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# COVID-19: Who should get vaccinated first?

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## Abstract

In this thesis we investigate what is the most effective vaccination strategy against an epidemic resembling the COVID-19 outbreak in the Stockholm region in Sweden, using a multi-type stochastic epidemic model with a fraction of initially vaccinated of each type, given a varied number of either *perfect* or 90% efficacious *all-or-nothing* vaccines. We extend the SEIR (Susceptible, Exposed, Infectious, Removed) model to an SEIRLD model with Recovered, Long-term ill or Dead as final states, and we vaccinate the population uniformly (where the same fraction of each type is vaccinated), in order of descending probability of early infection, and in descending age order with males first to protect risk groups. The final sizes of the outbreak are computed using a balance equation and the relative probabilities of long-term illness and death, computed from COVID-19 data from the Stockholm region.

The findings suggest that vaccinating the most vulnerable first protects risk groups the most, but reduces the spread the least for both vaccines and all fractions of available vaccines considered. Conversely, uniform vaccination protects the vulnerable groups the least, but reduces the spread more than the former approach. Vaccinating those with a higher risk of early infection reduces the spread quite similarly to the uniform vaccination for some fractions of vaccinated among the population, while also reducing death and long-term illness relatively sufficiently. Therefore, this strategy seems the most efficient overall.

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## Sammanfattning

I den här uppsatsen undersöker vi vad som är den mest effektiva vaccinationsstrategin mot en epidemi liknande utbrottet av COVID-19 i region Stockholm i Sverige, genom att använda en stokastisk epidemi-modell med flera typer och en andel initialt vaccinerade av varje typ, givet ett varierat antal antingen *perfekta* vaccin eller 90% effektiva *allt-eller-inget*-vaccin. Vi utökar SEIR-modellen (mottaglig, smittad, smittsam, borttagen) till en SEIRLD-modell med återhämtad, långtidssjuk eller avliden som slutgiltiga tillstånd och vi vaccinerar befolkningen likformigt (där samma andel av varje typ vaccineras), i fallande ordning av tidig smittorisk och i fallande åldersordning med män först för att skydda riskgrupper. Slutstorlekarna av utbrottet beräknas med hjälp av en balansekvation och de relativa sannolikheterna för långtidssjukdom och död, beräknat från data om COVID-19 i region Stockholm.

Resultaten tyder på att vaccinering av de mest sårbara först skyddar riskgrupper mest, men reducerar spridningen minst för båda vaccinen och alla andelar tillgängliga vaccin som beaktas. Omvänt skyddar likformig vaccination de sårbara grupperna minst, men reducerar spridningen mer än det tidigare tillvägagångssättet. Att vaccinera de med en högre risk att bli smittade tidigt minskar smittspridningen ganska liknande den likformiga vaccinationen för några andelar av vaccinerade i populationen, medan även dödsfallen och fallen av långtidssjuka minskas relativt tillräckligt. Därför verkar denna strategi vara den mest effektiva över lag.

### **Acknowledgments**

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# 1 Introduction

The current COVID-19 pandemic has caused a demand for the manufacturing and rapid distribution of vaccines to manage the worldwide spread and reduce the number of fatalities and severe infections. As of now, multiple vaccines have been assessed safe and efficacious enough for utilization and several countries are in the process of vaccination. However, determining who to vaccinate first is complicated as various vaccination strategies can affect the reduction of the spread, severity of cases and mortality rates differently depending on the type and allocation of vaccines. A range of contributing factors need to be regarded including for instance individual heterogeneities and structures in the population, and the optimal vaccination strategy in terms of decreasing the expected number of people a typical infectious individual in turn infects differs for these factors, as mentioned in Andersson & Britton [1, p.119-120]. If heterogeneity in the population is due to individual differences in susceptibility and infectivity as is considered in this thesis, those most susceptible should get vaccinated first if the infectivity is equal across types of individuals and otherwise the optimal strategy is unclear. Indeed, there is a trade-off in terms of high susceptibility and infectivity when determining the optimal vaccination strategy.

Furthermore, the purpose of vaccination influences the strategies as well, as vaccinating elderly and those with health issues may be more efficient to protect vulnerable people, whereas vaccinating younger individuals may reduce the spread more depending on who is the most susceptible to infection. As an intervention against the COVID-19 pandemic, The World Health Organization (WHO) [14] recommends vaccinating those most at risk of getting infected along with the elderly, those with other health conditions and health workers before vaccinating other risk groups and the general public. Similarly, in Sweden, the Public Health Agency [8] recommends first vaccinating those with the highest need of protection where age and socio-economic factors are considered, where those living or working at care homes and hospitals as well as those having close contacts to people living at care homes should be prioritized.

Research in relation to this has been done aiming to determine the optimal vaccine allocation as a means to reduce the consequences of the COVID-19 pandemic the most effectively. Ferranna, Cadarette & Bloom [7] evaluate several strategies considering different vaccines, including vaccinating the elderly first, the elderly as well as essential workers unable to work remotely, and younger individuals. They included age differences as well as essential worker status in their epidemic model and found that prioritizing the elderly reduced the number of deaths the most for most scenarios and also that vaccinating essential workers reduced the spread and years of life lost, while prioritizing younger people reduced fatalities the least. Earlier works considering optimal vaccination schemes include Duijzer, van Jaarsveld, Wallinga

& Dekker [6] who show that an optimal strategy can require notably fewer vaccines compared to commonly suggested allocation methods, and Wallinga, van Boven & Lipsitch [16] who show that vaccinating those with a higher risk of getting infected reduces transmission the most.

As the vaccination strategy against COVID-19 is a topical matter, this thesis aims to investigate the effectiveness of vaccination strategies in terms of reducing the severity of the outbreak and protecting risk groups for an outbreak similar to COVID-19 in a population structured similar to Sweden, using COVID-19 data for the Stockholm region in Sweden as well as population distribution and contact data for Finland (since we do not have access to the detailed contact data from Sweden). We assume that the number of available vaccines is such that a certain fraction of the population is vaccinated which is varied for each strategy, and we use as a *perfect* vaccine resulting in lifelong immunity for all vaccinated individuals along with an *all-or-nothing* vaccine resulting in 90% of the vaccinated individuals being fully immune and leaving the remainder unaffected. The first strategy considers vaccinating the population uniformly in the sense that each type of individual has the same fraction vaccinated, while the second strategy vaccinates individuals in descending order of the respective probability of getting infected early on in the outbreak. The final method vaccinates males before females in descending order of age to protect vulnerable groups, similar to the recommended strategies mentioned above.

Keeping in mind that all models are oversimplifications of reality but can be beneficial and applicable by being aware of the assumptions and reasons for deviations from real-life, we use a stochastic epidemic model with the population divided into types depending on age and sex, in order for the model to be as realistic as possible yet simple enough to comprehend. We extend the basic SEIR model for a homogeneous population mentioned by Britton & Pardoux [5, p.5-6], where each individual can either be susceptible (S), latently infected and hence exposed (E), infectious (I) or removed (R) through recovery or death, to a multi-type SEIRLD model using Andersson & Britton [1, p.51-52] where the last state of an individual is either recovered (R), long-term ill (L) or dead (D). This may be unrealistic considering a long-term ill individual would eventually either die or hopefully recover, but it allows for simpler computations of the final fractions of long-term ill.

Furthermore, we consider a population where a fraction of each type is vaccinated prior to the outbreak and the final percentages of infected are computed from the final size equation derived and presented in Section 2.2.2. The percentages of long-term ill and dead are computed from the relative probabilities of long-term illness and death computed in Section 3.2 from the COVID-19 data from the Stockholm region in Sweden. To compare the reduction of the spread we compute the effective reproduction number in Section 4.2.4 for each strategy and both vaccines, which is the expected number of further infections caused by an infectious individual in



a population with both susceptible and immune individuals, as is the case after vaccination.

As we show below, the results suggest that the strategy prioritizing vulnerable groups is the most effective in reducing the fatalities and severe cases but the least effective in reducing the spread. Vaccinating uniformly is the least effective in protecting the risk groups but reduces the spread more than vaccinating the risk groups first. The most effective strategy in terms of reducing the spread is the one first vaccinating those most likely to get infected early in the outbreak, which also keeps the vulnerable people fairly protected as the older age groups are the most likely to get infected early and are hence prioritized. As 70% of the population is vaccinated with the perfect vaccine, both vaccinating uniformly and based on early contribution to the spread achieve *herd immunity* where the entire population is protected from the disease even with unvaccinated individuals. When vaccinating with the all-or-nothing vaccine, only the latter strategy achieves herd immunity, clearly suggesting that this is the most effective in reducing the spread in this case.

## 2 Theoretical aspects

### 2.1 Epidemic models

This section introduces a basic stochastic epidemic model which is extended to a stochastic model for a multi-type population with added possible final states in order to capture the notion of risk groups in the model.

#### 2.1.1 The SEIR model

A simple model describing an epidemic outbreak is the stochastic SEIR model which is described by Britton & Pardoux [5, p.5-6], where we assume a closed population of  $N + 1$  individuals where  $N$  are initially susceptible to the disease and one person is already infected. During the epidemic, an individual can either be susceptible (S), exposed (E) and hence latently infected, infectious (I) or removed (R) by either recovering or dying of the disease. The possible movements of an individual between different states are displayed in Figure 1. We let  $S(t)$ ,  $E(t)$ ,  $I(t)$  and  $R(t)$  respectively represent the number of susceptible, exposed, infectious and removed individuals at time  $t$ , and it follows from the assumption of the population being closed that  $N + 1 = S(t) + E(t) + I(t) + R(t)$  at any time point  $t$ . Using this notation and that the epidemic starts at  $t = 0$  we can describe the initial state as  $(S(0), E(0), I(0), R(0)) = (N, 0, 1, 0)$ .

Furthermore, following Britton & Pardoux [5, p.5-6], an infectious individual is assumed to have infectious contacts following a Poisson process with rate  $\beta$ , where each of these contacts is chosen uniformly and randomly

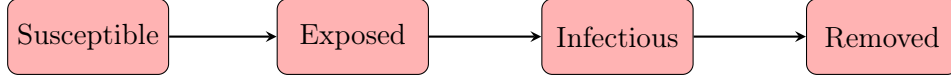


Figure 1: Diagram of the SEIR model with arrows showing the possible directions of movement between compartments for an individual.

from the population. Thus, to keep this rate constant independently of the population size, an infectious individual has contact with another specific individual at rate  $\beta/N$  as mentioned in Andersson & Britton [1, p.11]. Given that the contacted individual is susceptible they become infected and otherwise they are left unaffected. If infected, an individual  $i$  is first latently infected (exposed) for a period denoted by  $X_{(i)}$  which is distributed as the random variable  $X$  with expectation  $\mathbb{E}(X)$ . The individual is infectious for a period  $Y_{(i)}$ , distributed as the random variable  $Y$  with expectation  $\iota_Y$ . After infection the individual either dies or becomes immune and is hence not susceptible to reinfection. We also assume that all Poisson processes, uniformly chosen contacts and durations are mutually independent.

Since the population is closed it follows that eventually there will be no more exposed or infectious individuals and the epidemic will stop at some time point  $\tau$ , where  $\tau := \min\{t; E(t) + I(t) = 0\}$ . In this case there are only susceptibles and removed individuals left and therefore the final size of the outbreak is given by the number of infected (now removed) among the initially susceptible at time point  $\tau$ , computed as  $Z = R(\tau) - I(0) = N - S(\tau)$  as in Britton & Pardoux [5, p.6]. This topic is further discussed in Section 2.2.

In addition, as a supplement we also mention the deterministic SEIR model where each susceptible individual can become exposed, then infectious and finally removed as in the stochastic model and the rates of these events are respectively given by  $\beta$ ,  $\rho$  and  $\gamma$ . As in the stochastic model the population is still closed, but we now assume fractions instead of numbers such that  $s(t) + e(t) + i(t) + r(t) = 1$  at all times  $t$ , where  $s(t) = S(t)/N$ ,  $e(t) = E(t)/N$ ,  $i(t) = I(t)/N$ , and  $r(t) = R(t)/N$ . This means that we now assume that the initial case is  $(s(0), e(0), i(0), r(0)) = (1 - \varepsilon, 0, \varepsilon, 0)$  for a small fraction  $\varepsilon$ . In the case where the transmission rates between compartments are exponential it can be shown that the deterministic model approximates the stochastic model, where the following system of differential equations

$$\begin{aligned}
 s'(t) &= -\beta s(t)i(t), \\
 e'(t) &= \beta s(t)i(t) - \rho e(t), \\
 i'(t) &= \rho e(t) - \gamma i(t), \\
 r'(t) &= \gamma i(t)
 \end{aligned}$$

describes the deterministic model as in Britton & Pardoux [5, p.6, 22]. The

definitions of the symbols used are summarized in Table 1.

Table 1: Definitions of symbols used in the deterministic SEIR model.

Symbol	Definition
$s(t)$	Fraction of population that is susceptible at time $t$
$e(t)$	Fraction of population that is exposed at time $t$
$i(t)$	Fraction of population that is infected at time $t$
$r(t)$	Fraction of population that is removed at time $t$
$\beta$	Rate at which a susceptible individual becomes infected
$\rho$	Rate at which a latent individual becomes infectious
$\gamma$	Rate at which an infectious individual becomes removed

Nevertheless, the SEIR model relies on assumptions such that the population is homogeneous where every individual has equal rate of infectious contacts with other individuals and that each individual has equal rates of recovery (or death). In reality, there may be differences in susceptibility, social structures and activity, and some individuals could be more predisposed to severe illness due to for instance age or chronic diseases. Additionally, as the purpose of this thesis is to compare different vaccination strategies in terms of risk groups and social activity, it is necessary to include those aspects in the epidemic model. Therefore, we now extend the SEIR model to include individual differences within the population, as well as distinguishing between long-term ill, recovered and dead individuals.

### 2.1.2 The extended multi-type SEIRLD model

The SEIR model above is now extended to an SEIRLD model with the  $R$  compartment divided into recovered ( $R$ ), long-term ill ( $L$ ) and dead ( $D$ ) individuals. The  $R$ ,  $L$  and  $D$  compartments are absorbing states without possible intercompartmental movement, as seen in the diagram in Figure 2. This may be considered unrealistic as a long-term ill individual would probably either eventually recover or die, however we assume this is not the case since it is of interest to study the final fraction of long-term ill individuals in order to compare vaccination strategies in terms of risk groups.

Moreover, the population is assumed to be closed and is now divided into  $k \in \{1, 2, \dots, K\}$  different types with varying combinations of age and sex. Additionally, we let the outbreak develop for a period of one year in order to keep the ages of the individuals constant. Following Andersson & Britton [1, p.51-52], we now assume that initially there are  $n_k$  susceptible  $k$ -individuals and  $m_k$  infectious  $k$ -individuals with no exposed, recovered, long-term ill or dead individuals. We denote the total number of initially susceptible by  $n = \sum_{k=1}^K n_k$  and the total number of initially infectious by  $m = \sum_{k=1}^K m_k$ . In addition, we assume that each  $j$ -individual has infectious

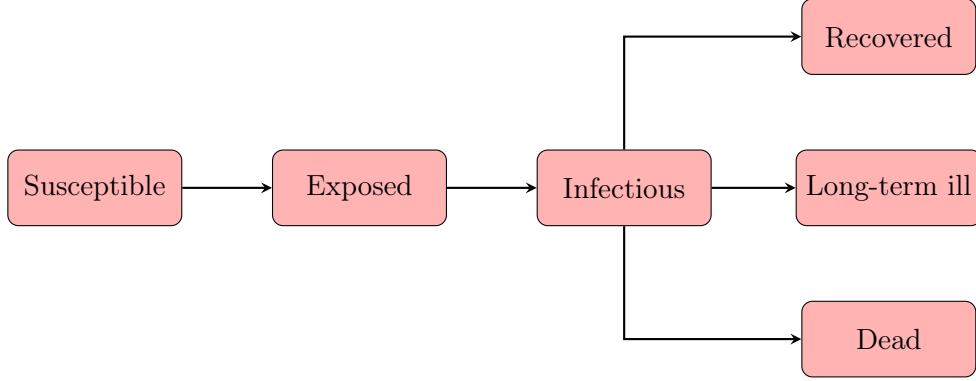


Figure 2: Diagram of the SEIRLD model with arrows showing the possible directions of movement between compartments for an individual.

contacts with a specific  $k$ -individual following a homogeneous Poisson process with rate  $\beta_{jk}/n$  and we note that  $\beta_{jk}$  and  $\beta_{kj}$  need not coincide, as the disease transmission depends both on the infectivity of the infectious individual and the susceptibility of the contacted individual.

Similar to the stochastic SEIR model, each infected individual  $i$  first becomes latent (exposed) with random duration  $X_{(i)}$  distributed as  $X$  with expectation  $\mathbb{E}(X)$ , while each  $k$ -individual is infectious for a random duration  $Y_k$  distributed as  $Y$  with expectation  $\iota_{Y_k}$ . This way we assume that the final state of either recovery, long-term illness or death for an individual is independent of the time spent in the infectious state. However, it is possible to assume otherwise if for instance individuals who die of the disease are assumed to do so at a faster rate than other individuals recover as they are more vulnerable, or they could be assumed to die at a slower rate if they for example are in a coma prior to death. Furthermore, we again assume that all Poisson processes, random choices of contacts and durations are mutually independent.

Moreover, we mention the deterministic SEIRLD model for the sake of completeness and using the supplementary materials for Britton, Ball and Trapman [4, p.3] as a main reference, we describe the model by the following system of differential equations

$$\begin{aligned}
s'_k(t) &= -s_k(t) \sum_{j=1}^K \pi_j \beta_{jk} i_j(t), \\
e'_k(t) &= s_k(t) \sum_{j=1}^K \pi_j \beta_{jk} i_j(t) - \rho e_k(t), \\
i'_k(t) &= \rho e_k(t) - (\gamma_k^r + \gamma_k^l + \gamma_k^d) i_k(t), \\
r'_k(t) &= \gamma_k^r i_k(t), \\
l'_k(t) &= \gamma_k^l i_k(t), \\
d'_k(t) &= \gamma_k^d i_k(t)
\end{aligned}$$

where  $\pi_j = n_j/n$  is the fraction of  $j$ -individuals and the rest of the symbols are defined in Table 2. In addition, as the population is assumed closed it follows in accordance with the SEIR model that the sum of the fractions of  $k$ -individuals in each state is given by  $1 = s_k(t) + e_k(t) + i_k(t) + r_k(t) + l_k(t) + d_k(t)$  for any time point  $t$  and  $\sum_{k=1}^K \pi_k = 1$ . Finally, similar to the SEIR model, the deterministic SEIRLD model approximates the stochastic SEIRLD model if the transmission rates between states are exponentially distributed. Nonetheless, Britton & Pardoux [5, p.22] mention that there are other possible distributions of the rates of leaving states, such as a gamma distribution with an integer valued shape parameter such that we have a sum of independent exponential distributions. Another special case they mention is when the infectious and latent periods are non-random.

Table 2: Definitions of symbols used in the SEIRLD model.

Symbol	Definition
$s_k(t)$	Fraction of susceptibles in group $k$ at time $t$
$e_k(t)$	Fraction of exposed individuals in group $k$ at time $t$
$i_k(t)$	Fraction of infectives in group $k$ at time $t$
$r_k(t)$	Fraction of recovered individuals in group $k$ at time $t$
$l_k(t)$	Fraction of long-term ill individuals in group $k$ at time $t$
$d_k(t)$	Fraction of dead individuals in group $k$ at time $t$
$\beta_{jk}$	Infection rate for an individual from group $k$ getting infected by an individual from group $j$
$\rho$	Rate at which a latent individual becomes infectious
$\gamma_k^r$	Recovery rate for an individual from group $k$
$\gamma_k^l$	Long-term illness rate for an individual from group $k$
$\gamma_k^d$	Disease-induced death rate for an individual from group $k$

## 2.2 Final size equations

In this section we introduce the final size equation for a single-type population where the whole population is initially susceptible, as well as when a fraction is initially immune and then extend the equations to a multi-type setting in the final subsection.

### 2.2.1 The single-type case

When the epidemic has stopped at some time point  $\tau$  and there are no more exposed or infectious people, it is possible to compute the final size of a major outbreak by evaluating the final fraction of infected among the initially susceptible. To do this, we first consider a population with single-type individuals for simplicity and utilize the SEIR model mentioned above. We note that in this model, each infected individual will have become removed at the end of the epidemic and thus the fraction of those not infected could be computed from the amount of susceptibles left when the epidemic has stopped. Therefore, as we are solely interested in the final outcome we can disregard the progression of the outbreak and this is a consequence of the infectious contacts following a Poisson process, which is a special case of a continuous time Markov process where each state in the process is independent of the past.

In order to do this, we first introduce the basic reproduction number, denoted by  $R_0$ . As in Heesterbeek & Dietz [9, p.89], this is the expected number of people that an infectious individual in turn infects during their infectious period, in a fully susceptible population. A major outbreak in a large population is possible if and only if  $R_0 > 1$ , since if  $R_0 = 1$  each infected person only infects one other person on average and if  $R_0 < 1$ , the expected number of infected people will decline and hence the outbreak will diminish quickly. This can be observed through that the expectation of a non-negative integer-valued random variable is greater than the probability that it is positive, since if the expected number of infected is declining, the probability of an outbreak decreases as well. Additionally, as each individual has infectious contacts at rate  $\beta$  with expected infectious time period  $\iota_Y$ , we get that  $R_0$  can be expressed as

$$R_0 = \mathbb{E}(\beta Y) = \beta \iota_Y$$

as stated by Britton & Pardoux [5, p.8-9].

Now we can derive the final size equation using Britton [3, p.6] as a main reference. We denote the final fraction infected among the initially susceptible by  $z$  and assume a large population such that we can neglect the initially infected single individual and assume a population size of  $N$  instead of  $N + 1$ , meaning that the final number of infected is  $zN$ . Additionally, as the infectious contact rate between an infectious individual and another

susceptible individual is  $\beta/N$ , we have that the number of infected individuals by an individual  $i$  during their infectious period is given by  $Y_{(i)}\beta/N$ . As the infectious contacts follow a Poisson process, the number of infectious contacts is Poisson distributed with parameter  $Y_{(i)}\beta/N$  and we get that the probability of escaping infection from an individual  $i$  is

$$\mathbb{E}\left(\frac{(Y_{(i)}\beta/N)^0 e^{-Y_{(i)}\beta/N}}{0!}\right) = \mathbb{E}(e^{-Y_{(i)}\beta/N}).$$

Furthermore, we assume that the fraction not getting infected is approximately the same as the probability of not getting infected by assuming that the fraction converges as the population size increases to infinity and hence that it is non-random. Supposing that this is approximately equal to the probability of escaping infection from all  $zN$  infectious individuals at the end of the epidemic, we get that

$$\begin{aligned} 1 - z &= \text{fraction not getting infected} \\ &\approx \text{probability not getting infected} \\ &\approx \text{probability of escaping infection from all } zN \text{ infected} \\ &\approx \mathbb{E}\left(e^{-\frac{Y_{(1)}\beta}{N}}\right) \mathbb{E}\left(e^{-\frac{Y_{(2)}\beta}{N}}\right) \cdot \dots \cdot \mathbb{E}\left(e^{-\frac{Y_{(zN)}\beta}{N}}\right) \\ &= \mathbb{E}\left(e^{-\frac{\beta}{N} \sum_{i=1}^{zN} Y_{(i)}}\right) \\ &\approx e^{-\beta z \mathbb{E}(Y)} \\ &= e^{-R_0 z} \end{aligned}$$

where we used that the probabilities of escaping infection from specific individuals are independent as well as that  $\sum_{i=1}^{zN} Y_{(i)}/(zN)$  converges to  $\mathbb{E}(Y)$  almost surely as  $zN \rightarrow \infty$  by the strong law of large numbers. To summarize, the final size equation in the single-type case is given as

$$1 - z = e^{-R_0 z}$$

which is a balance equation for  $z$ , where solving for  $z$  yields the final fraction of infected among the initially susceptible. We wish to determine the largest solution for  $z$ , as this can either be 0 in the case where  $R_0 \leq 1$  and for a minor outbreak if  $R_0 > 1$ , or between 0 and 1 for a major outbreak if  $R_0 > 1$ .

Furthermore, if there is a fraction  $v$  of initially immune individuals due to for instance vaccination, then the fraction of initially susceptible reduces to  $1 - v$  as the fraction of initially infectious is very small and negligible in a large population. The contact rate between an infectious individual and other initially susceptible individuals  $\beta/N$  is unchanged but the population size changes to  $N(1 - v)$ , which means that the new reproduction number is

given by

$$R_v = \mathbb{E}\left(\frac{\beta}{N}YN(1-v)\right) = (1-v)\beta\iota_Y = (1-v)R_0,$$

as in Britton & Pardoux [5, p.17-18]. As a result, we can express the final size equation in this case as

$$1 - z = e^{-R_v z}$$

and solving for  $z$  again yields the final fraction of infected among the initially susceptible but in the case of a fraction  $v$  being initially immune. Similar to the case without initially immune individuals, if  $R_v \leq 1$  the solution is 0 and if  $R_v > 1$  the final fraction is either 0 for a minor outbreak and otherwise between 0 and 1 for a major outbreak.

### 2.2.2 The multi-type case

In the case of a multi-type population where the infectious contacts again follow a Poisson process such that we can neglect the development of the outbreak, we can extend the above equations to find the final fraction of infected for each type of individual. We do this similarly as above by now considering the final fraction of infected  $k$ -individuals, which we denote  $z_k$  and the contact rate between an infectious  $j$ -individual and a susceptible  $k$ -individual is now  $\beta_{jk}/n$  where  $n$  denotes the population size. This means that the number of infected during the infectious period of the  $j$ -individual is  $Y_j\beta_{jk}/n$ . Since the infectious contacts are independent Poisson processes, the number of contacts follow a Poisson distribution with parameter  $Y_j\beta_{jk}/n$  and we get that the probability for a  $k$ -individual to escape infection from a  $j$ -individual is

$$\mathbb{E}\left(\frac{(Y_j\beta_{jk}/n)^0 e^{-Y_j\beta_{jk}/n}}{0!}\right) = \mathbb{E}(e^{-Y_j\beta_{jk}/n}).$$

Nevertheless, we need to find the probability that a  $k$ -individual avoids infection by all the  $j$ -individuals. We assume that the number of  $j$ -individuals  $n_j$  is very large and hence that the difference between  $n_j$  and  $n_j - m_j$  is negligible for the number of initially infectious  $j$ -individuals  $m_j$ , which results in the final number of infected  $j$ -individuals being  $z_j n_j$ . Thus, the probability of escaping infection from all  $j$ -individuals for a  $k$ -individual is given as the following

$$\mathbb{E}(e^{-Y_{j(1)}\beta_{jk}/n})\mathbb{E}(e^{-Y_{j(2)}\beta_{jk}/n}) \cdot \dots \cdot \mathbb{E}(e^{-Y_{j(z_j n_j)}\beta_{jk}/n}) = \mathbb{E}(e^{-\beta_{jk}/n \sum_{i=1}^{z_j n_j} Y_{j(i)}})$$

where we again utilized the independence between probabilities. Using the strong law of large numbers as in the single-type case, we get that the above is approximately

$$e^{-\beta_{jk} n_j z_j \mathbb{E}(Y_j)/n} = e^{-\beta_{jk} \pi_j z_j \iota_Y}$$



where we used that  $\pi_j = n_j/n$ .

However, this is merely the probability for a  $k$ -individual to escape infection from the individuals of type  $j$ . As there are  $K$  different types of individuals and as we assume that the rates of infectious contacts between groups are independent, we get that the probability of escaping infection from *all* individuals for a  $k$ -individual is given as

$$\prod_{j=1}^K e^{-\beta_{jk}\pi_j z_j \iota_{Y_j}} = e^{-\sum_{j=1}^K \beta_{jk}\pi_j z_j \iota_{Y_j}}.$$

Thus, we can finally express the final size equation in the multi-type case as

$$1 - z_k = e^{-\sum_{j=1}^K \iota_{Y_j} \beta_{jk}\pi_j z_j}$$

which is a balance equation for  $z_k$  and it is in accordance with Equation (6.2) in Andersson & Britton [1, p.54], since  $1 - z_k$  is the fraction among the initially susceptible who escape infection. In line with the single-type case, we wish to find the largest solution  $z_k$  which is 0 for a minor outbreak and between 0 and 1 for a major outbreak.

Additionally, as in the single-type case we also consider the scenario with a fraction  $v_j$  of already immune  $j$ -individuals and susceptible  $k$ -individuals, where  $j \neq k$ . Similar to in the single-type case, the contact rate  $\beta_{jk}/n$  between a  $j$ -individual and a  $k$ -individual is unchanged but the population size of  $j$ -individuals changes to  $(1 - v_j)n_j$ , as only the fraction  $1 - v_j$  of  $j$ -individuals are initially susceptible and this transforms the final size equation into

$$1 - z_k = e^{-\sum_{j=1}^K \iota_{Y_j} \beta_{jk}(1-v_j)\pi_j z_j}. \quad (1)$$

Furthermore, as the solution  $z_k$  denotes the final fraction of infected  $k$ -individuals among the initially susceptible we can use this in the SEIRLD model since this fraction is divided into the recovered, long-term ill and dead individuals. Hence, to find the final fractions of recovered, long-term ill and dead we can multiply  $z_k$  with the corresponding probabilities of these events for a  $k$ -individual.

As in the single-type case, we can compute  $R_0$  before vaccination which in the multi-type case is given as the largest eigenvalue of the next generation matrix whose entries are given by  $\beta_{jk}\pi_k \iota_{Y_k}$ , as stated in Andersson & Britton [1, p.54]. After a fraction  $v_k$  of  $k$ -individuals is vaccinated, the effective reproduction number  $R_v$  is given as the largest eigenvalue of the matrix with entries  $\beta_{jk}\pi_k \iota_{Y_k} (1 - v_k)$  since the fraction of initially susceptible  $k$ -individuals is now  $(1 - v_k)\pi_k$ .

Moreover, we use the supporting information by Wallinga, van Boven & Lipsitch [16, p.1-2] as a main reference to note that for the next generation matrix with entries  $\beta_{jk}\pi_k \iota_{Y_k}$ , the left eigenvector belonging to  $R_0$  is approximately proportional to the number of individuals of each type among the

infected in the generation. That is if we assume that no interventions have been introduced prior to the time period of this generation, as well as that the distribution of new infections for each type is similar to that during the time period of this generation. Thus, if the next generation matrix is denoted by  $N$  and the left eigenvector belonging to  $R_0$  is denoted by  $a$ , we get that  $aN = R_0a$  and this suggests that the fractions of infected are according to the normalized eigenvector  $a$  such that the entries add up to 1 and that the fractions are increased by the factor  $R_0$  each generation. Therefore, we assume that the normalized eigenvector  $a$  approximates the fractions of infected in the early stages of the epidemic.

To get an idea of how likely individuals are to get infected in the epidemic, we divide the entries of the eigenvector  $a$  by the respective fractions of individuals of each type in the population. As this is the fraction of infected of each type divided by the fraction of individuals of each type, it follows that this is proportional to the number of infected of each type divided by the number of people of each type, which is the probability for each type to get infected in the early stages. We can hence use this to decide who to vaccinate first by vaccinating in descending order of the size of  $a_k/\pi_k$  for each type  $k$ , where  $k = 1, 2, \dots, K$ .

## 2.3 Vaccination

In this section we describe different vaccination strategies to implement against an epidemic outbreak as well as how the properties of vaccines can differ. We also mention the assumptions we make considering vaccinations and the vaccines in this thesis.

### 2.3.1 Vaccination strategies

There are various strategies to combat an epidemic outbreak such as for instance introducing quarantine, closing down parts of the community and imposing travel restrictions, which all intend to reduce contact rates in order to reduce  $R_0$  as pointed out by Britton [3, p.7-8]. Another strategy is vaccination, which instead of reducing contact rates aims to reduce the amount of susceptibles in the population and there are numerous possible vaccination strategies with different purposes. However, in some circumstances some strategies may be impossible to implement due to for instance a shortage of vaccines or a lack thereof, as in the early stages of the COVID-19 pandemic prior to the manufacturing of vaccines.

Although, given that a vaccine is already available a proportion of the population could either be previously vaccinated, immediately vaccinated at the beginning of the outbreak or the vaccinations could be initiated at a later time point. The vaccination process could also be distributed over a longer period and in practice the process requires time and therefore this

would be more realistic than the assumption we make that a proportion of the population is already vaccinated. Moreover, a significant topic of interest is the allocation of the vaccines and its order which depend on the severity of the epidemic and the purpose of the vaccinations. One possible strategy is to prioritize individuals with high social activity to reduce the spread, while another is to focus on those with individual risk factors such as age or chronic diseases that could affect the recovery rate, and severity of illness. This would protect those most vulnerable in the population.

In this thesis we are interested in analyzing how the fractions of severely ill and deceased individuals at the end of the outbreak are affected by different vaccination strategies as well as how the size of the outbreak is affected. Moreover, it is also relevant to examine how the probability of an introduction of another outbreak is affected by different vaccination strategies, if for instance an outbreak has already occurred and parts of the community are already immune from being infected during the first outbreak.

### 2.3.2 Types of vaccines

Another significant aspect of vaccination is the action of the vaccine as its ability to reduce susceptibility and infectivity is essential. In this thesis we consider vaccines that reduce the susceptibility of individuals. Different types of vaccines can have varying efficacy, which as in Andersson & Britton [1, p.123] is the relative decrease in the rate of infection of vaccinated individuals by infectious individuals, in comparison with that of completely susceptible and unvaccinated people.

However, the efficacy could either impact how protected a vaccinated individual is from becoming infected or the proportion of completely immune individuals among those vaccinated, meaning that some vaccinated individuals remain fully susceptible to infection in that case. Thus, different vaccines can affect susceptibility differently and some examples include an *all-or-nothing* vaccine that independently provides a fraction  $E$  of individuals with complete lifelong immunity and leaves the rest unaffected, a *leaky* vaccine that independently reduces the risk of infection given exposure to an infectious contact by a fraction  $E$ , as well as a *perfect* vaccine which provides lifelong immunity to all vaccinated individuals with 100% efficacy. These definitions of different types of vaccines are in line with those stated by Halloran, Haber & Longini, as cited by Ball, Britton & Lyne [2, p.20,23]. We take these differences into account by comparing the use of a perfect vaccine and an all-or-nothing vaccine in this thesis.

Furthermore, some vaccines could require multiple doses with different time intervals, but for simplicity we solely consider single dose vaccines. Additionally, for a multi-type setting we assume a fraction  $v_k$  of already vaccinated  $k$ -individuals at the start of the epidemic, which as aforementioned is a simplification of reality since sometimes a vaccine is unavailable at the

start of an epidemic outbreak. However, this could be applicable to when new outbreaks occur in a population with a fraction already immune from the first outbreak.

When considering the fractions of available vaccines, we also keep in mind the ability to achieve *herd immunity* where the whole population, including unvaccinated individuals, is protected from the epidemic as in Andersson & Britton [1, p.118]. They also state that for a fraction  $v$  of initially vaccinated in the whole population with uniform vaccine allocation across groups, we need

$$v \geq \frac{1}{E} \left( 1 - \frac{1}{R_0} \right) \quad (2)$$

to reach herd immunity where  $v = v_c$  is the critical vaccination coverage to achieve this. In the case of a large  $R_0$  and a vaccine of low efficacy, it is possible that  $v_c$  exceeds 1 which means that vaccination alone is insufficient in preventing the outbreak as the population is not protected even if everyone is vaccinated.

### 3 Data

#### 3.1 Next generation matrix

In this section we compute a next generation matrix of the expected number of contacts between each type of individual, where the population is divided into 10 groups depending on age and sex with age groups 0-19, 20-39, 40-59, 60-79 and 80+ years. To do this, we use the data from all the reported contacts in Finland in 2008 in the form of the average number of contacts per day found in Table S8.3a in the supporting information in Mossong et al. [11], as well as the population distribution across age groups and sexes in 2008 made available by Statistics Finland [10].

As the rows of the table of average number of contacts represent the age groups of the contacted individuals and the last age group is 70+ years, we transform the data by first adding an extra row, dividing the last row of 70+ years old individuals into the age groups 70-79 and 80+ years. This since the average number of contacted people older than 79 years is included in the values for those older than 69 years, and we therefore assume that the expected number of contacts is the same for both groups. Similarly, as the same issue occurs for the columns which represent the individual making contacts, we divide the 70+ years column into age groups 70-79 and 80+ years by first computing the fraction of those older than 79 years among the individuals aged 70+ years from the population distribution table, which was found to be about 0.374. We then multiply the values in the 70+ years column by 0.374 to get our 80+ years column and by 0.626 to get the 70-79 years column.

Next, as the given age groups only span five years, we group the rows and columns together into our desired groups by taking the average of the average contacts for each combined age group. For instance, the values for contacts between the different combinations of the age groups of 0-4, 5-9, 10-14 and 15-19 years are combined to become the average number of contacts between the age group of 0-19 years and itself by adding the average number of contacts and dividing by the total number of values, which is 16. Repeating this for each of our selected age groups results in a matrix of the average number of contacts per day, but we still need to incorporate the sexes into the matrix. We do this by computing the fraction of males and females in each age group and multiplying the fraction of the sex of the individual making contacts by the fraction of the sex of the contacted individuals and the average number of contacts made between these age groups. We have now produced a contact matrix  $M$  of the average number of contacts between our selected groups by age and sex. The population distribution table with the fractions of males and females in each age group used to compute  $M$  are found in Table 17 in Appendix A.

Moreover, we can express the entries in the contact matrix  $M$  as  $\beta_{jk}\pi_k$  since a  $j$ -individual has close contacts with  $k$ -individuals at rate  $n_k\beta_{jk}/n = \beta_{jk}\pi_k$  (note that  $j$  is represented by the columns and  $k$  by the rows in the matrix as the individual making contacts is represented by the columns and the contacted individuals are represented by the rows in the data from Mossong et al. [11]). Therefore, we can use this to compute the basic reproduction number following Andersson & Britton [1, p.54] as the basic reproduction number in the multi-type case is given by the largest eigenvalue of the matrix whose entries are computed as  $\iota_{Y_j}\beta_{jk}\pi_k$ , which is the expected number of contacts made with  $k$ -individuals by an infectious  $j$ -individual during their infectious period. We assume that the mean infectious period  $\iota_{Y_j}$  is 4 days for all  $j$  as in Britton et al. [4, p.2] and further multiply the matrix  $4M$  by the constant 0.5 to get the largest eigenvalue to be approximately 2.89 to get  $R_0 = 2.9$  in order to have a large outbreak but still resembling the COVID-19 outbreak. Therefore, the next generation matrix is  $2M$  and is found in Table 18 in Appendix A.

Finally, in order to compute the final size equations, we need the matrix with entries  $\beta_{jk}$  which we obtain by dividing the entries  $\beta_{jk}\pi_k$  of matrix  $M$  by the fraction  $\pi_k$  of contacted  $k$ -individuals. However, as we rescaled the next generation matrix to decrease our  $R_0$ , we need to rescale the matrix with entries  $\beta_{jk}$  as well by multiplying each entry by 0.5 in order to keep the same contact intensity. The computed matrix can be found in Table 19 and the values for  $\pi_j$  for all  $j$  are found in Table 20 and both tables are located in Appendix A.

### 3.2 Probabilities of death, long-term illness and recovery

To compute the final fractions of deceased, long-term ill and recovered individuals, we obtain the respective probabilities of dying, becoming long-term ill and recovering for each age group and sex. As Finland and Sweden are geographically adjacent and similar in a sociocultural sense, we use data on the total number of reported cases and deaths due to COVID-19 in the Stockholm region in Sweden as of March 28th 2021 made available by Vårdgivarguiden Region Stockholm [15] to compute the probabilities of dying. We use Figure 5 in the report showing the total number of reported cases, including deaths, by age group and sex in the Stockholm region, and we combine the age groups in pairs by adding the values to obtain our selected age groups, as for instance the first two groups represent the age group 0-19 years and so on. Nevertheless, we note that there were 147 cases mentioned in Figure 5 with unknown details about age and sex.

Using the above as well as Figure 9 in the report showing the total number of reported deaths by age groups and sex, we can compute the probabilities of dying. However, the first age group in the figure is 0-49 years and hence to divide this into our desired groups we compute the fraction of deaths among the total number of cases in the 0-49 years age group and use this same ratio for ages 0-19, 20-39 and 40-49 years. We further compute the fractions of deaths for ages 50-59, 60-79 and 80+ years by adding the number of deaths for ages 60-69 and 70-79 years, as well as for 80-89 and 90+ years and dividing by the total number of cases for the respective age groups. Finally, we obtain the fraction of deaths in the 40-59 years age group by taking the average of the two fractions obtained for ages 40-49 and 50-59 years. The obtained probabilities can be found in Table 3.

Furthermore, we compute the probabilities of becoming long-term ill using data on the reported number of COVID-19 patients by age groups and sex in the Stockholm region as of March 28th 2021 made available by The Swedish Intensive Care Registry (SIR) [13], assuming that this represents the number of long-term ill individuals. We only consider patients who did not end up dying in order to distinguish between long-term ill individuals and disease-induced deaths, as well as only those who remained in hospital care for a minimum of four days to ensure long-term illness. Additionally, we consider both risk groups and non-risk groups and the individuals are divided into age groups spanning 10 years, meaning that we need to add the total number of patients in each age group for each sex in pairs to get our selected age groups spanning 20 years, as we did previously for the total number of cases. The probabilities of long-term illness by age group and sex are computed from the fractions of long-term ill individuals among the total number of cases found, which can be found in Table 3.

Finally, we compute the probabilities of recovering from COVID-19 by age groups and sex by assuming that only those not long-term ill or dead

recover and hence we compute the fractions of the remaining individuals when excluding the reported number receiving hospital care and the dead individuals. The results are found in of Table 3 and illustrated in Figure 3, where we note that the males and females aged 60 years and above can be viewed as risk groups as they have a higher probability of dying and becoming long-term ill than the rest of the population. In addition, the 40- to 59-year-olds have some probability of becoming long-term ill and even dying while almost all individuals in the younger groups are expected to recover in probability.

Table 3: The probabilities of death, long-term illness and recovery by sex and age group in years based on the COVID-19 data from the Stockholm region in Sweden as of March 28th 2021.

		Age groups				
		0-19	20-39	40-59	60-79	80+
Death	Males	0.0005	0.0005	0.0033	0.0623	0.3728
	Females	0.0003	0.0003	0.0010	0.0348	0.2536
Long-term illness	Males	0.0004	0.0019	0.0161	0.0930	0.0182
	Females	0.0001	0.0011	0.0062	0.0360	0.0063
Recovery	Males	0.9991	0.9976	0.9806	0.8448	0.6090
	Females	0.9996	0.9986	0.9929	0.9292	0.7401

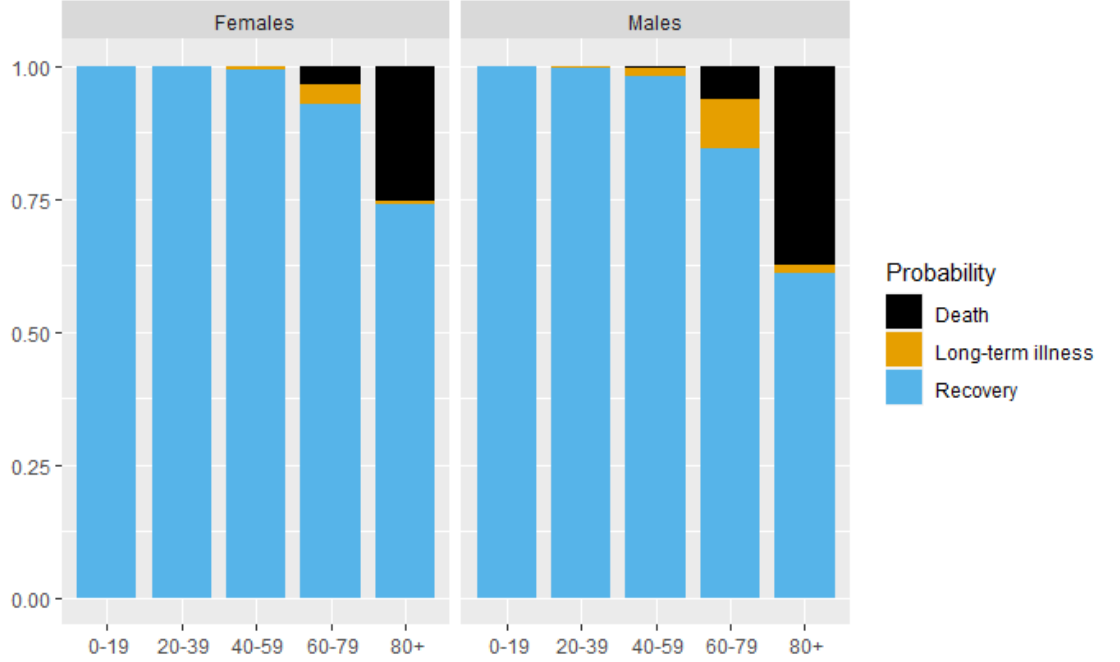


Figure 3: The probabilities of death, long-term illness and recovery by sex and age group in years based on the COVID-19 data from the Stockholm region in Sweden as of March 28th 2021.

## 4 Computations

### 4.1 Methods

We compute the final fractions of infected among the initially susceptible for each type of individual in the population by computing the final size equation in Equation (1) for each type with different fractions of initially immune due to vaccination. The types of vaccines we use are a perfect vaccine and an all-or-nothing vaccine with efficacy  $E = 90\%$ , which is incorporated into the final size equation by substituting  $v$  for  $Ev$  as the fraction  $E$  of the fraction  $v$  gets completely immune from an all-or-nothing vaccine. The final fractions of dead, long-term ill and recovered are computed by multiplying the final fractions with the respective probabilities of death, long-term illness and recovery for each type of individual computed in Section 3.2.

For comparative purposes we first compute the final fraction of infected for each group and their respective probabilities of death, long-term illness and recovery when nobody is vaccinated. Then we vary the different combinations of vaccinated groups as well as the vaccine used, while only vaccinating individuals such that the overall fraction of vaccinated is close to



the critical vaccination coverage for uniform vaccination in the population, according to Equation (2). For a perfect vaccine we get that the critical vaccination coverage is

$$v_c = 1 - \frac{1}{R_0} = 1 - \frac{1}{2.9} \approx 0.66 \quad (3)$$

and for an all-or-nothing vaccine with efficacy 90% the critical vaccination coverage is

$$v_c = \frac{1}{E} \left( 1 - \frac{1}{R_0} \right) = \frac{1}{0.90} \left( 1 - \frac{1}{2.9} \right) \approx 0.73.$$

Furthermore, we assume that there are  $V < n$  vaccines available such that  $V/n = c < 1$  is the fraction of the population we can vaccinate. We therefore assume scenarios where  $c = 0.1, 0.2, \dots, 0.7$  for both the perfect vaccine and the all-or-nothing vaccine in order to stay close to the critical vaccination coverage in both cases.

## 4.2 Results

As the main purpose of this thesis is to compare vaccination strategies in terms of effectiveness in reducing the spread and protecting vulnerable individuals, we first compute the final fractions of infected as well as the probabilities of dying, becoming long-term ill and recovering for each type of individual in an unvaccinated population for comparison. The results are found in Table 4 where we note that almost all individuals aged 80+ years are infected and that the percentages of infected for the ages 0-19, 20-39 and 40-59 years are all above 90% for both sexes, whereas the 60-79 years age group has a significantly lower percentage of 78.92% for both sexes. This could be due to that many individuals in the 80+ years age group may live at retirement homes and have many interactions with other residents and the staff, meanwhile individuals in the 60-79 years age group may be less interactive with other age groups since a large proportion are at the age of retirement and do not attend school or work daily. Furthermore, we note that the final percentages of infected males and females are almost identical across all age groups.

The table also shows that almost all infected individuals under the age of 60 years recover while the corresponding percentage for individuals aged 60+ years is lower, where we notice that men have a lower probability of recovering than females overall and that this difference is more prominent for the elderly. We also note that individuals from the 40-59 years age group have a slightly lower probability of recovering than the younger age groups.

Additionally, it is clear that individuals under the age of 60 years have a minimal risk of dying as the percentages are below 1% for both sexes, while the percentages increase to 4.92% for males and 2.75% for females for the 60-79 years age group and further increase significantly for the 80+ years age

group, where the males are 37.26% likely to die and the females are 25.35% likely to die. Thus, to reduce the number of deaths it seems necessary to vaccinate a larger fraction of those aged 60+ years. The findings also suggest that men are more likely to die than females for all age groups which may indicate a need for vaccinating a higher fraction of males than females.

Similarly, the percentages of long-term ill illustrate how men are more susceptible than females to long-term illness for all age groups, which again suggests that vaccinating a higher fraction of men would be beneficial for the population. The probability of long-term illness for men in the 60-79 years age group is considerably higher than in any other age group, and thus they may need a higher fraction of vaccinated. We also note that the percentages of long-term ill among those younger than 20 years are below 0.1% for both sexes.

Moreover, the 40-59 and 80+ years age groups show a similar percentage of long-term ill individuals, which could be attributed to a large fraction of those older than 79 years dying rather than staying long-term ill. Additionally, as the probability of long-term illness was based on data on the number of hospitalized patients, this result could also be explained by how individuals aged 80+ years may be less likely to get admitted into hospital than younger people who are more likely to recover from intensive care. However, as the highest percentage of long-term ill individuals is 7.34% for the males in the 60-79 years age group which is significantly lower than the highest percentage 37.26% of dead for the males older than 79 years, it may be more important to prioritize the latter group for an overall strategy to protect vulnerable individuals. This however depends on the main aim of the vaccinations.

As we now know the outcomes of the epidemic without any interventions, we can implement different vaccination strategies to compare the results to the outcomes in the unvaccinated case.

Table 4: The final percentages of infected, recovered, dead and long-term ill within each group by age groups in years and sex when the entire population is unvaccinated.

		Age groups				
		0-19	20-39	40-59	60-79	80+
Infected (%)	Males	93.39	94.43	92.53	78.92	99.94
	Females	93.39	94.43	92.53	78.92	99.95
Recovered (%)	Males	93.31	94.20	90.74	66.67	60.87
	Females	93.36	94.29	91.87	73.33	73.98
Dead (%)	Males	0.0467	0.0472	0.3054	4.92	37.26
	Females	0.0280	0.0283	0.0925	2.75	25.35
Long-term ill (%)	Males	0.0374	0.1794	1.49	7.34	1.82
	Females	0.0093	0.1039	0.5737	2.84	0.6297

#### 4.2.1 Vaccinating equal fractions of each group

Assuming we can vaccinate a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population, we first decide to vaccinate the same fraction  $c$  of all types. The final percentages of infected, recovered, dead, and long-term ill are presented in Table 5 after vaccination with a perfect vaccine and in Table 6 after vaccination with the all-or-nothing vaccine. Additionally, the ratios between the final sizes when the same fractions of each group are vaccinated and for the unvaccinated population are presented in Table 7. Since the final fractions of recovered, dead and long-term ill are computed from the final sizes of infected by multiplying with the respective probabilities for each type of individual, the ratios are the same as for the ratios of the fractions of infected which are displayed in the table.

In the case where a perfect vaccine is used, we see in Table 5 that the percentages of recovered are approximately the same for the 0-19, 20-39 and 40-59 age ranges for females and males which indicates that the percentages of dead and long-term ill individuals are small, and we note that they are all close to or below 1%. More significant differences between the percentages of infected and recovered are displayed for the 60-79 years age groups as well as for those aged 80+ years since larger proportions of these types become long-term ill or die.

Furthermore, if only 10% of each group is vaccinated then the final percentages of infected decrease to just under 90% for the individuals older than 79 years, to about 80% for the age groups under 60 years and to about 67% for the 60- to 79-year-olds. The final fractions of dead and long-term ill also decrease slightly, but the percentages of dead individuals aged 80+ years are still above 30% for the males and above 20% for the females. This corre-

sponds to about 90% of the final sizes in the unvaccinated population. We also note that when  $c$  is less than or equal to 0.5 at least 25% of individuals below the age of 60 years are infected for both sexes and when  $c = 0.5$  the proportions are about a third of the final fractions in the unvaccinated case, which is a decent reduction of cases. However, more than 40% of males and females aged 80+ years are still infected which is almost 45% of the infections without a vaccine, indicating that these types of individuals are not well protected by this strategy. This is also shown by how 16% of the 80+ years old males and 11% of the 80+ years old females die of infection when half of the population is vaccinated equally. The percentages of dead 60- to 79-year-olds are close to or below 1% when  $c = 0.5$  which means that this age group is quite well protected by this strategy, although the percentages of dead in the unvaccinated case for this age group were already quite low to begin with as they were below 5%. Additionally, the percentages of long-term ill are all below 2% when  $c \geq 0.5$ .

Moreover, all percentages of infected are close to or below 10% when  $c = 0.6$  except for the 80+ years groups, where the percentages have decreased to 21.7% and 22% which is a large decrease in the number of cases since almost the entire age group was infected in the unvaccinated case. In addition, about 8.1% of the males and 5.6% of the females die which means that less than 10% of those groups die, but these percentages might be reduced further by using another vaccination strategy. However, the percentages of dead 60- to 79-year-olds are below 0.5% for  $c = 0.6$ , indicating that this strategy is already sufficient in protecting this age group. Furthermore, since herd immunity is reached when roughly 66% of the population has been vaccinated with the perfect vaccine according to Equation (3), the final percentages of infected are zero when 70% of the population has been vaccinated.

Table 5: The final percentages of infected, recovered, dead and long-term ill within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with a perfect vaccine. In each group the same fraction  $c$  of individuals of that type is vaccinated.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Infected (%)	0.1	81.60	82.74	80.60	66.53	89.86	81.60	82.74	80.60	66.53	89.88
	0.2	69.30	70.52	68.16	54.03	79.65	69.30	70.52	68.16	54.03	79.69
	0.3	56.32	57.56	55.07	41.52	69.09	56.32	57.56	55.07	41.52	69.18
	0.4	42.41	43.59	41.12	29.14	57.62	42.41	43.59	41.12	29.14	57.80
	0.5	27.21	28.16	26.05	17.11	43.59	27.21	28.16	26.05	17.11	43.91
	0.6	10.15	10.60	9.54	5.75	21.67	10.15	10.60	9.54	5.75	22.01
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Recovered (%)	0.1	81.53	82.54	79.04	56.20	54.72	81.57	82.62	80.03	61.82	66.52
	0.2	69.24	70.35	66.84	45.65	48.50	69.27	70.42	67.68	50.21	58.98
	0.3	56.27	57.43	54.00	35.08	42.08	56.30	57.48	54.68	38.58	51.20
	0.4	42.37	43.49	40.32	24.62	35.09	42.40	43.53	40.82	27.08	42.78
	0.5	27.19	28.10	25.54	14.46	26.55	27.20	28.12	25.86	15.90	32.50
	0.6	10.14	10.58	9.35	4.86	13.20	10.14	10.59	9.47	5.34	16.29
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Dead (%)	0.1	0.0408	0.0414	0.2660	4.14	33.50	0.0245	0.0248	0.0806	2.32	22.79
	0.2	0.0346	0.0353	0.2249	3.37	29.69	0.0208	0.0212	0.0682	1.88	20.21
	0.3	0.0282	0.0288	0.1817	2.59	25.76	0.0169	0.0173	0.0551	1.44	17.54
	0.4	0.0212	0.0218	0.1357	1.82	21.48	0.0127	0.0131	0.0411	1.01	14.66
	0.5	0.0136	0.0141	0.0860	1.07	16.25	0.0082	0.0084	0.0260	0.5955	11.14
	0.6	0.0051	0.0053	0.0315	0.3581	8.08	0.0030	0.0032	0.0095	0.2000	5.58
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Long-term ill (%)	0.1	0.0326	0.1572	1.30	6.19	1.64	0.0082	0.0910	0.4997	2.40	0.5662
	0.2	0.0277	0.1340	1.10	5.03	1.45	0.0069	0.0776	0.4226	1.95	0.5020
	0.3	0.0225	0.1094	0.8866	3.86	1.26	0.0056	0.0633	0.3414	1.49	0.4358
	0.4	0.0170	0.0828	0.6620	2.71	1.05	0.0042	0.0480	0.2549	1.05	0.3641
	0.5	0.0109	0.0535	0.4193	1.59	0.7934	0.0027	0.0310	0.1615	0.6160	0.2766
	0.6	0.0041	0.0201	0.1535	0.5345	0.3944	0.0010	0.0117	0.0591	0.2069	0.1387
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Moreover, for the all-or-nothing vaccine we see in Table 6 that the percentages of infected aged 80+ years are above 90%, that the percentages of infected younger than 60 years are above 80% and that almost 70% of the 60- to 79-year-olds are infected for both sexes when 10% of the population is vaccinated, corresponding to about 90% of the infections in the unvaccinated case. The percentages of dead individuals under the age of 60 years are below 0.5% for both sexes, while the percentages of dead people aged 80+ years are still above 30% for the males and over 20% for the females. The final fractions of dead 60- to 79-year-olds did not change significantly either and similar results are found for the percentages of long-term ill as well.

Additionally, the proportion of infected individuals younger than 60 years are at least above 30% for both sexes for  $c$  less than or equal to 0.5, while about 20% of the 60- to 79-year-olds and about half of the 80+ years group are infected for both sexes, showing a similar trend to when a perfect vaccine

is used. The proportion of long-term ill individuals are less than or close to 2% when half the population in each group is vaccinated, and we also see that only about 1% of the 60- to 79-year-old males and less than 1% of the females die but that the percentages of deceased in the 80+ years age group are about 20% for the males and 13% for the females, resulting in a high number of deaths in a large population.

When 60% of the population is vaccinated, the proportion of deceased who are 80+ years old is closer to 10% and the remaining groups have percentages less than 1%, while the percentages of long-term ill individuals are minimal across all groups. The final proportions of infected are now all below 40%, where the younger age groups are down to about 20% infected and the 60- to 79-year-olds have closer to 10% infected. As we approach the critical vaccination coverage for the all-or-nothing vaccine with 70% of the population vaccinated, we note that all percentages of infected individuals younger than 80 years are below 5% whereas about 11% of the group aged 80+ years is infected. This results in about 4% and around 3% of the males and females aged 80+ years dying, respectively.

Table 6: The final percentages of infected, recovered, dead and long-term ill within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with an all-or-nothing vaccine of efficacy 90%. In each group the same fraction  $c$  of individuals of that type is vaccinated.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Infected (%)	0.1	82.80	83.93	81.82	67.77	90.87	82.80	83.93	81.82	67.77	90.89
	0.2	71.81	73.01	70.70	56.54	81.71	71.81	73.01	70.70	56.54	81.74
	0.3	60.30	61.54	59.07	45.27	72.31	60.30	61.54	59.07	45.27	72.39
	0.4	48.11	49.33	46.81	34.06	62.38	48.11	49.33	46.81	34.06	62.52
	0.5	35.00	36.09	33.74	23.06	51.11	35.00	36.09	33.74	23.06	51.36
	0.6	20.65	21.44	19.63	12.47	36.36	20.65	21.44	19.63	12.47	36.72
	0.7	4.56	4.79	4.26	2.51	11.14	4.56	4.79	4.26	2.51	11.36
Recovered (%)	0.1	82.73	83.73	80.23	57.25	55.34	82.77	83.81	81.23	62.97	67.27
	0.2	71.74	72.84	69.33	47.76	49.76	71.78	72.91	70.20	52.53	60.50
	0.3	60.24	61.39	57.93	38.24	44.04	60.27	61.46	58.66	42.06	53.57
	0.4	48.06	49.21	45.91	28.78	37.99	48.09	49.26	46.48	31.65	46.27
	0.5	34.97	36.01	33.09	19.48	31.13	34.99	36.04	33.50	21.43	38.01
	0.6	20.63	21.39	19.25	10.53	22.14	20.64	21.41	19.49	11.59	27.18
	0.7	4.56	4.78	4.18	2.12	6.78	4.56	4.78	4.23	2.33	8.41
Dead (%)	0.1	0.0414	0.0420	0.2700	4.22	33.88	0.0248	0.0252	0.0818	2.36	23.05
	0.2	0.0359	0.0365	0.2333	3.52	30.46	0.0215	0.0219	0.0707	1.97	20.73
	0.3	0.0301	0.0308	0.1949	2.82	26.96	0.0181	0.0185	0.0591	1.58	18.36
	0.4	0.0241	0.0247	0.1545	2.12	23.26	0.0144	0.0148	0.0468	1.19	15.86
	0.5	0.0175	0.0180	0.1113	1.44	19.05	0.0105	0.0108	0.0337	0.8026	13.02
	0.6	0.0103	0.0107	0.0648	0.7768	13.55	0.0062	0.0064	0.0196	0.4332	9.31
	0.7	0.0023	0.0024	0.0141	0.1563	4.15	0.0014	0.0014	0.0043	0.0873	2.88
Long-term ill (%)	0.1	0.0331	0.1595	1.32	6.30	1.65	0.0083	0.0923	0.5073	2.44	0.5726
	0.2	0.0287	0.1387	1.14	5.26	1.49	0.0072	0.0803	0.4383	2.03	0.5150
	0.3	0.0241	0.1169	0.9511	4.21	1.32	0.0060	0.0677	0.3663	1.63	0.4560
	0.4	0.0192	0.0937	0.7537	3.17	1.14	0.0048	0.0543	0.2902	1.23	0.3939
	0.5	0.0140	0.0686	0.5432	2.14	0.9302	0.0035	0.0397	0.2092	0.8303	0.3235
	0.6	0.0083	0.0407	0.3161	1.16	0.6617	0.0021	0.0236	0.1217	0.4489	0.2313
	0.7	0.0018	0.0091	0.0686	0.2333	0.2028	0.0005	0.0053	0.0264	0.0903	0.0716

Table 7: The ratio of final sizes within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with a perfect or an all-or-nothing vaccine with 90% efficacy, compared to in an unvaccinated population. In each group the same fraction  $c$  of individuals of that type is vaccinated.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Perfect	0.1	0.87	0.88	0.87	0.84	0.90	0.87	0.88	0.87	0.84	0.90
	0.2	0.74	0.75	0.74	0.68	0.80	0.74	0.75	0.74	0.68	0.80
	0.3	0.60	0.61	0.60	0.53	0.69	0.60	0.61	0.60	0.53	0.69
	0.4	0.45	0.46	0.44	0.37	0.58	0.45	0.46	0.44	0.37	0.58
	0.5	0.29	0.30	0.28	0.22	0.44	0.29	0.30	0.28	0.22	0.44
	0.6	0.11	0.11	0.10	0.07	0.22	0.11	0.11	0.10	0.07	0.22
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
All-or-nothing	0.1	0.89	0.89	0.88	0.86	0.91	0.89	0.89	0.88	0.86	0.91
	0.2	0.77	0.77	0.76	0.72	0.82	0.77	0.77	0.76	0.72	0.82
	0.3	0.65	0.65	0.64	0.57	0.72	0.65	0.65	0.64	0.57	0.72
	0.4	0.52	0.52	0.51	0.43	0.62	0.52	0.52	0.51	0.43	0.63
	0.5	0.37	0.38	0.36	0.29	0.51	0.37	0.38	0.36	0.29	0.51
	0.6	0.22	0.23	0.21	0.16	0.36	0.22	0.23	0.21	0.16	0.37
	0.7	0.05	0.05	0.05	0.03	0.11	0.05	0.05	0.05	0.03	0.11

#### 4.2.2 Vaccinating in descending order of contribution to spread

In order to find the types of individuals contributing most to the spread we consider the probabilities of getting infected in the early stages of the epidemic by first computing the eigenvector corresponding to  $R_0$  according to Section 2.2.2. However, as our next generation matrix in Table 18 in Appendix A is transposed with the columns representing the infectious  $j$ -individual making infectious contacts with the  $k$ -individuals in each row, we instead compute the right eigenvector corresponding to  $R_0$ . That is, we compute the positive right eigenvector  $a$  normalized with the entries adding up to 1 such that  $Na = R_0a$  where  $N$  is our next generation matrix. This eigenvector is given in Table 20 in Appendix A and the computed values of  $a_k/\pi_k$  can be found in the same table.

According to Table 20 we now vaccinate groups in the following order

1. Females aged 80+ years
2. Males aged 80+ years
3. Females aged 20-39 years
4. Males aged 20-39 years
5. Females aged 0-19 years
6. Males aged 0-19 years
7. Females aged 40-59 years
8. Males aged 40-59 years
9. Males aged 60-79 years
10. Females aged 60-79 years

where we vaccinate the entire group when possible and iteratively move on to give any remaining vaccines to the next group in the list, if the proportion



of vaccines is greater than the proportion of individuals in the former group. The fractions vaccinated using this strategy when different fractions of the whole population can be vaccinated are found in Table 8 and the results when a perfect vaccine is used can be found in Table 9. The results for the all-or-nothing vaccine are found in Table 10 and the ratio of the final sizes compared to the unvaccinated case are presented in Table 11.

Table 8: The fractions vaccinated by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated. The order of vaccinating individuals is determined by the computed  $a_k/\pi_k$  in Table 20.

$c$	Males					Females				
	0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
0.1	0	0	0	0	1	0	0.46	0	0	1
0.2	0	0.28	0	0	1	0	1	0	0	1
0.3	0	1	0	0	1	0.08	1	0	0	1
0.4	0	1	0	0	1	0.97	1	0	0	1
0.5	0.82	1	0	0	1	1	1	0	0	1
0.6	1	1	0	0	1	1	1	0.56	0	1
0.7	1	1	0.26	0	1	1	1	1	0	1

When 10% of the population is vaccinated with a perfect vaccine according to the above strategy none of the individuals aged 80+ years are infected and the final percentages of infected 60- to 79-year-olds are about 75% for both sexes. About 90% of the individuals younger than 60 years are infected except for the 20- to 39-year-old females, as only about 49.7% of this group is infected. Additionally, the highest percentage of deaths in this case is 4.67% for the 60- to 79-year-old males which significantly reduces the overall deaths as the most vulnerable 80+ years age group is completely protected from long-term illness and death. However, the final percentages of infected in the younger age groups are still high and the percentages of long-term ill 60- to 79-year-olds are not significantly affected as this group is yet to be vaccinated.

As we move from vaccinating 30% of the population to 40%, we note that the proportion of infected 0- to 19-year-old females decreases from about 79% to 2.01% as we vaccinate almost this whole group. This also decreases the percentages of infected in the remaining groups and especially for the 0- to 19-year-old males whose percentage is 68% of that in the unvaccinated case. The final fractions of dead and long-term ill decrease slightly for the unvaccinated individuals aged 40-59 and 60-79 years. As half of the population has been vaccinated, none of the age groups 20-39 years and 80+ years or the females younger than 20 years are infected and only about 5.7% of the 0- to 19-

year-old males are infected, which is 6% of the unvaccinated case and the remaining unvaccinated groups have about 62 – 66% of the infected in an unvaccinated population. Thus, this strategy is quite effective in reducing the spread if half of the population is vaccinated.

Nevertheless, the percentage of dead 60- to 79-year-old males is only reduced to below 3.5% when at least 50% of the population is vaccinated and the proportion of deceased females is not reduced significantly either until half the population is immune. However, the percentages were rather low to begin with and compared to the unvaccinated population the infections were reduced to 62% for this age group. Furthermore, it is also notable that the 60- to 79-year-olds remain unvaccinated for all values of  $c$  but the final fractions of infected are reduced from about 75% to about 24.8% for both sexes by vaccinating the other groups when  $c = 0.6$ . Although, about half of the 60- to 79-year-olds are still infected as  $c = 0.5$  and the reduction of infections is quite slow until we vaccinate at least half of the population. Moreover, when  $c = 0.7$  no one is infected as herd immunity is achieved for the perfect vaccine and no outbreak takes place, similar to when equal fractions were vaccinated in every group in the previous subsection.

Table 9: The final percentages of infected, recovered, dead and long-term ill within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with a perfect vaccine. The order of vaccinating individuals is determined by the computed  $a_k/\pi_k$  in Table 20.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Infected (%)	0.1	92.25	92.03	90.63	74.94	0.00	92.25	49.68	90.63	74.94	0.00
	0.2	89.83	61.69	86.51	70.56	0.00	89.83	0.00	86.51	70.56	0.00
	0.3	85.82	0.00	80.59	65.15	0.00	78.99	0.00	80.59	65.15	0.00
	0.4	63.67	0.00	71.60	57.24	0.00	2.01	0.00	71.60	57.24	0.00
	0.5	5.65	0.00	61.01	48.79	0.00	0.00	0.00	61.01	48.79	0.00
	0.6	0.00	0.00	28.71	24.83	0.00	0.00	0.00	12.75	24.83	0.00
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Recovered (%)	0.1	92.16	91.81	88.88	63.31	0.00	92.21	49.61	89.99	69.63	0.00
	0.2	89.75	61.54	84.83	59.61	0.00	89.80	0.00	85.89	65.57	0.00
	0.3	85.74	0.00	79.03	55.03	0.00	78.96	0.00	80.02	60.53	0.00
	0.4	63.61	0.00	70.21	48.36	0.00	2.01	0.00	71.09	53.19	0.00
	0.5	5.64	0.00	59.83	41.22	0.00	0.00	0.00	60.58	45.33	0.00
	0.6	0.00	0.00	28.15	20.97	0.00	0.00	0.00	12.66	23.07	0.00
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Dead (%)	0.1	0.0461	0.0460	0.2991	4.67	0.00	0.0277	0.0149	0.0906	2.61	0.00
	0.2	0.0449	0.0308	0.2855	4.40	0.00	0.0270	0.00	0.0865	2.46	0.00
	0.3	0.0429	0.00	0.2659	4.06	0.00	0.0237	0.00	0.0806	2.27	0.00
	0.4	0.0318	0.00	0.2363	3.57	0.00	0.0006	0.00	0.0716	1.99	0.00
	0.5	0.0028	0.00	0.2013	3.04	0.00	0.00	0.00	0.0610	1.70	0.00
	0.6	0.00	0.00	0.0947	1.55	0.00	0.00	0.00	0.0127	0.8640	0.00
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Long-term ill (%)	0.1	0.0369	0.1749	1.46	6.97	0.00	0.0092	0.0547	0.5619	2.70	0.00
	0.2	0.0359	0.1172	1.39	6.56	0.00	0.0090	0.00	0.5363	2.54	0.00
	0.3	0.0343	0.00	1.30	6.06	0.00	0.0079	0.00	0.4997	2.35	0.00
	0.4	0.0255	0.00	1.15	5.32	0.00	0.0002	0.00	0.4439	2.06	0.00
	0.5	0.0023	0.00	0.9823	4.54	0.00	0.00	0.00	0.3783	1.76	0.00
	0.6	0.00	0.00	0.4622	2.31	0.00	0.00	0.00	0.0790	0.8938	0.00
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Further implementing this strategy with the all-or-nothing vaccine when 10% of the population is vaccinated results in just under 10% of the individuals aged 80+ years getting infected for both sexes, and about 54% of the 20- to 39-year-old females getting infected. This already reduces the final percentages of dead people aged 80+ years to below 4% for both sexes. In addition, we note that for  $c = 0.4$ , each of the vaccinated groups has less than 10% infected and the remaining unvaccinated groups each has about 80% or less of the infected in an unvaccinated population, which could be reduced further. As 60% of the population is vaccinated only the 60- to 79-year-olds and the males aged between 40-59 years are left unvaccinated, and each group now has less than half infected. The final percentages of long-term ill and dead 60- to 79-year-olds are about half of that in the unvaccinated population and the percentages of long-term ill and dead are all under 4% for all groups.

Moreover, as 70% of the population is vaccinated, we have reduced the reproduction number to below 1 as no one is infected. Hence, we can protect the population and the vulnerable individuals if we vaccinate only 70%, even with an all-or-nothing vaccine of efficacy 90% where none of the 60- to 79-year-olds and only 26% of the 40- to 59-year-olds are vaccinated. This proves more efficient than vaccinating the same fraction of each group, as people were still infected when 70% of the population was vaccinated with the all-or-nothing vaccine.

Table 10: The final percentages of infected, recovered, dead and long-term ill within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with an all-or-nothing vaccine of efficacy 90%. The order of vaccinating individuals is determined by the computed  $a_k/\pi_k$  in Table 20.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Infected (%)	0.1	92.37	92.31	90.85	75.38	9.98	92.37	54.08	90.85	75.38	9.99
	0.2	90.27	65.04	87.29	71.57	9.97	90.27	8.68	87.29	71.57	9.98
	0.3	86.84	7.81	82.31	66.91	9.94	80.62	7.81	82.31	66.91	9.95
	0.4	69.47	6.93	75.37	60.62	9.88	8.92	6.93	75.37	60.62	9.89
	0.5	10.64	5.84	66.47	53.31	9.76	4.09	5.84	66.47	53.31	9.77
	0.6	2.14	3.63	42.32	35.58	8.88	2.14	3.63	21.15	35.58	8.89
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Recovered (%)	0.1	92.29	92.09	89.09	63.68	6.08	92.33	54.01	90.20	70.04	7.39
	0.2	90.19	64.89	85.60	60.47	6.07	90.23	8.67	86.68	66.51	7.38
	0.3	86.76	7.79	80.71	56.53	6.06	80.59	7.80	81.72	62.17	7.37
	0.4	69.40	6.91	73.91	51.21	6.02	8.91	6.92	74.84	56.33	7.32
	0.5	10.63	5.83	65.18	45.04	5.94	4.09	5.84	66.00	49.53	7.23
	0.6	2.14	3.63	41.50	30.06	5.41	2.14	3.63	21.00	33.06	6.58
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Dead (%)	0.1	0.0462	0.0462	0.2998	4.70	3.72	0.0277	0.0162	0.0908	2.62	2.53
	0.2	0.0451	0.0325	0.2881	4.46	3.72	0.0271	0.0026	0.0873	2.49	2.53
	0.3	0.0434	0.0039	0.2716	4.17	3.71	0.0242	0.0023	0.0823	2.33	2.52
	0.4	0.0347	0.0035	0.2487	3.78	3.68	0.0027	0.0021	0.0754	2.11	2.51
	0.5	0.0053	0.0029	0.2193	3.32	3.64	0.0012	0.0018	0.0665	1.86	2.48
	0.6	0.0011	0.0018	0.1397	2.22	3.31	0.0006	0.0011	0.0211	1.24	2.25
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Long-term ill (%)	0.1	0.0369	0.1754	1.46	7.01	0.1817	0.0092	0.0595	0.5633	2.71	0.0629
	0.2	0.0361	0.1236	1.41	6.66	0.1815	0.0090	0.0095	0.5412	2.58	0.0628
	0.3	0.0347	0.0148	1.33	6.22	0.1810	0.0081	0.0086	0.5103	2.41	0.0627
	0.4	0.0278	0.0132	1.21	5.64	0.1798	0.0009	0.0076	0.4673	2.18	0.0623
	0.5	0.0043	0.0111	1.07	4.96	0.1777	0.0004	0.0064	0.4121	1.92	0.0615
	0.6	0.0009	0.0069	0.6814	3.31	0.1617	0.0002	0.0040	0.1311	1.28	0.0560
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Table 11: The ratio of final sizes within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with a perfect or an all-or-nothing vaccine with 90% efficacy, compared to in an unvaccinated population. The order of vaccinating individuals is determined by the computed  $a_k/\pi_k$  in Table 20.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Perfect	0.1	0.99	0.97	0.98	0.95	0.00	0.99	0.53	0.98	0.95	0.00
	0.2	0.96	0.65	0.93	0.89	0.00	0.96	0.00	0.93	0.89	0.00
	0.3	0.92	0.00	0.87	0.83	0.00	0.85	0.00	0.87	0.83	0.00
	0.4	0.68	0.00	0.77	0.73	0.00	0.02	0.00	0.77	0.73	0.00
	0.5	0.06	0.00	0.66	0.62	0.00	0.00	0.00	0.66	0.62	0.00
	0.6	0.00	0.00	0.31	0.31	0.00	0.00	0.00	0.14	0.31	0.00
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
All-or-nothing	0.1	0.99	0.98	0.98	0.96	0.10	0.99	0.57	0.98	0.96	0.10
	0.2	0.97	0.69	0.94	0.91	0.10	0.97	0.09	0.94	0.91	0.10
	0.3	0.93	0.08	0.89	0.85	0.10	0.86	0.08	0.89	0.85	0.10
	0.4	0.74	0.07	0.81	0.77	0.10	0.10	0.07	0.81	0.77	0.10
	0.5	0.11	0.06	0.72	0.68	0.10	0.04	0.06	0.72	0.68	0.10
	0.6	0.02	0.04	0.46	0.45	0.09	0.02	0.04	0.23	0.45	0.09
	0.7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

#### 4.2.3 Vaccinating in descending order of vulnerability

According to Figure 3, the most vulnerable groups in terms of death and long-term illness are the males and females aged 80+ years as well as the 60- to 79-year-old males and females, followed by the 40- to 59-year-old males and females. The remaining groups have the same probability of dying, where the males are slightly more likely to die than the females. Thus, to reduce the percentages of dead and long-term ill we vaccinate groups in descending order of age, focusing on vaccinating males before females. We distribute the fractions  $c = 0.1, 0.2, \dots, 0.7$  of vaccines similarly as in Section 4.2.2, where the whole group is vaccinated when possible and any remaining vaccines are given to the group next in line until there are no more vaccines left.

The fractions of vaccinated in each group are shown in Table 12 and the results for the perfect vaccine are presented in Table 13, while the results for the all-or-nothing vaccine are presented in Table 14. The ratios of final sizes compared to an unvaccinated population are displayed in Table 15.

Table 12: The fractions vaccinated by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated. The individuals are vaccinated in descending order of age with males vaccinated before females.

$c$	Males					Females				
	0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
0.1	0	0	0	0.62	1	0	0	0	0	1
0.2	0	0	0	1	1	0	0	0	0.63	1
0.3	0	0	0.43	1	1	0	0	0	1	1
0.4	0	0	1	1	1	0	0	0.13	1	1
0.5	0	0	1	1	1	0	0	0.83	1	1
0.6	0	0.60	1	1	1	0	0	1	1	1
0.7	0	1	1	1	1	0	0.42	1	1	1

When vaccinating 10% of the population with the perfect vaccine none of those aged 80+ years are infected and only about 27.7% of the 60- to 79-year-old males are infected as these are the first to get vaccinated. Thus, none of the individuals 80+ years old die and less than 2% of the males and less than 2.6% of the females aged 60-79 years are deceased. When 30% of the population is vaccinated there are no infected individuals above the age of 60 years and hence the highest percentage of dead individuals is about 0.16% for the 40- to 59-year-old males and the highest percentage of long-term ill is just over 0.79% for this same group. Hence, already when 30% of the population is vaccinated the most vulnerable groups are protected and there are next to no cases of deaths and long-term illness. However, as only the 40- to 59-year-olds are vaccinated among the younger age groups, the final percentages of infected are still high for these groups. In fact, over 80% of both sexes of those younger than 20 years and almost 66% of the 20- to 39-year-old females are still infected even when 60% of the population has been vaccinated and about 26% of the 20- to 39-year-old males are infected.

As 70% of the population is vaccinated, we note that about 77.5% of both sexes aged 0-19 years are infected which is more than 80% of the infections in this group in an unvaccinated case and about 26.7% of the 20- to 39-year-old females are infected, even as 42% of this group has been vaccinated. We also see that none of the individuals younger than 20 years were vaccinated for any value of  $c$  and that herd immunity was not achieved, as we still have infections even after vaccinating more than the critical vaccination coverage for uniform vaccination with a perfect vaccine. Therefore, while this strategy managed to protect the most vulnerable well by reducing the final fractions of deaths and long-term ill for these groups, it is ineffective in minimizing the spread even for a perfect vaccine.

Table 13: The final percentages of infected, recovered, dead and long-term ill within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with a perfect vaccine. The individuals are vaccinated in descending age order with males vaccinated before females.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Infected (%)	0.1	93.03	93.80	91.68	27.70	0.00	93.03	93.80	91.68	72.53	0.00
	0.2	92.56	92.91	90.49	0.00	0.00	92.56	92.91	90.49	23.02	0.00
	0.3	91.35	90.57	49.11	0.00	0.00	91.35	90.57	86.40	0.00	0.00
	0.4	89.32	86.18	0.00	0.00	0.00	89.32	86.18	67.84	0.00	0.00
	0.5	87.00	80.40	0.00	0.00	0.00	87.00	80.40	11.13	0.00	0.00
	0.6	82.71	26.16	0.00	0.00	0.00	82.71	65.97	0.00	0.00	0.00
	0.7	77.54	0.00	0.00	0.00	0.00	77.54	26.72	0.00	0.00	0.00
Recovered (%)	0.1	92.95	93.58	89.90	23.40	0.00	92.99	93.67	91.03	67.40	0.00
	0.2	92.48	92.69	88.73	0.00	0.00	92.52	92.78	89.84	21.39	0.00
	0.3	91.27	90.36	48.15	0.00	0.00	91.31	90.45	85.79	0.00	0.00
	0.4	89.24	85.97	0.00	0.00	0.00	89.29	86.06	67.36	0.00	0.00
	0.5	86.92	80.21	0.00	0.00	0.00	86.96	80.29	11.05	0.00	0.00
	0.6	82.64	26.10	0.00	0.00	0.00	82.68	65.88	0.00	0.00	0.00
	0.7	77.47	0.00	0.00	0.00	0.00	77.51	26.68	0.00	0.00	0.00
Dead (%)	0.1	0.0465	0.0469	0.3025	1.73	0.00	0.0279	0.0281	0.0917	2.52	0.00
	0.2	0.0463	0.0465	0.2986	0.00	0.00	0.0278	0.0279	0.0905	0.8013	0.00
	0.3	0.0457	0.0453	0.1620	0.00	0.00	0.0274	0.0272	0.0864	0.00	0.00
	0.4	0.0447	0.0431	0.00	0.00	0.00	0.0268	0.0259	0.0678	0.00	0.00
	0.5	0.0435	0.0402	0.00	0.00	0.00	0.0261	0.0241	0.0111	0.00	0.00
	0.6	0.0414	0.0131	0.00	0.00	0.00	0.0248	0.0198	0.00	0.00	0.00
	0.7	0.0388	0.00	0.00	0.00	0.00	0.0233	0.0080	0.00	0.00	0.00
Long-term ill (%)	0.1	0.0372	0.1782	1.48	2.58	0.00	0.0093	0.1032	0.5684	2.61	0.00
	0.2	0.0370	0.1765	1.46	0.00	0.00	0.0093	0.1022	0.5610	0.8289	0.00
	0.3	0.0365	0.1721	0.7906	0.00	0.00	0.0091	0.0996	0.5357	0.00	0.00
	0.4	0.0357	0.1637	0.00	0.00	0.00	0.0089	0.0948	0.4206	0.00	0.00
	0.5	0.0348	0.1528	0.00	0.00	0.00	0.0087	0.0884	0.0690	0.00	0.00
	0.6	0.0331	0.0497	0.00	0.00	0.00	0.0083	0.0726	0.00	0.00	0.00
	0.7	0.0310	0.00	0.00	0.00	0.00	0.0078	0.0294	0.00	0.00	0.00

Moving on to the all-or-nothing vaccine, we observe that almost 10% of both sexes of the group aged 80+ years are infected and that 32.5% of the males aged 60-79 years are infected when 10% of the population is vaccinated, corresponding to about 40% of the infected 60- to 79-year-old males in an unvaccinated population. The unvaccinated groups have over 90% infected, except for the 60- to 79-year-old females who have about 73% infected. As almost all the most vulnerable groups are vaccinated already, the percentages of deaths and long-term illness are minimal and below 4% for all types and hence the vulnerable are already quite well protected.

Nonetheless, as in the case of the perfect vaccine the final fractions of infected in the younger age groups are still high and as half of the population has been vaccinated, we still have over 80% infected for both sexes of the ages 0-19 and 20-39 years. Actually, when 70% of the population is vaccinated there are still about 80% infected for both sexes younger than 20 years

and just below 33% infected 20- to 39-year-old females, while the remaining vaccinated groups have less than 7% infections. The final percentages of long-term ill and dead are also minimal. Therefore, this strategy is highly effective in protecting the vulnerable, but much less effective in reducing the spread for the all-or-nothing vaccine as well.

Table 14: The final percentages of infected, recovered, dead and long-term ill within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with an all-or-nothing vaccine of efficacy 90%. The individuals are vaccinated in descending age order with males vaccinated before females.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Infected (%)	0.1	93.07	93.87	91.77	32.50	9.97	93.07	93.87	91.77	73.23	9.98
	0.2	92.64	93.06	90.69	6.47	9.89	92.64	93.06	90.69	27.77	9.91
	0.3	91.56	91.00	53.27	5.65	9.71	91.56	91.00	87.11	5.65	9.75
	0.4	89.79	87.26	7.99	4.81	9.34	89.79	87.26	70.63	4.81	9.44
	0.5	87.74	82.35	7.02	3.91	8.60	87.74	82.35	17.60	3.91	8.81
	0.6	84.09	32.22	5.82	3.06	7.59	84.09	70.50	5.82	3.06	7.94
	0.7	79.42	5.27	4.50	2.28	6.30	79.42	32.96	4.50	2.28	6.81
Recovered (%)	0.1	92.98	93.64	89.99	27.45	6.07	93.03	93.73	91.11	68.04	7.38
	0.2	92.55	92.83	88.93	5.46	6.03	92.60	92.92	90.04	25.80	7.33
	0.3	91.47	90.78	52.24	4.78	5.91	91.52	90.88	86.49	5.25	7.22
	0.4	89.71	87.05	7.84	4.06	5.69	89.75	87.14	70.13	4.47	6.98
	0.5	87.66	82.15	6.88	3.30	5.24	87.71	82.24	17.47	3.63	6.52
	0.6	84.01	32.14	5.71	2.59	4.62	84.05	70.40	5.78	2.84	5.88
	0.7	79.35	5.25	4.41	1.93	3.84	79.39	32.91	4.46	2.12	5.04
Dead (%)	0.1	0.0465	0.0469	0.3028	2.02	3.72	0.0279	0.0282	0.0918	2.55	2.53
	0.2	0.0463	0.0465	0.2993	0.4028	3.69	0.0278	0.0279	0.0907	0.9663	2.51
	0.3	0.0458	0.0455	0.1758	0.3521	3.62	0.0275	0.0273	0.0871	0.1967	2.47
	0.4	0.0449	0.0436	0.0264	0.2996	3.48	0.0269	0.0262	0.0706	0.1674	2.39
	0.5	0.0439	0.0412	0.0232	0.2435	3.21	0.0263	0.0247	0.0176	0.1360	2.23
	0.6	0.0420	0.0161	0.0192	0.1907	2.83	0.0252	0.0212	0.0058	0.1065	2.01
	0.7	0.0397	0.0026	0.0148	0.1421	2.35	0.0238	0.0099	0.0045	0.0794	1.73
Long-term ill (%)	0.1	0.0372	0.1783	1.48	3.02	0.1815	0.0093	0.1033	0.5689	2.64	0.0629
	0.2	0.0371	0.1768	1.46	0.6013	0.1801	0.0093	0.1024	0.5622	0.9996	0.0624
	0.3	0.0366	0.1729	0.8576	0.5257	0.1767	0.0092	0.1001	0.5401	0.2035	0.0615
	0.4	0.0359	0.1658	0.1287	0.4473	0.1699	0.0090	0.0960	0.4379	0.1731	0.0595
	0.5	0.0351	0.1565	0.1130	0.3635	0.1566	0.0088	0.0906	0.1091	0.1407	0.0555
	0.6	0.0336	0.0612	0.0937	0.2846	0.1382	0.0084	0.0776	0.0361	0.1102	0.0500
	0.7	0.0318	0.0100	0.0724	0.2121	0.1147	0.0079	0.0363	0.0279	0.0821	0.0429



Table 15: The ratio of final sizes within each group by age groups in years and sex for different allocations of vaccines when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the population can be vaccinated with a perfect or an all-or-nothing vaccine with 90% efficacy, compared to in an unvaccinated population. The individuals are vaccinated in descending age order with males vaccinated before females.

	$c$	Males					Females				
		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Perfect	0.1	1.00	0.99	0.99	0.35	0.00	1.00	0.99	0.99	0.92	0.00
	0.2	0.99	0.98	0.98	0.00	0.00	0.99	0.98	0.98	0.29	0.00
	0.3	0.98	0.96	0.53	0.00	0.00	0.98	0.96	0.93	0.00	0.00
	0.4	0.96	0.91	0.00	0.00	0.00	0.96	0.91	0.73	0.00	0.00
	0.5	0.93	0.85	0.00	0.00	0.00	0.93	0.85	0.12	0.00	0.00
	0.6	0.89	0.28	0.00	0.00	0.00	0.89	0.70	0.00	0.00	0.00
	0.7	0.83	0.00	0.00	0.00	0.00	0.83	0.28	0.00	0.00	0.00
All-or-nothing	0.1	1.00	0.99	0.99	0.41	0.10	1.00	0.99	0.99	0.93	0.10
	0.2	0.99	0.99	0.98	0.08	0.10	0.99	0.99	0.98	0.35	0.10
	0.3	0.98	0.96	0.58	0.07	0.10	0.98	0.96	0.94	0.07	0.10
	0.4	0.96	0.92	0.09	0.06	0.09	0.96	0.92	0.76	0.06	0.09
	0.5	0.94	0.87	0.08	0.05	0.09	0.94	0.87	0.19	0.05	0.09
	0.6	0.90	0.34	0.06	0.04	0.08	0.90	0.75	0.06	0.04	0.08
	0.7	0.85	0.06	0.05	0.03	0.06	0.85	0.35	0.05	0.03	0.07

#### 4.2.4 The effect on $R_0$

After vaccination with the perfect vaccine, we see from Table 16 that for  $c = 0.1, 0.2, 0.3$ , vaccinating those contributing the most to the spread in the early stages first reduces  $R_0$  slightly more than when the vulnerable individuals are vaccinated first, but vaccinating the same fraction of individuals reduces  $R_0$  the most. These values are quite similar, though, and this trend continues for  $c = 0.5, 0.6$ , but for  $c = 0.4$  and  $c = 0.7$  we observe that the strategy of vaccinating those most socially active in the early stages seems to reduce  $R_0$  the most. Therefore, in order to reduce the spread when only 40% of the population can be vaccinated, it might be more effective to prioritize socially active individuals. However, the differences between these values are quite small, especially for when  $c = 0.7$  and in this case, we further observe that  $R_v < 1$  for both strategies, meaning that the outbreak will end which is shown by the final sizes for these strategies in the previous sections where we had no cases of infections for the perfect vaccine in Table 5 and Table 9.

In addition to this, we recognize that vaccinating the vulnerable individuals first does not result in  $R_v < 1$  even as 70% of the population is vaccinated. This is in line with the results in Table 13 as we still have a high percentage of infected among the younger age groups when the maximum percentage of vaccinated in the population is reached. This could seem odd as we vaccinate the group aged 80+ years first which is the group with the

otherwise highest percentage of infected, yet  $R_0$  is not substantially reduced since  $R_v$  is still above 2 for  $c = 0.6$ . However, this could be explained by how this group only constitutes a small portion of the population and by how these individuals have the most contacts with their own group and the older age groups, as seen in the contact matrix in Table 19 in Appendix A. The values of  $\beta_{jk}$ , which is proportional to the contact rate, for the infectious males aged 80+ years are below 0.3 for all contacted age groups younger than 80 years of both sexes, while the values are greater than 2 for both sexes of the contacted individual aged 80+ years. The same trend is observed for the infectious females of this age group, where the values for all contacted individuals younger than 80 years are below 0.5, while the values for the contacted males and females older than 79 years are almost 4.9.

This suggests that those in the 80+ years age group mainly infect individuals in their own age groups and that the infection stays within this group instead of spreading through interactions with other types, which is reasonable considering that many aged 80+ years might be accommodated in care homes or similarly where interactions mainly occur with other residents from the same age groups, or the staff who are likely to be part of the 20-39 and 40-59 years age groups. Thus, vaccinating people older than 79 years protects solely this group whereas the number of infections of the other groups is not as significantly affected.

Moreover, this could also explain why vaccinating the same fraction of individuals and vaccinating those of higher probability of becoming infected in the early stages have a similar effect in reducing the spread, as illustrated by the values of  $R_v$  in Table 16. Since the people aged 80+ years are the first to get vaccinated in the latter strategy and the groups ending up with the highest percentages of infected in the unvaccinated case are not fully or mostly vaccinated until at least half of the population has been vaccinated, it seems to be slightly more effective to vaccinate the same fraction of each group if fewer vaccines are accessible such that less than 40% of the population can be vaccinated.

Similarly, this might be the reason why vaccinating the socially active first reduces  $R_0$  more when  $c = 0.4$ , as this is when almost all the 0- to 19-year-old females are vaccinated and this group has one of the highest percentages of infected when the outbreak occurs in an unvaccinated population. Additionally, when  $c = 0.5$  the strategies have the same values of  $R_v$  for the all-or-nothing vaccine, as vaccinating the same fraction of all groups includes the 40- to 59-year-olds who have a high percentage of infected in the outbreak with fully susceptible individuals and these are unvaccinated still for the other strategy until when  $c = 0.6$  and 56% of these females are vaccinated, causing  $R_v$  to be lower for the all-or-nothing vaccine. As  $c = 0.7$  only the least socially active 60- to 79-year-olds are left unvaccinated when prioritizing the socially active individuals and 26% of the 40- to 59-year-old males are vaccinated with the rest of the groups fully vaccinated, resulting

in most to all groups otherwise entirely infected being immune. As these younger groups also have more interactions with each other and the group aged 80+ years as seen in the contact matrix, we have reached herd immunity when parts of or all of these more active groups are immune before the outbreak and hence no outbreak occurs.

Furthermore, we note that the differences in the reduction of  $R_0$  when  $c$  is greater than 0.4 are more significant for the all-or-nothing vaccine, suggesting that in a more realistic setting and with more vaccines available it is more effective to vaccinate those most likely to get infected in the early stages than to vaccinate the population uniformly, whereas when a perfect vaccine can be assumed these differences are less prominent and influential. This could be interpreted as there being more individuals still making many infectious contacts in the groups vaccinated by the less efficacious vaccine through uniform vaccination than when allocating vaccines first to the entire group of those getting infected early on, thus preventing these groups from making further infectious contacts and protecting the rest of the population.

Table 16: The effective reproduction number  $R_v$  when a fraction  $c = 0.1, 0.2, \dots, 0.7$  of the whole population is vaccinated for different allocation strategies including vaccinating the same fraction of each group, prioritizing those most likely to get infected in the early stages of the outbreak, as well as vaccinating in descending order of vulnerability.

		$c$						
		0.1	0.2	0.3	0.4	0.5	0.6	0.7
Perfect	Same fraction	2.60	2.31	2.02	1.73	1.44	1.15	0.87
	Socially active	2.64	2.37	2.13	1.67	1.50	1.17	0.86
	Vulnerable	2.79	2.74	2.57	2.36	2.22	2.02	1.89
All-or-nothing	Same fraction	2.63	2.37	2.11	1.85	1.59	1.33	1.07
	Socially active	2.66	2.41	2.19	1.77	1.59	1.28	0.98
	Vulnerable	2.80	2.75	2.59	2.40	2.26	2.07	1.93

## 5 Conclusions

From the results it is clear that the strategy of vaccinating those most vulnerable first reduces the cases of deaths and long-term illness the most overall, hence protecting the vulnerable individuals more than the other strategies and this holds for when  $c = 0.1, 0.2, \dots, 0.7$  for both the perfect and the all-or-nothing vaccine. However, it is the least effective strategy in reducing the overall spread for all values of  $c$  and for both vaccines as the younger age groups are not prioritized, and these groups contribute most to the spread in the unvaccinated case, together with the people aged 80+ years. Instead,

when less than 40% of the population can be vaccinated it would be more useful to implement uniform vaccination or focus on those contributing most to the early spread in order to decrease the infections in the population.

When 40% or more are vaccinated it is more effective to vaccinate the socially active individuals first with the all-or-nothing vaccine instead of using uniform allocation, and it is even possible to achieve herd immunity and preventing the outbreak if 70% of individuals are vaccinated this way. This is significant as one would expect that uniform vaccination is less effective overall, and since the all-or-nothing vaccine can reduce  $R_0$  below 1 when vaccinating the socially active first. When assuming a perfect vaccine, the differences are however less distinguished and both strategies prevent the outbreak for the highest fraction of vaccinated individuals, suggesting that these strategies have similar effectiveness in reducing the spread.

Nevertheless, vaccinating the same fractions of each type with the perfect and all-or-nothing vaccines resulted in high fatality rates for all values of  $c$  as the 60- to 79-year-olds and those aged 80+ years were not all vaccinated, causing these more vulnerable groups to be less protected than when the individuals contributing most to the spread were vaccinated primarily. This since the females and males older than 79 years are the most likely to get infected early on and simultaneously have the highest mortality rates. Therefore, in order to reduce the spread and concurrently protect those at risk of dying it would be more effective to vaccinate in descending order of contribution to the spread at the early stages. Although the percentages of long-term ill 60- to 79-year-olds are slightly higher for this strategy, it is arguably still more practical as there is no dramatic difference between the final sizes of long-term ill for these strategies, yet vaccinating the socially active first reduces the amount of deaths and infections substantially more.

To summarize, if the purpose of vaccination is to reduce the number of deaths and cases of long-term illness it is evidently the most advantageous to vaccinate those most vulnerable first, whereas if the purpose is to reduce the spread it is more effective to either vaccinate uniformly or in descending order of contribution to the early spread. Interestingly enough, for lower numbers of available vaccines it seems like vaccinating the same fraction is slightly more effective, while when a larger proportion of the population is vaccinated it is more effective to use the other strategy and the reduction of the spread is more distinguishable for the all-or-nothing vaccine than the perfect vaccine. However, to reduce both the infections and the fatality rates, it is the most effective to vaccinate those with a higher probability of getting infected early in the outbreak for all values of  $c$ , as this reduces the risk of a large outbreak the most.

## 6 Discussion

As the results in this thesis rely on data and on mathematical models simplifying reality, it is essential to take into account the assumptions made with such an approach. For instance, the extended multi-type SEIRLD model neglects births and deaths through assuming a constant population and while some individual heterogeneities are accounted for by dividing the population into types by age groups and sex with varying contact rates, there could be differences in individual susceptibility due to for example short-term antibodies or natural predisposition to becoming infected, violating the assumption that all unvaccinated individuals are susceptible. Additionally, realistically most individuals have high rates of interactions with certain individuals at their workplace, school, household or similar as these places are regularly visited and usually the same people are encountered there. However, this model does not take this into account and doing so could be a possible improvement of the applicability to a real-life setting.

In addition, the model assumes constant contact rates over time whereas in reality people may be more socially active and thus more susceptible to infection around holiday seasons and due to other seasonalities, however as we are mainly interested in the final size of the outbreak which is independent of the development of the outbreak over time this is not of major concern. The model also assumes that all Poisson processes, choices of contacts and durations are independent and while this may not be realistic it greatly simplifies computations and in particular the derivation of the final size equation. Moreover, the SEIRLD model includes the exposed state of being latently infected which is realistic but does not impact the results due to the aforementioned independence of past states.

Furthermore, we also suppose that recovery and vaccination induce life-long full immunity to the disease, thus rejecting the possibility of reinfection through for instance viral mutations, short-term antibodies or vaccines having short-term effectiveness. In addition to this, some vaccines may only reduce the probability of getting infected instead of protecting the individual fully and one such vaccine is the leaky vaccine mentioned in Section 2.3.2. Thus, a possible extension is to compare the outcomes when a leaky vaccine is used since in this case vaccinated individuals are not completely immune and could still contribute to the spread. One could also possibly combine the all-or-nothing and leaky vaccine to get an all-or-leaky vaccine, such that vaccinated individuals are either fully immune or the risk of infection is reduced by the efficacy of the vaccine, or to get a leaky-or-nothing vaccine where some people are not protected at all from the disease.

Other assumptions made about the vaccines include that all the available vaccines are used and hence that people do not reject the opportunity of getting vaccinated which may occur in real-life and that vaccination is the sole preventive measure used. Thereby assuming that no lockdowns,

quarantines, social distancing or even masks are used which are all widely implemented strategies across countries during the ongoing COVID-19 pandemic. However, although assuming that vaccination is used exclusively of other preventive measures is unrealistic in relation to the current pandemic, it allows for comparison between different allocations and types of vaccines without the interference of other simultaneous strategies. Therefore, the isolation of this preventive measure is a strength of the method.

Moreover, it is also important to consider that the results could have been impacted by the assumptions made in order to compensate for unavailable data, such as when transforming the contact matrix to our age groups and computing the probabilities of death, long-term illness and recovery. We also used data on social contacts relevant to disease transmission in Finland in 2008 and the reported number of COVID-19 cases in the Stockholm region in Sweden. Consequently, we assume that people in Finland over a decade ago have similar contact patterns as people in Stockholm during the COVID-19 outbreak where additional restrictions have been implemented other than vaccination. However, as Sweden did not introduce severe restrictions and the vaccination process was still in its early stages when the data were retrieved, the data are still useful for computing the relative probabilities of dying, becoming long-term ill and recovering for each type of individual. Even so, we do assume that the long-term ill individuals do not recover and that they are represented by those admitted into hospitals for a period of four days or longer which does not include those who were not admitted into hospital and also only includes the reported cases. Similarly, the data on infections only include the reported cases and the reporting procedure has changed throughout the epidemic; consequently, the data may not be representative of the true number of cases.

Furthermore, other vaccination strategies could be investigated as well and as we took into consideration the combined the risk of becoming long-term ill or dying when vaccinating in descending order of vulnerability, this could be further extended by a separate strategy to vaccinate those with a higher probability of becoming long-term ill, thus further distinguishing between risk groups. We also chose to prioritize vaccinating the males as opposed to the females in this strategy when the probabilities of dying were equal for the sexes of the younger groups and we could also have divided the vaccines equally among both sexes, although this may not have a profound influence on the results considering the equal probabilities of dying and similar contact rates. However, the proportion of females and males is almost the same across age groups except for the people aged 80+ years, where about two thirds are females and this could have impacted the order of vaccination for this strategy, as evidently more of the fatalities in the 80+ years group are females considering this composition of the sexes.

Another possible extension is to determine the most advantageous strategy to employ in a population where the main goal is to prevent the intro-

duction of an outbreak, even if  $R_0 > 1$ . Doing this could protect vulnerable individuals by minimizing the probability that an introduction occurs, and given that  $R_0 > 1$  the probability of a major outbreak through the explosion of the branching process of infections is given by  $1 - \prod_k q_k^{m_k}$ , where  $m_k$  is the number of initially infectious  $k$ -individuals and  $q_k$  is the extinction probability of type  $k$  individuals, as in Andersson & Britton [1, p.54]. Hence, it would be of interest to minimize this probability through vaccination to prevent a large outbreak. Some examples of countries where  $R_0 > 1$  but the probability of introduction is low are Taiwan, New Zealand and Australia and if an introduction would occur in these countries other interventions than vaccination would be implemented to manage the outbreak.

During the writing process, a preprint was released by Sjödin, Rocklöv and Britton [12] where different vaccination strategies are evaluated for COVID-19 from December 1st 2020 to October 1st 2021 through a deterministic SEIR model with similar structure and characteristics to Sweden, in contrast to our use of a stochastic model. Similar to in this thesis, they also consider an all-or-nothing vaccine but with the slightly higher efficacy of 95% and in addition to that they assume that only 90% of people older than 19 years get vaccinated with strategies varying the transmission rates, if there is a delay of vaccine deliveries, whether antibody-positive individuals are down-prioritized or not and whether vaccination is in descending age order or not. As in this thesis, they consider fatalities and infections but in contrast they further differentiate between regular and critical care. Their model is also more applicable to reality, since vaccination occurs over time instead of before the outbreak and there are also more possible states an individual can be in, such as in self-quarantine at home and recovery at different locations.

Additionally, the results of the preprint suggest that the best strategy is to vaccinate in descending age order after risk groups have been vaccinated since even though ascending age order decreases transmission rates more, the strain on healthcare is greater than for vaccinating in descending age order as the 50+ year-olds are left unvaccinated until later on. The results in this thesis are partly in line with this, as prioritizing vulnerable people does not reduce the infections as much as vaccinating those most likely to get infected early on or even as vaccinating uniformly, but uniform vaccination does not reduce deaths and long-term illness adequately. Instead, vaccinating those most likely to get infected early reduces both the infections and fatalities sufficiently, as the highly infectious 80+ years group which is considered a risk group is vaccinated early on. However, this strategy vaccinates those younger than 40 years in descending order and those aged between 40-59 and 60-79 years in ascending order, which clearly is a mixture of both strategies mentioned in the preprint. Not only that, but we also consider vaccinating ages 0-19 years in contrast to the preprint and consequently we can thereby reduce infections by prioritizing this group as well, which may be unrealistic

considering only those aged 18+ are vaccinated against COVID-19. Thus, this may partly explain the differences in our results, and we also did not compare descending age order with ascending age order, but rather with the probability of early infection.

Furthermore, our results could also differ due to that we only consider cases of critical hospital care exceeding a period of 4 days to represent long-term illness, while they consider not only critical care but also regular care. Thus, an improvement of our study could be to consider additional cases of hospitalization and to distinguish between the status of care. Another major difference is that our results are based on the final sizes where the final fractions of dead and long-term ill are computed from the relative probabilities of these events occurring, whereas the preprint considers the effect of strategies on the development of the outbreak and the outcomes over time.

## Appendix

### A Tables

This appendix includes the tables showing the population distribution, the next generation matrix and the rescaled contact matrix based on the data from Finland in 2008, as well as the table presenting the fractions of individuals of each type along with the right eigenvector corresponding to  $R_0$  and the ratio between these two as mentioned in Section 4.2.2.

Table 17: Population distribution by sex and age group in years for data from Finland in 2008.

	Total	0-19	20-39	40-59	60-79	80+
Total males	2 611 653	625 136	672 313	764 654	476 134	73 416
Total females	2 714 661	599 203	639 548	757 637	553 337	164 936
Faction males	0.490	0.511	0.512	0.502	0.463	0.308
Fraction females	0.510	0.489	0.488	0.498	0.537	0.692



Table 18: Next generation matrix for data from Finland in 2008 where the columns indicate the sex and age group in years of the individual making the contacts and the rows indicate the sex and age group in years of the contacted individuals.

		Males					Females				
Age groups		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Males	0-19	0.960	0.250	0.182	0.064	0.014	0.920	0.238	0.180	0.076	0.034
	20-39	0.318	0.708	0.408	0.162	0.028	0.304	0.674	0.404	0.188	0.064
	40-59	0.318	0.434	0.658	0.186	0.036	0.306	0.412	0.650	0.216	0.082
	60-79	0.084	0.096	0.160	0.308	0.078	0.080	0.092	0.158	0.358	0.178
	80+	0.034	0.058	0.116	0.242	0.120	0.052	0.056	0.114	0.282	0.270
Females	0-19	0.920	0.240	0.176	0.062	0.014	0.882	0.228	0.174	0.072	0.032
	20-39	0.302	0.674	0.388	0.154	0.028	0.290	0.642	0.384	0.180	0.060
	40-59	0.316	0.430	0.650	0.184	0.036	0.302	0.410	0.646	0.214	0.082
	60-79	0.096	0.112	0.186	0.358	0.092	0.092	0.106	0.184	0.416	0.206
	80+	0.124	0.132	0.260	0.544	0.270	0.118	0.124	0.258	0.632	0.606

Table 19: Rescaled contact matrix for data from Finland in 2008 where the columns indicate the sex and age group in years of the individual making the contacts and the rows indicate the sex and age group in years of the contacted individuals.

		Males					Females				
Age groups		0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
Males	0-19	2.045	0.534	0.389	0.138	0.032	1.960	0.508	0.386	0.160	0.071
	20-39	0.630	1.404	0.807	0.322	0.057	0.604	1.335	0.799	0.375	0.127
	40-59	0.555	0.756	1.144	0.324	0.064	0.532	0.719	1.134	0.376	0.144
	60-79	0.233	0.271	0.446	0.862	0.221	0.223	0.258	0.442	1.001	0.496
	80+	0.603	1.060	2.105	4.388	2.179	0.957	1.008	2.086	5.099	4.894
Females	0-19	2.045	0.534	0.389	0.138	0.032	1.960	0.508	0.386	0.160	0.071
	20-39	0.630	1.404	0.807	0.322	0.057	0.604	1.335	0.799	0.375	0.127
	40-59	0.555	0.756	1.144	0.324	0.064	0.532	0.719	1.134	0.376	0.144
	60-79	0.233	0.271	0.446	0.862	0.221	0.223	0.258	0.442	1.001	0.496
	80+	0.999	1.060	2.105	4.388	2.179	0.957	1.008	2.086	5.099	4.894

Table 20: The fractions  $\pi_k$  of individuals in each group by sex and age groups in years in the whole population for data from Finland in 2008, the entries  $a_k$  of the right eigenvector and  $a_k$  divided by the fractions  $\pi_k$  of people of each type for  $k = 1, 2, \dots, 10$ .

	Males					Females				
	0-19	20-39	40-59	60-79	80+	0-19	20-39	40-59	60-79	80+
$\pi_k$	0.117	0.126	0.144	0.089	0.014	0.112	0.120	0.142	0.104	0.031
$a_k$	0.119	0.135	0.135	0.049	0.039	0.114	0.128	0.134	0.057	0.090
$a_k/\pi_k$	1.013	1.069	0.942	0.547	2.833	1.014	1.069	0.943	0.546	2.898

## B Code

The code used to compute the final size equations is inspired by the code used in Britton et al. [4] and is available at a public GitHub repository at <https://github.com/veraandersson/Thesis>. The data and the remainder of the code used to compute the fractions vaccinated in each group, the ratios of the outcomes compared to the unvaccinated scenario and  $R_v$  are available there as well.

## 7 References

- [1] ANDERSSON, H., AND BRITTON, T. *Stochastic epidemic models and their statistical analysis*, vol. 151. Springer Science & Business Media, 2012.
- [2] BALL, F., BRITTON, T., AND LYNE, O. Stochastic multitype epidemics in a community of households: estimation and form of optimal vaccination schemes. *Mathematical biosciences* 191, 1 (2004), 19–40.
- [3] BRITTON, T. Stochastic epidemic models: a survey. *Mathematical biosciences* 225, 1 (2010), 24–35.
- [4] BRITTON, T., BALL, F., AND TRAPMAN, P. A mathematical model reveals the influence of population heterogeneity on herd immunity to sars-cov-2. *Science* 369, 6505 (2020), 846–849.
- [5] BRITTON, T., PARDOUX, E., BALL, F., LAREDO, C., SIRL, D., AND TRAN, V. C. Stochastic epidemics in a homogeneous community. In *Stochastic epidemic models with inference*, T. Britton and E. Pardoux, Eds. Springer, 2019.
- [6] DUIJZER, E., VAN JAARSVELD, W., WALLINGA, J., AND DEKKER, R. The most efficient critical vaccination coverage and its equivalence with maximizing the herd effect. *Mathematical biosciences* 282 (2016), 68–81.
- [7] FERRANNA, M., CADARETTE, D., AND BLOOM, D. E. Covid-19 vaccine allocation: Modeling health outcomes and equity implications of alternative strategies. *Engineering* (2021).
- [8] FOLKHÄLSOMYNDIGHETEN (FOHM). Rekommendationer om prioriteringsordning för vaccination mot covid-19. <https://www.folkhalsomyndigheten.se/smittskydd-beredskap/utbrott/aktuella-utbrott/covid-19/vaccination-mot-covid-19/rekommendationer-for-vaccination-mot-covid-19/>. Last accessed on 2021-05-09. Original document in Swedish.

- [9] HEESTERBEEK, J., AND DIETZ, K. The concept of  $R_0$  in epidemic theory. *Statistica neerlandica* 50, 1 (1996), 89–110.
- [10] HELSINKI: STATISTICS FINLAND. Official statistics of finland (OSF): Population structure. [http://www.stat.fi/til/vaerak/meta\\_en.html](http://www.stat.fi/til/vaerak/meta_en.html). ISSN: 1797-5395. Last accessed on 2021-03-31.
- [11] MOSSONG, J., HENS, N., JIT, M., BEUTELS, P., AURANEN, K., MIKOLAJCZYK, R., MASSARI, M., SALMASO, S., TOMBA, G. S., WALLINGA, J., ET AL. Social contacts and mixing patterns relevant to the spread of infectious diseases. *PLoS Med* 5, 3 (2008), e74.
- [12] SJÖDIN, H., ROCKLÖV, J., AND BRITTON, T. Evaluating and optimizing covid-19 vaccination policies: a case study of sweden. *medRxiv* (2021).
- [13] SVENSKA INTENSIVVÅRDSREGISTRET (SIR). Ålder- och könsfördelning på vårdtillfällen med coronavirus. <https://portal.icuregswe.org/siri/report/corona.alderkon>. Last accessed on 2021-04-02. Original document in Swedish.
- [14] THE WORLD HEALTH ORGANIZATION (WHO). Covid-19 vaccines. <https://www.who.int/westernpacific/emergencies/covid-19/covid-19-vaccines>. Last accessed on 2021-05-09.
- [15] VÅRDGIVARGUIDEN REGION STOCKHOLM. Covid-19 – statistik till och med 2021-03-28, Region Stockholm. <https://vardgivarguiden.se/globalassets/kunskapsstod/smittskydd/statistik/covid-19/antal-fall.pdf>. Last accessed on 2021-04-01. Original document in Swedish.
- [16] WALLINGA, J., VAN BOVEN, M., AND LIPSITCH, M. Optimizing infectious disease interventions during an emerging epidemic. *Proceedings of the National Academy of Sciences* 107, 2 (2010), 923–928.