A counterfactual model of the 2020 COVID-19 Pandemic in the Netherlands.

Modelling the health outcomes if no public or private measures had been taken to combat the COVID-19 pandemic in the Netherlands.

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Abstract. In this paper, I model the health outcomes of the pandemic in the hypothetical scenario where no countermeasures are taken to limit the spread of COVID-19. This means no lockdown, no social distancing, no working from home, or any other organised response. However, this puts many with existing diseases at an additional risk, and places a heavy strain on healthcare capacity. I specifically model how these two factors affect the outcomes of this scenario.

Method. I estimate the number of infections in this hypothetical scenario with an SEIR model. I then estimate the number of deaths using data from the ICU monitoring body NICE, as well as the total years of life lost by calculating the remaining life expectancy for deaths based on age and comorbidity.

Results. My model estimates 279,619 deaths in this scenario (sensitivity range 138,275 - 365,338). The total loss of life years I estimate to be 3 million years (Range1 – 5 million). This would in turn translate to between 13,050 and 60,446 full lives lost (of 82.37 years each). The average remaining life years would be 10.7 years (range 7.8 – 13.6).

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Introduction

At the time of writing, many western countries are reaching the presumable end of the COVID-19 pandemic. With vaccination efforts increasing, public measures and lockdowns are gradually eased and lifted across the world. Hopefully the return to normalcy will soon reach all corners of the globe so people everywhere can pick up their normal lives again. With the "wartime" of the pandemic drawing to a close, it can be easy to forget the delicate balance between public health and private freedoms every government has had to protect.

There is a strange paradox that the most successful policies in combatting the pandemic have the unfortunate side-effect in that they reduce the public perception of how dangerous it truly was. Threats to public health which are successfully mitigated are easily overshadowed by other threats. While it is tempting to think of infectivity and mortality as inherent properties of the disease, they are also partially determined by human behaviour; as the population socially distances themselves, infections drop, and the quality and capacity of healthcare can have a strong impact on the mortality. In short, a pandemic could play out in many different ways, depending on how we, the people, respond to it.

In this thesis I model what would have happened in one of such possible responses, namely one where no individual or public measures are taken, not even from common sense. Contextually, this could be because nobody is aware of the disease, or perhaps people have simply resigned to the fact that the disease is there, similarly to the flu. Regardless, in this hypothetical scenario the government instigates no lockdowns or procedures, and people do not socially distance themselves; everybody continues their ordinary lives. In modelling this, I pose the question: *what are the health outcomes in this scenario? How many people would die as a result, and what is the associated loss of life years?*

This question is partially shaped by the public debate surrounding the pandemic. Lockdown-sceptics have posted claims that suggest that the number of deaths is inflated, or that the deaths do not represent a serious loss of potential life-years. For example, PolitiFact (2020) discussed one particularly popular social media post which claimed among other things that only 'bad healthcare and weak immune systems' cause COVID-19 deaths. Another website called VirusWaarheid (Virus Truth) propogates many articles along similar lines. One such article (2020) claims that only those with severe pre-existing morbidities, or aged above 70 die from the disease. A direct line from this article claims that 'even with these pumped up numbers (fatalities), the virus is no worse than a flu-season'.There is a lot to say about such claims, from the perspective of ethics and ableism (the notion that less healthy people are implicitly less valuable), the perspective from effective public governance, or techno-sociological of how such claims proliferate through social media. In this thesis, I will focus on estimating the health costs, e.g. the number of deaths and the resulting loss of life years, that could have resulted from an absence of public and private response. In this hypothetical worst case scenario, I will account for both the age of people who died, their comorbidities (concurrent diseases such as cancers, lung disease, etc), and the healthcare capacity.

Likewise, framing the pandemic around health care capacity is often found in news articles, and was implicitly placed on the centre stage from the earliest moments in the pandemic. In March 2020 the 'flattening the curve' strategy became the dominant ideology when containment of the virus was no longer possible. (NOS, 2020). This strategy was directly aimed at maintaining the *demand* for healthcare below the available *supply*. This was also accompanied with a public interest in how the available supply (of ICU beds) could be scaled up (Klaassen, 2021). Implicitly and explicitly, healthcare capacity was often an important indicator of the pandemic's dangers.

These factors are mostly selected by the public debate. At the start of the pandemic, governmental health officials have repeatedly stated the need to “flatten the curve” in order not to overwhelm the healthcare system. In modelling this healthcare capacity, I can estimate how much mortality increases if this capacity is exceeded. At the same time, by considering the comorbidities of patients this paper can be critically contrasted with such statements claiming that ‘only sick people die from COVID-19’ or that ‘it is no worse than the flu’.

This thesis is structured as follows. In the first section I discuss the other studies that have described the COVID-19 pandemic along the mortality, the loss of years of life lost (e.g. the hypothetical life potential), and the infectivity. The second section discusses my methodology and data selection. In this thesis I use a SEIR model to estimate the number of infections, from which I estimate mortality. I rely on reported data from healthcare organisations to estimate the probabilities of disease outcomes. In the third section I discuss the results of this model. First I will describe the number of deaths that occur according to the model, as well as the years of life lost (YLL). I also present the sensitivity of this model to comorbidity, healthcare capacity, and infectivity, among other things. Lastly, I present my conclusions in the fourth section. I will also discuss the limitations of this study, such as the restrictive assumptions that are in my model. I also discuss some broader policy implications in this section.

Background

In this paper, I attempt to estimate the number of deaths and the loss of life years that would have occurred in the Netherlands if no measures had been taken by the state or the public during the first wave of the COVID-19 pandemic. By looking at both the number of deaths, as well as the number of Years of Life Lost (YLL) this counterfactual should give some idea on what the true impact could have been. In this section, I discuss some other articles and studies that have looked at the number of deaths, as well as the number of infections. This background helps to decide on a good methodology to estimate the counterfactual. The health outcomes (e.g. deaths) come from the interplay between biological factors (how COVID for example interacts with existing diseases) and psycho-social factors (state policy, medical treatments, etc). For this reason, I will specifically discuss some articles that deal with such factors.

Estimating the number of deaths

Estimating the number of deaths is not an easy task. Even counting the number of actual deaths has proved difficult in the real world. There are different ways to define COVID-19 deaths, and different ways to count or estimate them. For example, the economist published an article that highlights this difficulty (The economist, 2021). The official reported deaths due to COVID-19 globally was over 3 million, but the reported excess mortality over the pandemic was over 4 million. Moreover, their own estimation of the excess mortality was over 10 million, roughly 2.5 times as much (The economist, 2021). This number however includes both direct and indirect deaths, i.e. deaths in patients who did not have COVID-19. Still, numbers like these can be hard to interpret, as nobody can be sure that such excess mortality is truly due to COVID, nor how many people would have died in that timespan without COVID. In the case of the Netherlands, the official death toll was 16,622 at the end of March. However, there is also an excess mortality over and

above the reported COVID-19 deaths (CBS, 2021). There is another pattern that is also important to note from this data. Periods with high excess mortality (observed deaths from any cause – expected deaths for that period) are followed by a period with less than expected mortality. This is true for the whole of Europe as well (EUROMOMO, 2021). This could mean that COVID may have 'hastened' mortality for some people that might otherwise have died weeks later. This is always difficult to account for in models, but it is one of the reasons why studies also look at YLL. Second, it also possible that official figures underreport COVID-19 deaths. This is also noted in other global studies, for example by I Arolas et al (2020), who speak of 'systemic undercounting'. This can be explained by inadequate testing materials, or time constraints in healthcare settings. The number of deaths however is crucial for determining the mortality rate of COVID-19.

One measure of reporting the mortality rate is the Case Fatality Rate (cfr). This is chance of death, conditional on being infected. It is calculated by dividing the number of deaths by the number of cases. Unsurprisingly, many studies attempted to estimate this rate in the early days of the pandemic, because it plays a crucial role in planning the governmental response. Verity et al (2020) discussed the cfr for all countries in April 2020. From their data, they estimated a global cfr between 0.87% and 9.26%. In the initial phases of the pandemic, the cfr was incredibly high, reaching as high as 14% in Italy and 11% in Spain for May 2020. Of course, both of these countries were experiencing exceptionally high first waves of the pandemic. The world average meanwhile was around 7% for this period, but is now around 2% (Ritchie, 2021). All this is to say that the cfr varies over time, as well as between countries.

This can be partially explained by how 'we' got better at identifying and treating COVID over time. Better treatments reduce the cfr by reducing the number of deaths, while better testing reduces the cfr by dividing the number of deaths with a greater number of cases. Sorci et al (2020) also discuss why the cfr

differs *between* countries by using an autoregressive model on different national variables. The results indicate that the cfr is strongly associated with population morbidities, such as cardiovascular disease, cancer, and chronic respiratory diseases. It was meanwhile negatively associated with the healthcare capacity. These same factors are also noted by Verity et al, as well as the demographic composition of countries. For example, countries with older populations, or with higher rates of smoking also frequently observed higher case fatality rates. In this thesis, I will primarily use rates based on the officially reported COVID-19 deaths, as these rates are generally reported with less uncertainty.

In the introduction, I have noted that some of the public debate centres around these factors as well, in the form of 'flattening the curve' and claims about the comorbidity levels of deaths. It is widely reported that comorbidity (secondary diagnoses) does play an important role in determine patient outcomes. For example, an article by the Economist (2021) discusses a list of 29 comorbidities that all the risk of hospitalisation, as well as the case fatality rate. The article noted that the added risk of comorbidities was similar for *both* the risk of death *and* the risk of hospitalisation. (For example, chronic heart conditions was the second strongest predictor for both hospitalisations and risk of death.) Such comorbidities could, depending on age and gender increase such risks by a factor of 1.5 to 5, Another article published in the Lancet by Bhaskaran et al (2021) provide further evidence, controlling for many other health and social factors such as BMI, Smoking, Ethnicity, and different comorbidities. Here, Malignancies, Diabetes, Respiratory disease, Cardiovascular disease, Kidney disfunctions, and autoimmune disorders are particularly noted as both very prevalent (occurring often in the observed COVID-19 patients) as well as significantly diminishing the patient outcome of COVID-19. However the bulk of the results are published as odds ratios, which cannot be easily quantified into more meaningful risks.

Ji et al (2020) note a different important factor which partly determines the cfr. The availability of healthcare resources, e.g. mediations, beds, and personnel, also has a substantial impact on mortality rates. This resource availability can vary strongly between and within countries, and is strongly associated with the overall affluence of the country. In the studies by I Arolas et al (2020, 2021) this can be observed as well. Namely, in more affluent countries (with presumedly better developed health care systems) median the age-at-death due to the disease lies primarily *above* 70 years. In many countries in the Latin Americas, Asia, and Africa, with less developed health care systems this is different. Here, significant portions of mortality (more than 60% in some cases) occurs in those *under* 55 years old. This could also be due to differences in the demographics the population, but I speculate that the health outcomes of younger individuals (around 55) is tied to the available healthcare. In simple terms, I expect that older patients have substantially lower chances for survival *irrespective* of available healthcare, and younger patient's survival rates are more responsive to the amount of available care they can receive. This would explain why in countries where there is presumably more healthcare scarcity, there are significantly more deaths at younger ages.

Based on this, I include both population morbidity and healthcare capacity in my models. Population morbidity however also plays an important role in estimating the years of life lost, as it lowers the life expectancy.

Years of Life Lost estimations

In order to calculate the YLL, we would need to estimate what the remaining lifespan would have been for each COVID-19 death. This is an often used measurement of the pandemic impact on a given region. Quast et al (2020) have done precisely this for the (actual) COVID-19 deaths in the USA, by taking the remaining life years per age-group and gender. Rommel et al (2021) used this

method as well for Germany. Both these studies draw the remaining life years from the Global Health Observatory (GHO) life tables for the respective countries. I Arolas et al (2021) use the same framework, using the observed deaths by COVID-19 to estimate the loss of life years, across a total of 81 countries. They have done an earlier study in 2020, where they compare the YLL for 42 countries. In their latest study, they have studied over 1,279,866 deaths, which are responsible for a total of 20,507,518 lost life years.

These studies have not taken pre-existing morbidities of patients into account. In many cases, this was not possible with the amount of available data. Rommel et al (2021) argue that these pre-existing morbidities are already implicitly applied in the average remaining life. This may however be insufficient in the case of COVID-19, as COVID deaths may substantially impact specific comorbidities. Hanlon et al (2020) do take the comorbidity of patients implicitly into account, albeit at a flat rate (in the form of a reduction on the life expectancy). They note that this only had a limited effect on the YLL; accounting for the comorbidity in patients and the resulting loss of remaining life expectancy reduces the average YLL per patient with one year. Devleesschauwer et al (2020) note that the method by Hanlon et al could be made more accurate by using life tables based on mortality risks to estimate the effect of comorbidity on the remaining life years. A study by Ferenci (2021) using such morbidity adjusted lifetables to estimate the impact of comorbidity, and finds an overall reduction in average YLL of about 2 years. Wouterse et al (2021) do model the specific impacts of comorbidities on mortality and loss of life years using this method. This study is aimed at the Dutch population, which makes it very applicable to this model. Their results indicate a larger impact of comorbidities: from an average of 8.72 years without accounting for comorbidity, 7.4 when adjusting for comorbidity, and 5.53 years when considering selective comorbidities in long-term care facilities where a lot of deaths take place.

While it may initially seem surprising, both Devleesschauwer et al and Green (2021) comment on finding very different average YLLs. In the selected literature I have discussed, the range is certainly considerable: I Arolas (2021) suggest the highest unadjusted average of 16 years, while Wouterse et al provide an adjusted average of 5.53 years. Green notes that the decision to include or omit different social and health related factors can create significant differences in the estimates. Adjusting for comorbidity also captures the implicit effect of healthcare which is apt to be different between countries. I would also add to this that this difference also captures the succes of healthcare. This is the paradox I have mentioned in the introduction: successful strategies lower the impact of the pandemic, and thus lower statistics that measure the 'seriousness' of the disease. Devleeschauwer et al meanwhile note that even when the same methodology is used, there are likely to be significant differences between the average YLLs of different cities within the same country. All this suggests that we should not be too quick in comparing YLLs directly. Small differences can have large impacts on the outcome, potentially voiding many insights that stem from a direct comparison.

Stichting NICE, reporting specifically on the situation in Dutch hospitals, have reported on 5 different comorbidities: lung disease, kidney disfunction, cardiovascular disease and malignancy, as well as immunodeficiency. Their report (2021) notes these comorbidities for their pronounced effect on mortality, as well as provide statistics on adjusted risk ratios and bed occupancy rates. While there are other comorbidities that have been noted (diabetes mellitus, obesity, and others) the impact of these is harder to quantify in a model, especially given the unclear comorbidity between these and other comorbidities. The report by NICE also highlights the key role of healthcare capacity, as patients with COVID-19 can stay on ICU wards upwards of 19 days. This is substantially longer than the non-COVID average of 6.3 days.

Predicting the counterfactual number of infections

This leads to the last piece of the puzzle, namely how many people would become infected in the absence of any public policy or private behavioural response to the pandemic. This counterfactual scenario where everyone carries on 'business as usual' determines primarily the number of infections, with disease outcomes such as deaths being the secondary outcomes. So I would argue that this is a natural place to start modelling the counterfactual; by modelling the number of infections.

While there are several mathematic models, delineating the different merits of each type of model is outside of the scope of this paper. However, out of several model types a SEIR model seems most suitable. This is a compartmental model that can estimate infection curves with relatively little data, which makes it well suited for simulating epidemics. It is also self-limiting by taking the amount of un-infected people into account (e.g. when there are not enough susceptible people left, the spread halts). It is also a predictive model, meaning that it is often applied to predict how large an infective disease can spread.

Certainly, these SEIR has been applied many times in the COVID-19 pandemic (Anderson et al, 2020; Fan et al, 2020; He et al, 2020; Tang et al, 2020). These studies all predicted how the pandemic would develop if no further actions are taken at the time of their model. Based on the model inputs, these models have predicted infection levels reaching between 60-95% of the population. Usually these models do not become reality, as further measures are generally taken to further limit the spread.

These models are also highly dependent on the initial inputs. Small changes in for example the time someone stays infectious, or how long the incubation takes all influence the outcome of such models. In the next section, I will discuss this methodology further.

Methodology

In this section, I will detail the methodology I employ to estimate a counterfactual of an unchecked spread of COVID-19. First, I will be discussing the SEIR model which produces the number of infections per day. From the number of infections, I estimate disease outcomes with reported mortality rates. In the second part of this chapter, I discuss how I take the number of infections and calculates the total number of deaths, and the resulting years of life lost. I will also discuss how I take comorbidities and the available healthcare capacity into account. Lastly I will discuss how data on comorbidities, hospitalizations, mortality, and life expectancy.

SEIR MODEL

$$
\begin{aligned}
S_{t+1} &= S_t - (S_t \cdot I_t \cdot \beta_{rate}) \\
E_{t+1} &= E_t + (S_t \cdot I_t \cdot \beta_{rate}) - (E_t \cdot f_{rate}) \\
I_{t+1} &= I_t + (E_t \cdot f_{rate}) - (I_t \cdot R_{rate}) \\
R_{t+1} &= R_t + (I_t \cdot R_{rate})
\end{aligned}
$$

$$
\begin{aligned}
\beta_{rate} &= rate\ at\ which\ Susceptibles\ are\ infected\ by\ Infected. \\
f_{rate} &= rate\ at\ which\ Exposed\ become\ infectious \\
R_{rate} &= rate\ at\ which\ Infected\ are\ removed
\end{aligned}
$$

Table 1: Mathematical structure of the SEIR MODEL

The SEIR Model – Predicting infections

As I discussed in the previous chapter, to estimate the number of infections (in the scenario where no individual or public preventative measures are taken) I will be using a SEIR model. In its simplest form, the SEIR model estimates the number of newly exposed on ($t + 1$) based on the number of infected at day t with rate β. These newly exposed individuals become infectious with rate f, which is simply the inverse of the incubation period. Finally, these infectious individuals stop being infected with rate R, which includes both those recovered and those who die as a result of the disease. Table 1 shows the set of equations that make up the model.

It is important to note one disadvantage of this method however. I have not been able to model 'asymptomatic' carriers. While the SEIR model allows for a latency period, effectively every infection is at least a symptomatic case.

It is worth noting that the β-rate in this model is closely tied to the basic reproductive number, or R_0. The reproductive number is typically calculated as follows:

$$R_0 = Time_{Infectious} \cdot Daily\ Contacts\ \cdot \varphi$$

The average probability that a given contact results in a successful disease transmission is typically denoted with φ. φ multiplied by the average daily contacts results in the β-rate for the SEIR model. Multiplying this by the average time an infectious individual remains infectious results finally in the R_0, e.g. the average number of infections a new case would infect in a given susceptible population.

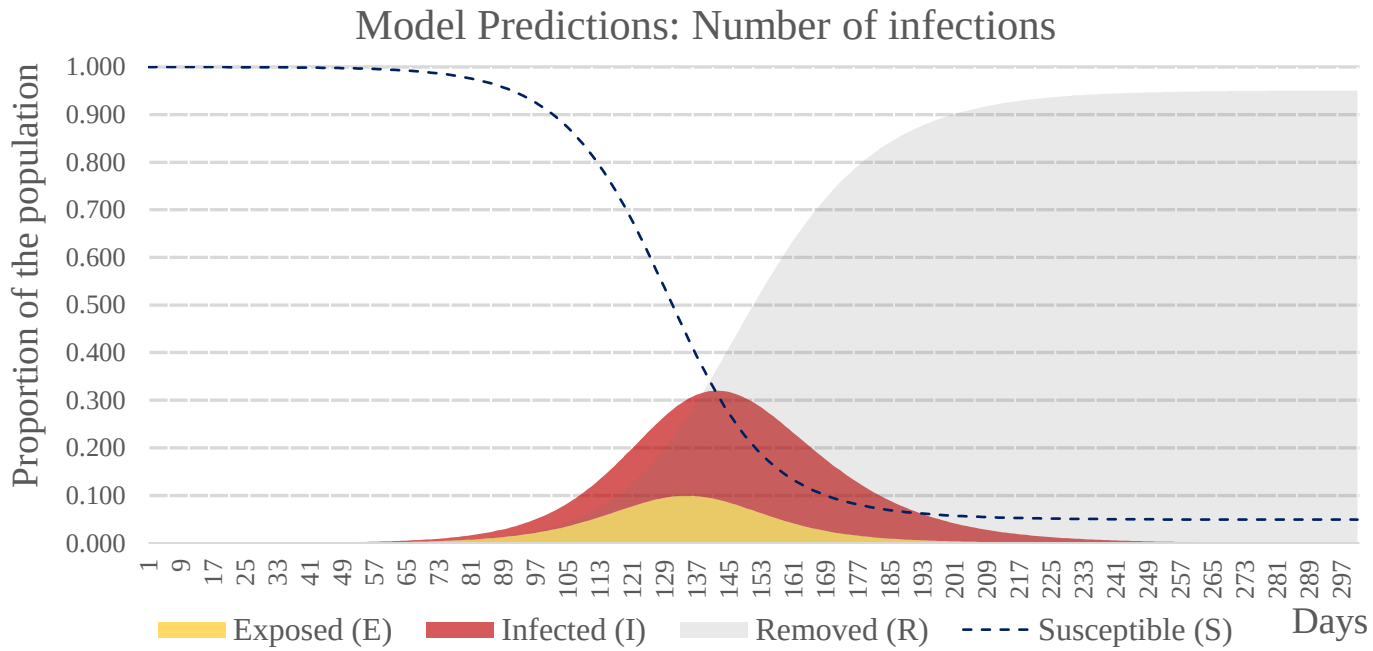
The most important inputs for the SEIR equations I have taken from the RIVM reports. The R_0 that was being reported in the early days of the pandemic ranged between 1 and 4 (RIVM, 2021), usually clustering somewhere around 2. This is likely a range that comes near the scenario in this paper, e.g. one where no public or individual measures have been taken. Reported estimates for the time until an infected individual becomes infectious (the incubation period) and the time they are then infectious vary between sources and times. A relatively early report (RIVM, 2020) gives the estimate of a 5.6 day incubation period (*f-rate*) and an infectious period of 10 days (δ). The average daily contacts (γ) that could allow for infection was pre-pandemic estimated at 16 per day (De Fada et al, 2020). Finally, for simplicity, I have rounded the country population to 17.000.000. These inputs for the baseline scenario are shown in the table below.

SEIR model inputs			description
γ	16		Average daily contacts
δ	10		Average days infectious
φ	0.0125		Prob. contact leads to infection
β	0.2	$(\gamma \cdot \varphi)$	Daily new infections per infected
f	0.1786	$(5.6)^{-1}$	Rate at which exposed become infectious
R	0.0641	$(15.6)^{-1}$	Rate at which infected get removed
Population	17,000,000		

Table 2: SEIR Model inputs (baseline scenario)

These inputs have pronounced effects on the 'shape' of the pandemic, e.g. the curve of the infections. The previous R_0 estimates from the RIVM data between 4 and 1.5 will not be fully discarded however. I will use these two values to also generate a range for the final results. An R_0 of 2 will however function as my 'best estimate' of what would have happened without measures. As can be seen in the bottom graph, around 95% of the population becomes infected in the first 300 days.

After this point, the virus cannot find enough susceptible individuals and dies out. This model provides not only the total number of infected people, but crucially also the timeframe of when they become infected. This timeframe ultimately determines if and when healthcare will be at capacity. In the next section I discuss how these infections are used to calculate concrete health outcomes for the population.



Graph 1: Number of infections predicted by SEIR model in the baseline scenario

Translating infections into YLL

The SEIR model estimates the total number of infections, but does in itself not specify how many deaths will occur. The main results of this paper will be the total number of deaths, and the number of years of life lost. To estimate this, I will need to transform the number of infections from the SEIR model into these outcomes. To do this, I assign each state a set of probabilities (based on empirical estimates). This method effectively 'sorts' the number of infections into states. Such states can be intermediary (e.g. admitted to a hospital) or final (e.g. recovery or death). Key here is the scalability of probabilities: if it is observed that 10 out of 1000 infected individuals get admitted to the hospital, we assume that that 1% also scales to a population of 17 million. That is essentially how the model works: on *t=100* there are 9.000 new cases. Of these cases, 13.5 will be admitted to the hospital, 2.9 to the ICU, and of these some will die depending on their age and comorbidities. This methodology of course misses a lot of nuances that determine disease outcomes and is only as good as the data that feeds into it.

Regardless of the initial simplicity of the method, the number of states in which infected individuals are sorted increases with the desired level of complexity. For example, to estimate the YLL, the number of infections need to be distributed across age groups. I have opted to group these ages as follows: 0-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, and >85. Then, to include the effects of comorbidities, I stratify every intermediary state for these. E.g. a substate for the number of infected at *t=x* who are: *Infected, aged between 40-44, have a prior lung condition, AND are sent to the ICU,* and so forth. The following page shows a diagram that shows the different states and their corresponding equations.

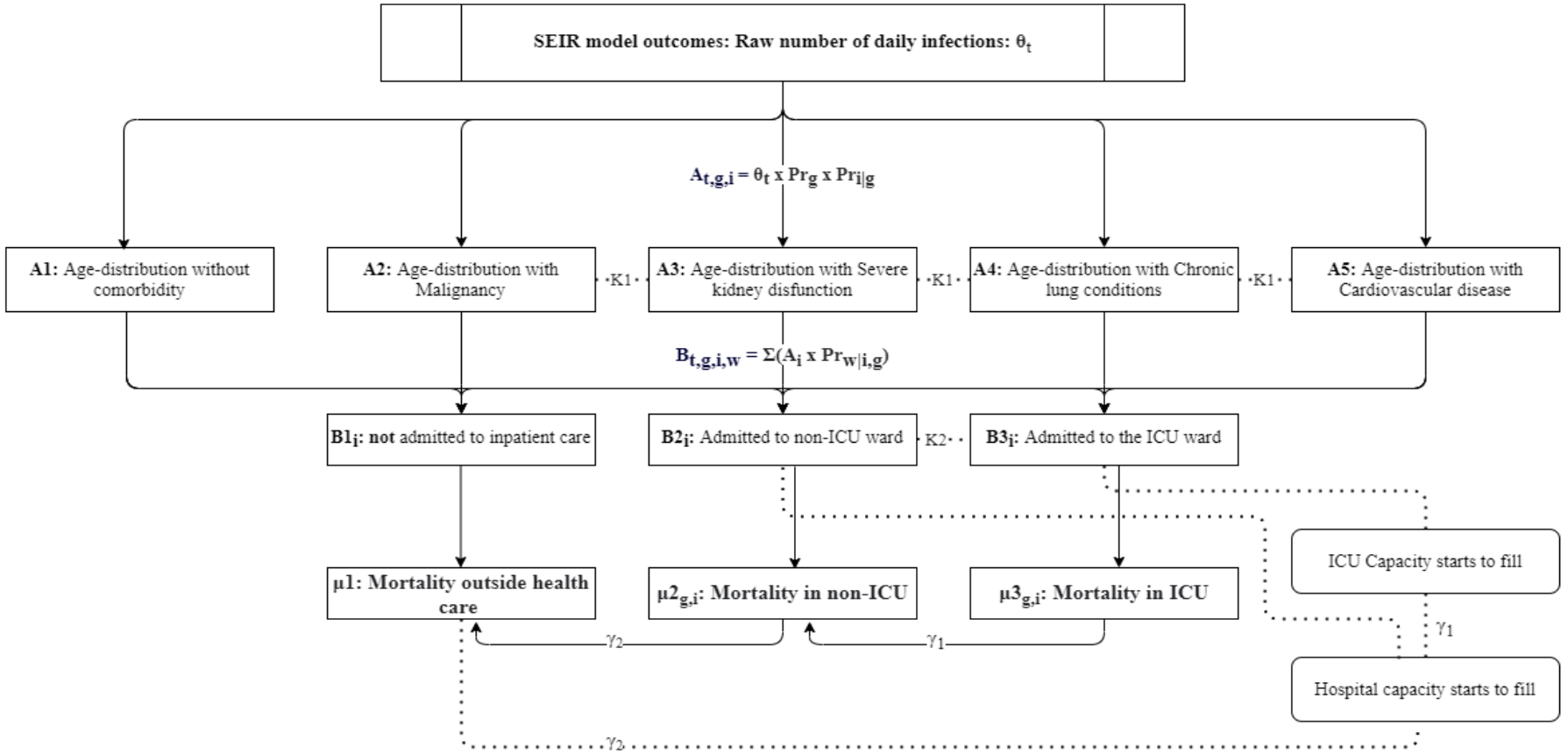


Diagram 1: Abstract representation of how infections are translated into number of deaths

Starting with the number of infections, I distribute these across age (*g*) and 4 different types of comorbidity (*i*): Malignancy, Kidney disfunction, Chronic lung conditions, and Cardiovascular disease. There is also some overlap in comorbidities, which I correct for with K1. This correction (as well as K2) is a flat rate correction that I apply to reduce 'double counting', e.g. overestimating the total morbidity level of the population. Additionally, this group with more than one comorbidity also has an increased mortality rate, which I have estimated with NICE data on plural comorbidities. Next, each state is sorted into another intermediate compartment

based on the type of care they will receive based on their age and morbidity. Again, there is some overlap between types of care, which I correct for with K2. Effectively, this correction accounts for patients that have been in both wards, otherwise inflating the hospitalization rates. Finally, each state empties into either recovery (not shown in diagram) or death (compartments μ 1 to 3). However, as patients are admitted into either general wards or the ICU, these capacities start to fill. At a certain point, no additional patients can be admitted until beds become free again. Starting with the ICU, once all beds are full, patients will be admitted into non-ICU beds instead. It is reasonable to assume that this will worsen the outcome of the patient; ICU admitted patients have a roughly 20% fatality rate (unweighted average, NICE 2021). When the ICU beds are at capacity, new ICU patients will not have access to that level of care. ICU patients that cannot get to an ICU bed will receive an additional mortality rate ($\gamma 1$). At this point regular wards will start to fill at an increased rate. When all other beds are also full, no patients will be admitted into healthcare at all. This too comes with an additional mortality rate ($\gamma 2$). Finally, there is also mortality outside of hospital care. This mostly includes long-term care facilities, and is effectively 0 at ages below 50. (note. This does not mean that there is no mortality at all below age 50. Only that all fatalities below that age have occurred in hospitals and ICU beds.) I discuss this in more detail in the next section.

The result is the number of deaths that accounts for age, comorbidity, and care required and received. This same framework also serves to estimate the YLL per death. The same grouping of age-group and comorbidity is relevant to estimate the

remaining life lost due to COVID-19. To estimate this YLL, I use the same methodology as Briggs et al (2020) and Wouterse et al (2020). To start, I collected data from the Global Health Estimates (Published by the WHO, 2016) on *age* and *cause* specified mortality in the Netherlands. I could have also used data from the Dutch CBS, as counts are almost identical. Starting with age specified mortality, I calculate the per-age probability of dying for any cause (*q(x)*). Then, for every age year *x* I take the product of the preceding *probability of surviving to age x+1.* Doing this for all age groups until the maximum lifespan of *82.37* years, yields the life expectancy (LE) at each age group. This remaining life years at a certain age are however not adjusted for morbidity.

To adjust this remaining life expectancy downwards for groups with comorbidity *i* I calculated Standardized Mortality Ratios (SMR) for four groups of comorbidity as shown in the equations below. This is essentially a disease-specific per-year mortality risk increase. Summing the product of the instantaneous death rate, adjusted for this SMR then provides the remaining life expectancy LE at age *x* for comorbidity *i.*

The equations at the bottom of this page shows in depth how I have calculated these morbidity adjusted life expectancies.

$$LE_{x,i} = \sum_{g=G}^{x} \left[\prod_{x=1}^{x} \left\{ e^{-\delta(x) \cdot SMR_i} \right\} \right] = \ remaining\ LE\ at\ agegroup\ g\ for\ disease\ i$$

$$\delta(x) = -ln\left\{1 - q(x)\right\} = Instantaneous\ death\ rate$$

$$SMR = \frac{Observed\ deaths\ \ for\ disease\ i}{Expected\ deaths\ \ for\ disease\ i} = \ Standardized\ Mortality\ Ratio$$

Table 3: Equations to calculate morbidity (i) adjusted life expectancy at age of death x.

The only remaining step is to sum up the lost life years for every death *z* at age *x* (age at death) as follows: $YLL_{x,i} = \sum_{z=1}^{z} \{LE_{x,i}\}$ The resulting sum represents the total loss of potential years of life. Additionally, the average of this sum represents how many years of life the average COVID-19 fatality lost. In Appendix A , I show the resulting remaining life expectancies.

A final note on a factor not included in this model. I have opted not to include gender in this model. The main reason was because not all the important data and figures were separated by gender. I will address this in more depth in the discussion section.

Modelling different scenarios

To show the effect of the R_0, as well as some other factors, I use a multivariate sensitivity matrix, the inputs of which are shown in the table below. In total, I create 9 different scenarios. Scenario A, the 'Baseline scenario' consists of an R_0 of 2, the frequencies for the comorbidities and the excess mortality from capacity constraints (the mortality 'penalty' for not getting the right care because no hospital beds are available) are the best estimates as discussed in the following section.

Scenario	**R_0**	**Excess mortality from capacity constraints**	**Comorbidity factor**
A - Baseline Scenario	**2.0**	***x 1***	***x 1***
B - Lower Infectivity	**1.5**	*x 1*	*x 1*
C - Higher Infectivity	**4.0**	*x 1*	*x 1*
D - No capacity mortality	*2.0*	***x 0***	*x 1*
E - Double capacity mortality	*2.0*	***x 2***	*x 1*
F - No Comorbidity prevalence	*2.0*	*x 1*	***x 0***
G - Double Comorbidity prevalence	*2.0*	*x 1*	***x 2***
Min - All inputs low	**1.5**	***x 0***	***x 0***
Max - All inputs high	**4.0**	***x 2***	***x 2***

Table 4: Different modelling scenarios with varying inputs. (x: factor change of the inputs)

Data and probabilities

In this section, I discuss which data I have used to estimate state probabilities. For example, the risk of being admitted to the ICU, and the risk of then dying in that ICU. Some distributions, such as the age-group distribution in the Netherlands, are relatively straightforward. I have applied the age-group structure of the year 2018, from the Dutch office for Statistics (CBS, 2020).

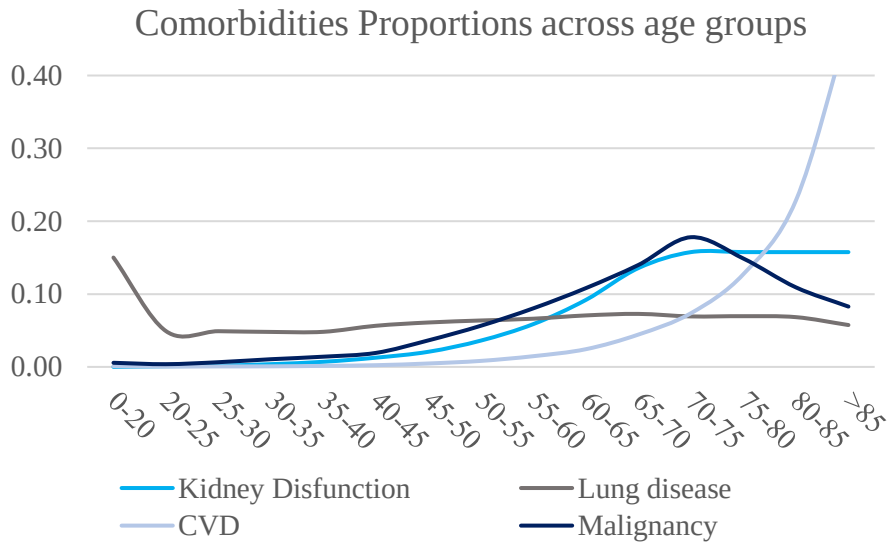
While I discuss mortality rates in more detail later, I had to decide early on how to model healthcare efficacy *over time*. As discussed previously, the cfr has started out high and then decreased over time. Similarly, hospital admission rates have fluctuated over time. It is hard to pinpoint the reasons for this. It is likely that there was perhaps initial confusion in testing and treatment, as well as a 'learning' effect. However, the reduced testing capacity in the early days of the pandemic also mean that the mortality rates are possibly upwardly biased as well. In the end I decided to simply use the average rates over the first year of the pandemic (until the end of march, 2021). This could potentially result in an underestimation of the number of deaths, because as I will show later, a substantial number of deaths occur early in the pandemic. In this, as with many other variables, I prefer to base my model on data that could potentially lead to an underestimation, as opposed to overestimations.

In the remainder of this section I first discuss the data I have used to estimate comorbidity levels. Then, I discuss healthcare admission rates, e.g. the likelihood that someone with COVID-19 is admitted to the hospital. Lastly, I also discuss the data mortality rates both inside and outside hospitals.

Comorbidities in the population.

To start, I found comorbidity distributions for the Dutch population, which are shown in the graph below. In this section, I will briefly discuss the sources for these comorbidities.

Starting with malignancy, it was reported that in 2020 roughly 604.000 people (or 3.5%) in the Netherlands had some form of cancer. I have opted to simply take the overall prevalence, which was reported by the IKNL (2021), which was also reported stratified by age.



Graph 2: distribution of the four selected comorbidities across the age groups

For lung diseases, I looked at the VTV (2020), which provides a ranked list of the most common diseases in the Netherlands. An article in The Lancet (Aveyard et al, 2021) reported that while data was still scarce, COPD, Asthma, and airway infections were notably linked to increased mortality rates. They also report that the severity of these conditions play an important role. Based on this, I only look at the prevalence for asthma classified as '*severe*' (3.6% of cases) and COPD grade 3 and 4 (severe and very severe, 15% and 3% of cases respectively). I also correct for the fact that around 32% of these patients have both Asthma and COPD, to avoid double counting. I further look at the incidence of lower airway infections, which appear to further cause severe complications. All three of these rates have been found in the VTV for the year 2018. Only COPD and Asthma were stratified according to age, and I proportionally distributed the incidence of lower airway infections.

For kidney disfunction I found that there were a large range of diseases responsible. To overcome this, I took aggregate data on kidney disfunction rates from the Nierstichting (2019). While this provided me with the total prevalence of kidney disfunction (rated severe or very severe) of 210,000, this was not stratified according to age. This was solved imperfectly by taking the age-stratified prevalence of Chronic Kidney Disease (CKD) (only grade 3 and higher) from the U.S. veteran affairs department (Korshak et al, 2019). I used these proportions to distribute the prevalence found in the Nierstichting data across the age-groups.

Cardiovascular diseases presented the largest overall prevalence, with also a large set of different diseases responsible. For practicality, I only looked at *strokes*, *Coronary Heart disease*, and *heart failure.* However, as there was not a lot of data comparing different cardiovascular diseases and the effect on COVID-19, I based this on general severity and prevalence. Again, there was a notable overlap of patients having both diseases. Data on the incidences came from the VTV, however I could only partially estimate the overlap in patients with data from the Hartstichting (2018), which ranged between 0.26 and 0.42.

Healthcare occupancy, admission, and mortality rates

There are many sources reporting on the various statistics of COVID patients, such as the RIVM, the GGD, Stichting NICE, and others. For tracking the healthcare aspect NICE produced the most complete range of statistics for the purposes of this study. Their reported data provided estimates of the average number of available beds (e.g. free beds that are not occupied by other non-COVID patients), age-stratified admission rates for both general wards and the ICU wards, and importantly mortality rates adjusted for comorbidity.

First I used data on the absolute number of positive tests by the GGD and the RIVM for the period up to February 2021. Using a higher number of positive cases when calculating healthcare

admission rates reduces any potential upwards bias so I ended up using the GGD data. I then used the NICE data on hospitalizations and ICU admissions, which divided by the number of positive tests produces the general healthcare admission rates per positive test. Lastly, I have used outcome data on mortality by comorbidity to extract the baseline (without comorbidity) mortality rates per ward, as well as mortality risk ratios for the 4 comorbidities. All these rates per age group are shown in Appendix B.

There also exists mortality outside of the hospital-care setting I have previously discussed. While I do not pinpoint this to the exact origins in this paper, I suspect that the majority of these deaths occur in long-term care facilities. This is corroborated by the fact that outside hospitals and ICUs no mortality in the ages below 50 is reported in the period up to February 2021 (RIVM, 2021). I have taken these figures, deflated them for underlying comorbidities, and age-distributed these across the age-groups (see Appendix B for these figures).

The final source of COVID-19 deaths in the model comes from patients requiring care when there are no more beds available. There is no immediate precedent which gives a good quantification of what mortality is involved with foregone care, so I have applied a flat mortality inflation factor. Effectively, when a patient requires care but is unable to receive it, I apply the ordinary mortality rates for that type of care multiplied by this factor. The main benefits of this method are transparency and proportionality. First, the method is clear for any reader to follow into the final results. Second, the method is proportional to the underlying risks, e.g. the additional mortality is proportional to the age, comorbidity, and type of foregone care.

I have settled on a factor of *four* as the lowest reasonable factor. This is an exceedingly conservative estimate, giving someone between 55 and 59 a 50% chance of survival when they go without the intensive care. I will however show the effects of this inflation factor in the sensitivity analysis later on.

Results

In this section, I will discuss the results of this model in terms of the number of deaths, as well as the years of life lost (YLL). In Appendix C I show specifically the SEIR model results. In the table at the bottom of this page the results are shown in absolute and relative terms. They may at first glance seem either substantially lower or substantially higher than one would first assume. In a timespan of 300 days an unchecked pandemic could, according to this model, have produced anywhere between 138,275 and 365,338 deaths (0.813% - 2.149% of the total population), with the best estimate landing on 279,619. This is a very substantial number of deaths for any country, especially so in a country with a population of 17 million. The case fatality rates in this model are however low compared to what has been reported by the global monitoring bodies (ranges between 1% – 5.6%, COVID-19 Health System Response Monitor 2020). Total loss of life years falls between 1 and 5 million years, with an average of 7.8 – 13.6 years per death.

Scenario	**Deaths** *(% of population)*	**Case fatality rate**	**Total YLL** *(Average)*	**Full lives lost**
A - Baseline Scenario	279,619 *(1.65%)*	1.79%	3,002,130 *(10.7)*	36,447
B - Lower Infectivity	253,172 *(1.49%)*	1.73%	2,621,576 *(10.4)*	31,827
C - Higher Infectivity	291,604 *(1.72%)*	1.72%	3,202,266 *(11.0)*	38,877
D - No capacity mortality	152,105 *(0.89%)*	0.97%	1,189,642 *(7.8)*	14,443
E - Double capacity mortality	348,816 *(2.05%)*	2.23%	4,630,933 *(13.3)*	56,221
F - No Comorbidity prevalence	275,601 *(1.62%)*	1.76%	4,826,017 *(10.8)*	58,589
G - Double Comorbidity prevalence	283,638 *(1.67%)*	1.81%	4,938,443 *(10.6)*	59,954
Min - All inputs low	138,275 *(0.81%)*	0.88%	1,074,962 *(7.8)*	13,050
Max - All inputs high	365,338 *(2.15%)*	2.34%	4,978,916 *(13.6)*	60,446

Table 5: The model results for all 9 scenarios. Full lives are calculated by dividing total YLL by 82.37 years, e.g. the maximum life expectancy at birth.

The results also suggest that, *ceteris paribus*, the comorbidity level (F and G) has the smallest impact on the total number of deaths. This too might seem initially very surprising, given the increased mortality rates associated with the comorbidities. While mortality and hospitalization rates are a lot higher for these individuals, they only make up a small number of the population. For example, the total number of cancer patients in the Netherlands comprise 3.5% of the population. In other words, the comorbidity effect is marginalized when the entire population gets infected.

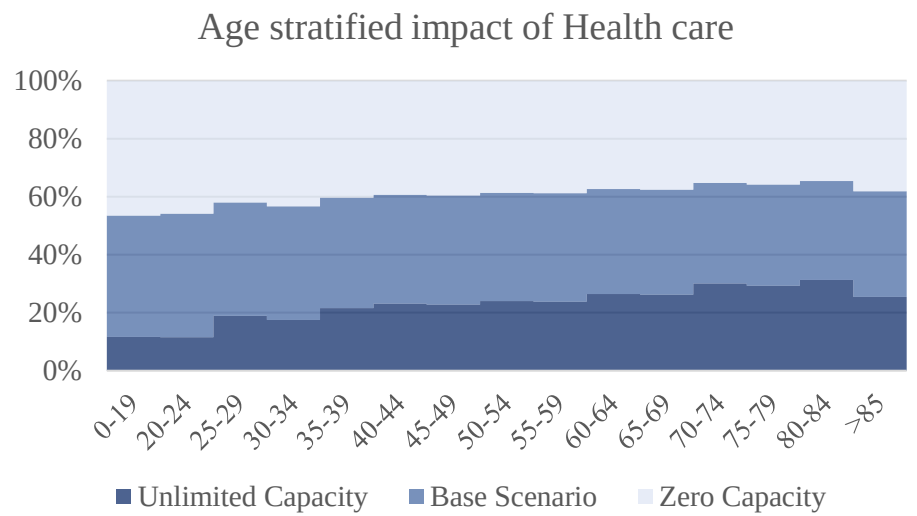
The R_0 (B and C) has a larger impact. Doubling the R_0 increases the number of deaths by slightly less than 12,000, while decreasing it to 1.5 reduces the number of deaths with over 26,000. This too is related to the total population, which in both cases reaches infection levels of 90% to 99% of the country. There is however an asymmetry in the reduction and increase. A reduction of 25% in R_0 has a larger effect on the number of deaths than an increase of 100%. This is mostly due to the most important factor: healthcare. By far the largest impact on the number of death is the available healthcare (or the excess mortality resulting from capacity constraints). There is a difference of almost 197,000 between scenario D (no excess mortality from foregone care), to scenario E. This highlights the key role that available healthcare has on mortality. In the model, healthcare becomes completely overwhelmed: 22 thousand people die within hospital settings, while the remaining 259,000 die elsewhere. This is due to both the extreme long time COVID-19 patients are stay in hospitals (ICU stays last 19 days on average).

Lastly, I have also calculated the full lives lost. This is simply to give the total YLL a more intuitive interpretation. It is the total YLL divided by the maximum life expectancy, hence ‘full lives lost’.

Lastly, it is worth mentioning the average years of life lost. At the end of the simulated pandemic, the Best Estimate scenario predicts

a loss of life years of 3,000,000 years. The sensitivity analysis estimates a range from 1,075,000 up to almost 5,000,000 years. The average YLL per death for the different scenarios is indicative of the remaining life the 'average' person who died has lost. Here, a higher number represents that a person had more years to live, and is a larger loss of potential life. The average YLL of the best estimate is around 10.74 years, with a range from 7.77 to 13.63 years.

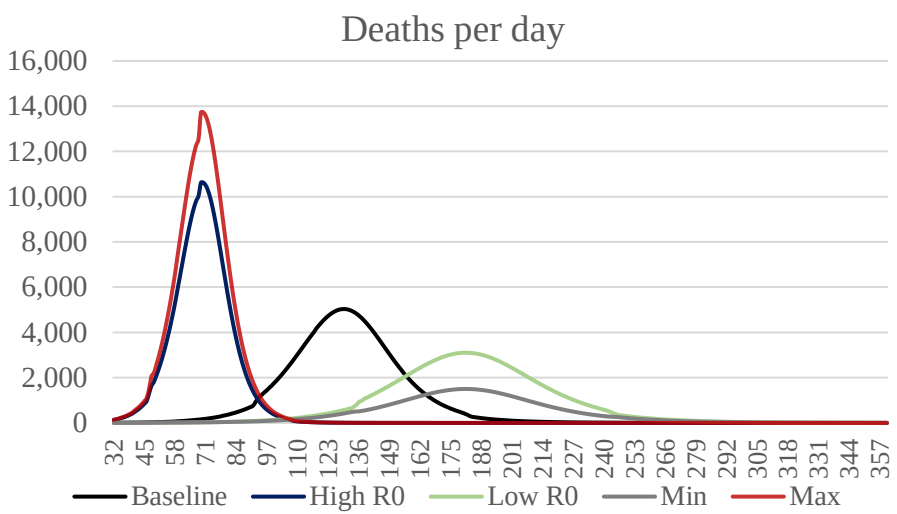
Varying the R_0 and the comorbidities a limited effect on the average YLL. Again, the greatest impact can be seen by capacity constraint related mortality (D and E). This shows the huge impact healthcare has on saving life potential. Those patients who can be saved through medical interventions generally have more than average remaining life years. This can further be seen in graph 3, which shows the relative portions of deaths by age group for 3 scenarios: one with unlimited healthcare capacity, the Best Estimate, and with no available beds at all. If the portions are roughly equal for an age group, the number of available beds has a limited impact on the relative mortality for that group. This graph further shows that healthcare has the most pronounced effect on (saving) younger age groups. In other words, a lower healthcare capacity would ultimately increase the average YLL, because less younger patients are saved.



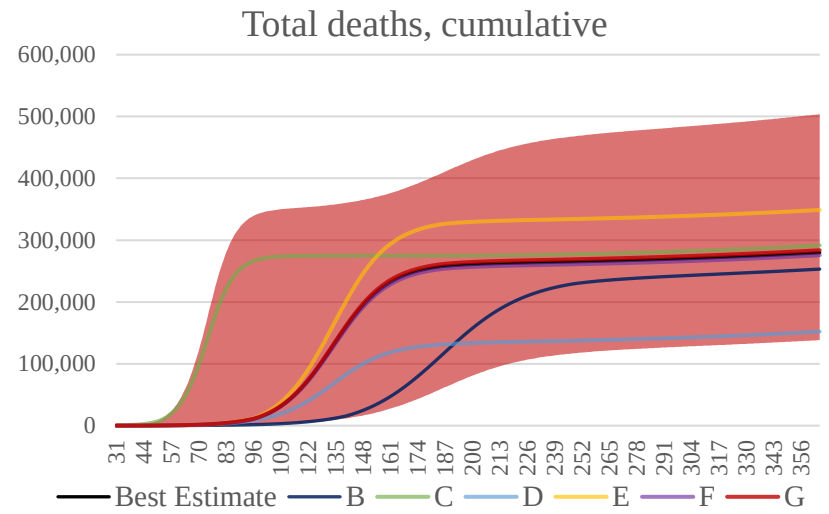
Graph 3: Proportional mortality over 3 scenarios. unequally sized bands for an age-group indicates that this age-group has more marginal benefits from healthcare capacity

The next page shows several complimenting graphs, which I will also discuss in more depth.

Model results shown over 4 graphs

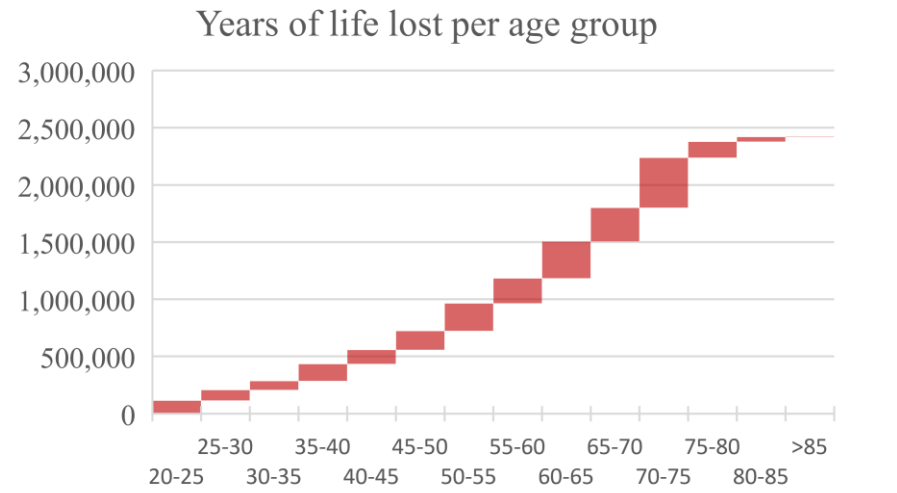


Graph 4: Daily deaths across selected scenarios

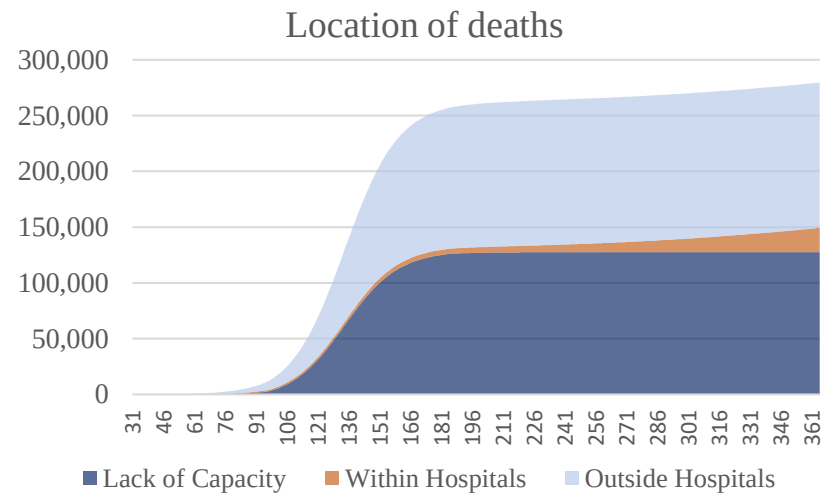


Graph 5: Cumulative deaths across all scenarios

B (High R_0), **C** (Low R_0), **D** (No Capacity Mortality), **E** (2x Capacity Mortality), **F** (No Comorbidities), **G** (2x Comorbidity prevalence), **Min**/**Max**: Red bands.



Graph 6: This graph shows the per age-group contribution to the total YLL (0-20 omitted for balance)



Graph 7: This graph shows where, over time the deaths occur

The top two graphs show the progression of deaths over time. The first graph shows how daily deaths rise exponentially over time, before quickly peaking and subsiding. One feature to note are the points where a sudden increase in deaths occur such as at day 45. This happens when one of the hospital wards reaches capacity. Depending on the scenario, this happens once for the ICU, and in the worse cases again for the general wards. Looking at cumulative deaths, we can see the influences of the variables on the timeline. The R_0 (B and C) has the strongest effect on when the deaths occur, while capacity mortality (D and E) significantly impact the total number of deaths most substantially. Meanwhile comorbidities seems to have only marginal effects on the number of deaths compared to the best estimate.

Graph 5 shows the contribution of each age-group to the total YLL. Different from the number of deaths, this shows that all age-groups substantially contribute to the total loss of life years. This can be contrasted against table 6, which shows the number of deaths per age-group. Essentially, while mortality is comparatively much lower among the younger ages, they have longer life expectancies that substantially contribute to the total YLL. Overall however, the age-group between 70 to 75 has the largest impact on YLL.

Age-group	Deaths
20-25	1,551
25-30	1,413
30-35	1,295
35-40	2,787
40-45	2,588
45-50	4,123
50-55	7,258
55-60	8,264
60-65	16,606
65-70	22,256
70-75	55,038
75-80	42,552
80-85	70,608
>85	19,680

Table 6: Number of deaths by age (0-20 omitted for balance)

Finally, graph 7 gives another insight. The large majority of deaths occur outside hospitals. The largest number of deaths occur outside hospitals, firstly (presumedly) in long-term care facilities. Then, as the healthcare capacity is reached, there is an almost equal number of deaths from people who couldn't get COVID treatment when they needed it. This further indicates the key role that available healthcare capacity plays in reducing the number of deaths. In the following section, I will present my conclusions and discuss this the limitations and implications in depth.

Conclusion and Discussion

In this study, I set out to model the health costs that could have occurred in the scenario where no public or private measures are taken against the COVID-19 pandemic. This is meant to portray an worst case scenario. Specifically, I estimate how many people could have died, and what the YLL then would have been.

As previously discussed, my best estimate scenario predicts 279,619 deaths due to COVID-19, with a sensitivity range of 138,275 to 365,338. The total loss of life years I estimate to be 3,002,130 years with a range between 1,074,962 - 4,978,916 years. This would in turn translate to between 13,050 and 60,446 complete lives lost (of 82.37 years each). The average remaining life years would be 10.7 years, with a sensitivity range between 7.8 and 13.6 years. Of course, this is a prediction based on very strict assumptions, but it is safe to say that these numbers are shockingly high. And yet, the estimations do not appear to be outlandish or anomalous. The case fatality rate based on these outcomes falls between 0.813% and 2.149%. This is actually on the lower end of the reported spectrum of fatality rates, which have been reported as high as 14% in the earliest stages of the pandemic (but more commonly between 1 and 5% worldwide). It is hard to say if this makes the number of deaths more plausible, given that the resulting cfr is on the lower end of the spectrum. I do however feel that it suggests that I have been conservative in my estimations.

The average YLL too falls within the spectrum of other reported averages (between 5.5 and 16 years). The average from my estimation is somewhere in the middle of this spectrum. This of course does not indicate that the results themselves are valid, something I will discuss more deeply in the limitations.

Limitations

We should be careful in making too direct comparisons with real life, because the model I have used is built upon very stringent

assumptions. While every mathematical model always abstracts away realistic complications to a certain degree, it is important to note that this is specifically true in this paper.

To start, I have used a modified SEIR model to estimate the number of infections. This assumes a freely mixed, homogeneous population. E.g. everybody travels freely, randomly, and everybody has an equal chance to become infected. I also could not include asymptomatic carriers in the model, a group that is substantially less susceptible to worse health outcomes. It accounts for exactly none of the psycho-social complexities that make up modern society, nor the spatial dynamics that allow for disease transmission. These are also strengths, depending on the circumstance, but these assumptions primarily mean that reality will always be different from the SEIR predictions.

In the next step in my study, I have used these infections to estimate the number of deaths. I think that I have been fortunate in the data I have been able to use, which are all based on Dutch observations (e.g. it comes from the same population), and reported with a relatively high degree of certainty. However, my method extracts static average rates from the empirical data, which further removes complexity. For example, the mortality rate in ICU wards is the average mortality rate over the entire year in the Netherlands. In reality, the mortality rate has decreased over time, as medical staff becomes better adapted to combat this disease. My model however just assumes a static rate. In other words, in my model medical professionals are completely consistent over time in treating patients. This may not be realistic, especially when we see that the timeline of the disease can vary between scenarios.

In estimating the deaths and YLL, I have also not accounted for the specific mortality in long time care facilities. This was partly because this was not compatible with the SEIR model as far as I am aware off, but it does pose a significant limitation in this paper. LTC facility inhabitants are living closely together, which makes

it likely that they will all get infected in short succession. They are generally also at a higher risk for mortality. At the same time, the remaining life expectancy is generally lower for somebody in an LTC facility, as compared to someone with a similar age not in such a facility. The study by Wouterse et al for example particularly notes how strong the YLL decreases when one accounts for this lower remaining life expectancy. I also have not accounted for gender. This can also lead to biases, as men have been observed as being more vulnerable to the disease, while also having a lower life expectancy.

Finally, the scenario itself was unrealistic. I have modelled a population which is either completely unaware of, or completely unwilling to measures that combat the disease. While there have been countries where the government has not imposed any strict public measures, my model goes still further than that. It assumes that people will not even self-quarantine, nor will they avoid the infected. It is very unlikely that societal behaviour as such will ever be observed in real life. All this is simply to say that this model is very abstract. While I think that the method and data are structurally sound, it does not present a 'true' prediction of the counterfactual of what would have happened.

Discussion

Still, even with these limitations it is interesting to discuss the implications of this model. Especially since some of these implications match so very closely with the official narrative of flattening the curve. I think first and foremost, these estimations give an impression of the difference between what has happened in the Netherlands, and what *could* have happened. As I mentioned earlier, public measures can become a victim of their own success. As measures slow down the spread and mortality of the virus, and mostly healthy people are being saved by healthcare, the pandemic may seem like it was 'not so bad after all'. Of course, this is *because* measures have been taken and sacrifices have been made.

It is precisely the point that predictions such as the model I have created do *not* come true. And it is by our measures and behaviours that we have, in real life, possibly staved off much, much worse.

Another thing we can learn from this model which fits the narrative of the public debate is the crucial role of healthcare. This can be noted in terms of the number of deaths, which sharply increases when healthcare capacity is exceeded. In my model, I have been conservative in how strong the "mortality penalty" is when you cannot get the care you need. Anecdotally, many healthcare professionals I have spoken to mention that it would have likely been between 90% and 100%, in the case of the ICU. And even with this conservative estimate of excess mortality, a substantial increase in mortality occurs when every ICU bed is filled.

At the same time, this also leads to a higher average YLL. Specifically, the average YLL from my estimations rises proportionally with the number of deaths. Or, put differently, as the number of deaths increases, more and more younger people end up dying. This suggests that as capacity is reached, fewer younger patients can be saved. This all leads to larger average YLLs. This indicates the important role that healthcare plays in this pandemic in keeping the death toll low, and the YLL proportionally lower. In real life, we have skirted against this threshold several times, but we have been able to avoid a collapse of the healthcare system.

This shows from the official death count over a similar period, 16,662. This pandemic has been tragic and devastating, for both the victims and the survivors, mentally and physically. But it seems that it could have been much worse, by a factor between 8 and 22. If these numbers are realistic, they suggest that the bulk of our public health measures have been exceedingly successful in reducing the death toll and overall loss of life substantially. This does not reduce the tragedy that has occurred and indeed is still occurring in many places, but at least we can be sure that our efforts are not in vain.

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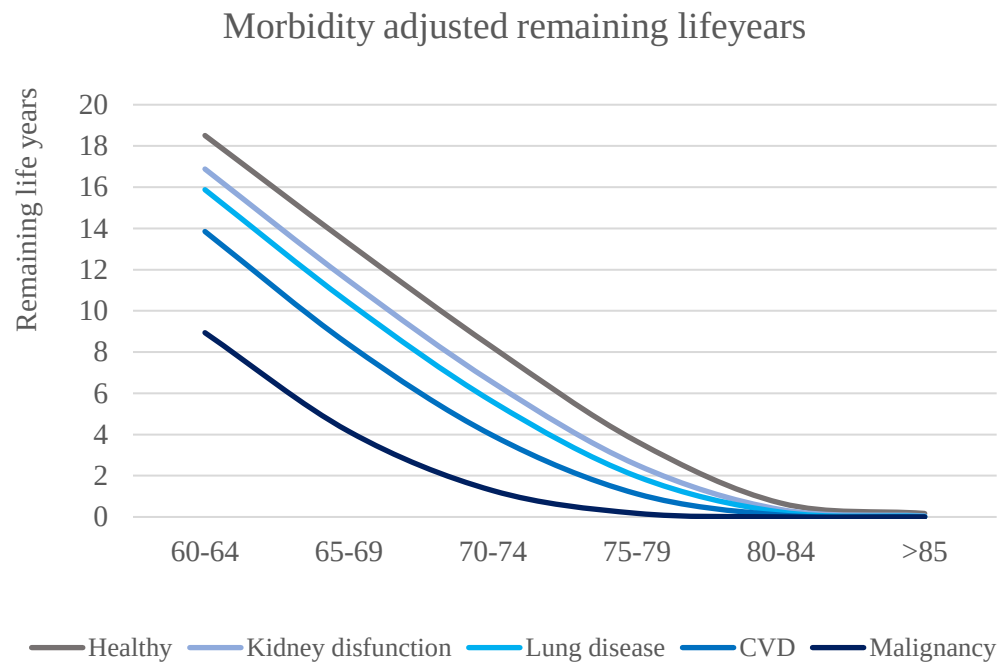
APPENDIX A – Comorbidity levels



Graph A1: Different remaining life expectancies for the selected comorbidities

This graph shows the resulting morbidity adjusted remaining life years for the ages 60 to >85, based on the methodology discussed on pg. 21. It gives a good indication of the relative effects of each comorbidity on the remaining life expectancy, and as such, on the years of life lost.

APPENDIX B – mortality and admission rates

Age group	0-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	>85
Positive tests	114122	36509	109528	28937	86811	31379	94137	35611	106834	20842	62528	12671	38013	8903	39039
ICU admission rate	0.0002	0.0009	0.0006	0.0034	0.0014	0.0059	0.0043	0.0195	0.0095	0.0626	0.0226	0.1257	0.0262	0.0339	0.0014
GW admission rate	0.0078	0.0080	0.0045	0.0244	0.0094	0.0344	0.0191	0.0815	0.0342	0.1942	0.0657	0.4265	0.1527	0.6138	0.1350
ICU mortality	0.0540	0.0458	0.0360	0.0229	0.0699	0.0691	0.0816	0.1046	0.1253	0.1862	0.2616	0.3437	0.3983	0.4859	0.4762
GW Mortality	0.0034	0.0026	0.0031	0.0053	0.0075	0.0056	0.0088	0.0151	0.0192	0.0346	0.0720	0.1124	0.1755	0.2281	0.2614

Table A1: Hospital admission rates and the corresponding mortality rates, stratified by age.

This table shows the age-stratified hospital admission rates and corresponding mortality rates as discussed on pg. 25 and pg. 26. The rates are not always strictly increasing between age groups, but I have decided to use them as-is, without further adjustments.

	Lung disease	Kidney disfunction	CVD	Malignancy
RR	1.36	1.98	1.89	1.81

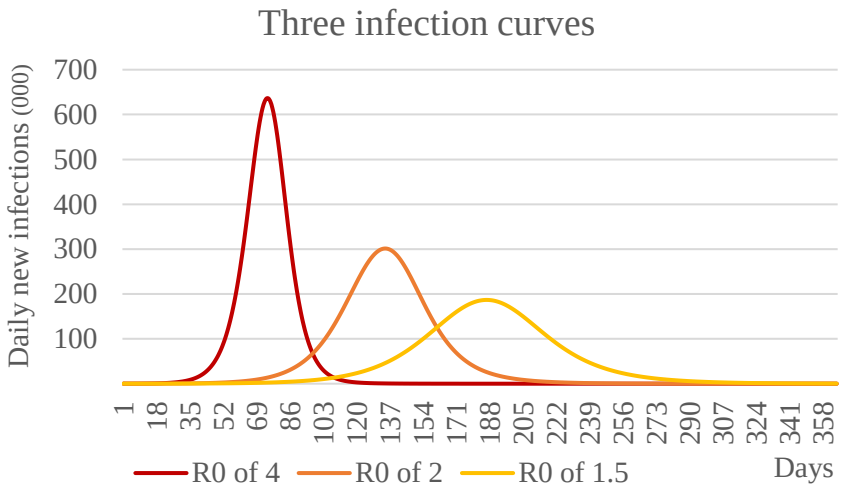
Table A2: Risk Ratios (RR) for death for the four selected comorbidities.

This table shows the risk ratios of the four comorbidities. This can be read as ‘a risk ratio of 2 doubles the mortality risk on the ICU’.

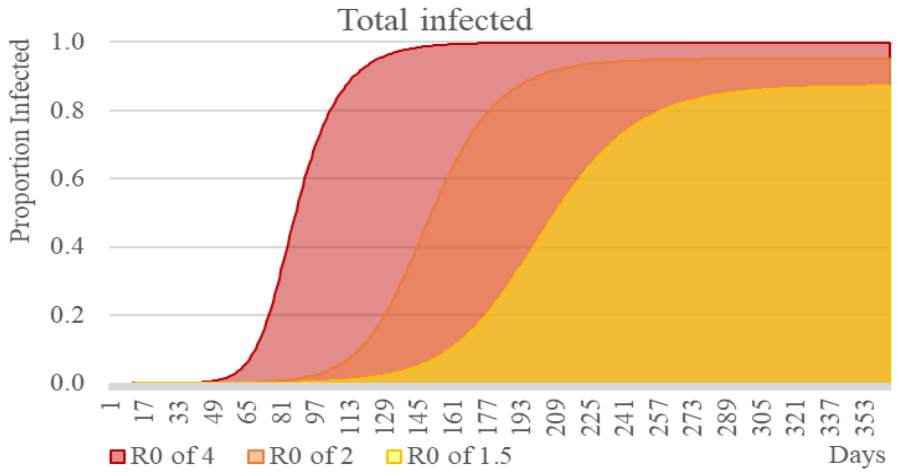
Age	<50	50-54	55-59	60-64	65-69	70-74	75-79	80-84	>85
Mortality	0.000	0.001	0.001	0.006	0.006	0.046	0.046	0.151	0.008

Table A3: Mortality rates outside the hospital setting.

APPENDIX C – SEIR MODELS



Graph A2: daily infections for three different R0 values.



Graph A3: Total number of infections for three different R0 values.

Different R_0 values would result in different infection curves. An R_0 of 4 (the highest estimation by the RIVM) would result in a steep rise in infections. With this R_0 the disease is capable of spreading faster, which would infect (nearly) the entire population, with 16,976,217 becoming infected. An R_0 of 1.5 however is still able to spread exponentially, albeit at a slower pace. Not only does the pandemic take longer, the decreased speed also causes it to 'die out' before the entire population can become infected. This would result in 14,671,629 becoming infected. For my main scenario, I will model a pandemic with an R_0 of 2, as a middle ground. This results in a total number of 15,637,206 infections over the course of the pandemic.