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A Simple, Non-Recursive Model of the Spread of Covid-19 with Applications to Policy

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Abstract

The paper presents early research on problems that make the use of the basic SIR-model of epidemiology difficult for short- and medium-run policy. The model essentially generalizes the simple exponential model in two respects. First, it considers the structure of the infectious population in more detail and introduces the concept of the "cohort composition kernel" that generalizes the aggregate transmission function and renders the transmission model non-recursive. Second, it shows how policy measures such as testing, social distancing, or quarantine rules can affect this kernel and how this can provide estimates for the impact and lag of non-pharmaceutical policy interventions.

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Covid-19 “might be a one-in-a-century evidence fiasco"
(J. Ioannidis, STATnews 17/3/20)

1 Introduction

Much of the rapidly developing economic literature on the spread of the Covid-19 pandemic builds on the classic SIR-model of contagion.[1] The simplest version of this model derives the dynamics of transmission in a recursive framework, in which the number of newly infectious individuals in a population (ΔI or $\frac{d}{dt}I$) depends on the number of non-infected individuals (S) who are susceptible to an infection in a reduced-form model of personal encounters.[2] At the early stage of the epidemic where we are currently, the mitigating effect of having increasing numbers of individuals removed (R) from the susceptible group through immunization or death can be neglected. Hence, if Y_t denotes the number of infectious individuals and β the net transmission rate (per period), then new infections are $n_t = \beta Y_{t-1}$, yielding simple exponential growth. This is what we should focus on - if the disease has reached the feedback region in the S-I phase diagram too quickly, our countries' public health systems will have collapsed.

While the model is a useful basis for longer-run macroeconomic analyses,[3] Covid-19 presents (at least) two problems that make an application of the basic exponential model difficult for short- and medium-run policy. First, transmission does not simply depend on the number of infectious individuals, but on the composition of this group, which is influenced by policy. And second, the data at our disposal are quite inadequate to evaluate the evolution of the disease and thus the effectiveness of policy. In fact, in most countries we do not even know the number of infected, not even their magnitude, let alone that of infectious individuals. This state is very unsatisfactory, as politicians must make real-time decisions with dramatic economic and societal consequences based on insufficient data.

This paper presents a simple model that addresses both of these problems by looking in more detail into the structure of the transmission process. It is therefore related to the so-called “compartmental models" of epidemiology (see, e.g., Brauer, 2008), but it differs from them by introducing a non-recursive

[1] The model goes back to Kermack and McKendrick (1927). A timely review of this and related models is Allen (2017) and, just in time, as I have just learned, Stock (April 5, 2020). My favorite description is in Braun (1978, Ch. 4).

[2] Unfortunately, sometimes the acronym is interpreted as “susceptible-infected-recovered". This is too narrow, and the original model (e.g., in Braun, 1978) divides the population more generally in susceptible, infectious, and removed agents. Removal may be recovery, but it is much broader, as it also includes quarantine and other policy measures. On the other hand, infectious is narrower than infected. Most applications of the model that I know of, however, assume that the infected are immediately infectious. The fact that this is not the case with Covid-19 is one of the aspects of the model developed in this paper.

[3] Such as Atkeson (2020) and Eichenbaum, Rebelo, Trabant (2020). This literature grows like $n_t = \beta Y_{t-1}$.

structure, i.e. by abandonning the usual exponentially distributed duration framework. The model identifies the variables which we need to understand and measure better, establishes relations between them that can be used to make efficient use of the data that we can observe, and points to several mechanisms by which policy influences these variables. The model essentially generalizes the relation $n_t = \beta_t Y_t$ in two respects. First, it considers the structure of the infectious population and introduces the concept of the "cohort composition kernel" that generalizes the aggregate transmission function and renders the transmission model non-recursive. Second, it shows how policy measures such as mass testing, social distancing, or quarantine rules can affect this kernel and how this can provide estimates for the impact and lag of non-pharmaceutical policy interventions.

The research presented here is very preliminary and uses data sources until early April. My emphasis is on policy and, of course, I am most familiar with the German data and policy. But the structural problem is the same all over Europe and probably more broadly.[4]

The model presented here is at the same time much simpler than the influential model by Ferguson, Cummings et al. (2005) that is the basis for the simulations recently conducted by Ferguson et al. (March 16, 2020, the "Imperial Study"), and more detailed in some respects, as it explicitly considers the working of parameters that can be used for policy. It thus tries to bridge the gap between the mathematical theories of dynamical systems, the clinical evidence, and the reduced form models used by economists to evaluate the economic consequences of the crisis.

2 Individual Evolution of the Disease

Time is discrete, $t = 0, 1, ...$, and measured in days.

At the individual level, the disease and its consequences evolve in stages after the infection. Suppose infection is at time $t = 0$. The different stages of the disease are then given by the following random times T^z, measured in days after infection.

- T^o (outbreak): time of first clear symptoms (if at all) or undetected outbreak for mild or asymptomatic cases
- $T^n = T^o + \tau^n$ (no more infectious): time until no more contagious if no severe symptoms and not treated

[4] There is a rapidly growing body of clinical evidence, mostly evaluating small sample experiences from China in January and February 2020; and not being an expert, I am much indebted to the summaries provided by the websites of the Centers for Disease Control and Prevention, its German counterpart, the Robert-Koch Institute, or public discussions by virologists, such as Christian Drosten of the Charité at Humboldt University. These are, respectively, https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html, https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Steckbrief.html, and the remarkable podcast https://www.ndr.de/nachrichten/info/Corona-Podcast-Alle-Folgen-in-der-Uebersicht,podcastcoronavirus134.html.

- $T^s = T^o + \min(\tau^n, \tau^s)$ (severe): time until severe symptoms if any
- $T^{ex} = T^s + \tau^{ex}$ (exit): time until end of infection after severe symptoms
- $T^d = T^s + \tau^d$ (death): time until death after severe symptoms

T^o and the τ^z are positive-valued random variables. In case of no clear symptoms or no symptoms at all, T^o is an artificial date to make the subsequent timing comparable.[5]

The above events refer to the evolution of the disease only, not to any interventions. Diagnoses at the time of outbreak are grouped into three types:

- a: asymptomatic (resp., unnoticed by patient)
- m: mild, but clear symptoms (fever, cough, etc.)
- s: severe, potentially life-threatening (acute respiratory distress (ARD), severe pneumonia, lung failure, cytokine release syndrome, etc.)

To simplify the presentation, the model does not distinguish between patients with severe symptoms and critically ill patients. In functioning medical environments the former is usually associated with hospitalization,[6] the latter with progression to ICUs. Clinical data from January/February 2020 indicate that this progression has occured in approximately 30% of all hospitalizations.[7]

If the individual outbreak immediately produces symptoms, we have $\tau^s = 0$, i.e. $T^s = T^o$. For asymptomatic and mild cases, we have $\tau^s > 0$ if severe symptoms occur later, and $\tau^s = \infty$ if not. See Figure 1 for an illustration of the evolution after the outbreak.

Given the current experience, the following probabilities seem reasonable benchmark estimates for the evolution at the time of the initial outbreak, T^o:

$$\theta_a = 0.2 - 0.4 \quad (1)$$

$$\theta_m = 0.5 - 0.6 \quad (2)$$

$$\theta_s = 0.01 - 0.02 \quad (3)$$

Remark: These parameters are highly subgroup-specific (for example, severe cases are highly prevalent in the over 70 yr group), and sub-group-specific estimates are strongly biased.[8] Furthermore, it is not clear how the probabilities can be estimated at all, because the underlying population (all infected individuals) is unobservable.[9] See, e.g., Verity, Okell, Dorigatti et al. (2020) for some discussion of "crude CFRs".

[5] Completely asymptomatic cases seem to be rare, though. Upon careful investigation, most patients are able to identify at least some very mild signs (as in the "Munich study" by Böhmer, Buchholz et al., SSRN preprint 31/3/2020. See Drosten Podcast 24, 30/3).

[6] This was different during the early spread of the disease in Wuhan in January, where hospitalization was also used as a isolation device.

[7] See Ferguson et al. (Imperial College, 16/3/2020) or Centers for Disease Control and Prevention at https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html

[8] This is particularly true for the Wuhan data from January, where the health system was quickly overwhelmed.

[9] There is some important information from natural experiments.
Most impressive is the experiment in Vò (Italy) where the entire population of 3 300 was

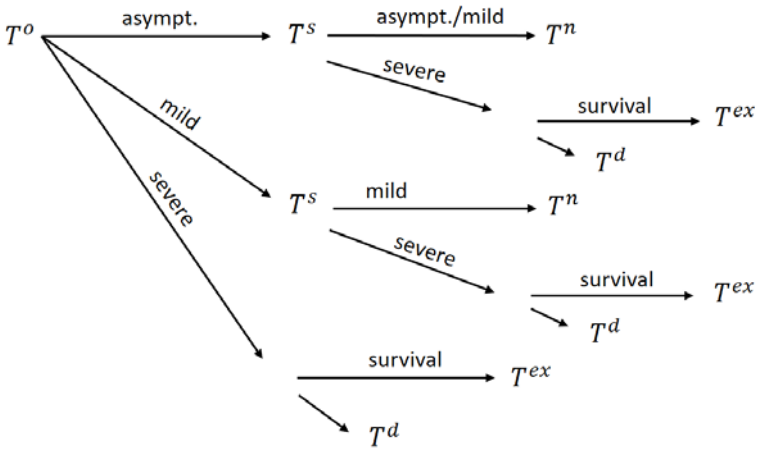


Figure 1: The basic dynamics

The conditional probability of progressing to severe symptoms from initially no or mild symptoms is not systematically documented it seems.[10] Given estimated overall probabilities of developing severe symptoms eventually, it seems realistic to put this probability at

$$\theta_{ms} = 0.02 - 0.05 \tag{4}$$

Together with (3), (4) gives an interval for the overall probability of severe symptoms conditional on infection of $\theta = \theta_s + (1 - \theta_s)\theta_{ms} \in [0.03, 0.07]$.[11]

tested in late February and then again 10 days later. The findings in Vò seem to be consistent with $\theta_a \in [0.3, 0.5]$. Source: Financial Times, 17/3/20.

A well known natural experiment is the case of the cruiseship *Diamond Princess* that was quarantined between Feb. 3 and 20 in Yokohama after a passenger who had disembarked on Jan. 25 tested positive on Feb. 1. Almost all passengers and crew were tested subsequently, and 18% of all positive passengers seem to have had no symptoms (Mizumoto et al., 2020). Given that the average age of passengers was 58, this is consistent with θ_a being well above 20% in broader populations. Numbers differ across reports and studies (perhaps because they were written before the full extent of the data was known). A sufficiently informed (but not complete) report seems to be https://www.cdc.gov/mmwr/volumes/69/wr/pdfs/mm6912e3-H.pdf

According to Ferguson et al. (the Imperial Study), Verity, Okell, Dorigatti et al. (MedArxiv, 09/03/2020) find 40 - 50% unrecognized cases on repatriation flights from China in late January. This finding is not reported in the Verity et al. paper, though.

Interesting estimates along the above lines can be obtained by comprehensive tests in special institutions such as care facilities (see, e.g., Kimball, Arons et al. (2020) for the case of a long-term care facility in Washington).

[10] There is signifiant evidence from Wuhan for the clinical progression of Covid-19 during hospitalization. This is informative for θ_{ms} because in the early phase of the outbreak in Hubei many patients with relatively mild symptoms were hospitalized. An interesting example is Zhou, Yu, Du et al. (2020).

[11] Remember: I do not distinguish between “severe" and “critical". The former is often

A good current estimate for the median incubation period (time until T^o after infection) seems to be[12]

$$T^o = 5 - 6 \tag{5}$$

with most of the mass on the interval $[3, 11]$. I assume that the events during the evolution of the disease are i.i.d. across individuals; in other words, the distributions of all events are not patient specific (while the outcomes are).[13] Let $p^o = (p^o_0, p^o_1, p^o_2, ...)$ be the (discrete density of the) probability distribution of outbreak days after infection (over $\mathbb{N}_0$, $t = 0$ is the day of infection).

For patients with mild symptoms at outbreak, severe symptoms are observed, if at all, τ^s days later, with most of the mass on days $[5, 11]$ after incubation.[14] Let $\pi^s = (\pi^s_0, \pi^s_1, \pi^s_2, ...)$ be the corresponding probability distribution.[15]

For severe cases, probabilities and time to death depend on the clinical environment and can therefore not be pinned down universally and without reference to specific policy. The Hubei studies of January/February 2020 report mortality rates of 10 - 25 % conditional on hospitalization.[16] In any case, I am less interested in hospitalization and fatality ratios, because once in hospital, the dynamics are mechanical (as concerns the spread of the disease).

It is critically important to know when infected patients are contagious. One of the most striking findings of the early literature is that the virus can be transmitted before an outbreak is noticed.[17] The time span over which

associated with hospitalization, the latter with life support on ICUs. But even in published studies the distinction is not always clear. The estimates given here are on the low side if all hospitalized cases are included.

Ferguson et al. (16/3/2020, the Imperial Study), building on Verity, Okell, Dorigatti et al. (2020), give an estimate of 4.4% for θ, arguing that their original data from China are likely to be biased. On the cruiseship "Diamond Princess" 52 of the 697 infected cases developed critically severe symptoms. These 7.5% are high compared to what one can expect for the general population, as the overall group on the ship was relatively high risk (and by far most infections occured in the over-60 age group). Data from Russel et al. (2020), with some preliminary background.

Again, careful: reports such as that until 2/4/2020, "13% of all Covid 19 patients were hospitalized" (https://www.n-tv.de/infografik/Coronavirus-aktuelle-Zahlen-Daten-zur-Epidemie-in-Deutschland-Europa-und-der-Welt-article21604983.html) are misleading. The denominator is wrong.

[12] See Wall Street Journal, March 9, 2020, https://flexikon.doccheck.com/de/SARS-CoV-2, https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html, the references therein and many others in the same ball park.

[13] This seems to be common in the mathematical epidemiological literature. It is, of course, possible to condition the distributions on obervable characteristics.

[14] Relevant data seem to be mostly from Hubei, summarized by Wu, McGoogan (2020). Early cases in Wuhan were documented by Huang, Wang, Li et al. (2020). Here and on other topics the documentations by the Robert-Koch-Institute at https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Steckbrief.html and the CDC at https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html are useful.

[15] I am using the convention that probability distributions over days since infection (the events $T^{\cdots}$) are denoted by $p^{\cdots}$, and those for days since the last event (the incremental lags $\tau^{\cdots}$) by $\pi^{\cdots}$.

[16] See Verity, Okell, Dorigatti (2020) and the references therein. They model Case Fatality Rates (CFRs) and other mortality indicators based on early data.

[17] See, e.g., https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-

transmission can occur is usually stated with reference to the outbreak date T^o and probably individual-specific, say $[T^o - \tau^o, T^o + \tau^n]$. Current estimates for the onset of contagiousness, T^c, suggest values $\tau^o \in [0, 2]$.[18] For the end date of untreated cases, $T^o + \tau^n$, there seems to be a window of $\tau^n \in [6, 12]$ days after the outbreak, with even longer times possible for children.[19] This implies that, in the absence of mass testing, the vast majority of infections occur when the transmitter either is completely unaware of her disposition or has mild symptoms for which she has not been tested.[20] Combined with the evidence for T^o, this yields an overall broad time interval of $[1, 23]$ days after infection, during which (at least some) individuals can be contagious, with little or no mass on $t = 1, 2$ and in the far right tail.

Let $p^c = (p^c_0, p^c_1, p^c_2, ...)$ be the probability distribution of the day T^c of the onset of contagiousness since infection, and $\pi^n = (\pi^n_0, \pi^n_1, \pi^n_2, ...)$ the probability distribution of the final day of infectiousness since incubation, both conditional on not being quarantined or hospitalized. According to the previous remarks we should have $p^c_k = 0$ for $k \geq T^o + 1$ and $\pi^n_k = 0$ for $k \leq 5$.

Remark 1: There does not seem to be much evidence on these distributions. Probably, they are not independent of each other.[21] The papers I have read usually provide means or medians and estimates of the support (min/max). This makes it difficult, in particular, to calculate confidence intervals or p-values in empirical studies.

The following tools from probability theory are useful to put these distributions to work.

- Cumulative lags: If the distributions of subsequent lags are independent, the density (probability mass) function of subsequent events can be obtained by the usual convolution of the densities. Example: if the distribution π^o of days τ^o from the onset of infectiousness to incubation is independent of the distribution p^c of the duration from infection to the onset of contagiousness T^c, then

$$p^o_k = \sum_{m=0}^{k} p^c_m \pi^o_{k-m} \tag{6}$$

management-patients.html, and the discussion in Section 4 below.

[18] Ferguson et al. (March 16, 2020, the Imperial Study), assumes a point estimate of $\tau^o = 0.5$, which implies a larger upper bound for the actual distribution of τ^c, consistent with other findings. For evidence, see the discussion in Section 4 below and the references given there.

[19] See https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html, https://flexikon.doccheck.com/de/SARS-CoV-2#cite_note-50 and the references therein. An interesting small sample result is in Wölfel, Corman, Guggemos et al. (2020).

[20] This is currently the case in most countries. South Corea, Taiwan, and Hong Kong seem to be notable exceptions.

[21] For example, the distribution of the time from the outbreak of symptoms until exit from infectiousness (τ^n) is likely to depend on the day of the outbreak (T^o).

- Time intervals: Again, an example suffices to show the general principle. The probability that an individual on day k after infection is already contagious (i.e., she is on or past day T^c), but has not yet had symptoms (before T^o) is

$$\Pr(T^c \leq k < T^c + \tau^o) = \sum_{n=k+1}^{\infty} \sum_{m=0}^{k} p_m^c \pi_{n-m}^o \tag{7}$$

  Given the evidence cited above, in calibrations the first sum (summation over n) will not extend much further than 12 or 13.

3 The Population Dynamics

In the following model, capital letters will denote stocks (numbers measured at the end of the day) of current cases, lower case letters denote flows (during day t). Script letters denote subgroups of individuals in a population.

Consider a given population (which may be a sub-population of another population, such as the population of a certain region, or the above 70-years olds in that region). A key group of interest in the population is the group of all currently infected individuals, called $\mathcal{X}_t$, of which there are (at the end of day t) X_t. The increment during day t is $\Delta X_t = X_t - X_{t-1}$. The size of the inflow into $\mathcal{X}_t$ (the new infections on day t) is $i_t^{\mathcal{X}}$. Since this variable is of central importance, we let $n_t = i^{\mathcal{X}}$. Note that, despite the common use of language in the media or politics, neither X_t nor ΔX_t nor n_t appear in any of the official statistics.

In order to define the subgroups of $\mathcal{X}_t$ that are relevant for policy, we must consider one important policy variable, testing. I assume that the population can be tested for the virus and that tests are correct.[22] Furthermore, to simplify the exposition and, in fact, as is the case in all somewhat functioning health systems, I assume that all severe cases are automatically tested and hospitalized, and that people only die in hospital.[23] Finally, the base model will not consider mass testing of asymptomatic patients. This can be introduced into this model fairly easily, but is left for future research. Targeted testing of asymptomatic individuals based on special tracing procedures is a further, more elaborate option that must be modelled explicitly. Hence, the only relevant policy variable explicitly considered here is the intensity with which patients with mild symptoms are tested. Thus the model corresponds to the practice in Germany and many other European countries until early April (where, of course, the test intensity has varied across countries and time).

[22] This assumption is not at all innocuous, in particular in times of extreme stress of the system.

[23] This does not mean that all hospitalized cases receive the same treatment. In fact, several current examples show that the mortality rate in hospitals depends on the hospitalization rate (however defined).

The spread of the infection can now be described as in Figure 2, which extends Figure 1. It includes the outcomes "mild - no test" and "mild - test" after the outbreak date T^o, where the probability of testing, conditional on m, is the policy variable $\lambda \geq 0$.

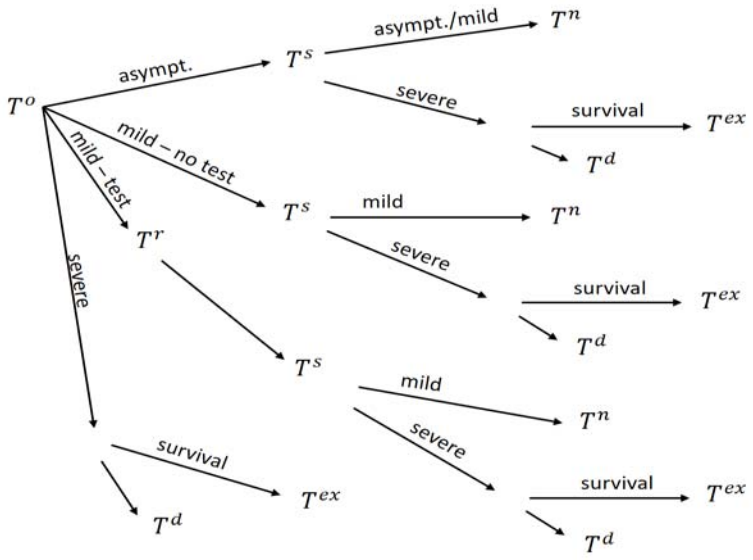


Figure 2: The dynamics with testing

The other new element in Figure 2 is an additional time lag due to testing:[24]

- $T^r = T^o + \tau^r$ (result): time until test result available if tested.

Up to now, in Germany τ^r is largely exogenous and has several additive components: (i) time until individual realizes that the symptoms are potentially problematic,[25] (ii) time until appointment with GP, (iii) time until tested,[26] (iv) time until test result. Overall we probably have, with little randomness,

$$\tau^r \in [2.5, 3]$$

[24] To economize on space, the time lag is not included for severe symptoms, as it does not matter for future events in the model (of course, it is necessary for the clinical treatment).

[25] This time span was probably substantial in the early phases of the Corona wave and may even have exceeded τ^s in some cases. This has certainly changed now. I therefore assume that $\tau^r \leq \tau^s$.

[26] Until now in Germany, tests have been adminstered only after referral by the GP.

The subgroups of $\mathcal{X}_t$ of interest now are:

	Definition	**Size**	**Increment**
$\mathcal{N}_t$	newly infected on day t	n_t	
$\mathcal{E}_t$	early infections: not yet contagious	E_t	ΔE_t
$\mathcal{Y}_t$	contagious and not in quarantine or hospitalized	Y_t	ΔY_t
$\mathcal{Q}_t$	confirmed positive and in quarantine at home	Q_t	ΔQ_t
$\mathcal{H}_t$	confirmed positive and hospitalized	H_t	ΔH_t
$\mathcal{D}_t$	dead	D_t	ΔD_t
	exits from $\mathcal{Y}_t$ as no longer contagious		$\ell_t^{\mathcal{Y}}$
	exits from hospital, healed		$\ell_t^{\mathcal{H}}$
	exits from quarantine, healed		$\ell_t^{\mathcal{Q}}$
$\mathcal{A}_t$	confirmed currently infected	A_t	ΔA_t
	new confirmed infected	c_t	

Table 1: Sub-groups of $\mathcal{X}_t$

Some remarks may be useful to put these definitions in perspective:

- Most of these groups are not documented in official statistics.
- The total inflow into $\mathcal{X}_t$ at date t is the group $\mathcal{N}_t \subset \mathcal{E}_t$. Its size is $i_t^{\mathcal{X}} = n_t$.
- Unfortunately, even the numbers of healed exits $\ell_t^{\mathcal{H}}$ and $\ell_t^{\mathcal{Q}}$ (from hospital or from home-quarantine) are not officially documented in Germany.[27] The exits from undetected outbreaks, $\ell_t^{\mathcal{Y}}$, are of course unobservable. Given the administrative cost of tracking the group $\mathcal{Q}_t$ of confirmed positives in quarantine at home, I doubt whether such numbers are accurate where provided in countries outside East Asia.
- Hence, even the number of confirmed currently infected cases (A_t) is not observable. That is very unfortunate, in particular as the number is sometimes publicly reported.[28]
- The media report $Z_t = \sum_{k=0}^{t} c_k$, total confirmed infected cases until t. I am not sure how useful this historical number is for modelling the dynamics (it is informative in general, of course), because the healed exits $\ell^{\mathcal{Q}}, \ell^{\mathcal{H}}, \ell^{\mathcal{Y}}$ are not known.

[27] See the official website of the Robert-Koch Institut, at https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Situationsberichte/2020-03-25-en.pdf?__blob=publicationFile.
The RKI has begun reporting estimates of recovered cases in early April, see https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Situationsberichte/2020-04-08-en.pdf?__blob=publicationFile
There are many other estimates, and it is not clear how reliable they are for future scientific work.

[28] E.g., on https://www.worldometers.info/coronavirus/#countries

For each sub-group $\mathcal{G}$ the net increment at date t is the difference between inflow $i_t^{\mathcal{G}}$ and outflow. If a group has several inflows then we let $i^{\mathcal{MN}}$ denote the inflow from $\mathcal{M}$ into $\mathcal{N}$. The flow accounting is as follows (where we suppress the time indices):

- $\Delta E = n - i^{\mathcal{Y}}$
- $\Delta Y = i^{\mathcal{Y}} - i^{\mathcal{YH}} - i^{\mathcal{YQ}} - \ell^{\mathcal{Y}}$
- $\Delta Q = i^{\mathcal{YQ}} - i^{\mathcal{QH}} - \ell^{\mathcal{Q}}$
- $\Delta H = i^{\mathcal{YH}} + i^{\mathcal{QH}} - \ell^{\mathcal{H}} - \Delta D$

The outflows $i^{\mathcal{YH}}$ and $i^{\mathcal{YQ}}$ are observable. I have not seen them reported separately, but health authorities must have them.[29] Publicly available (and widely reported) is[30]

$$c_t = i_t^{\mathcal{YH}} + i_t^{\mathcal{YQ}} \tag{8}$$

Summing the above 4 flow equations yields the total net flow equation

$$\Delta E + \Delta Y + \Delta Q + \Delta H = n - \Delta D - \ell^{\mathcal{Q}} - \ell^{\mathcal{H}} - \ell^{\mathcal{Y}} \tag{9}$$

We have the following fundamental counting relations for total infections (using (9)):

$$X_t = E_t + Y_t + H_t + Q_t \tag{10}$$
$$\Delta X_t = n_t - \Delta D_t - \ell_t^{\mathcal{Q}} - \ell_t^{\mathcal{H}} - \ell_t^{\mathcal{Y}} \tag{11}$$

Hence, if (11) is positive, the number of infected increases ($\Delta X_t > 0$); if it is negative it decreases. Unfortunately, except for ΔD_t, none of these variables is observed, either because of intrinsic difficulties or because the administrative infrastructure and public planning have been insufficient.

4 Transmission Dynamics

Under the assumption that there are no nosocomial infections and people observe home-quarantine,[31] new infections depend on the uncontrolled contagious population $\mathcal{Y}_t$ and the transmission rate(s). As noted in the Introduction, at the early stage of an epidemic, as currently in Europe and the US, in a stationary model without intervention, transmission would simply occur according to

$$n_t = \beta Y_{t-1} \tag{12}$$

[29] Hopefully - simply aggregating total hospital admissions will not be sufficient. One needs days of first symptoms.

[30] Note that this number depends on the test intensity λ and that it contains cases of different cohorts.

[31] This assumption can be relaxed in a fuller model. In fact, the proportion ν of tested individuals who ignore the quarantine, is an important variable, partially policy (how is quarantine enforced?), partially behavioral (how cooperative are people?).

where $\beta = \widehat{\beta} - \widehat{\gamma} > 0$ is the net transmission rate, $\widehat{\beta}$ the gross infection rate, $\widehat{\gamma}$ the gross removal rate (through recovery, isolation or death), and infectiousness and removal occur homogenously across cohorts.

This model corresponds to the standard SIR model in epidemiology (see, e.g., Allen (2017)) when the number of infectious cases (the I in SIR) is small relative to the total population The naive transmission model is not well suited for policy analyses for at least four reasons. First, transmission is stochastic. Second, population size is affected directly by policy intervention, such as testing and quarantining. Third, even if one assumes that the population mixes homogeneously, transmission depends on the composition of $\mathcal{Y}_t$, not just on its total size, and fourth, at the individual level the transmission rate is probably not constant over time.[32] In particular, as discussed above, individuals are not contagious immediately after the infection, but typically they are before the onset of symptoms. For example, evidence from the Munich group of early German infections by Woelfel, Corman, Guggemos et al. (2020) indicate that the viral load in the throat - an important determinant of individual infectivity - decreases from the time of the outbreak on and that the virus actively replicates in the throat until date $T^o + 5$, but not much thereafter. The Guandong study by He, Lau, Leung et al. (2020) confirm that the viral load peaks before day T^o, and that approx. 44% of all infections take place before T^o (the transmission probability is highly left-skewed).[33]

Overall, it seems difficult to estimate transmission rates from population data, because neither n_t nor Y_t, let alone its composition, are observable. Even observations from natural experiments, such as the cruise ship *Diamond Princess*, are difficult to interpret, as transmission onboard was massively interrupted from the early days of the outbreak on because (i) all confirmed infected cases were continuously evacuated and (ii) passengers (not crew members) were quarantined (but not fully isolated).[34]

Given these qualifications, let $\beta(k)$ denote the average daily transmission rate per active person on day $k \geq 0$ after infection, which we assume to be constant across (infection) cohorts.[35] It is not clear whether the transmission rate is age-

[32] An obvious further impediment for policy analysis is that the group of infectious but not isolated cases ($\mathcal{Y}_{t-1}$) is unobservable.

[33] The evidence on individual infectivity as measured by viral loads is increasing. An interesting observational study on 23 patients in Hong Kong is To, Tsang, Leung et al. (2020). For more evidence, see https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html#Asymptomatic and the studies cited there, and (as usual) the Drosten Podcast (20, 24/3/2020).

[34] See Government of Japan, Ministry of Health, Labour and Welfare, at https://www.niid.go.jp/niid/en/2019-ncov-e/9407-covid-dp-fe-01.html

[35] Potentially, there are at least two ingredients going into the construction of the individual transmission rate. First, the degree of biological individual infectivity (i.e., the viral load spread per day), which has a distribution over the time interval $[T^c, T^n]$, as described previously. Second, transmission depends on individual behavior, which in turn depends on personal traits and social rules (see, e.g., Blackwood and Childs (2018)). $\beta(k)$ compounds these two dimensions into one single number, describing the average infectivity per cohort member over time (Ferguson et al., 2020, assume i.i.d. Gamma distributions). Then these intensities are weighted by the numbers (frequencies) of infectious individuals of a given cohort, given by the p^c and p^n introduced in Section 2.

specific. Ferguson et al. (2020) assume that it differs between asymptomatic and symptomatic cases.[36] This could easily be incorporated into this model by distinguishing between β^a and β^m. From the discussion of the current empirical evidence in Section 2, we quite certainly have (conservatively estimated) $\beta(0) = \beta(1) = 0$ and $\beta(k) = 0$ for $k \geq 24$.

Conceptually, n_t is a random variable with a probability distribution $f_t\,|\mathcal{Y}_{t-1}$ on $\mathbb{N}_0$. If we limit our attention to average new infections, the associated transmission dynamics is governed by

$$E\left[n_t\,|\mathcal{Y}_{t-1}\right] = \sum_{k=1}^{t} \beta(k)\,|\mathcal{N}_{t-k} \cap \mathcal{Y}_{t-1}| \tag{13}$$

where $|\mathcal{Z}|$ denotes the size (number of elements) of a set $\mathcal{Z}$. (13) states that at the end of day $t-1$, the expected number of new infections on day t is equal to the sum over all previous days $t-k$ of the number of infections caused by the members that were infected in $t-k$ and are still infectious and not hospitalized or home-isolated at the end of day $t-1$. Note that (13) is a generalization of (12) to the case where not only the size but also the composition of $\mathcal{Y}_{t-1}$ matters. In fact, (13) describes an expectation of day t conditional on the full state of infections in $t-1$. In this sense (13) would in principle be useful for daily forecasting, if the information about $\mathcal{Y}_{t-1}$ were known in $t-1$ (which it is not).

If we want to derive an ex ante law of motion similar to the naive model (12), we must use the expected composition of $\mathcal{Y}_{t-1}$. Building on Fig. 2, for each cohort $\mathcal{N}_t$ the duration in $\mathcal{Y}$ is of different length for the different branches of the event tree:

sub-group	path	cond. probability at T_o	end time
$\mathcal{Y}^c$	between T^c and T^o		T^o
$\mathcal{Y}^{am}$	a, then a/m	$\theta_a(1-\theta_{ms})$	T^n
$\mathcal{Y}^{as}$	a, then s	$\theta_a\theta_{ms}$	T^s
$\mathcal{Y}^{mNT}$	m, not tested, then m	$(1-\lambda)\theta_m(1-\theta_{ms})$	T^n
$\mathcal{Y}^{ms}$	m, not tested, then s	$(1-\lambda)\theta_m\theta_{ms}$	T^s
$\mathcal{Y}^{mT}$	m, tested	$\lambda\theta_m$	T^r

Table 2: Sub-groups of $\mathcal{Y}_t$

By construction,

$$\mathcal{Y} = \bigcup_{j\in\{c,am,as,mNT,ms,mT\}} \mathcal{Y}^j$$

[36] The assumption being that symptomatic cases are 50 % more infectious than asymptomatic ones. No source is given for this assumption.

Using the distributions of the evolution of single cohorts constructed in Section 2, the transmission equation (13) becomes

$$E\left[n_t \left|\mathcal{Y}_{t-1}\right.\right] = \sum_{k=1}^{t} \beta(k) w(k) n_{t-k} \tag{14}$$

where the "cohort composition kernel" is given by

$$\begin{aligned} w(k) &= \Pr(T^c \leq k-1 < T^o) && (15)\\ +[\theta_a + (1-\lambda)\theta_m](1-\theta_{ms}) \Pr(T^o &\leq k-1 < T^n) && (16)\\ +[\theta_a + (1-\lambda)\theta_m]\theta_{ms} \Pr(T^o &\leq k-1 < T^s) && (17)\\ +\lambda\theta_m \Pr(T^o &\leq k-1 < T^r) && (18) \end{aligned}$$

Here the first term of the sum gives the mass of the cohort that at the end of day $k-1$ is contagious without having developed symptoms yet (the "pre-symptomatic transmitters"), the second term the fraction with no or mild symptoms that have not been tested and that do not develop severe symptoms (the "long-term stealth transmitters"), the third the fraction with no or mild symptoms that have not been tested and develop severe symptoms later (the "short-term stealth transmitters"), and the fourth the fraction of mildly symptomatic cases that are tested (and sent into home-quarantine once the test result is available).

Note that the $w(k)$ do not sum to 1. They describe the size of subgroups of any given cohort that are not necessarily distinct as they evolve over time; they are bounded by 1 for each k and not more. They depend on the interaction of policy and the different durations in (15)-(18). This latter fact is different from the basic recursive model (12), in which the $w(k)$ simply decrease exponentially, driven by the uniform removal rate from the infectious population, and from its compartmental generalization SEIR (where E stands for "exposed"), where two such exponential decreases are folded into each other.

5 A Simple Calibration

To illustrate the dynamics derived above, this section presents a simple parametric calibration. The basic assumptions are in lign with the preliminary medical evidence presented in Section 2 and so are the derived distributions, as far as this can be judged from the limited empirical evidence. But the calibration is not optimized and the example meant to be illustrative rather than descriptive.

In order to be able to use the simple composition rules (6) and (7) above, assume that the distributions of subsequent events are independent of each other. The building blocks of the model are therefore the distributions

- p^c for the onset of infectiousness T^c,
- π^o for the time τ^o between T^c and the time of first symptoms T^o

- π^n for the time τ^n between T^o and the end of infectiousness on day T^n
- π^s for the time τ^s between T^o and the display of severe symptoms (resulting in hospitalization), if any.

In order to simplify the exposition I assume that τ^n and τ^s are governed by the same distribution π^e. Hence, there is one single day T^e on which patients exit from the group $\mathcal{Y}$, either "healed", which means no longer contagious, or with severe symptoms, which takes them to hospital.[37]

Under this assumption, the cohort composition kernel w takes the following simple form:

$$w(k) = p^{co}_{k-1} + \lambda\theta_m p^{or}_{k-1} + (1 - \theta_s - \lambda\theta_m) p^{oe}_{k-1} \tag{19}$$

where

Time	Group	State
$p^{co}_k = \Pr(T^c \leq k < T^o)$	$\mathcal{Y}^c$	contagious, but still pre-symptomatic
$p^{or}_k = \Pr(T^o \leq k < T^r)$	$\mathcal{Y}^{mT}$	tested after mild symptoms, but without home-quarantine yet
$p^{oe}_k = \Pr(T^o \leq k < T^e)$	all other in$\mathcal{Y}$	no or mild symptoms, not tested, and neither healed nor hospitalized yet

Note that these probabilities do not sum to 1 because of double counting and the group $\mathcal{E}$ of early infections is missing.

Assumption 1: T^c is distributed on $k = 2, ..., 8$, with probability mass function p^c_k given by

k :	0	1	2	3	4	5	6	7	8
p^c_k :	0	0	0.1	0.1	0.2	0.2	0.2	0.1	0.1

Hence, contagiousness begins between day 2 and 8 after infection with most of the mass on days 4 to 6.

Assumption 2: τ^o is distributed on $k = 0, 1, 2$, with probability mass function

k :	0	1	2
π^o_k :	0.25	0.5	0.25

[37] The apparent similarity of the two distributions π^n and π^s discussed in Section 2 has given rise to the hypothesis that τ^n and τ^s are actually driven by the same event ("up or out").

Hence, first symptoms appear on the day of the onset of contagiousness or up to two days afterwards, with most of the mass on the next day.[38]

Assumption 3: τ^e is distributed on $k = 6, ..., 10$, with probability mass function

k :	0	1	2	3	4	5	6	7	8	9	10
π^e_k :	0	0	0	0	0	0	0.125	0.25	0.25	0.25	0.125

Hence, patients with initially no or mild symptoms either develop severe symptoms or stop being infectious between day 6 to 10 after the outbreak with a median at day 8.

By standard calculations using (6), the distribution of the length of the incubation period T^o follows from Assumptions 1 and 2 and has the following density (probability mass) function on $\{2, ..., 10\}$:

0	1	2	3	4	5	6	7	8	9	10
0	0	0.025	0.075	0.125	0.175	0.2	0.175	0.125	0.075	0.025

Table 3: The distribution of T^o, $k = 2, ..., 10$

Table 3 shows that in the example, 80 per cent of all outbreaks occur 4 - 8 days after infection.

Using Assumptions 1 and 2 and using (7), one can also calculate the first term of the cohort composition kernel w in (19). The fraction of the cohort that is contagious but not yet symptomatic on day k after the infection, $p^{co}_k = \Pr(T^c \leq k < T^o)$, $k \geq 0$,[39] is given by

0	1	2	3	4	5	6	7	8	9
0	0	0.075	0.1	0.175	0.2	0.2	0.125	0.1	0.025

Table 4: The cohort fractions $\Pr(T^c \leq k < T^o)$, $k = 2, ..., 9$

According to Table 4, in our example 90 per cent of all "pre-symptomatic transmitters" are to be found 3 - 8 days after infection, with some left skewness.

For the fraction of the cohort that has experienced mild symptoms, is being tested but not yet in home-isolation on day k after the infection, we assume that it takes 3 days until the positive test result has been established and resulted in home quarantine. Hence, the delay τ^r has a point distribution with mass

[38] Remember that T^o is an imputed value for completely asymptomatic cases. See also footnote 5.

[39] Again: remember that T^o is an imputed value for completely asymptomatic cases. Hence, the date T^o is defined for each cohort member.

$\pi^r_3 = 1$ and $\pi^r_k = 0$ for all $k \neq 3$.[40] Using (7), the fraction of the cohort between T^o and T^r on day k is therefore given by

0	1	2	3	4	5	6	7	8	9	10	11	12
0	0	0.025	0.1	0.225	0.375	0.5	0.55	0.5	0.375	0.225	0.1	0.025

Table 5: The cohort fractions $\Pr(T^o \leq k < T^r)$, $k = 2, ..., 12$

The distribution of values is symmetric,[41] just as that of T^o, and peaks one day after that of T^o.

Finally, the fraction of the cohort that has experienced no or mild symptoms, has not been tested, is infectious, but not in hospital on day k after the infection (the "stealth transmitters"), $\Pr(T^o \leq k < T^e)$, has the following size

0	1	2	3	4	5	6	7	8	9	
0	0	0.025	0.1	0.225	0.4	0.6	0.775	0.897	0.956	
10	11	12	13	14	15	16	17	18	19	
0.941	0.863	0.741	0.584	0.416	0.259	0.138	0.056	0.019	0.003	

Table 6: The cohort fractions $\Pr(T^o \leq k < T^e)$, $k = 2, ..., 19$

According to Table 6, if not tested and isolated, between days 6 and 13 after infection these stealth transmitters constitute the vast majority of each cohort (approx. 60% or more).

To illustrate the impact of the composition of $\mathcal{Y}_{t-1}$ on En_t given by (14) in our example, let us assume that the average individual transmission rate $\beta(k)$ is constant over the course of the infection, just as in the basic SIR model. As discussed in Section 4 (in particular footnote 33), this is probably not correct, but it helps to make the point.

Consider the following baseline scenario:

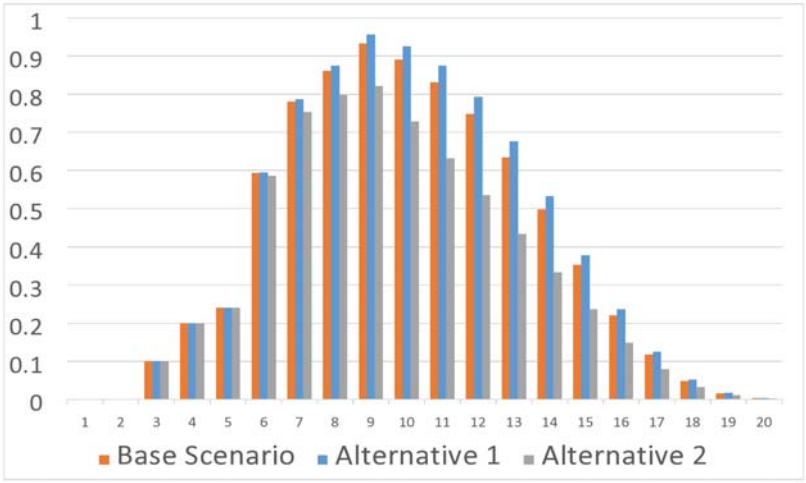
$$\begin{aligned} \theta_s &= 0.01,\ \theta_m = 0.7, \\ &\lambda = 0.2 \end{aligned}$$

In this scenario, 70 per cent of all infected cases develop mild symptoms at the time of the (potentially unobserved) outbreak, 29 per cent are asymptomatic, and 20 per cent of all mild cases are tested. Given the lack of data on the evolution of $\mathcal{Y}_{t-1}$, it is difficult to translate these percentages into observables, but at least the values of θ_a and θ_m are consistent with what we seem to know

[40] As noted in Section 3, in Germany this delay has unfortunately been relatively long for too long. In particular in the very early days of the disease, in March, when (at least in Germany) testing capacity was overwhelmed, practices not yet established, and patients not used to preventive self-quarantine, $\tau^r = 3$ may be an underestination.

[41] Remember: this is not a probability distribution.

from the natural experiments discussed above. The time structure of the cohort composition kernel w for this scenario is given by the orange bars in Figure 3. Almost 80 per cent of the total mass lies between days 6 and 13. after infection, less than 10 per cent between days 1 and 5. Hence, policies affecting new infections will show very little effect in the first 5 days, and one will have to wait for almost 2 weeks to see most of the impact. This is very different from the case of exponential growth in which most of the mass is concentrated on the early days.



Structure of $w(k)$ under three scenarios

This picture changes little for the alternative scenario 1, where we assume that the fraction of asymptomatic cases among the infected is much larger:

$$\begin{aligned} \theta_s &= 0.01,\ \theta_m = 0.4, \\ &\lambda = 0.2 \end{aligned}$$

The composition of w for this scenario is given by the blue bars in Figure 3. The mass of active transmitters increases slightly overall, with most of the increase between days 8 and 15. This is mainly due to the fact that testing occurs only for the mildly symptomatic, whose the number is now lower. The finding that the two scenarios differ only little is interesting because the fraction of aymptomatic patients, θ_a, is difficult to estimate and not yet well understood.

Alternative scenario 2 describes a policy experiment, by assuming that the test frequency is drastically increased compared to the baseline:

$$\begin{aligned} \theta_s &= 0.01,\ \theta_m = 0.7, \\ &\lambda = 0.6 \end{aligned}$$

Not surprisingly, the result, given by the grey bars in Figure 3, shows a strong decrease of new infections, which occurs mostly between 8 and 17 days after the change. More importantly, the analytical expression for the cohort composition kernel w in (19) makes it possible to quantify this effect. This is important because the gain in lives and treatment costs brought about by the mitigation of the transmission activity can now be compared to the considerable costs of expanding the testing capacity.

In a next step, the improved transmission dynamic (14) can be integrated in dynamic economic models. We need better data and a more granular model to do these estimates reliably, but first simulations already suggest that the cohort composition kernel reacts non-trivially to economic policy.

6 Conclusions

We currently know far too little about the epidemic. While the empirical evidence on small samples of patients or from unplanned natural experiments is rising rapidly, aggregate data are very problematic. Hardly any of the basic numbers in the fundamental counting relation (10) is known. The model of this paper is one step in understanding and using the available data better. Better data can be obtained from large-scale public testing, but will also require the intelligent use of selective random tests. To organize and interpret such data collection, it is important to understand the underlying structure of the population and its dynamics. The research described in this paper may help on both these fronts: understanding the available data and organizing the collection of new data.

Testing is important, but the question is what testing. In the early stage of an epidemic, mass testing is likely to be very expensive and relatively uninformative, since outbreaks are random and so are observed clusters. But controlled mass testing of specific populations can be very useful to identify key theoretical parameters, such as the θ_z in the above model. As the model has shown, such tests must control carefully for the timing of interventions, for example to be able to distinguish asymptomatic cases from pre-symptomatic ones. Given the great uncertainty and the different needs and views with respect to policy, it is now the time for controlled experiments.

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