Understanding the COVID-19 Pandemic with High School Mathematics

Dr. Patrick Mangat*

Dated: 2021-02-26

Abstract

The goal of this document is to explain the COVID-19 pandemic from a mathematical perspective. In order to make this note as accessible as possible, we will merely use mathematical methods taught in high school. First, we give an intuitive explanation of the basic reproduction number and explain how it relates to the more publicly known interest rate. Afterwards we introduce the SIR-model of epidemiology where we will recover the basic reproduction number in a more rigorous way. We will then extensively exploit this model to address very practical questions such as: where does the number of $60-70\%$ for herd immunity come from? How many people will get infected without any measures (such as shutdowns or vaccinations)? What is the effective reproduction number and how can we estimate its size from published data? Why is it important to suppress the reproduction number below the critical value of one? (And why is this value critical at all?) What does "flattening the curve" mean from a mathematical perspective? How small does the reproduction number need to be to make this work? How can we estimate the duration of a shutdown until the incidence drops below some target value? How many vaccinations per day are needed to prevent further increase of the incidence values? What could happen if the immunity only holds for one year?

Those and some more questions will be addressed from a practical and mathematical point of view. Political opinions or statements will be avoided.

Essential prerequisites – High school mathematics, especially exponential functions, logarithms, basic calculus.

Contents

*Send comments, questions, typos to: patrick.mangat@gmail.com

1 Introduction

Due to the COVID-19 pandemic, the general population was suddenly confronted with elementary mathematical concepts (such as exponential functions) and the basics of epidemiology. In the reporting the increasing number of infections was initially assessed on the basis of the doubling time. Subsequently, the effective reproduction number $\mathcal{R}_{\text{eff}}$ has moved into our focus. Eventually, the 7-day incidence value per 100 000 inhabitants has become an important figure. While those three parameters may have appeared to be quite different, they are actually closely related. Unfortunately, their relation is not immediately obvious, but requires some knowledge of exponential functions, logarithms and basics of epidemiology. During the shutdowns in Germany, one frequently asked question among the population was about the duration of the shutdown. Although it is not possible to precisely calculate the duration due to many uncertainties, there is a fairly simple formula to estimate the order of magnitude of the duration of shutdowns.

These issues serve as a motivation to introduce a well-known and relatively simple but powerful epidemiological model to the broader audience. Since more sophisticated epidemiological models can easily become arbitrarily complicated, we will mostly focus on the so-called *SIR-model*. It has been proposed in 1927 by A. G. McKendrick and W. O. Kermack [1]. Although the SIR-model is only applicable to the COVID-19 pandemic with some caveats, it captures the most important aspects of the current pandemic. Being mathematically accessible to high school graduates it is certainly *the easiest model* in order to understand the fundamentals of the COVID-19 pandemic. Nevertheless, it should be stressed that any model has its limitations when it comes to applications to the real world. In 2020, more realistic scenarios have been developed to model the COVID-19 pandemic, see e.g. [2–16] and many more papers. In particular, the references [3; 4] provide the source code for their models as supplementary material, and are therefore highly recommended for mathematically inclined readers. The discussion of details of their models goes beyond the scope of this note. Nevertheless we

will see that the SIR-model allows for simple estimations yielding similar results. We hope that this note turns out to be useful for a first step into the mathematics of epidemiology.

Unless otherwise stated, concrete numbers and case studies are usually referred to Germany.

Outline and brief summary

In Section 2 we begin with an intuitive argument for the origin of the exponential growth of infection numbers at an early stage of a pandemic. We use an analogy to the interest rate model and heuristically guess the exponential law of a pandemic at an early stage. It will be illustrated intuitively, why this exponential growth cannot be eternal. In this context we introduce the concept of herd immunity.

In Section 3 we rediscover these heuristically obtained results by studying the SIR-model of epidemiology. We motivate the underlying differential equations and qualitatively derive the dynamical behaviour of this model. The basic and effective reproduction numbers are introduced and its relations to the doubling time, incidence values and the concept of herd immunity are carefully explained. Moreover, we highlight and demonstrate that the expected number of total infections at the end of the pandemic is significantly higher than the often quoted $60-70\%$ due to herd immunity. The SIR-model is then applied to compare the swine influenza with COVID-19 based on epidemiological parameters. At the end of this section we discuss several limitations of the SIR-model.

In Section 4 we briefly explain how to calculate the effective reproduction number from actual infection data. For instance, we find that the average effective reproduction number in Germany in October 2020 was $\mathcal{R}_{\text{eff}} = 1{,}72$.

In Section 5 we explain the concept of *flattening the curve*. We give three reasons why the infection numbers should be kept as small as possible during the COVID-19 pandemic and then estimate by how much the curves need to be flattened. Amongst other things we find that if the pandemic were to continue with $\mathcal{R}_{\text{eff; max}} > 1{,}09$ for sufficiently long time the estimated ICU capacities in Germany may be exhausted at the peak.

Moreover, in Section 6 we study the effect of shutdowns by looking at the data of some countries. Based on these results we estimate the duration of a typical shutdown using a formula derived from the SIR-model. We also estimate the duration between the end and the beginning of two subsequent shutdowns if contact tracing and other measures fail to work.

In Section 7 we provide a simplified estimate on the minimum vaccination frequency (i.e. number of vaccinations per day) in order to immediately slow down the infection dynamics. Afterwards, the results of a more realistic numerical model are discussed. It is shown that too many relaxations almost certainly lead to further shutdowns in 2021, unless more than approximately 400 000 people get vaccinated every day. But even then the chances for further shutdowns increase with growing effective reproduction number. It is therefore crucial to avoid the spread of more contagious mutations.

At the end, in Section 8 we dare to look into the future. We extend the SIR-model by the possibility of reinfections. Assuming that the immunity from infection or vaccination against COVID-19 lasts for one year on average we find that there will be further sudden outbreaks in this decade until the system stabilises to a non-vanishing incidence value. The size of this stabilised incidence value depends on the vaccination frequency. For instance, less than 200 000 vaccinations per day will result in a long-term 7-days incidence per 100k of at least 500. However, for a sufficiently large vaccination frequency, these future infection waves can be avoided and the incidence value can be sustainably suppressed to zero. In case of Germany this can be achieved by vaccinating at least 340 000 people every day.

2 Exponential growth in early stages of a pandemic

2.1 Interest rates and exponential growth

In high school we learned that exponential functions can be described by the following function:

$$f(t) = a \cdot e^{bt} \ , \tag{2.1}$$

where $a, b \in \mathbb{R}$ are real numbers and $e = 2{,}71828...$ is Euler's number.

One well-known application of the exponential function is the computation of interests on some deposit value D_0. Imagine you put some money D_0 on a bank account for an annual interest rate p. After one year you receive an interest of pD_0. Hence, the deposit value growths to

$$D_1 = D_0(1+p) \ . \tag{2.2}$$

After two years the deposit value will increase to

$$D_2 = D_1(1+p) = D_0(1+p)^2 \ . \tag{2.3}$$

This logic keeps going and we can generally write

$$D_n = D_0(1+p)^n \ . \tag{2.4}$$

Let us now go one step further: we assume a compounding frequency k (e.g. quarterly compounding means $k = 4$). We then have for one year:

$$D_1 = D_0 \left(1 + \frac{p}{k}\right)^k \ , \tag{2.5}$$

because the interest is calculated k times and added to the deposit. After n years we have

$$D_n = D_0 \left(1 + \frac{p}{k}\right)^{n \cdot k} \ . \tag{2.6}$$

Now we want to make the step towards *continuous compounding*. This means that the interest is continuously added to the deposit. Mathematically this means that the frequency k goes to infinity. The mathematical notation is then:

$$D_n = D_0 \cdot \lim_{k \to \infty} \left(1 + \frac{p}{k}\right)^{n \cdot k} \ , \tag{2.7}$$

There is a non-trivial result from calculus the reader either has to know or accept:

$$e^x = \lim_{k \to \infty} \left(1 + \frac{x}{k}\right)^k \ . \tag{2.8}$$

Therefore, we get

$$D_n = D_0(e^p)^n = D_0 e^{p \cdot n} \ . \tag{2.9}$$

Hence, we have established that a continuously compounded deposit value follows an exponential function.

Figure 1: A susceptible person can get infected. This person will eventually recover from the infection and become immune against the disease.

Case Study – Let us assume a deposit $D_0 = 10\,000$ EUR and an interest rate of $p = 2{,}0\%$ per year with continuous compounding. After $n = 10$ years we have

$$D_{10} = 10\,000 \text{ EUR} \cdot e^{0{,}02 \cdot 10} = 12\,214{,}03 \text{ EUR} \,. \tag{2.10}$$

One can see that the value has increased more than linear. The effect becomes stronger for larger interest rate p or duration n.

It is also instructive to calculate the time it takes to double the deposit value. Demanding $D_n = 2D_0$, we have to solve (2.9) for n by taking the logarithm:

$$n = \frac{1}{p} \cdot \ln\left(\frac{D_n}{D_0}\right) = \frac{\ln(2)}{0{,}02} \simeq 34{,}7 \text{ [years]} \,. \tag{2.11}$$

By the way: this is precisely the computation we will later need to relate the basic reproduction number with the doubling time.

2.2 Exponential growth in early stages of a pandemic

We now make first steps towards epidemiology. The population is assumed to be composed of susceptible, infectious and recovered people only. The latter are assumed to be (eternally) immune against the virus. Hence, recovered people can no longer become susceptible. The model is depicted in Figure 1. This is the chain of the SIR-model which we will introduce in more detail in Section 3.

Let us begin with one single infectious person within a population while every other person is assumed to be susceptible. To be as explicit as possible, let us further assume that any infectious person will infect two other people during the pandemic. We then introduce the parameter $\mathcal{R}_0 = 2$ and refer to $\mathcal{R}_0$ as the *basic reproduction number*. Just for illustration we assume that infections occur once per week. Moreover, an infectious person becomes immune after one week. The number of infectious people after n weeks can be expressed as

$$I_n = I_0 \cdot \mathcal{R}_0^n \,, \tag{2.12}$$

where $I_0 = 1$ in our example because we begin with just one single infectious person. The infection numbers are going to increase as follows:

Week (n)	0	1	2	3	4	5	6	7	8	9	10
No. of infectious people (I_n)	1	2	4	8	16	32	64	128	256	1024	2048

As one can see the numbers are rising tremendously with increasing time. The reader can check that after two weeks, i.e. $n = 14$, we have $I_{14} = 16\,384$ infectious people and after three weeks we would arrive at $I_{21} \simeq 2$ Mio. people. Of course, assuming that the exponential growth keeps going. We are going to comment on this assumption in a moment.

Let us first relate (2.12) to our model for interest rates. For the moment we write

$$\mathcal{R}_0 := 1 + p \,. \tag{2.13}$$

Here, p has the interpretation of an *infection rate per week*. We can then rewrite (2.12) as follows:

$$I_n = I_0(1+p)^n \ . \tag{2.14}$$

Mathematically this is identical to our formula for calculating the deposit value after n years with annual compounding, see (2.4). The only difference is that we have the analogy of weekly compounding.

But the virus is continuously spreading through the population. Therefore, continuous compounding applies and we can write in analogy to (2.9):

$$I_n = I_0 e^{p\cdot n} = I_0 e^{(\mathcal{R}_0-1)\cdot n} \ . \tag{2.15}$$

This is almost the formula that we will derive in Section 3 from the SIR-model. We were assuming that the infection cycle is once per week. This is reflected as follows. We write

$$n = \gamma \cdot t \ , \tag{2.16}$$

where t is the time and γ defines the characteristic time period for infections. In our example we have

$$\gamma = 1 \text{ week}^{-1} \simeq 0{,}14 \text{ d}^{-1} \ , \tag{2.17}$$

where d is the unit for days.

Consequently, we obtain the following exponential law for the spreading of a virus within a population:

$$I(t) = I_0 e^{\gamma(\mathcal{R}_0-1)t} \ . \tag{2.18}$$

This equation is of great importance for the remainder of this note. Without having dived too deeply into epidemiology, we can already infer an important observation:

If $\mathcal{R}_0 > 1$ then the infection numbers grow exponentially, because the exponent is then positive. If $\mathcal{R}_0 < 1$ we have an exponential decline in the infection numbers. For $\mathcal{R}_0 = 1$ the infection numbers remain constant. This is why it is important to suppress the effective reproduction number, which we will define later in this note, needs to be below unity to contain the pandemic.

Now we want to illustrate the limitation of the law (2.18). We return to our scenario with $I_0 = 1$ and $\mathcal{R}_0 = 2$. The situation is shown in Figure 2. In week 1 we have $I_1 = 2^1 = 2$ infectious people. They had been infected by the person (coloured in green) in the upper right corner. In week 3 there are only two susceptible people left. Therefore, the pandemic can no longer continue exponentially. Instead, *herd immunity* is reached (see discussion below). Exponential growth only occurs at the beginning of a pandemic as long as the number of infectious people is negligible compared to the size of the susceptible population.

2.3 Herd immunity: The end of exponential growth

The concept of *herd immunity* can be understood with very little mathematics. In epidemiology we say that herd immunity is reached once an infectious person statistically meets one

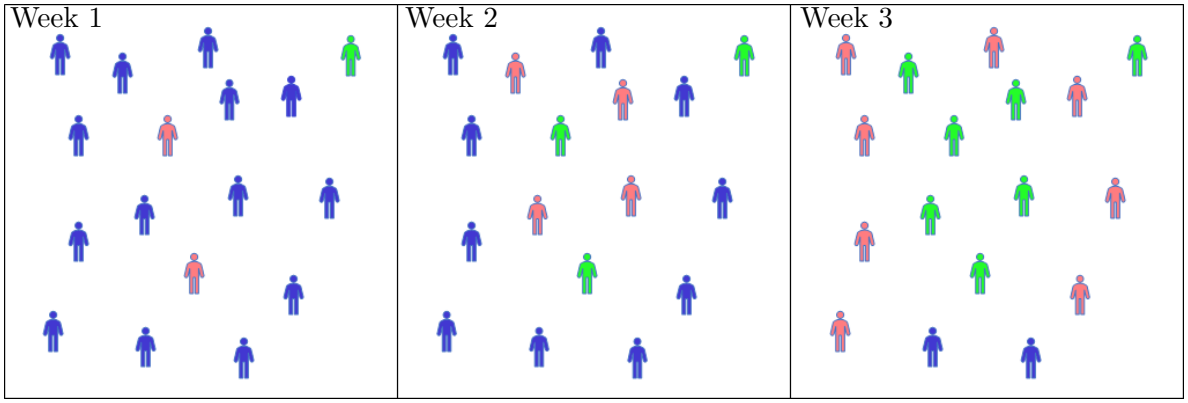


Figure 2: A pandemic with $\mathcal{R}_0 = 2$ and $I_0 = 1$. Susceptible people are coloured in blue, infectious people in red and recovered/immune people in green. In the first week we have $I_1 = 2^1 = 2$ infectious people (who got infected by the recovered person in the upper right corner). Each of them will infect two other people while they recover the week after. Hence, in week 2 we have $I_2 = 2^2 = 4$ infectious and three recovered people. In week 3 we have $I_3 = 2^3 = 8$ infectious people and seven recovered people. From now on the exponential law fails to hold because there are not enough susceptible people left.

susceptible person out of a group of $\mathcal{R}_0$ people. This means that herd immunity is reached once a fraction of at least

$$q_{\mathrm{H}} = 1 - \frac{1}{\mathcal{R}_0} \tag{2.19}$$

has gotten infected during the pandemic.

Now we are in the position to understand the origin of the frequently quoted number of herd immunity at 67%. For SARS-CoV-2 the basic reproduction number is often estimated to be approximately $\mathcal{R}_0 \simeq 3$. Then, (2.19) yields $q_{\mathrm{H}} \simeq 1 - 1/3 \simeq 67\%$. From (2.19) we can read off that herd immunity is harder to reach the bigger the basic reproduction number is.

It is important to understand that *herd immunity does not imply the end of infections*! Instead further infections occur at continuously decreasing rates. Taking the example of $\mathcal{R}_0 \simeq 3$, more than 67% will eventually have contact to the virus. We will compute the total number of infections in the subsequent section on the SIR-model.

3 The SIR-model

In this section we introduce the SIR-model [1] and follow textbook presentations such as [17; 18].

3.1 The differential equations

We have introduced a simplified scenario of the SIR-model in Section 2.2. The chain depicted in Figure 1 remains valid but is slightly developed as shown in Figure 3. We will subsequently refer to the R-population the *removed* population. This is because a disease may result in long-term impairments to health. Furthermore, for diseases like COVID-19 there is a non-vanishing infection fatality rate. In the SIR-model deaths can be (roughly) treated as subset

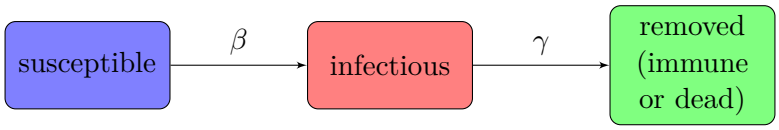


Figure 3: In the SIR-model susceptible people get infected at a transmission rate β. Infectious people become immune (or die) at a rate γ.

of the removed population.

We introduce the variables S, I and R, which denote the susceptible, infectious and removed population, respectively. The transmission rate of the disease is denoted by β. The number of infections per unit time is then given by βSI. This is because the number of infections is both proportional to the number of susceptible and currently infectious people. Furthermore, the number of people losing their infectivity is γI.

Therefore, we can write down the following system of differential equations:

$$\dot{S} = -\beta SI \ , \tag{3.1}$$

$$\dot{I} = \beta SI - \gamma I \ , \tag{3.2}$$

$$\dot{R} = \gamma I \ . \tag{3.3}$$

Here, $\dot{S}, \dot{I}$ and $\dot{R}$ are the derivatives of S, I and R with respect to time t. The left hand side of these three equations therefore denote the rate of change in the respective quantities.

The total population is

$$N = S + I + R \ . \tag{3.4}$$

In the SIR-model N is a conserved quantity. This is because from eqs. (3.1) to (3.3) it follows that the time derivative of N vanishes:

$$\dot{N} = \dot{S} + \dot{I} + \dot{R} = -\beta SI + \beta SI - \gamma I + \gamma I = 0 \ . \tag{3.5}$$

It is, of course, possible to include births, deaths[1] and migration. But we will refrain from doing so, because we want to highlight the very fundamentals while maintaining the model as simple as possible.

If we want to obtain the temporal evolution, i.e. functions $S = S(t)$, $I = I(t)$ and $R = R(t)$, we have to solve the system of differential equations. Unfortunately, it can only be solved numerically. (However, it is possible to derive functions such as $I = I(S)$ analytically.) The good news is that this model can easily be implemented in any programming language and even in Excel.

3.2 The (basic) reproduction number

The eqs. (3.1) to (3.3) involve the transmission rate β and the removal rate γ, but not the (famous) basic reproduction number $\mathcal{R}_0$, which we introduced in Section 2.2. We now explain how to express $\mathcal{R}_0$ in terms of β and γ. The basic reproduction number is the average of the number of infections caused by a single infectious person. The average duration of infectivity $\Delta t_{\text{infectious}}$ is simply

$$\Delta t_{\text{infectious}} = \frac{1}{\gamma} \ . \tag{3.6}$$

[1] As mentioned above, we will include deaths only as a subset of the removed population.

Aside – If this formula is not immediately convincing it can be motivated as follows. For a *single* infectious person equation (3.3) reads

$$\dot{R} = \gamma \cdot 1 \ . \tag{3.7}$$

Let Δt be the time it takes to remove this person from the infectious population. By definition it must hold

$$1 = \dot{R} \cdot \Delta t \ . \tag{3.8}$$

Hence, combining these two equations,

$$1 = \gamma \cdot \Delta t \tag{3.9}$$

or

$$\Delta t = \frac{1}{\gamma} \ , \tag{3.10}$$

as asserted.

Using probability distributions such as the exponential distribution this can be made even more rigorous, see e.g. [19].

Now we can compute the average number of susceptible people who get infected by one single person. Initially we have $S(0)$ susceptible people. Therefore, one infectious person induces

$$\beta \times S(0) \times 1 \times \Delta t_{\text{infectious}} = \frac{\beta}{\gamma} S(0) \tag{3.11}$$

infections on average.

Hence, by definition, the basic reproduction number $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \frac{\beta}{\gamma} S(0) \ . \tag{3.12}$$

Due to (3.1) the number of susceptible people will decrease with time. Consequently, it also makes sense to define a *time-dependent* or often called *effective reproduction number* $\mathcal{R}_t$:

$$\mathcal{R}_t = \frac{\beta}{\gamma} S(t) \ . \tag{3.13}$$

We can then rewrite the differential equations of the SIR-model as follows:

$$\dot{S} = -\gamma \mathcal{R}_t I \ , \tag{3.14}$$

$$\dot{I} = \gamma \left(\mathcal{R}_t - 1 \right) I \ , \tag{3.15}$$

$$\dot{R} = \gamma I \ . \tag{3.16}$$

The remainder of this note will be built on these three equations.

3.3 Characteristic dynamics of the SIR-model

We can qualitatively anticipate the characteristic dynamics of the SIR-model: Since $S(t)$ is a strictly decreasing function in time, so is the time-dependent reproduction number. This follows immediately from (3.13). As long as $\mathcal{R}_t > 1$ we have $\dot{I} > 0$ from (3.15). At $\mathcal{R}_t = 1$ we reach herd immunity which occurs due to the effect illustrated in Figure 2. We will elaborate on herd immunity once again in more detail in Section 3.4. At herd immunity we have $\dot{I} = 0$. This means that the number of actively infected people reaches its maximum at herd immunity. Afterwards, $I(t)$ will strictly decrease. Indeed, $\mathcal{R}_t$ will drop below unity, hence $\dot{I} < 0$. Since $I(t) \geqslant 0$ we can infer that $I(t) \to 0$ as $t \to \infty$. We can also deduce the shape of $R(t)$. Since $I > 0$ we have $\dot{R} > 0$ due to (3.16). Therefore, $R(t)$ is a strictly increasing function with time. Since $I(t) \to 0$ as $t \to \infty$ it follows that $\dot{R} \to 0$ as $t \to \infty$. Hence, the $R(t)$-curve will flatten towards the end of the pandemic and converge to a value smaller than N.

In Figure 4 we present the numerical solutions to eqs. (3.14) to (3.16) for $\gamma = 0{,}1\ \mathrm{d}^{-1}$ and $\mathcal{R}_0 = 3$. These are approximately parameters that are related to the COVID-19 pandemic. The first graph shows the full solution. Indeed, we precisely observe the functional behaviour described above. The second plot demonstrates that at an early stage of the pandemic the function $I(t)$ can be approximated by an exponential law given by (2.18).

The latter observation can be justified as follows: as long as $S(t) \simeq S(0)$ we can approximate $\mathcal{R}_t \simeq \mathcal{R}_0$. Then, (3.15) reads

$$\dot{I} = \gamma\,(\mathcal{R}_0 - 1)\,I \tag{3.17}$$

Since for a short period of time $\gamma\,(\mathcal{R}_t - 1)$ is approximately constant, this differential equation has a very simple solution. The solution is precisely (2.18) as we can readily confirm:

$$I(t) = I(0)e^{\gamma(\mathcal{R}_0-1)t} \implies \dot{I}(t) = \gamma(\mathcal{R}_0 - 1)I(0)e^{\gamma(\mathcal{R}_0-1)t} = \gamma(\mathcal{R}_0 - 1)I(t)\ . \tag{3.18}$$

Recall that when taking the derivative with respect to time we need to use the chain rule. Hence, we proved that (2.18) is a solution to (3.17).

3.4 Herd immunity

An important feature of the SIR-model is *herd immunity*. An intuitive explanation of this phenomenon has been presented in Section 2.3. Now we are in the position to give a more general definition. We say that herd immunity is reached once the number of new infections is balanced by the number of people losing infectivity. That is, herd immunity begins when

$$\gamma\mathcal{R}_t I = \gamma I\ ,$$

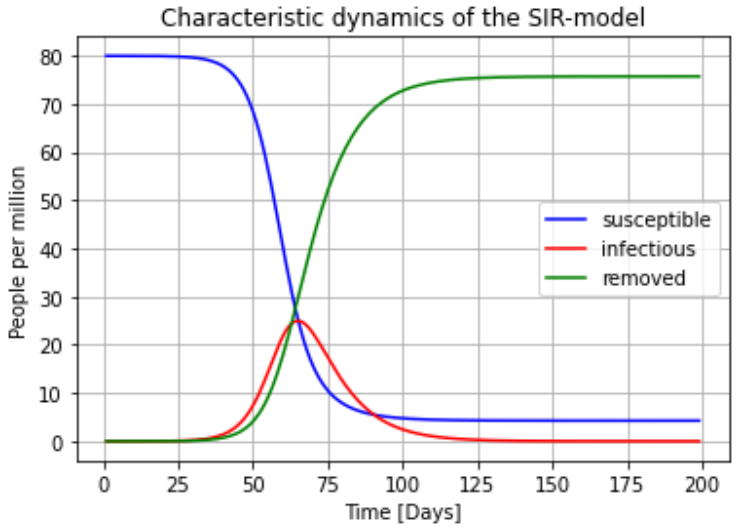
or,

$$\mathcal{R}_t = 1 \tag{3.19}$$

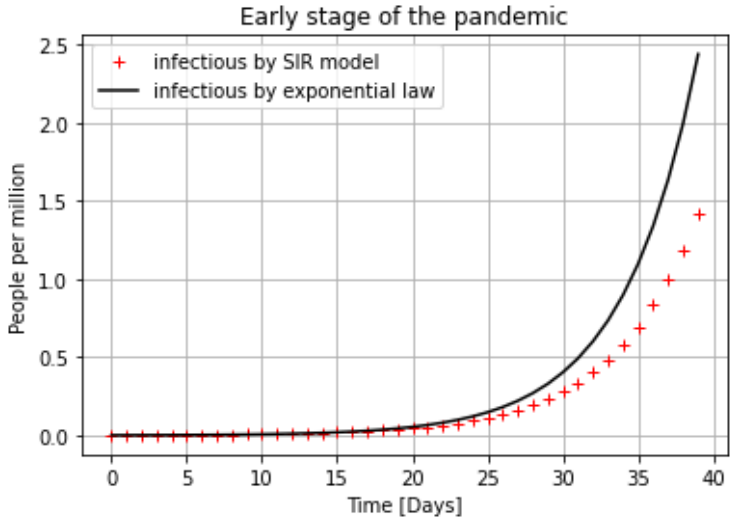
by *natural* dynamics of the pandemic.[2] By (3.13) this translates into

$$\frac{S(t)}{N} = \frac{1}{\mathcal{R}_0}\ . \tag{3.20}$$

[2]Of course, herd immunity is not reached if the reproduction number is lowered by means of containment policies such as shutdowns.



(a) Time evolution of the number of susceptible, infectious and removed people.



(b) At an early stage of the pandemic the function $I(t)$ of the SIR-model (red crosses) can be approximated by an exponential function (black curve). Once the function $S(t)$ starts to drop the approximation fails to hold and the exponential law overestimates the number of infectious people predicted by the SIR-model.

Figure 4: Plots of the characteristic dynamics of the SIR-model for parameter values $\gamma = 0{,}1\ \mathrm{d}^{-1}$ and $\mathcal{R}_0 = 3{,}0$ with initial conditions $N = 80 \cdot 10^6$, $I = 10^3$ and $R = 0$.

Therefore, herd immunity is reached once at most a fraction $1/\mathcal{R}_0$ of the population is susceptible. Or, in other words, herd immunity is reached if a fraction $1 - 1/\mathcal{R}_0$ of the population has had contact with the virus. Again, it follows (2.19):

$$q_{\mathrm{H}} = 1 - \frac{1}{\mathcal{R}_0} \tag{3.21}$$

Herd immunity can be easily read off from Figure 4. Since (3.19) implies $\dot{I} = 0$ (see (3.15))

herd immunity is reached at the maximum of $I(t)$. In the scenario shown herd immunity would be reached within approximately two months. At that time, $S(t)$ has dropped to about $N/3$ as expected for $\mathcal{R}_0 = 3$.

3.5 Doubling time

We just showed that the early stage of a pandemic is governed by an exponential growth in the infectious population. The rapidity of the growth is determined by $\mathcal{R}_0$ (which implicitly contains the parameter γ as well). A more intuitive parameter for laymen may, however, be the *doubling time* t_d. It is defined by the time span during which the infection numbers have doubled. Hence, $I(t_\mathrm{d}) = 2I(0)$, and from (2.18) it follows

$$2 = e^{\gamma(\mathcal{R}_0 - 1)t_\mathrm{d}} \tag{3.22}$$

Taking the natural logarithm on both sides we can solve for t_d:

$$t_\mathrm{d} = \frac{\ln(2)}{\gamma(\mathcal{R}_0 - 1)} \tag{3.23}$$

Hence, the doubling time is not at all a new parameter, but just a re-expression of existing parameters of the SIR-model. It is certainly much less abstract than the basic reproduction number. For instance, in our example of Figure 4 with $\mathcal{R}_0 = 3{,}0$ and $\gamma = 0{,}1\ \mathrm{d}^{-1}$ we get $t_\mathrm{d} \simeq 3{,}5$ d. In particular, we can then easily infer that the infectious population grows by a factor of four within one week.

3.6 The incidence

An important quantity during a pandemic is the number of new infections per day. In the SIR-model the rate of new infections is given by

$$\dot{I}_\mathrm{new}(t) = \gamma \mathcal{R}_t I(t)\ . \tag{3.24}$$

A countries health care capacity may easily get exhausted once $\dot{I}_\mathrm{new}$ exceeds some critical value.

In epidemiology the rate of new infections is normalised by the population size N per 10^5 inhabitants within some time span. This is the *incidence* $\mathcal{I}$. The time span in the COVID-19 pandemic is chosen to be one week, hence:

$$\mathcal{I} = \dot{I}_\mathrm{new} \times \frac{10^5}{N} \times 7\ \mathrm{d}\ . \tag{3.25}$$

Note that $\mathcal{I}$ is essentially determined by $\mathcal{R}_t$ and γ. The plot in Figure 5 shows the incidence curve $\mathcal{I}(t)$ for the same parameters and initial conditions as in Figure 4. One can read off that the 7-days incidence can grow as large as $3 \cdot 10^4$. The peak is reached *before* herd immunity.

Aside – We explain mathematically why the peak of $\mathcal{I}(t)$ is reached before herd immunity. This is just a technical argument that can be skipped.

The incidence $\mathcal{I}(t)$ is extremal for $\dot{\mathcal{I}} = 0$. This implies $\ddot{I}_\mathrm{new} = 0$. Then, by applying product rule to (3.24),

$$0 = \dot{\mathcal{R}}_t I(t) + \mathcal{R}_t \dot{I}(t)\ . \tag{3.26}$$

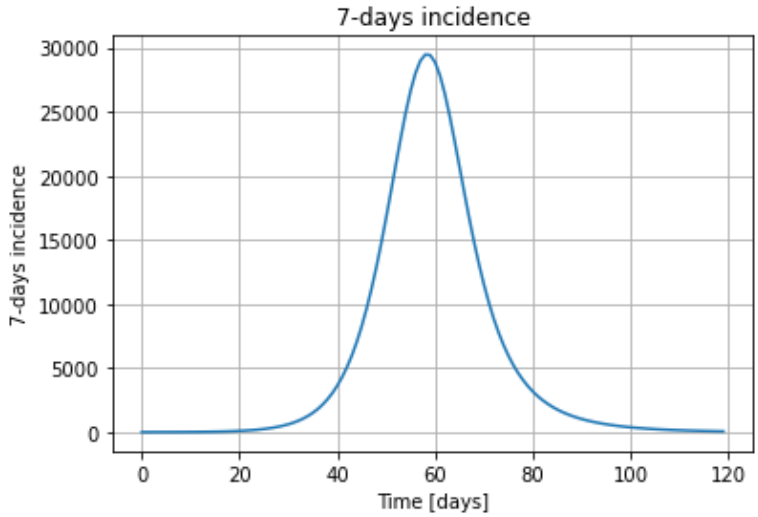


Figure 5: The 7-days incidence from the SIR-model for $\gamma = 0{,}1\ \mathrm{d}^{-1}$ and $\mathcal{R}_0 = 3{,}0$ with initial conditions $N = 80 \cdot 10^6$, $I = 10^3$ and $R = 0$.

Hence,

$$\dot{I}(t) = -\frac{\dot{\mathcal{R}}_t}{\mathcal{R}_t} I(t) > 0 \ . \tag{3.27}$$

The inequality follows because $\mathcal{R}_t > 0, I(t) > 0$ and $\dot{\mathcal{R}}_t < 0$ (recall that this follows from the fact that $S(t)$ is strictly decreasing).

Hence, we proved that $\dot{I}(t) > 0$ when the incidence curve peaks. Therefore, the incidence becomes maximal before herd immunity.

3.7 Final total number of infections

A common misconception is the assumption that the total number of infections at the end of a pandemic is given by $q_{\mathrm{H}} N$, i.e. by the fraction for herd immunity. This is wrong as already explained in Section 2.3. It can also be seen by looking at the first plot in Figure 4. The function $S(t)$ converges to approximately $4{,}3 \cdot 10^6$ which corresponds to 5,4%. Consequently, 94,6% of the population are getting infected for $\mathcal{R}_0 = 3$. This is significantly more than $q_{\mathrm{H}} = 67\%$.

A systematic computation of the total number of infections during a pandemic requires to find a solution to the following equation:

$$1 - \alpha - e^{-\mathcal{R}_0 \alpha} = 0 \ , \tag{3.28}$$

where

$$\alpha := \lim_{t \to \infty} \frac{R(t)}{N} \tag{3.29}$$

is the ratio of the total population getting infected during the pandemic. Note that taking $t \to \infty$ is precisely the strategy how we read off $\alpha = 94{,}6\%$. The derivation of (3.28) is explained in the aside below.

Aside – We derive (3.28). The key idea is to divide (3.1) by (3.3) to get a differential equation for S and R:

$$\frac{dS}{dR} = -\frac{\beta}{\gamma} S \ . \tag{3.30}$$

We can solve this differential equation by separation of variables and integration:

$$\int_{S(0)}^{S(t)} \frac{dS}{S} = -\frac{\beta}{\gamma} \int_{R(0)}^{R(t)} dR \ . \tag{3.31}$$

We simply get

$$\ln\left(\frac{S(t)}{S(0)}\right) = -\frac{\beta}{\gamma} \left(R(t) - R(0)\right) \ . \tag{3.32}$$

We choose the initial conditions $R(0) = 0$ and $S(0) \simeq N$. Using (3.12) it follows:

$$\frac{S(t)}{N} = \exp\left(-\mathcal{R}_0 \frac{R(t)}{N}\right) \ . \tag{3.33}$$

Now we take $t \to \infty$ on both sides,

$$\lim_{t\to\infty} \frac{S(t)}{N} = \lim_{t\to\infty} \exp\left(-\mathcal{R}_0 \frac{R(t)}{N}\right) . \tag{3.34}$$

Note that $\lim_{t\to\infty} S(t) = 1 - \lim_{t\to\infty} R(t)$. Applying (3.29) yields

$$1 - \alpha = e^{-\mathcal{R}_0 \alpha}, \tag{3.35}$$

as asserted.

The easiest way to solve (3.28) is by numerical methods. The plot in Figure 6 shows the solutions as a function $\alpha = \alpha(\mathcal{R}_0)$. The plot starts for basic reproduction numbers $\mathcal{R}_0 > 1$ because below unity no pandemic can occur. This plot illustrates the danger of virus infections with large basic reproduction numbers: a large $\mathcal{R}_0$ does not only accelerate the infection rate, it also results in a larger number of total infections and hence more deaths. This can lead to the paradoxical situation that a virus infection with high case fatality rate but small basis reproduction number results in fewer deaths than a virus infection with small case fatality rate but large basis reproduction number.

3.8 Comparison of COVID-19 and Influenza

Now we can collect our main results and compare COVID-19 with typical types of influenza. To be more concrete we choose to compare with influenza A virus subtype H1N1 (swine influenza). Note that the comparison will be made only from the viewpoint of epidemiology without taking into account medical aspects (such as long-term damages).

From Table 1 we see that, according to the current knowledge, COVID-19 may result in approximately $2-20$ times more deaths than swine flu. This is not only because of the larger infection fatality rate of COVID-19 but also due to the larger basic reproduction number resulting in a higher total number of infections.

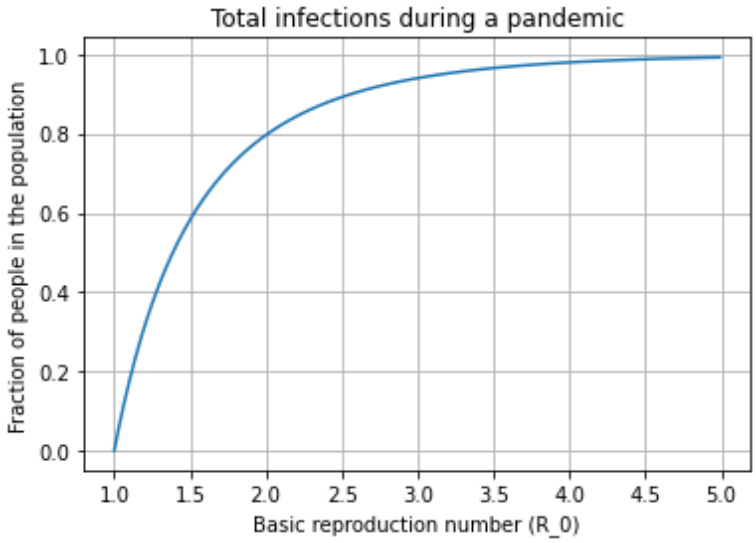


Figure 6: Solution to eq. (3.28) as a function $\alpha = \alpha(\mathcal{R}_0)$.

	Influenza (H1N1)	COVID-19
$\mathcal{R}_0$ [20–24]	1,4 – 1,6	1,4 – 5,7
Days of infectivity [25]	7 d	10 d
Doubling time	8,1 – 12,1 d	1,5 – 17,3 d
Herd immunity q_{H}	28,6% – 37,5%	28,6% – 82,5%
Total fraction of infections α	51,1% – 64,2%	51,1% – 99,7%
Infection fatality rate [26]	0,1%	0,23% – 1,15%
Potential deaths in Germany per 1 000	42 – 53	97 – 950

Table 1: Comparison of epidemiological parameters for swine flu (H1N1) and COVID-19.

3.9 Limitations of the SIR-model

Like any model the SIR-model is only an approximation of the real world. In this section we discuss some of the main limitations of the SIR-model:

1. Usually it takes some time before a newly infected person becomes infectious. In the SEIR-model the SIR-model is extended by the state "*exposed*", in which a person is infected but not yet infectious.

2. The (plain-vanilla) SIR-model does not make any statement about the course of infection. In particular, it does not involve asymptomatic cases and deaths.

3. The SIR-model ignores the possibility of re-infections. We will cover this scenario in Section 8.

4. The SIR-model ignores the propagation of the infection by motion (diffusion). In particular, the SIR-model assumes that any two persons of a population can get in contact with each other. This is clearly not realistic.

5. The SIR-model ignores demographic structures such as population dynamics, urbanity etc.

6. The SIR-model assumes an equal basic reproduction number for every individual of a population. More realistic is some distribution of values of $\mathcal{R}_0$ reflecting different behaviour of individuals.

7. The SIR-model ignores reactions of the population on the pandemic: a population may start to react with containment policies such as quarantine, social distancing etc. This is not reflected in the original SIR-model.

Despite these limitations the SIR-model can be seen as a basic framework for more sophisticated models tailored to the COVID-19 pandemic, see for instance [3; 4].

The SIR-model can still be useful: it captures the basic dynamics of the COVID-19 pandemic, see e.g. the phenomenon of herd immunity. Furthermore, it can be used to make rough estimates such as the doubling time or the duration of a shutdown. Nevertheless, it is too naive to consider calculations such as those in Figure 4 as predictions.

4 The effective reproduction number from actual data

This section demonstrates how one can read off the effective reproduction number from actual data on active infections. Since the test activities on weekends are lower we will only calculate the 7-days (geometric) moving average of the effective reproduction number.

Note that within one week $S(t)$ will not change significantly compared to the total population N. Therefore, we can apply (2.18) replacing $\mathcal{R}_0$ by $\mathcal{R}_t$:

$$I(t) = I(t - \Delta t)e^{\gamma(\mathcal{R}_t - 1)\Delta t} \tag{4.1}$$

In this equation $\mathcal{R}_t$ is the Δt-days moving average of the reproduction number. We solve for $\mathcal{R}_t$ and set $\Delta t = 7$ d:

$$\mathcal{R}_t = 1 + \frac{1}{\gamma \Delta t} \ln\left(\frac{I(t)}{I(t - \Delta t)}\right) = 1 + \frac{1}{\gamma \cdot 7 \text{ d}} \ln\left(\frac{I(t)}{I(t - 7 \text{ d})}\right) \ . \tag{4.2}$$

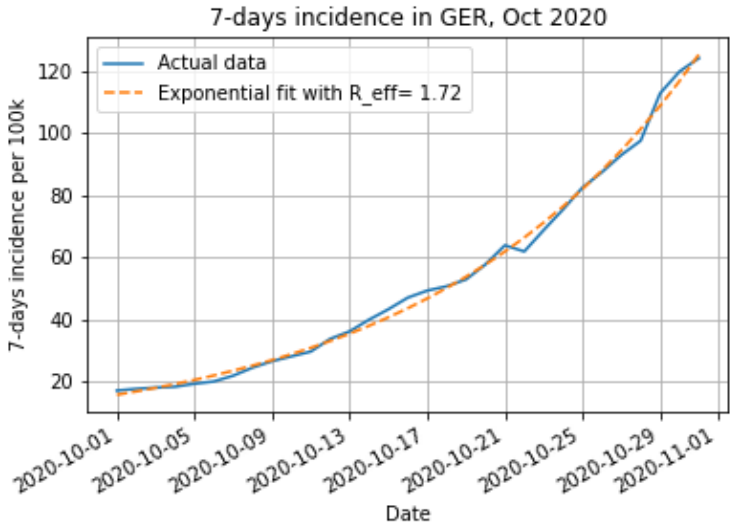
For COVID-19 one can estimate $\gamma \simeq 0{,}1 \text{ d}^{-1}$. From the data one can read off $I(t - 7 \text{ d})$ and $I(t)$. Of course, the data only contain recorded cases. But within this short period we can assume that the proportion of unrecorded cases is approximately the same during one week.

Because of (3.24) we can also obtain $\mathcal{R}_t$ from the incidence:

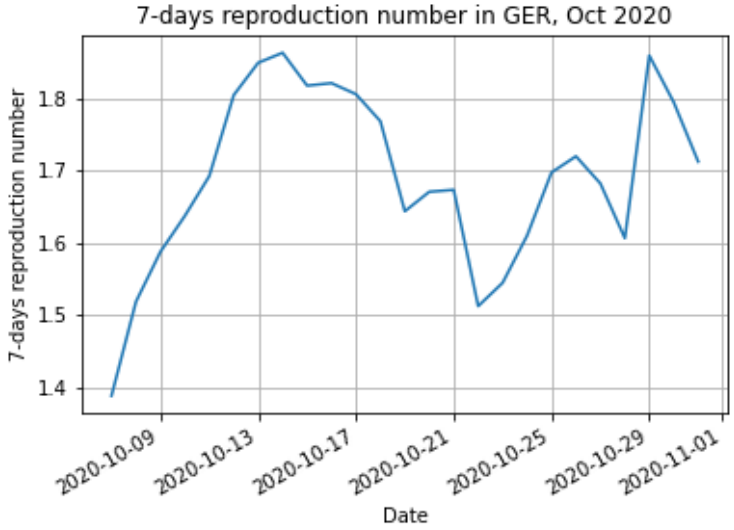
$$\mathcal{R}_t = 1 + \frac{1}{\gamma \cdot 7 \text{ d}} \ln\left(\frac{\mathcal{I}(t)}{\mathcal{I}(t - 7 \text{ d})}\right) \ . \tag{4.3}$$

We evaluate the 7-days reproduction number for the published data [27] for Germany, October 2020, see Figure 7. We observe that in October 2020 the incidence values followed an exponential function with average effective reproduction number of $\mathcal{R}_{\text{eff}} = 1{,}72$. However, the 7-days reproduction number has fluctuated a lot in this period (approximately between $1{,}4 - 1{,}9$). This fluctuations explain the small deviations of the actual incidence values from the exponential fit curve.

Of course, the method presented here relies on various simplifications. For a more precise evaluation one also needs to account for testing activities and estimates on the unrecorded cases. Nevertheless the results roughly coincide with [3].



(a) 7-days incidence per 100 000 inhabitants in Germany during October 2020. The orange curve represents an exponential fit.



(b) 7-days reproduction number in Germany during October 2020.

Figure 7: Incidence and reproduction number in Germany during October 2020.

5 The concept of flattening the curve

In Section 3.8 we have seen that COVID-19 is indeed a threatening disease. Therefore, containment policies are necessary. One containment policy since the outbreak of the pandemic has been the concept of *flattening the curve* shown in Figure 5.

5.1 Why flattening the curve?

First of all we want of argue why the incidence curve in Figure 5 needs to be flattened at all. We focus on three major reasons:

Capacities of the health care system

To define capacities for the health care system is clearly difficult. In particular, it is actually necessary to define different capacities for family doctors, hospitals, ICUs etc. Moreover, capacities are determined by various parameters, such as available staff or infrastructure.

For the purpose of estimating the order of magnitude of the largest acceptable incidence value, we make the following simplification. We only consider ICUs and assume that the number of available beds (plus medical equipment) is the limiting factor (see also [19]). We assume that an infected person has a 5% chance of ending in an ICU staying there for 10 days. Furthermore, we assume that Germany's ICUs can treat at most 30 000 patients, from which at least 50% are non-COVID-19 patients. Then, the upper bound on the incidence value is

$$\mathcal{I}_{\text{critical}} = \frac{50\% \cdot 30\,000}{5\% \cdot 10} \times \frac{7 \cdot 10^5}{83{,}9 \cdot 10^6} \simeq 250 \ . \tag{5.1}$$

For comparison: in Germany the situation in hospitals has been critical during December 2020 where the peak in the incidence values was at $\mathcal{I} \simeq 215$. Therefore, our coarse estimate of $\mathcal{I}_{\text{critical}} = 250$ seems to be the right order of magnitude. From Figure 5 it is easy to see that this critical value would be easily exceeded by a factor of 100 without any containment measures. Once the incidence values exceed the above critical value, a rise in the infection fatality rate is very likely.

Absence at work due to sickness of professionals

An infection dynamics such as illustrated in Figure 5 would result in significant absenteeism due to sickness. Here we want to estimate the order of magnitude of sick professionals during the pandemic. We work with the parameters used in Figure 4 and Figure 5, i.e. $\mathcal{R}_0 = 3{,}0$. Furthermore, we assume that 50% of the population are professionals. In addition, we assume 40% of asymptomatic cases. We assume that those professionals will be unaware of their infection and keep going to work. Moreover, in 30% of cases, the infected persons are assumed to be sick for one and two weeks, respectively. In Figure 8 it is shown the number of absent professionals (in million) due to sickness from COVID-19 infection. At the peak there would be approximately 2,25 million sick professionals just due to COVID-19. There would be a period of 29 days during which more than 1 million professionals are absent from work. If infections take place in systematically relevant departments, the pandemic might cause huge (socio-)economic damages. Consequently, it would also be harmful for the economy to completely ignore the pandemic (see [28] for an extensive discussion of the impact of the pandemic on health and economy).

Contact tracing

Contact tracing may allow to control the pandemic, but it obviously requires a rather small incidence value. We try to estimate an upper bound on the incidence value with a simple model. We denote by τ the time needed for successful contact tracing of one single case. We then assume that the infectious people involved in one case will be separated from the susceptible population and modify (3.15) as follows:

$$\dot{I}(t) = \gamma \left(\mathcal{R}_t - 1\right) I(t) - \frac{1}{\tau} I(t) \ . \tag{5.2}$$

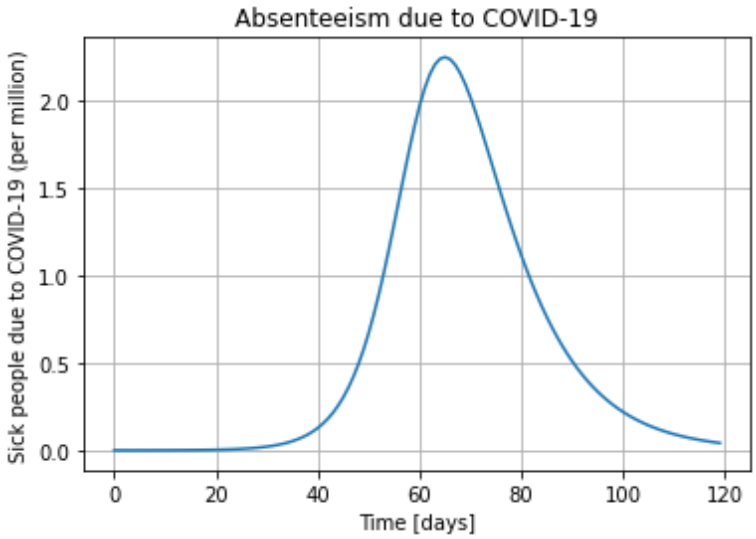


Figure 8: Absenteeism at work with $\mathcal{R}_0 = 3{,}0$ and no containment measures.

In order to achieve $\dot{I}(t) < 0$ by contact tracing we need

$$\tau < \min_{t \geqslant 0} \frac{1}{\gamma(\mathcal{R}_t - 1)} = \frac{1}{\gamma(\mathcal{R}_0 - 1)} \ . \tag{5.3}$$

Taking $\mathcal{R}_0 \simeq 3$ and $\gamma \simeq 0{,}1\ \mathrm{d}^{-1}$, we can deduce

$$\tau \lesssim 5 \text{ days} \ . \tag{5.4}$$

This result means that one case needs to be solved within 5 days of the day of infection. The efficiency of contact tracing therefore needs to be

$$\text{efficiency} > \frac{I(t)}{\tau} > \frac{I(t)}{5 \text{ days}} \tag{5.5}$$

cases per day.

Case Study – As of January 25th, 2021 the number of actively infected Germany is $I = 275\,432$. Therefore, the efficiency of contact tracing would need to be

$$\text{efficiency} > \frac{275\,432}{5 \text{ days}} \simeq 55\,000 \text{ cases per day} \ . \tag{5.6}$$

Hence, the number of actively infected people at that date is still too large to control the pandemic.

What is the largest possible incidence value in Germany to control the pandemic by contact tracing only? If we assume that the largest possible efficiency is 1 000 per day, then $I(t) < 5\,000$. By (3.24) with $\mathcal{R}_0 = 3$ and $\gamma = 0{,}1\ \mathrm{d}^{-1}$ we get $\dot{I}_{\text{new}} < 0{,}1\ \mathrm{d}^{-1} \times 3 \times 5\,000 = 1\,500\ \mathrm{d}^{-1}$. This corresponds to a 7-days incidence of $\mathcal{I} \lesssim 12{,}5$. Note that this result is sensitive to factors of order one. Nevertheless, this rough estimate suggests that the incidence must be smaller than $\mathcal{I} < 3 - 50$ (accounting for factors of order one).

From (5.5) it becomes clear that the number of active infections must be kept below some value given by the efficiency of the contact tracing system. The case study for Germany suggests that the incidence should be kept below $\mathcal{I} < 3 - 50$ to make contact tracing work.

5.2 How to flatten the curve?

Having provided three arguments supporting the concept of flattening the curve, we now explain how this idea may work in principle. Since the reproduction number has significant impact on the dynamics of a pandemic, we focus on methods how to suppress the effective reproduction number.

We recall (3.13):

$$\mathcal{R}_t = \frac{\beta}{\gamma} S(t) \ . \tag{5.7}$$

A priori there are three independent options to reduce $\mathcal{R}_t$:

1. Reduction of $S(t)$.
2. Reduction of the transmission rate β.
3. Reduction of the duration of infectivity, i.e. increasing γ.

Option 1: Examples for reducing $S(t)$ would be via isolation. If Q susceptible people are isolated from the remaining population the reproduction number will be reduced by $-\beta Q/\gamma$. Another example is via vaccination. If V people get vaccinated, then the reproduction number is reduced by $-\beta V/\gamma$.

Option 2: The transmission rate β can be reduced e.g. by wearing masks, keeping enough distance from other people or by following certain rules on hygienic standards.

Option 3: The parameter γ can be increased for instance by suitable medication or other medical treatment lowering the duration of infectivity.

The containment policies in Germany mainly combine options 1 and 2. In particular, the concept of social distancing reduces the number of new infections βSI, because there is less social interaction.

5.3 Flattening the curve by how much?

Having listed some options how to reduce the reproduction number, the subsequent question to answer is the following: by how much do we need to reduce $\mathcal{R}_t$? Clearly, $\mathcal{R}_t < 1$ is sufficient, because this ensures $\dot{I} < 0$ right away (this option is studied in Section 6). But what is the largest possible value of $\mathcal{R}_t$ to keep the curve sufficiently flat?

In order to answer this question we assume that the major criterion is to avoid the collapse of the health care system. As estimated in Section 5.1 we work with an upper bound of $\mathcal{I}_{\text{critical}} = 250$.

From Figure 9 we can read off that $\mathcal{R}_t = 1{,}09$ is the largest possible effective reproduction number to remain within the capacities of the German health care system. Remarkably, the height of the peaks is very sensitive to fluctuations of the reproduction number by 1%. In [19] this is referred to the *fine-tuning problem* of the strategy flattening the curve. Furthermore, the time scale of the pandemic for $\mathcal{R}_t < 1{,}09$ is very large (more than 2 years). Therefore, flattening the curve requires huge efforts from the society for a long period of time.

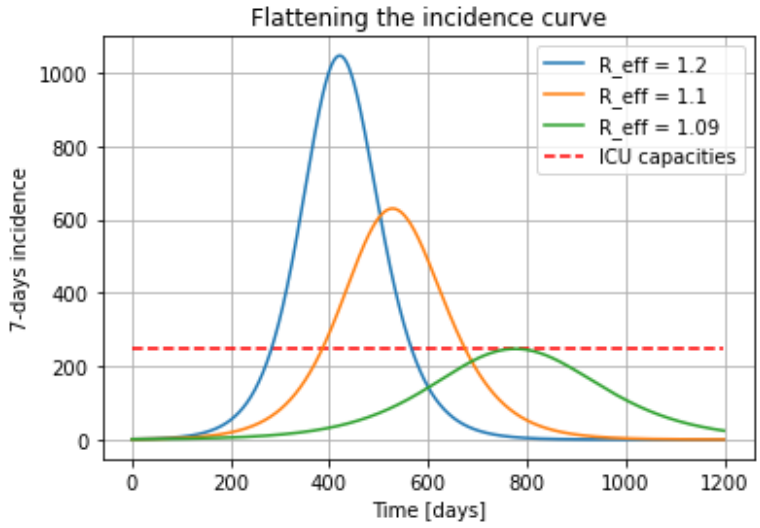


Figure 9: Incidence curves for $\mathcal{R}_t$ between 1,09 and 1,2. For $\mathcal{R}_t > 1{,}09$ the pandemic will exceed ICU capacities.

6 The hammer: Stopping the curve with shutdowns

In this section we consider containment strategies aiming at $\mathcal{R}_t < 1$. This can be achieved by shutdowns. These shutdowns then aim at (temporally) *stopping the curve*. In [29] such shutdowns have also been labelled as *the hammer*. We will analyse the effect of shutdowns in data for several countries. The results are then used to establish a formula to estimate the duration of a shutdown. Finally, we discuss exit strategies from shutdowns.

6.1 Effect of shutdowns in data

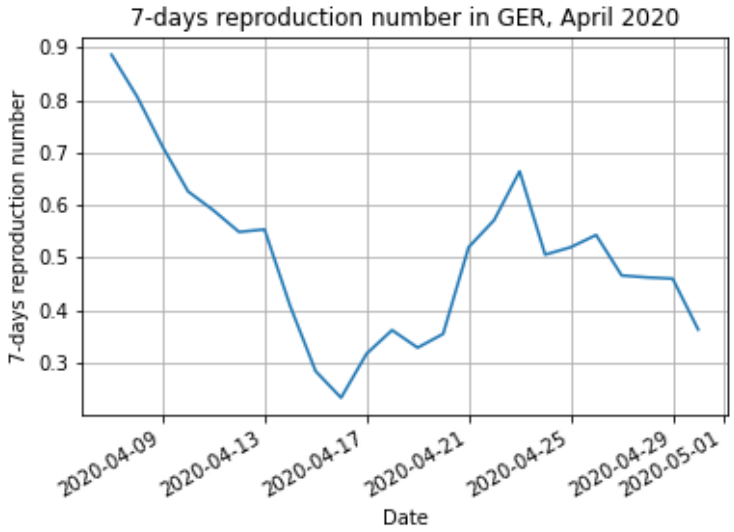
It is a priori not easy to estimate the effective reproduction number during a shutdown. Hence, we determine $\mathcal{R}_{\text{eff}}$ during the shutdown in Germany during April 2020 using the methods presented in Section 4.

We find that the 7-days reproduction number has fluctuated between less than 0,3 to 0,9 during the shutdown in April 2020 in Germany, see Figure 10. The mean effective reproduction number can be obtained by fitting the exponential law (2.18) into the data on the 7-days incidence. The result is $\mathcal{R}_{\text{eff}} \simeq 0{,}71$ assuming that $\gamma = 0{,}1\ \text{d}^{-1}$. This corresponds to a half-life time of

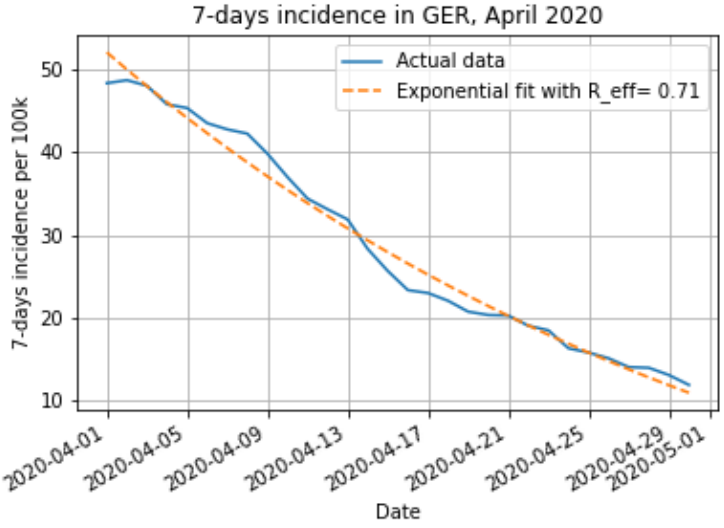
$$t_{\text{h}} = \frac{\ln(2)}{\gamma(1-\mathcal{R}_{\text{eff}})} \simeq 24 \text{ days} . \tag{6.1}$$

This formula can be obtained in the same way as the doubling time (3.23).

Naively, we would then conclude that within 48 days the incidence value has dropped from 50 to 12,5. But in fact, on May 18th 2020 the 7-days incidence has dropped to 5. Indeed, the average effective reproduction number for the time span of April 1st to June 15th was $\mathcal{R}_{\text{eff}} \simeq 0{,}48$ (see Figure 11), implying a half-life time of $t_{\text{h}} \simeq 13$ days. Therefore, during spring/early summer 2020 the half-life time of the incidence curve in Germany had fluctuated significantly.



(a) 7-days effective reproduction numbers during the shutdown in Germany in spring 2020.



(b) Mean effective reproduction number during the shutdown in Germany in spring 2020 from an exponential fit on the 7-days incidence.

Figure 10: Data for the shutdown in Germany in spring 2020.

To conclude, we have seen that a shutdown can suppress the reproduction number below the critical value of 1, leading to a decline of the incidence values.

6.2 A formula for the duration of shutdowns

Due to the large fluctuations in the data it is notoriously hard to make precise predictions for the dynamics of the pandemic. Nevertheless, we want to provide a formula to estimate the rough order of magnitude of the duration of a shutdown.

Let $\mathcal{R}_{\text{eff}} < 1$ be the effective reproduction number during a shutdown starting at an

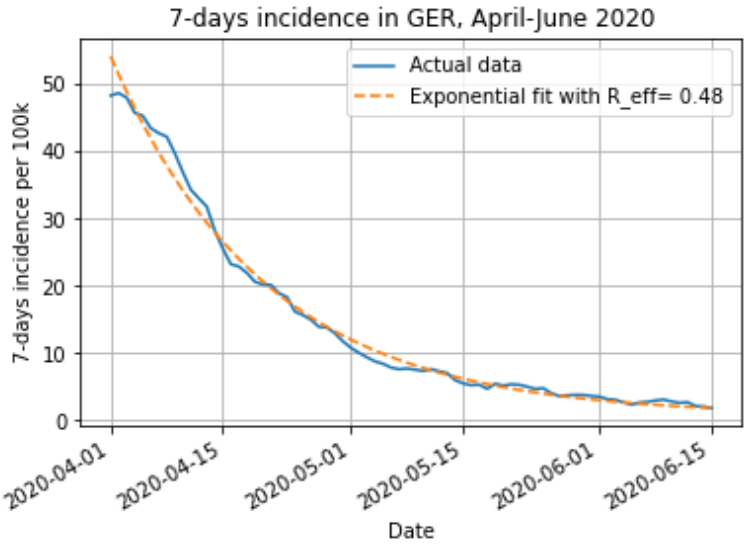


Figure 11: 7-days incidence from April to June 2020 in Germany.

incidence of $\mathcal{I}(0)$. If $\mathcal{I}_{\text{target}}$ is the target incidence, then the exponential law (2.18) reads:

$$\mathcal{I}_{\text{target}} = \mathcal{I}(0) e^{-\gamma(1-\mathcal{R}_{\text{eff}})\Delta t} \ , \tag{6.2}$$

where Δt is the duration of the shutdown. We obtain the formula

$$\Delta t_{\text{shutdown}} = \frac{1}{\gamma(1-\mathcal{R}_{\text{eff}})} \ln\left(\frac{\mathcal{I}(0)}{\mathcal{I}_{\text{target}}}\right) \ . \tag{6.3}$$

Case Study – On December, 23rd 2020 the 7-days incidence per 100 000 inhabitants was $\mathcal{I}(0) = 215$. Assuming $\mathcal{R}_{\text{eff}} = 0{,}7$ the duration of a shutdown with the goal $\mathcal{I}_{\text{target}} = 50$ is

$$\Delta t = \frac{1}{0{,}1 \text{ d}^{-1} \times (1-0{,}7)} \ln\left(\frac{215}{50}\right) \simeq 49 \text{ days} \ . \tag{6.4}$$

Hence, the shutdown would terminate on February 9th 2021. Obviously, on December 23rd the assumption of $\mathcal{R}_{\text{eff}} = 0{,}7$ is rather speculative (and roughly motivated by experience from the first shutdown). Nevertheless, this computation provides an idea for the order of magnitude of the duration of the shutdown.

The reader may check as an exercise that $\mathcal{I}_{\text{target}} = 25$ would be reached on March 4th 2021, and $\mathcal{I}_{\text{target}} = 10$ on April 4th 2021.

Case Study – Assuming that $\mathcal{I}_{\text{target}} = 10$ what is largest tolerable 7-days incidence before starting a shutdown with $\mathcal{R}_{\text{eff}} = 0{,}7$ and maximum duration of $\Delta t = 30$ days? The answer is given by

$$\mathcal{I}(0) = \mathcal{I}_{\text{target}} e^{\gamma(1-\mathcal{R}_{\text{eff}})\Delta t} = 10 \times e^{0{,}1 \text{ d}^{-1} \times (1-0{,}7) \times 30 \text{ days}} \simeq 25 \ . \tag{6.5}$$

This result means that we would then have to start the shutdown once the 7-days incidence has exceeded the value of 25 per 100k. This is probably hard to justify politically. Hence, this computation suggests that a short shutdown of one month either has to be much tighter

aiming at a much smaller reproduction number than $\mathcal{R}_{\text{eff}} = 0{,}7$ or a much higher target incidence has to be accepted.

Even though the formula (6.3) is not able to make precise predictions it allows us to draw the following conclusions:

1. The more we suppress $\mathcal{R}_{\text{eff}} < 1$ the faster we can reach the target incidence number.
2. In particular, $\mathcal{R}_{\text{eff}} \lesssim 1$ implies restrictions for a very long time. Hence, one really needs to strive for a very small reproduction number.
3. The bigger $\mathcal{I}(0)$ the longer the shutdown. In other words: the duration of the shutdown or its severity will increase as we delay the beginning of the shutdown (at rising incidence).

6.3 Shutdown after shutdown?

Once a shutdown is imposed to contain the spread of the virus one needs to define some exit strategy. A very simple (and maybe naive) strategy is to terminate the shutdown once the incidence has dropped below some target value $\mathcal{I}_{\text{target}}$, which is chosen such that contact tracing is fully possible. In Section 5.1 we estimated $\mathcal{I}_{\text{target}} < 3 - 50$ (for Germany). The three examples Israel, Spain and Ireland illustrate that it is crucial to suppress the incidence as much as possible to avoid a further wave (see Figure 12). Otherwise a subsequent wave is expected within 1-2 months. Indeed, in analogy to (6.3) we can write down a formula to estimate the time span between exit from a shutdown and the subsequent beginning of a new shutdown:

$$\Delta t_{\text{next shutdown}} = \frac{1}{\gamma(\mathcal{R}_{\text{eff}} - 1)} \ln\left(\frac{\mathcal{I}_{\text{critical}}}{\mathcal{I}_{\text{target}}}\right) . \tag{6.6}$$

Case Study – Suppose that $\mathcal{I}_{\text{critical}} = 215$ (see Section 5.1) and $\mathcal{I}_{\text{target}} = 50$. These are realistic figures for Germany. We also assume that $\mathcal{R}_{\text{eff}} = 1{,}2$ (with $\gamma = 0{,}1\ \text{d}^{-1}$) once the shutdown terminates. (This is justified by data on daily infections from July 1st to August 31st 2020 in Germany.) With these figures we obtain

$$\Delta t_{\text{next shutdown}} = \frac{1}{0{,}1\ \text{d}^{-1} \times (1{,}2 - 1)} \ln\left(\frac{215}{50}\right) \simeq 73\ \text{days} . \tag{6.7}$$

If the shutdown terminates once $\mathcal{I}_{\text{target}} = 25$ the same assumptions yield

$$\Delta t_{\text{next shutdown}} \simeq 108\ \text{days} , \tag{6.8}$$

i.e. more than one additional month without shutdown. This is particularly important to notice if the containment strategy aims at bridging the time between winter and spring (assuming that the virus is sensitive to seasonal effects).

However, a more infectious mutation may significantly reduce the bridging time. The reader may check that for $\mathcal{R}_{\text{eff}} = 1{,}7$ we get $\Delta t_{\text{next shutdown}} \simeq 21$ days and $\Delta t_{\text{next shutdown}} \simeq 31$ days for $\mathcal{I}_{\text{target}} = 50$ and $\mathcal{I}_{\text{target}} = 25$, respectively.

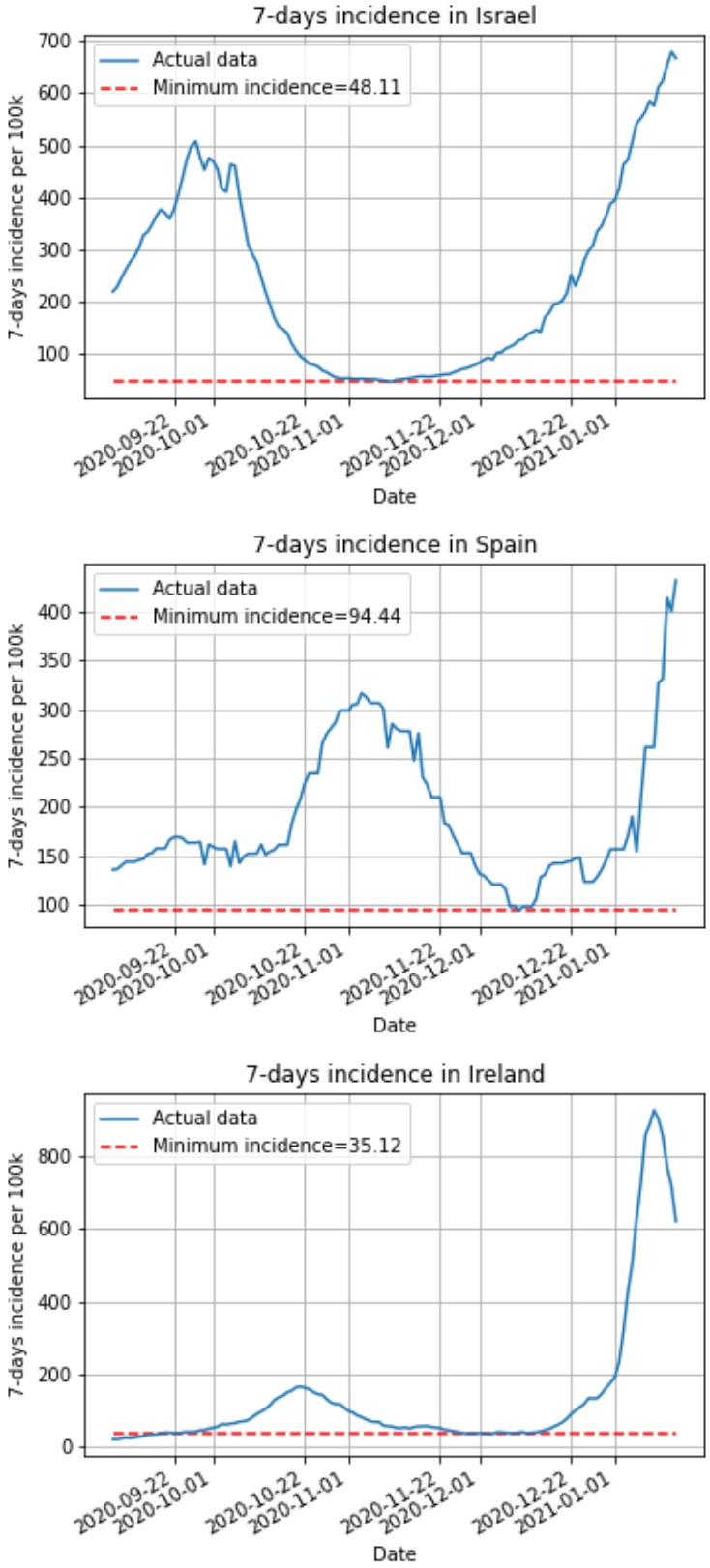


Figure 12: Subsequent waves in Israel, Spain and Ireland from September 2020 to January 2021. The blue curve represents the 7-days incidence per 100 000 inhabitants and the red dashed line represents the minimum incidence *between* two waves.

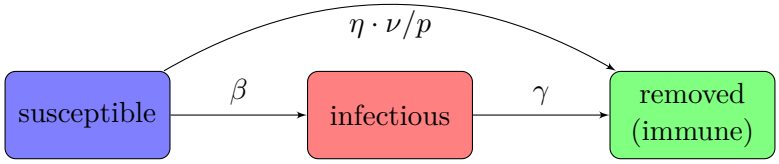


Figure 13: Extension of the SIR-model by vaccination of the susceptible population at a constant rate of $\eta \cdot \nu/p$ with vaccination frequency ν and efficacy η. Here, p is the number of doses needed.

7 The impact of vaccines on the infection dynamics

In this section we estimate the impact of the vaccines on the dynamics of the SIR model. We start with an analytical approach aiming at an estimate on the order of magnitude of the vaccination frequency (i.e. number of vaccinations per day) to slow down the infection dynamics right away. Afterwards we make more precise estimations using a numerical approach.

In the following we assume for simplicity that the vaccines provide sterile immunity and yield eternal protection. Therefore, successfully vaccinated people will be counted to the removed population, see Figure 13.

7.1 An analytical estimate

Let ν be the vaccination frequency and η the efficacy of the vaccine hat requires p doses (e.g. for the BioNTech vaccine $p = 2$). Then the SIR-model can be extended as follows:

$$\dot{S} = -\gamma \mathcal{R}_t I - \eta\nu/p \ , \tag{7.1}$$

$$\dot{I} = \gamma \left(\mathcal{R}_t - 1\right) I \ , \tag{7.2}$$

$$\dot{R} = \gamma I + \eta\nu/p \ . \tag{7.3}$$

Now we want to address the following question: What is the required order of magnitude of ν to achieve a decline in the incidence values?

Recall the formula (3.24) for the number of new infections:

$$\dot{I}_{\text{new}} = \gamma \mathcal{R}_t I \ , \tag{7.4}$$

where $\mathcal{R}_t$ is the current effective reproduction number. We assume $\mathcal{R}_t > 1$ and ask for the size of ν to ensure $\ddot{I}_{\text{new}} < 0$ (the slope of the number of new infections per day $\dot{I}_{\text{new}}$ should be negative).

By product rule we get

$$\ddot{I}_{\text{new}} = \gamma \dot{\mathcal{R}}_t I + \gamma \mathcal{R}_t \dot{I} \ . \tag{7.5}$$

By (3.12) and (3.13) we get:

$$\dot{\mathcal{R}}_t = \mathcal{R}_0 \frac{\dot{S}}{N} \tag{7.6}$$

Hence,

$$\ddot{I}_{\text{new}} = \gamma \frac{\mathcal{R}_0}{N} \left(-\gamma \mathcal{R}_t I - \frac{\eta\nu}{p}\right) I + \gamma^2 \mathcal{R}_t \left(\mathcal{R}_t - 1\right) I < 0 \ . \tag{7.7}$$

For $I > 0$ we can solve this condition for ν and obtain:

$$\nu > p\frac{N\gamma\mathcal{R}_t}{\eta\mathcal{R}_0}\left[(\mathcal{R}_t - 1) - \mathcal{R}_0\frac{I}{N}\right] \simeq p\frac{N\gamma}{\eta}\frac{\mathcal{R}_t(\mathcal{R}_t - 1)}{\mathcal{R}_0} \ . \tag{7.8}$$

In the last step we assumed that $I \ll N$. Note that this result is very sensitive to the precise value of $\mathcal{R}_t$.

Case Study – We apply this formula (7.8) to Germany ($N = 83{,}9 \cdot 10^6$ people). We assume a 2-dose vaccine ($p = 2$) with efficacy $\eta = 95\%$ (e.g. BioNTech vaccine).

In the first case we assume that $\mathcal{R}_t = 1{,}1$. The number of vaccinations every day in order to achieve a decreasing incidence is (for $\mathcal{R}_0 = 3$):

$$\nu > 2 \times \frac{83{,}9 \cdot 10^6 \times 0{,}1 \text{ d}^{-1}}{0{,}95} \times \frac{1{,}1 \times 0{,}1}{3} \simeq 650\,000 \text{ per day} \ . \tag{7.9}$$

Our estimate would double already for $\mathcal{R}_t = 1{,}2$, because ν grows proportional to $(\mathcal{R}_t - 1)$.

This case study demonstrates that the vaccination of the population against COVID-19 has no chance to reveal an immediate effect on the dynamics of the pandemic.

However, the vaccine is essential to reach herd immunity. It is necessary to vaccinate a fraction of

$$q_\text{V} \geqslant \frac{1}{\eta}\left[\left(1 - \frac{1}{\mathcal{R}_0}\right) - q_\text{infected}\right] \tag{7.10}$$

of the population. Recall that the first term is the standard expression for herd immunity (2.19). We subtract the fraction q_infected of people who got immune due to infection. Since the effectiveness of vaccinations is less than 100% we need to multiply the difference by $1/\eta$.

Case Study – In Germany there are about $2{,}3 \cdot 10^6$ known COVID-19 infections as of February 08, 2021. This implies $q_\text{infected} \simeq 2{,}3/83{,}9 \simeq 2{,}7\%$. Ignoring unregistered cases and assuming $\mathcal{R}_0 = 3$ and $\eta = 95\%$, we obtain $q_\text{V} \geqslant 67{,}3\%$. However, if $\eta = 70\%$ (e.g. AstraZeneca) then $q_\text{V} \geqslant 91{,}3\%$.

A dominant mutation may have huge impact on these figures. Suppose $\mathcal{R}_0 = 4$ (more infectious mutation) and $\eta = 85\%$ (smaller efficacy against the mutation) yields $q_\text{V} \geqslant 85\%$. Any vaccine with $\eta < 72\%$ would then require vaccination of the full population.

How long does it eventually take to reach herd immunity by vaccination? The answer depends on the vaccination frequency ν. Let ν be constant. Then the time it takes towards herd immunity is

$$\Delta t_\text{herd immunity} = \frac{q_\text{V} N}{\nu} p \ . \tag{7.11}$$

Case Study – What is the required vaccination frequency ν in order to reach herd immunity in Germany by September 21st 2021? For simplicity, we assume that $q_\text{V} = 70\%$ using a 2-dose vaccine. Furthermore, we assume that 28 days need to elapse until the vaccine takes full effect. Starting from February 5th this implies $\Delta t_\text{herd immunity} = 200$ days. Consequently,

$$\nu = \frac{70\% \times 83{,}9 \cdot 10^6}{200 \text{ days}} \times 2 \simeq 587\,000 \text{ per day} \tag{7.12}$$

or 0,7% of the population per day. Israel has reached 2,1% (end of January 2021) of the population per day, i.e. our estimate is a priori certainly not unrealistic. However, as of early February 2021 Germany has reached a vaccination frequency of 0,12% per day only, i.e. there is still a lot of potential for improvement.

7.2 A numerical estimate

Now we turn to a numerical analysis of the impact of vaccinations in the dynamics of the SIR-model.

For the simulation we assume that the largest acceptable incidence value is $\mathcal{I}_{\text{critical}} = 215$ (see Section 5.1). Once this value is reached, a shutdown is imposed until the incidence drops below $\mathcal{I} = 35$. During the shutdown we assume $\mathcal{R}_{\text{eff}} = 0{,}7$ and $\mathcal{R}_{\text{eff}} = 1{,}2$ otherwise. The latter value is justified by looking at the incidence values of August 2020 in Germany. Note that in this simulation there is no smooth transition between $\mathcal{R}_{\text{eff}} = 0{,}7$ and $\mathcal{R}_{\text{eff}} = 1{,}2$. This will result in some discontinuity in the incidence curves. Due to the threat of various more contagious mutations we will analyse also the range $\mathcal{R}_{\text{eff}} = 1{,}2 - 1{,}6$.

Furthermore, we include vaccination of the population by some vaccine such as BNT162b2, i.e. two doses are required to unfold an efficacy of $\eta = 95\%$ within 28 days after the first dose. We assume that successful vaccination implies eternal sterile immunity. For the simulation we suppose that 70% of the population are willing to get vaccinated.

We divide the population into vulnerable and non-vulnerable people with the fraction of vulnerable people being $\alpha_0 = 30\%$. This high-risk group will be vaccinated first. Hence, those successfully vaccinated people will be subtracted from the susceptible population. In the simulation the fraction α of vulnerable people will be updated by computing

$$\alpha(t) = \frac{S_{\text{vulnerable}}(t)}{S_{\text{total}}(t)} \ . \tag{7.13}$$

As α decreases we allow $\mathcal{I}_{\text{critical}}$ to increase, because the fraction of vulnerable people among the new infections is going to decrease. The critical incidence is updated as follows:

$$\mathcal{I}_{\text{critical}}(t) = \mathcal{I}_{\text{critical}}(0)\frac{\alpha_0}{\alpha(t)} \ . \tag{7.14}$$

Consequently, as more people get infected or vaccinated the critical incidence increases over time. It is important to emphasise that this assumption is highly debatable because a sufficiently high incidence among the less vulnerable population has some risky implications, too (see e.g. Section 5.1 and [28]).

The results are depicted in a heatmap in Table 2. Needless to say, a larger vaccination frequency can avoid further weeks of shutdown. We see that as of February 19th, 2021 we expect three more weeks of shutdown until an incidence $\mathcal{I} = 35$ is reached. Depending on the maximum tolerable effective reproduction number $\mathcal{R}_{\text{eff}}$ and the vaccination frequency ν there may be further weeks of shutdown expected in 2021. The number of expected weeks are shown in the heatmap. We observe that for $\mathcal{R}_{\text{eff}} \lesssim 1{,}3$ a vaccination frequency of $\nu \gtrsim 150\,000$ per day is sufficient to avoid further shutdowns. However, from the heatmap it becomes evident that a more contagious mutation of the virus can easily ruin any temporary success achieved by vaccination.

8 Reinfections? The SIRS-model

An important question during the COVID-19 pandemic is to what extent reinfections can occur. It is known that most of the recovered people enjoy some immunity against COVID-19 for an unknown time span.[3] However, the new mutation B 1.135 seems to enhance reinfection

[3]The reader shall be aware that immunity after COVID-19 infection is a highly non-trivial subject. While the initial immunity at early stages after the infection is driven by protection from antibodies, the role of T-cells may play a crucial role in long-term protection. A discussion of these issues goes much beyond the scope of this note.

		Max. $\mathcal{R}_{\text{eff}}$				
		1,2	1,3	1,4	1,5	1,6
Vaccination frequency ν	100.000	3	8	13	18	23
	150.000	3	3	8	13	18
	200.000	3	3	8	8	13
	400.000	3	3	3	8	8
	600.000	3	3	3	3	8

Table 2: Esimate on the total number of weeks in shutdown in 2021 based on the data from February 19th, 2021. It is assumed that the actual number of closed cases is twice as large as reported. Main results: Three further weeks of shutdown are unavoidable to reach $\mathcal{I} = 35$. Depending on the vaccination rate and the effective reproduction number outside the shutdown, additional weeks of shutdown are expected in 2021. For instance, if $\nu = 150\,000$ per day and if the maximum acceptable effective reproduction number outside the shutdown is $\mathcal{R}_{\text{eff}} = 1{,}5$ then there will be a total of 13 weeks of shutdown, i.e. ten more weeks beyond the current shutdown. The larger $\mathcal{R}_{\text{eff}}$ the more we are risking further shutdowns.

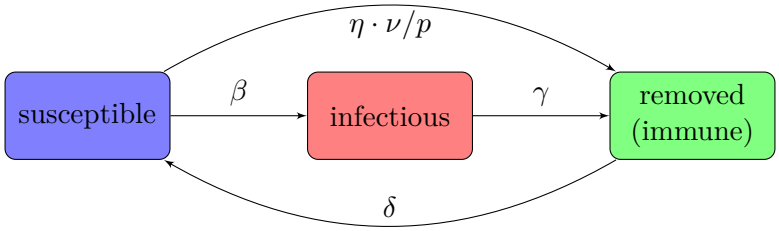


Figure 14: The SIRS-model is an extension of the SIR-model by introducing a characteristic time scale δ^{-1} for the immunity against COVID-19. It is also assumed that susceptible people are getting successfully vaccinated at a rate of $\eta \cdot \nu/p$.

events. Therefore, we extend the SIR-model by limiting the duration of immunity to a time scale of δ^{-1} on average. In addition, we take into account vaccination of the susceptible population at a frequency ν with a p-dose vaccine of efficacy η. The corresponding chain for the model is depicted in Figure 14.

The differential equations for the SIRS-model are a simple modification of the SIR-model:

$$\dot{S} = -\gamma \mathcal{R}_t I + \delta R - \eta\nu/p \ , \tag{8.1}$$

$$\dot{I} = \gamma \left(\mathcal{R}_t - 1\right) I \ , \tag{8.2}$$

$$\dot{R} = \gamma I - \delta R + \eta\nu/p \ . \tag{8.3}$$

The term $\delta \cdot R$ is the number of recovered people losing their immunity per unit time. Therefore, they need to be subtracted from the recovered and added to the susceptible population. The term $\eta \cdot \nu/p$ counts the number of people who are successfully vaccinated each day. They are subtracted from the susceptible and added to the removed population.

In the following we show that there is a dynamic equilibrium at $t \to \infty$ with $I \geqslant 0$. Equality $I = 0$ only holds for sufficiently large vaccination frequencies ν, whose lower bound we will compute. Moreover, for scenarios with too low vaccination frequency we will calculate the long-term fraction of infectious people in the population.

A dynamic equilibrium requires that $\dot{S} = \dot{I} = \dot{R} = 0$. From (8.2) it follows

$$0 = \gamma\,(\mathcal{R}_t - 1)\,I \quad \overset{I>0}{\Longrightarrow} \quad \mathcal{R}_t = 1\ . \tag{8.4}$$

Using (3.13) we find

$$S_{\text{eq}} = \frac{N}{\mathcal{R}_0} \tag{8.5}$$

at the equilibrium. Moreover, from (8.3) it follows

$$0 = \gamma I - \delta R + \frac{\eta\nu}{p} \quad \Longrightarrow \quad R = \frac{\gamma}{\delta} I + \frac{\eta}{p\delta}\nu\ . \tag{8.6}$$

Using

$$N = S + I + R \tag{8.7}$$

we obtain at equilibrium:

$$I_{\text{eq}} = \max\left(\frac{1}{\gamma+\delta}\left[N\left(1 - \frac{1}{\mathcal{R}_0}\right)\delta - \frac{\eta\nu}{p}\right]; 0\right)\ . \tag{8.8}$$

The max-function is needed to ensure that the infection number remains non-negative. One can mathematically show that this is indeed the limit of $I(t)$ as $t \to \infty$. We see that without vaccination we will always have $I > 0$. However, $I(t) \to 0$ for $t \to \infty$ if the daily vaccination rate exceeds

$$\nu \geqslant \frac{p}{\eta} \times N\left(1 - \frac{1}{\mathcal{R}_0}\right)\delta\ . \tag{8.9}$$

Case Study – We compute the vaccination rate ν for Germany if the immunity due to infection or vaccination only holds for one year, i.e. $\delta = 1/365\ \text{d}^{-1}$. Furthermore, we assume $\eta = 90\%$ and two doses are needed. For $\mathcal{R}_0 = 3$ we then obtain

$$\nu \geqslant \frac{2}{0{,}9} \times 83{,}9 \cdot 10^6 \cdot \left(1 - \frac{1}{3}\right) \times \frac{1}{365}\ \text{d}^{-1} \simeq 340\,000\ \text{per day}\ . \tag{8.10}$$

The formula (8.9) reveals the importance of why escape mutations are threatening: these mutations may be more contagious (i.e. bigger basic reproduction number), and by definition of escape mutations vaccinations may be less effective (i.e. η may become smaller) and the period of immunity may decrease (i.e. larger δ). All these aspects enforce a larger vaccination rate.

Next we determine the limit of the incidence $\mathcal{I}(t)$ for $t \to \infty$. We have

$$\lim_{t\to\infty} \dot{I}_{\text{new}}(t) = \lim_{t\to\infty} \gamma\mathcal{R}_t I(t) = \gamma \cdot 1 \cdot I_{\text{eq}} = \frac{\gamma}{\gamma+\delta}\left[N\left(1 - \frac{1}{\mathcal{R}_0}\right)\delta - \frac{\eta\nu}{p}\right] \tag{8.11}$$

at equilibrium. From the definition (3.25) of the 7-days incidence per 100 000 inhabitants we then get:

$$\mathcal{I}_{\text{eq}} = \frac{\gamma}{\gamma+\delta}\left[N\left(1 - \frac{1}{\mathcal{R}_0}\right)\delta - \frac{\eta\nu}{p}\right] \times \frac{10^5}{N} \times 7\ \text{d}\ . \tag{8.12}$$

Case Study – As in the previous case study we assume $\delta = 1/365\ \mathrm{d}^{-1}$, $\eta = 90\%$ and two doses required. However, we now assume that the vaccination rate is $\nu = 200\,000$ per day, i.e. less than the required rate to stop the pandemic. We then get

$$I_{\mathrm{eq}} = \frac{1}{0{,}1 + 1/365}\left[83{,}9 \cdot 10^6 \times \left(1 - \frac{1}{3}\right) \times \frac{1}{365} - 0{,}9 \times 200\,000 \times \frac{1}{2}\right] \simeq 610\,000\ . \tag{8.13}$$

The 7-days incidence at equilibrium is then

$$\mathcal{I}_{\mathrm{eq}} = 0{,}1 \times 610\,000 \times \frac{10^5}{83{,}9 \cdot 10^6} \times 7\ \mathrm{d} = 509\ . \tag{8.14}$$

All these analytical results can be confirmed numerically, see Figure 15. The first two plots show the dynamics for the case study with vaccination rate is $\nu = 200\,000$ per day. Indeed, the incidence curve converges to $\mathcal{I}_{\mathrm{eq}} \simeq 510$, as analytically predicted. The third and fourth plot depict the situation with just enough vaccinations per day, namely $\nu = 340\,000$ per day. Apparently, this suffices to suppress the incidence to (nearly) zero.

9 Summary

In this note we have presented the basics of the SIR-model. Although it relies on several simplifications it is useful to understand the basic mechanics of the COVID-19 pandemic. In particular, we have seen that the model predicts an exponential growth of the infection numbers at the beginning, which is consistent with the data. As the virus spreads out through the population the dynamics naturally slows down resulting in a peak of the infection numbers. This peak is the moment in which herd immunity is reached. For $\mathcal{R}_0 = 3$ the SIR-model predicts herd immunity once 67% of the population has become immune. From then on the virus continues to spread with exponentially decreasing pace until approximately 94% have become immune.

Due to containment policies such as masks, physical distancing and shutdowns the dynamics has slowed down significantly. By analysing various data sets we have seen that in Germany shutdowns were able to suppress the effective reproduction number to $\mathcal{R}_{\mathrm{eff}} \simeq 0{,}7$, which implies a half life time of 23 days. More practically, this implies that the incidence can be suppressed by a factor of two every 3-4 weeks.

In the last parts of this note we have extended the SIR-model by the inclusion of vaccinations. A numerical simulation, in which vulnerable people are getting vaccinated first, shows that more contagious mutations of the original virus may easily enforce further weeks of shutdown in 2021, even for vaccination frequencies of more than 400 000 per day.

Finally, we have started a thought experiment: we assumed that the immunity from infection or vaccination only holds for one year. We saw that this assumption leads to further sudden outbreaks during this decade. We derived a formula to estimate the minimum vaccination frequency to avoid further outbreaks like those. We found that in Germany it is then necessary to vaccinate more than 340 000 people each day in the next year(s).

Disclaimer

The author is not an epidemiologist. This note has been written out of private interest in this topic.

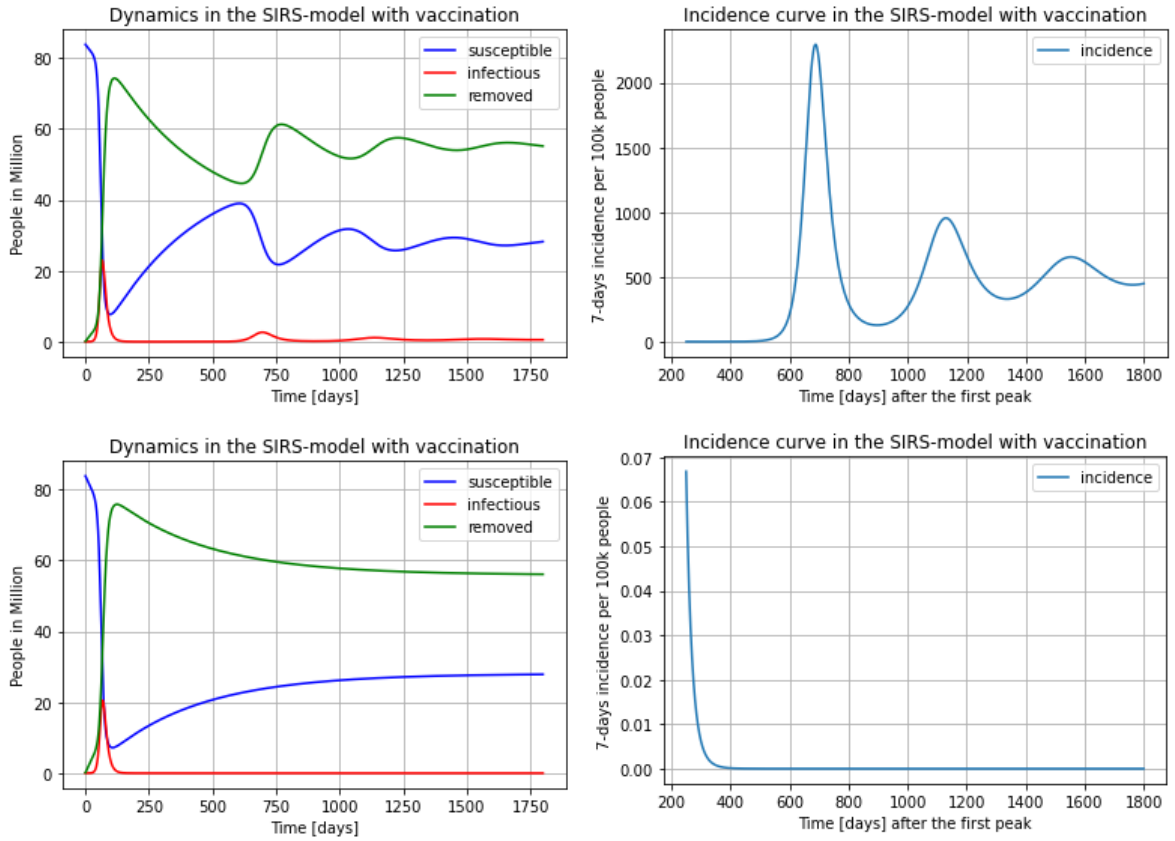


Figure 15: These plots show the dynamics of a SIRS-model with vaccination. Parameter choice: $N = 83{,}6 \cdot 10^6$, $I = 1000$, $R = 0$, $\gamma = 0{,}1$ d^{-1}, $\delta = 1/365$ d^{-1} and $\mathcal{R}_0 = 3$. Regarding the vaccination we assume that two doses are needed and efficacy is $\eta = 90\%$. In the upper example we assume a vaccination frequency of $\nu = 200\,000$ per day. This results in further waves within the next years converging to an incidence of approximately 500. In the lower example we choose $\nu = 340\,000$, which results in a dynamic equilibrium with nearly no further cases. In both examples the incidence curves are plotted for $t > 200$ days because the new features of this model are the subsequent peaks.

References

[1] W. O. Kermack and A. G. McKendrick, "A Contribution to the Mathematical Theory of Epidemics," *Proceedings of the Royal Society of London Series A* **115** no. 772, (Aug., 1927) 700–721.

[2] N. M. Ferguson *et al.*, "Report 9: Impact of non-pharmaceutical interventions (npis) to reduce covid-19 mortality and healthcare demand." https://www.imperial.ac.uk/media/imperial-college/medicine/sph/ide/gida-fellowships/Imperial-College-COVID19-NPI-modelling-16-03-2020.pdf. Imperial College COVID-19 Response Team; Accessed: 2020-05-03.

[3] S. Khailaie, T. Mitra, *et al.*, "Estimate of the development of the epidemic reproduction number Rt from Coronavirus SARS-CoV-2 case data and implications for political measures based on prognostics," *medRxiv* (April 2020) .

[4] J. Dehning, J. Zierenberg, F. P. Spitzner, M. Wibral, J. P. Neto, M. Wilczek, and

V. Priesemann, "Inferring change points in the spread of covid-19 reveals the effectiveness of interventions," *Science* **369** no. 6500, (May, 2020) eabb9789. http://dx.doi.org/10.1126/science.abb9789.

[5] G. Oliveira, "Refined compartmental models, asymptomatic carriers and covid-19,". http://dx.doi.org/10.1101/2020.04.14.20065128.

[6] J. Dolbeault and G. Turinici, "Heterogeneous social interactions and the covid-19 lockdown outcome in a multi-group seir model," arXiv:2005.00049 [q-bio.PE].

[7] D. Kai, G.-P. Goldstein, A. Morgunov, V. Nangalia, and A. Rotkirch, "Universal masking is urgent in the covid-19 pandemic: Seir and agent based models, empirical validation, policy recommendations," arXiv:2004.13553 [physics.soc-ph].

[8] R. Goel and R. Sharma, "Mobility based sir model for pandemics – with case study of covid-19," arXiv:2004.13015 [cs.SI].

[9] F. Ndairou, I. Area, J. J. Nieto, and D. F. Torres, "Mathematical modeling of covid-19 transmission dynamics with a case study of wuhan," *Chaos, Solitons and Fractals* **135** (Jun, 2020) 109846. http://dx.doi.org/10.1016/j.chaos.2020.109846.

[10] C. Bandt, "Transparent covid-19 prediction," arXiv:2004.04732 [q-bio.PE].

[11] H. M. Singer, "The covid-19 pandemic: growth patterns, power law scaling, and saturation," arXiv:2004.03859 [q-bio.PE].

[12] J. M. Carcione, J. E. Santos, C. Bagaini, and J. Ba, "A simulation of a covid-19 epidemic based on a deterministic seir model," arXiv:2004.03575 [q-bio.PE].

[13] G. C. Calafiore, C. Novara, and C. Possieri, "A modified sir model for the covid-19 contagion in italy," arXiv:2003.14391 [physics.soc-ph].

[14] J. E. Amaro, "The d model for deaths by covid-19," arXiv:2003.13747 [q-bio.PE].

[15] L. Dell'Anna, "Solvable delay model for epidemic spreading: the case of covid-19 in italy," arXiv:2003.13571 [q-bio.PE].

[16] L. Squillante, I. F. Mello, A. C. Seridonio, and M. de Souza, "Attacking the covid-19 with the ising-model and the fermi-dirac distribution function," arXiv:2003.11860 [q-bio.PE].

[17] J. D. Murray, *Mathematical Biology I: An Introduction*. Interdisciplinary Applied Mathematics. Springer, 3rd ed., 2007.

[18] J. Murray, *Mathematical Biology II Spatial Models and Biomedical Applications*. Springer, 3rd ed., 2003.

[19] P. Mangat, "A divide and conquer strategy against the covid-19 pandemic?!," *medRxiv* (2020) . https://www.medrxiv.org/content/early/2020/05/09/2020.05.05.20092155.

[20] B. J. Coburn, B. G. Wagner, and S. Blower, "Modeling influenza epidemics and pandemics: insights into the future of swine flu (h1n1)," *BMC medicine* **7** no. 1, (2009) 1–8.

[21] Q. Li *et al.*, "Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus-Infected Pneumonia," *The New England Journal of Medicine* **382** no. 13, (January, 2020) 1199–1207.

[22] C. L. A. Julien Riou, "Pattern of early human-to-human transmission of Wuhan 2019 novel coronavirus (2019-nCoV), December 2019 to January 2020," *Eurosurveillance* **25** no. 4, (2020) .

[23] J. T. Wu *et al.*, "Estimating clinical severity of COVID-19 from the transmission dynamics in Wuhan, China," *Nature Medicine* **26** no. 5, (March, 2020) 506–510.

[24] S. Sanche *et al.*, "High Contagiousness and Rapid Spread of Severe Acute Respiratory Syndrome Coronavirus 2," *Emerging Infectious Diseases* **26** no. 7, (April, 2020) 506–510.

[25] R. K. Institut, "Epidemiologischer steckbrief zu sars-cov-2 und covid-19." https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Steckbrief.html.

[26] N. Brazeau *et al.*, "Report 34: Covid-19 infection fatality ratio: estimates from seroprevalence." https://spiral.imperial.ac.uk/handle/10044/1/83545. Imperial College COVID-19 Response Team.

[27] J. Hasell, E. Mathieu, D. Beltekian, *et al.*, "A cross-country database of COVID-19 testing. ," *Scientific Data* **7** no. 345, (2020) .

[28] F. Dorn, S. Khailaie, M. Stöckli, S. Binder, B. Lange, P. Vanella, T. Wollmershäuser, A. Peichl, C. Fuest, and M. Meyer-Hermann, "Das gemeinsame interesse von gesundheit und wirtschaft: Eine szenarienrechnung zur eindämmung der corona-pandemie," *ifo Schnelldienst Digital* **1** no. 6, (2020) . http://hdl.handle.net/10419/223322.

[29] T. Pueyo, "Coronavirus: The hammer and the dance." https://tomaspueyo.medium.com/coronavirus-the-hammer-and-the-dance-be9337092b56.