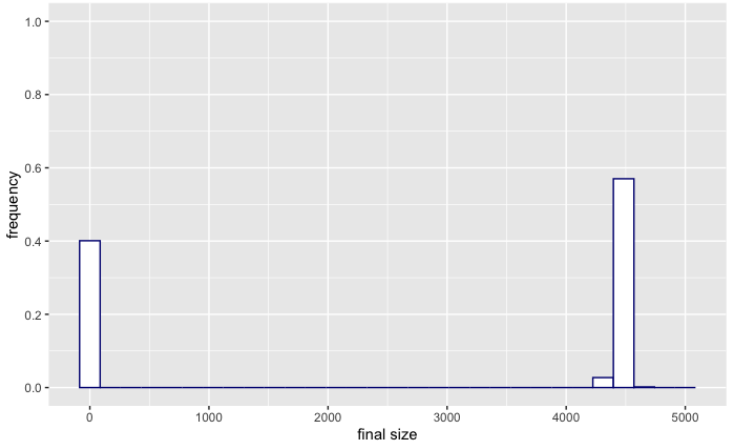

Figure 3: Empirical distribution of final size from $10,000$ simulations of the general stochastic model, without interventions, with $5,000$ susceptible individual, one initially infected and $R_0 = 2.5$.

3.2 SIR with interventions

We will now look at the results of the general stochastic SIR models with interventions. For clarity, we divide the two methods into two sections and, as mentioned in Section 2.5, call these *Piecewise interventions* and *Linear interventions*. Note that we for these two methods are interested in the effective reproduction number $R_e(t)$ and not the basic reproduction number R_0.

3.2.1 Piecewise interventions

For the general stochastic SIR model with piecewise interventions, we start by carrying out several simulations for different values of c_1, c_2, and different times of interventions to find the best-fit values.

We find that the most effective time points to introduce and relax the restrictions are at $t = 10$ and $t = 60$, respectively, for values $c_1 = 0.6$ and $c_2 = 0.8$. This means that we introduce restrictions that give a reduction of the contacts by 40%, keep the restrictions in place for 50 days and then ease them slightly so that we only have a reduction of 20%. Hence, $\beta_1 = 0.6\beta_0$ and $\beta_2 = 0.8\beta_0$.

If we instead loosen the restrictions at an earlier time or loosen them too much, we see that the curve does not significantly decrease and therefore does not avoid an obvious peak in the outbreak dynamic. It is instead only delayed. This result was also discovered by Michael Höhle and addressed in his article, which is not entirely surprising since this method is directly influenced by his.

We will now simulate and present the plot of the number of infected individuals during the course, i.e., $I(t)$. In this plot, we will also draw the two vertical lines representing the time of interventions.

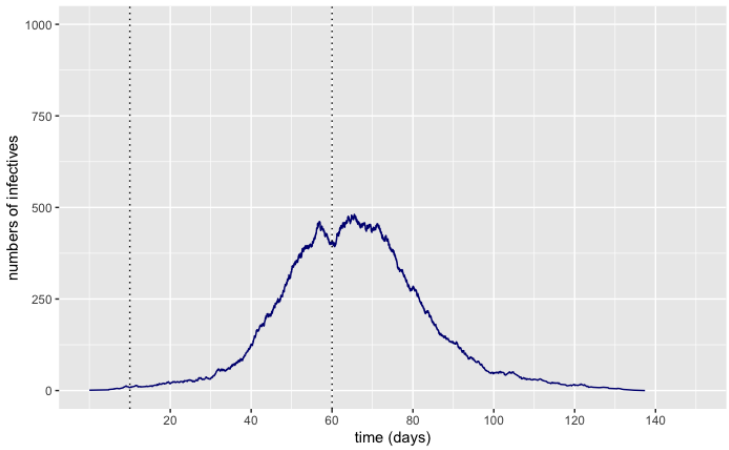

Figure 4: One simulation of the general stochastic SIR model in a population with $5,000$ initial susceptible and one initially infected with a time-varying $\beta(t)$ for interventions introduced piecewise.

In Figure 4 we can see that the outbreak extends close to 140 days and at most 500 individuals infected at the same time. Which is a clear improvement over what we observed previously in the model without interventions.

We will now look at the effective reproduction numbers. As mentioned previously, we have three different $\beta(t)$ values. And since the initial value β_0 is the same as we use in the calculation of the basic reproduction number R_0 and the fact that all non-infected individuals are susceptible in the community at time $t = 0$, it applies that $R_e(0) = R_0 = 2.5$. For the other $R_e(t)$ values, we must use the formula of the effective reproduction number given in Section 2.6. Therefore, we need to use the values of $\beta(t)$, as we presented at the beginning of this section, and $S(t)$ during the period. The latter we can obtain from our simulations. Note that $\gamma(t) = \gamma$ since we assume that γ does not change over time. We compute all the effective reproduction numbers and present these in a graph.

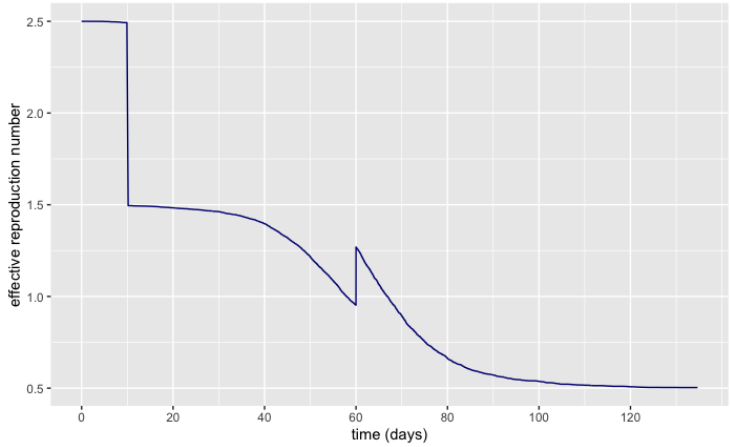

Figure 5: The values of the effective reproduction number as a function of time t for a model with piecewise interventions and initial value $R_e(0) = R_0 = 2.5$.

In Figure 5, we can now see the graph over the effective reproduction numbers as a function of time. If we look at Figure 4 and 5, we can see that the number of new infections decreases for $R_e(t) < 1$ and increases for $R_e(t) > 1$. A clear example of when we can see this is when we relax the restrictions at $t = 60$. For this time, we can see in Figure 4 that $R_e(t)$ goes from below 1 to be above 1. And at the same time, in Figure 2, we can see that also the number of new infections slightly increases immediately after $t = 60$ even though it decreased just before.

Lastly, we will find the final size Z using the previous approach. We thus get $Z = 5,001 - 1258 = 3,743$ and can immediately state that a smaller number of individuals in society were infected, i.e., carried out the jump $S \to I$, in this model than in the model without intervention. This also confirms our result that we, for this model, can see a clear improvement in comparison to the first model. We now look at the final size distribution plot for $10,000$ simulations of this model.

We can now see from Figure 6 that we will get either a minor or a major outbreak since in 40% of the simulations, we have a final size close to 0, and in all the other simulations, we have a final size between $3,000$ and $4,000$ individuals. The size of the large outbreaks also appears to be almost normally distributed between $3,000$ and $4,000$.

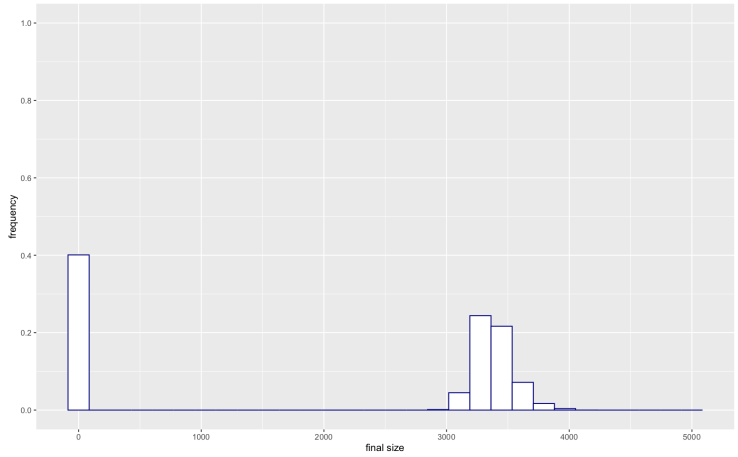

Figure 6: Empirical distribution of final size from $10,000$ simulations of the general stochastic model for $5,000$ susceptible individual and one initially infected, with piecewise interventions.

3.2.2 Linear interventions

For the general stochastic SIR model with linear interventions, we will also start with carrying out several simulations for different values of c_1, c_2, r_1, r_2 and different time and length of interventions, i.e., $[t_1, t_2]$ $[t_3, t_4]$. Note that a change in r_1 and r_2 corresponds to a change in t_2 and t_4, respectively, the reverse also applies since r_1 and r_2 are the slope of the straight lines $\beta(t) = r_1\beta(t-1)$ and $\beta(t) = r_2\beta(t-1)$ and a greater incline/decline means that we reach the end point faster.

We find that the most effective time intervals to impose and relax the restrictions on are $[10, 30]$ and $[60, 70]$, for values $c_1 = 0.6$, $c_2 = 0.8$ and $r_1 = r_2 = 0.2$. This means that we introduce restrictions linearly for 20 days with a reduction of the contacts by 2% for each day until we have a total reduction of 40%. We then keep the restrictions in place for 30 days. After this, we ease the restrictions linearly for 10 days with a reduction of 2% each day until we only have a reduction of 20%. Hence, $\beta_1 = 0.6\beta_0$, $\beta_2 = 0.8\beta_0$ and $\beta(t) = 0.98\beta(t-1)$.

If instead we introduce the interventions at a slower rate r_1 and over a longer period, we will reach a slightly higher peak of people infected at one point in time, meaning a higher $I(t)$ at some point t, in the outbreak. Or, if we instead loosen the restrictions at an earlier time or too much, we will end up with a bigger final size Z of the epidemic. Although it is not a huge change.

Now, we simulate and present the plot of infectives during the period, i.e., $I(t)$. We also draw four vertical lines in the plot to represent the time of intervention interval

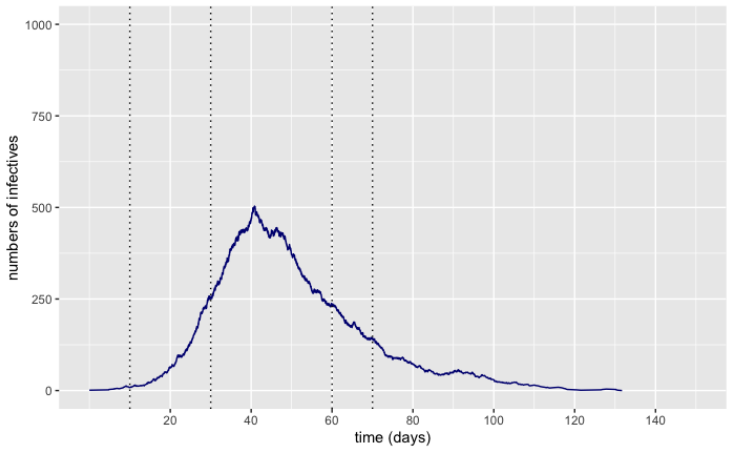

Figure 7: One simulation of the general stochastic SIR model in a population with $5,000$ initial susceptible and one initially infected with a time-varying $\beta(t)$ for interventions introduced linearly.

In Figure 7, we can see that the outbreak lasts for just over 130 days, with at most 500 infected individuals at the same time. This is a clear improvement compared to the first model without interventions but similar to the method of piecewise interventions.

We now look at the effective reproduction numbers. During the period in which we have linearly decreasing and increasing interventions, we obtain a new $\beta(t)$ for each time step t and therefore, we have several different $\beta(t)$ values. In addition, we also have three values that are held constant over a period of time. For the same reason as the method with piecewise interventions, mentioned in Section 3.2.1, the initial effective reproduction number is $R_e(0) = R_0 = 2.5$. For every other $R_e(t)$ value, we must use the formula given in Section 2.6. We compute these and present them in a graph.

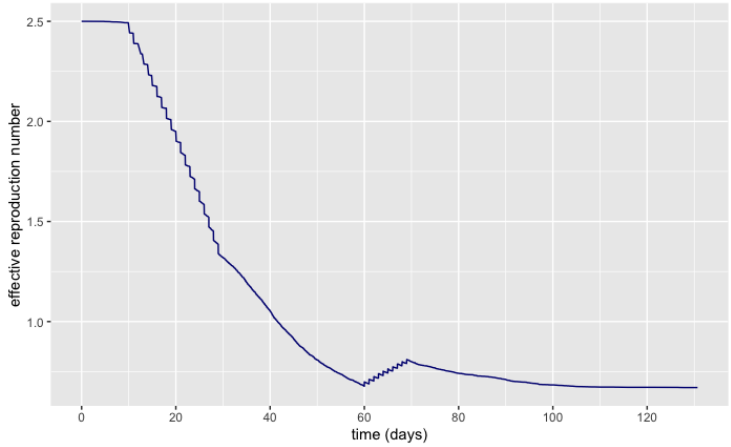

Figure 8: The values of the effective reproduction number as a function of time t for a model with linear interventions and initial value $R_e(0) = R_0 = 2.5$.

We can now see all the effective reproduction numbers over the outbreak in Figure 8. If wee look at Figure 7, we see that the number of new infections increases until $t = 40$ and then immediately decreases and do so until the end of the epidemic. This is consistent with the fact that we in Figure 8, can see that up until the same time $t = 40$, $R_e(t) > 1$, and after this we reach the critical limit $R_e(t) = 1$ and stay below this limit for for the rest of the epidemic. This agrees with what the epidemic threshold tells us.

Lastly, we look at the final size of the epidemic, which is given by $5,001-1676 = 3,325$ and is the lowest outbreak we have got so far in our simulations. We also look at the final size distribution plot for $10,000$ simulations and can see in Figure 9 that in just over 40% of our simulations we have a minor outbreak close to 0. In all the other simulations, we have a major outbreak where the final size appears to be normally distributed between $3,000$ and $4,000$ individuals.

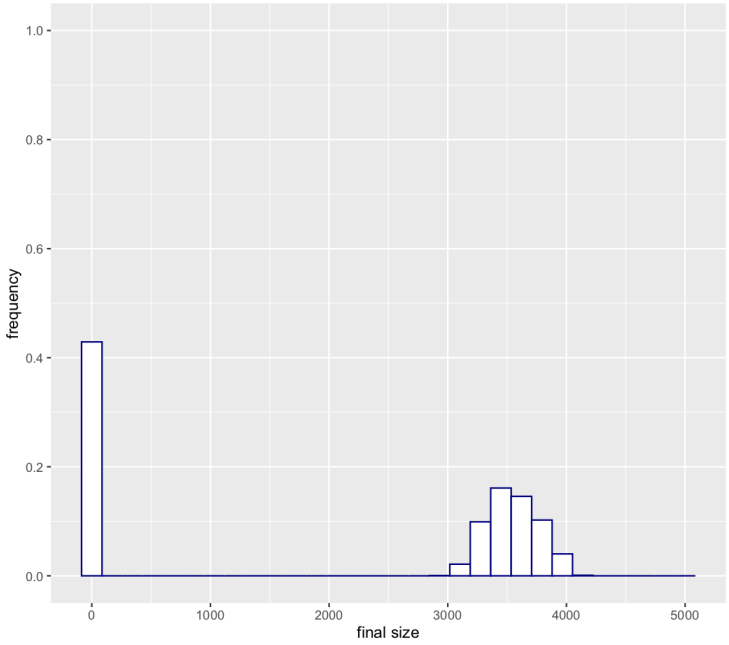

Figure 9: Empirical distribution of final size from $10,000$ simulations of the general stochastic model with $5,000$ susceptible individual and one initially infected, for linear interventions.

3.3 Epidemic curve

Now that we have presented the results for each model individually, let us look at the results in terms of the concept of flattening the epidemic curve. By interpreting the given results, we can see that both methods in the model with intervention slow down the outbreak dynamics and stretch out the outbreak over time.

We begin by presenting a graph of the epidemic curves for the model without interventions and the one with piecewise interventions. In this plot seen in Figure 10, we can see that when we introduce the preventive measures at $t = 10$ in the piecewise approach, there is a noticeable difference between the two models. While the model without interventions increases continuously rapidly, the one with (piecewise) interventions continues relatively steadily forward with only a slight incline. Consequently, the model without interventions has a much higher transmission in society, and thus, more jumps $S \to I$ are performed in the branching process. A consequence of this is that not as many susceptibles individuals are infected at the same time t during the course of the outbreak in the model with (piecewise) interventions. Hence we avoid a clear peak and thus increase the probability that all people who needs healthcare receive it. The curve is thus flattened.

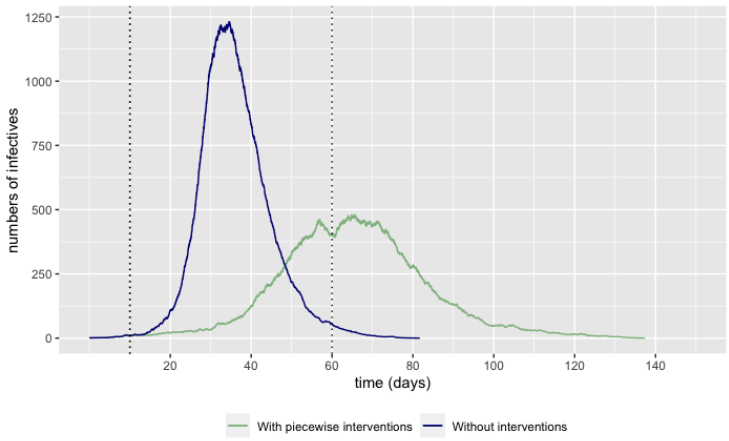

Figure 10: Two epidemic curves, one general stochastic SIR mode without interventions (blue) and one with piecewise interventions (green).

Figure 11 illustrates the similar pattern between the model with linear interventions and the model without interventions. For instance, the epidemic curve for the model with (linear) interentions also does not increase as much or at the same rate as the model without interventions at time $t = 10$.

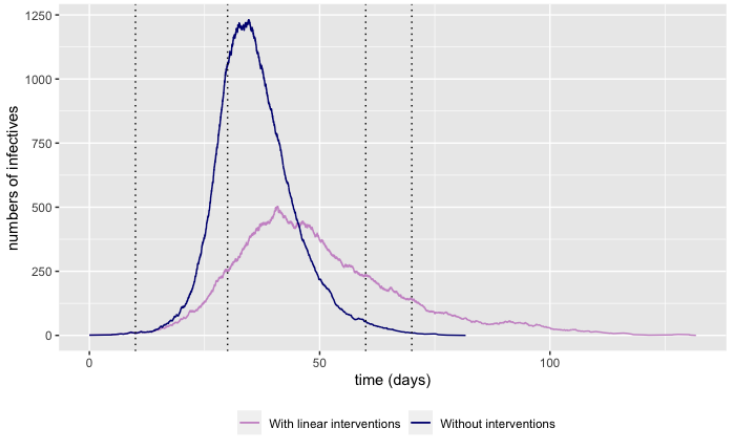

Figure 11: Two epidemic curves, one general stochastic SIR mode without interventions (blue) and one with linear interventions (purple).

We now present all three epidemic curves in the same plot. One of the first things we see in this plot, present in Figure 12, is that the piecewise and linear intervention models share many similarities. One of them being that both have at most $I(t) = 500$ at some time t and also that they stretched out the outbreak to about the same length. But if we look more closely at Figure 12, we see one potential difference. This is because the interval in which the epidemic curve assumes its largest values appears to be shorter in the case of linear interventions compared to the case with piecewise interventions. However, it is difficult to see with the naked eye if this is true, and it is impossible to draw any conclusions

as to whether one of the two methods is more effective than the other, but we can state that they both flatten the curve.

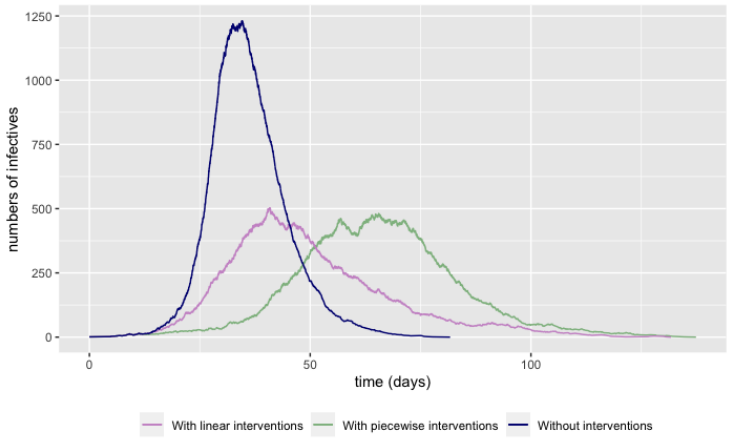

Figure 12: Three epidemic curves, one general stochastic SIR model without interventions (blue), one with piecewise interventions (green) and one with linear interventions (purple).

We now take the final size Z and the final size distribution into account for all models. We call the final size of each model Z_1, Z_2, and Z_3 where Z_1 is the model without interventions, Z_2 is the model with piecewise interventions, and Z_3 is the model with linear interventions. As mentioned in Section 3.2, we obtained values of $Z_1 = 4,426$, $Z_2 = 3,743$, and $Z_3 = 3,325$. From this, we can see that $Z_2 > Z_3$, but from Figure 6 and 9, we also see that out of $10,000$ simulations, we have more major outbreaks in the model with linear interventions, the difference in both of these results is also very little. Thus, we cannot draw any conclusion about the most effective method by looking at these results either. But we can conclude that both methods minimize the outbreak and stretch the epidemic.

4 Discussion

It is important to note that this is a strong simplification of reality. A more realistic model is one where the population is not assumed to be homogeneous mixing since one can assume with certainty that social groups or structures do exist. With this said, the values we assume are the best adapted in Section 3 are not always the ones that give the best results in our simulations. For example, if one in both methods instead introduces interventions directly at the start of the epidemic at time $t = 0$, this generate a better result since we lower the value of $\beta(t)$ right from the beginning. By better results, we mean a smaller final size Z, fewer people infected at the same time, etc. In such a simulation, the epidemic rarely takes off. But it is not likely that we know in advance that an epidemic will come, and therefore, we always choose values that are more similar to reality. Hence, we always choose the values that are closest to reality

when we examine which values are best adapted.

Another important note is that, regarding the best-fitted values, we get similar results for the different methods of introducing interventions. This may be because we assume the same epidemic, i.e., the same initial values $S(0)$, $I(0)$, $\beta(0)$ and γ, and that the two methods are relatively similar. Despite having many elements in common, one significant difference is that the method with piecewise interventions has more effects when we alter the period at which we remove the constraints or if we loosen them even further toward the end. Therefore, it appears that the gradual introduction and easing of restrictions is a more stable approach that can result in a community that can prevent significant spreads of infection in society once more even if we do not maintain the same level of protective measures or do not maintain them for the same period of time.

Another benefit of the method with linear interventions is that its effective reproduction number, $R_e(t)$, both reaches the critical limit 1 faster and does not appear to be significantly affected when we eventually relax the interventions as compared to the method with piecewise interventions. This is seen in Figure 8, for the linear interventions, where it applies that $R_e(40) = 1$, and it then stays below this value even when the interventions are relaxed at $t = 60$. At the same time, we can see in Figure 5, for the piecewise interventions, that $R_e(40) > 1$ and it isn´t below 1 until approximately $t = 50$ and even after this, it immediately increases again when the interventions are relaxed at $t = 60$. However, in some cases, we can see an advantage with the piecewise interventions. For example, from the $10,000$ simulations we generated for both methods the piecewise had a lower number of major outbreaks than the linear interventions which is seen in Figure 6 and 9. Because of this, it is impossible to determine which approach is more effective because both have advantages and disadvantages. It would be advantageous to examine these for a variety of epidemic models in the future if one wants to depict more differences and find the most effective one.

5 Conclusion

In this thesis, we have aimed to analyze how interventions affect an epidemic. We did this by considering two stochastic general epidemic models, one with a constant rate of infection (i.e., no interventions) and one with time-varying rate of infection. We also computed two different methods on how to implement these infections and called these *Piecewise interventions* and *Linear interventions*.

One main thing we can conclude from this essay is that both methods we used in the model with interventions produced similar results. Both flattened the curve, stretched the outbreak, and got a smaller final size Z of the outbreak. Both also had approximately at most $I(t) = 500$ at some time t. From this, it was not conceivable to draw any conclusions about the most effective method, but we can establish that both methods improved the outbreak dynamics.

If we instead looked at the two methods separately, we could draw the following conclusions. For the method with piecewise intervention, if we loosen the

restriction too early or loosen them too much we would only delay the peak of the outbreak and not significantly decrease it. And for the method with linear interventions, if we introduced the interventions at a slower rate r_1 and over a longer period of time, we would reach a higher value of $I(t)$ at some point t. And if we loosened the restrictions too early or too much, we would get a larger final size Z than what we got with our values.

References

[1] Britton, T. (2009). *Stochastic epidemic models: a survey.* Mathematical Biosciences.

[2] Andersson, H. and Britton, T. (2000). *Stochastic epidemic models and their statistical analysis.* Springer, New York.

[3] Britton, T. (2018). *Basic stochastic transmission models and their inference.* Mathematical Statistics, Stockholm University.

[4] Höhle, M. (2020). *Flatten the COVID-19 curve.* Matematiska Institutionen, Stockholm University.

[5] Ross, S. (2014). *Introduction to Probability Models.* Eleventh edition. Elsevier.

[6] Britton T., Trapman, P. and Ball, F. (2021) *The risk for a new COVID-19 wave and how it depends on R0, the current immunity level and current restrictions.* Royal Society Open Science.