Title: COVID-19 and the math behind epidemics

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Introduction

The year 2020 has been defined by the COVID-19 pandemic, which has already infected and killed millions globally. The novel coronavirus responsible for this disaster has joined the likes of HIV, Zika, Ebola, and many influenza strains - all viruses which have made the evolutionary jump from animals to humans and wreaked havoc around the world. The battle to get this infection under control and get back to life as we knew it continues.

When an outbreak is identified - usually through an anomalous spike in cases with similar symptoms - scientists rush to answer questions about the mysterious new illness. What type of microbe causes the infection? Where did it come from? How does it spread? What symptoms does it cause? What drugs could treat it? In the current epidemic, science has proceeded at a frenetic pace. Hundreds of “preprints” - drafts of scientific articles yet to be formally published - are shared each day, genetic sequences of viruses collected from patients around the world are analyzed in real-time, and cases and deaths are reported, visualized, and analyzed daily by anyone so inclined.

While some scientists may rush for their microscopes and lab coats to study a new infection, others leap for their calculators and code. A new outbreak can be characterized by a handful of metrics that are critical to guiding the public health response and to building more complex models to forecast the trajectory of the epidemic. Infectious disease epidemiologists, mathematical biologists, biostatisticians, and others with extensive training in both microbiology and math try to answer questions like: How quickly is the infection spreading? What fraction of transmission do we need to block to control the spread? For how long is someone infectious? How likely are infected people to die, or need to be hospitalized?

People tend to think of physics as being the most “mathematical” of the sciences, but the fields of ecology, evolutionary biology, and epidemiology have also been driven by theory and rigorous mathematical analysis [1]. Ideas and people constantly flow between physics and the “three E’s” of biology. In fact, the idea of using mathematics and models to understand the spread of infectious diseases is older than germ theory itself. David Bernoulli of fluid mechanics fame devised a model to predict the potential benefit of smallpox inoculations in 1760 [2], and Nobel prize-winning physician Ronald Ross created mathematical models to motivate the use of mosquito control to reduce malaria transmission [3]. Some of today’s most prolific infectious disease modelers, such as Neil Ferguson of Imperial College in London, who leads the COVID-19 Response Team that advises the UK government, and Vittoria Colizza of Sorbonne

University in Paris, a leader in network modeling of disease spread, originally trained as physicists.

This article will introduce some of the key mathematical quantities that characterize an outbreak, summarize how scientists try to calculate these numbers, and clarify the nuances and caveats in interpreting them. For COVID-19, these estimates are being shared, debated, and updated in real time, and regularly appear on the evening news or on your social media feed. Physicists are in a unique position to understand the subtle art of distilling real-world complexity into meaningful mathematical summaries and parsimonious models, and can serve as key allies in communicating these ideas to the public.

Summarizing transmission dynamics with the basic reproduction number, R_0

Few scientific fields have a single metric that both insiders and outsiders obsess over as much as the "basic reproductive number" of infectious disease epidemiology. Expressed as "R_0" and pronounced "R naught", this unitless number is defined as the average number of secondary infections caused by a typical, single infected individual when they are introduced into an otherwise completely susceptible population, before they die or recover (**Box 1**). It's a single quantity used to describe how infectious a given pathogen is and how difficult it will be to control.

An entire textbook could be devoted to all the fascinating mathematics behind R_0 [4]. In nearly all situations, infectious disease dynamics display some sort of criticality or threshold behavior, whereby a spreading process can only really take off if certain conditions are met. Absent these conditions the outbreak is destined to fizzle out, similar to a nuclear chain reaction. If we think about disease spread in terms of continuous differential equations, then R_0 tends to show up in the conditions that determine when a particular equilibrium condition is stable or unstable. If we instead think about the propagation of infection as a series of stochastic reactions, then R_0 is likely to determine when extinction is more likely than establishment.

Roughly speaking, R_0 depends on the product of three main factors characterizing an infection: the contact rate, which is the number of people an infected individual contacts each day, the transmissibility, which is the probability per time that any given contact results in transmission, and the duration of infection. The goal of most infectious disease control efforts is to reduce R_0 by altering one or more of its components. For example, the contact rate can be reduced by limiting the number of contacts an infected individual has (e.g. by general "social distancing" or targeted isolation of diagnosed cases), and the transmissibility can be reduced by limiting the chance of transmission during each interaction (e.g. by mask wearing. See article by Poulain & Bourouiba in May 2019 issue of Physics Today for more info on the physics of respiratory infection spread). The duration of infection can often be reduced by therapies that clear the disease-causing microbe (e.g. like antibiotics for strep throat), but we don't currently have drugs that can do this for COVID-19. Another way to decrease R_0 is to reduce the portion of uninfected individuals who are susceptible to infection, which is what we hope a vaccine will eventually do.

There are several important limitations of R_0 that are critical to keep in mind. Firstly, R_0 does not tell us anything about how deadly a disease is (what we call its *virulence*). There are infections with small R_0 values that are extremely lethal (e.g. SARS), and others with very high R_0 values that rarely lead to death (e.g. chickenpox). Others, like smallpox, have both a high R_0 and a high risk of death. Secondly, R_0 does not tell us anything about the *timescale* over which a disease spreads. The average number of new cases described by R_0 could occur over the period of a few days or many years. For example, a typical individual with untreated HIV transmits the virus to only a few individuals over the course of the 10-year period before they succumb to AIDS, whereas an individual with the common cold has only a few days to infect a few others before the virus is cleared from their body. Finally, contrary to popular belief, R_0 is not actually an intrinsic property of a particular infection any more than the Reynold's number is an intrinsic property of a particular fluid. R_0 is highly dependent on the population in which a disease is spreading and in particular, on human behavior. The same infection could have a high R_0 in a crowded population with poor hygiene and an immune system weakened due to malnutrition, but a very low R_0 in a population with better living conditions and baseline health. Even details of the demographics of a population can influence R_0, like the proportion of the population in high-risk age groups, or the pattern of social mixing (e.g. between different age groups, cliques, occupations, or neighborhoods). In addition, the average number of secondary infections can change dramatically over the course of an epidemic. This "effective reproduction number" (R_{eff}) responds in real-time as individuals change their behavior to avoid infection.

Despite these limitations, knowing the value of R_0 or R_{eff} has many practical uses during an outbreak. For example, Stephen Kissler and Christine Tedijanto at Harvard found that with an estimated R_0 of 2.2, we would need to have 60% effective social distancing in place at least 70% of the time in order to avoid overflowing the current ICU capacity of the US. Luca Ferretti and Chris Wymant at Oxford calculated that with their estimate of R_0 = 2.0, testing and contact tracing could only control the epidemic if we isolated 75% of confirmed and suspected cases within 2 days. Steven Sanche and Yen Ting Lin at Los Alamos National Laboratory estimated that with R_0 = 5.7 early in the outbreak in Wuhan, control would require quarantining 50% of infected people combined with 50% effective social distancing. Huaiyu Tian at Beijing Normal University and colleagues estimated that R_0 was on average 3.1 early in the outbreak in Chinese cities, but that it quickly decreased to ~1 in cities that rapidly implemented a set of control measures, before further decreasing to ~0.04 under more intense controls.

But where did these estimates of R_0 come from? Estimating R_0 is notoriously difficult. We rarely actually get to observe a complete chain of infection events starting from a single individual, which would be needed for a direct estimation. Sometimes, when infection is still relatively rare, the symptoms are relatively unique, we have good diagnostic tests, and when we can sample a high proportion of the population of interest, then we can recreate the path of transmission, correctly attributing the source of infection in each case (**Figure 1**). In *contact tracing studies*, as soon as an individual is diagnosed, public health professionals track down anyone they might have contacted over the duration of their infectious period and test them for infection, which also allows us to estimate R_0 for a single generation. However these direct ways of estimating R_0 can be biased: We are more likely to detect outbreaks in which a large number of individuals were

infected by a single source (i.e. a “superspreading” event), so our estimates are biased upwards. The source individuals may also be more likely than the average person to be diagnosed and isolated quickly, leading to underestimates of the true R_0.

For these reasons, indirect ways of estimating R_0 are more common and may give more representative values. A key type of indirect estimation involves observing the *growth rate* of an epidemic. As we explained earlier, R_0 alone does not determine how quickly a disease spreads - this also depends on how spread out in time the secondary infections a given individual generates are. However, if we can estimate the average duration of infection from other data, or more specifically, the distribution of the length of time that someone is infectious, then it is generally possible, with some mathematical tricks, to back out the R_0 values from the rate at which the disease is spreading at the population level (**Box 1**).

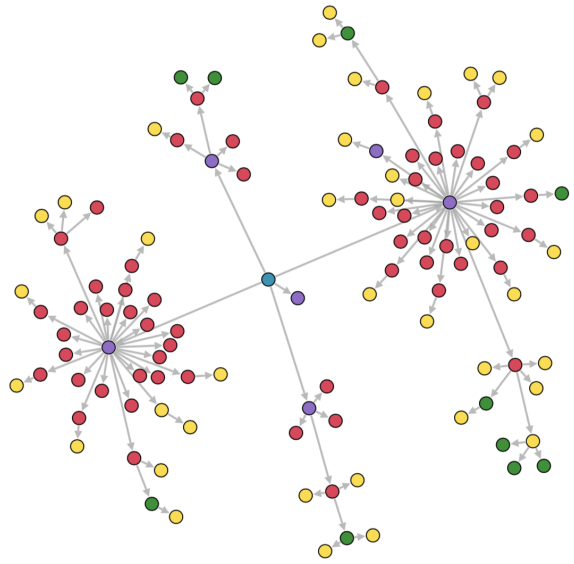


Figure 1: Example transmission network for COVID-19. This transmission cluster was seeded by an unknown infected individual (blue) who attended a training course with other fitness instructors (purple), who in turn spread the infection to students in their exercise classes (red), family (yellow), and coworkers (green) (created from data in [5]). Differences in the number of secondary transmissions likely occurred due to both circumstances (i.e. aerobic exercise in crowded indoor areas vs rapid isolation of cases by public health authorities) and individual biological variation in infectiousness.

Characterizing the timescale of infection: the exponential growth rate, incubation period, infectious period, and serial interval

Exponential growth is one of the defining features of epidemics early in their course (**Figure 2**). Estimates of the rate of exponential growth of the number of people infected, often labeled with the parameter r (units of “per day”), can be used to make short-term projections of the epidemic. It is more common in the popular press for these rates to be expressed as doubling times (defined as $T_2 = \log(2)/r$).

The exponential growth rate is not an intrinsic property of a particular infection but often varies across regions and over time. Most of the time, changes in r occur for the same reasons they do for R_0, for example due to changes in human behavior and adherence to policies to reduce spread. But since r must be estimated from data, it is also subject to other factors. Dramatic changes in testing capacity that alter the proportion of cases detected and reported, over the timescale that the exponential growth rate is being measured, can lead to biased estimates of r. Similarly, changes in reporting delays can alter estimates.

Observed exponential growth rates can be used to back out R_0, which has a more intuitive interpretation and is more directly connected to the underlying process of disease transmission. Researchers have derived mathematical equations to relate r to R_0 under different assumptions about disease transmission (**Box 1**). In general, these formulas require knowing how long a typical individual is infectious (often called the *infectious period*) and how long the delay is between when someone is initially infected and when they become infectious (often called the *latent period*) (**Figure 2**). A high observed exponential growth rate of infection would imply a high R_0 value if either the latent period or infectious period were known to be quite long, while it could imply a much smaller R_0 value if these intervals were known to be short. It is therefore critical to conduct studies following individual patients with known dates of exposure to infection, in order to estimate these intervals. Moreover, since mathematical analyses suggest it is not just the average duration of these intervals, but their entire distribution, that determines the r vs R_0 relationship, enough patients must be studied to get a reasonable estimate of the full distribution.

For many infections, it is easy to define these periods because they correspond with the disease symptoms. However, for COVID-19, this has turned out not to be the case: individuals often shed virus in their respiratory secretions, and are therefore highly infectious, before they develop symptoms like cough or fever. Thus, the *incubation period* (the time until symptoms develop) is generally longer than the latent period. Furthermore, it appears that many of the symptoms of COVID-19 extend much longer than the infectious period, due perhaps to a maligned immune response to the virus. Consequently, we often need to use epidemiological information, instead of just symptom tracking, to estimate when someone was infectious.

One common way that infectious disease epidemiologists try to back out the timing of infectiousness relative to the disease course is to use observed transmission chains to estimate quantities called the *generation time* or the *serial interval* (**Figure 2**). The generation time is defined as the time between when an index individual was infected and when they infected a secondary case, whereas the serial interval is defined as the time from when symptoms start in an index individual until symptoms start in a secondary individual they infected. The generation interval and serial interval are both mathematical convolutions of the latent and infectious periods and can both be used to back out one if the other is known. Because it is generally much easier to determine symptom onset time compared to infection time, the serial interval is much more commonly estimated in studies. Researchers have developed additional mathematical formulas that directly relate the serial interval distribution to r and R_0 (avoiding the need to recover the latent and infectious periods first), which has become the most common technique used to estimate R_0 (**Box 1**). However, these methods are subject to many sorts of biases in estimates of the serial interval. For example, individuals enrolled in research studies are often isolated shortly after diagnosis, reducing the time they have to infect others.

Estimating these intervals for new infections is useful for control policies beyond just estimating R_0. The incubation period distribution tells us how long we need to keep exposed individuals in quarantine before we can safely rule out (say, with 99% confidence) them developing

symptomatic infection. The infectious period distribution helps us understand how long infected individuals should be kept in isolation to prevent them from infecting others.

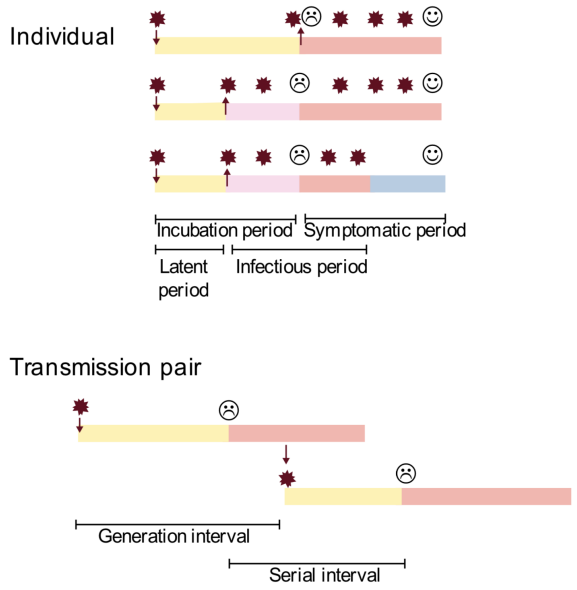
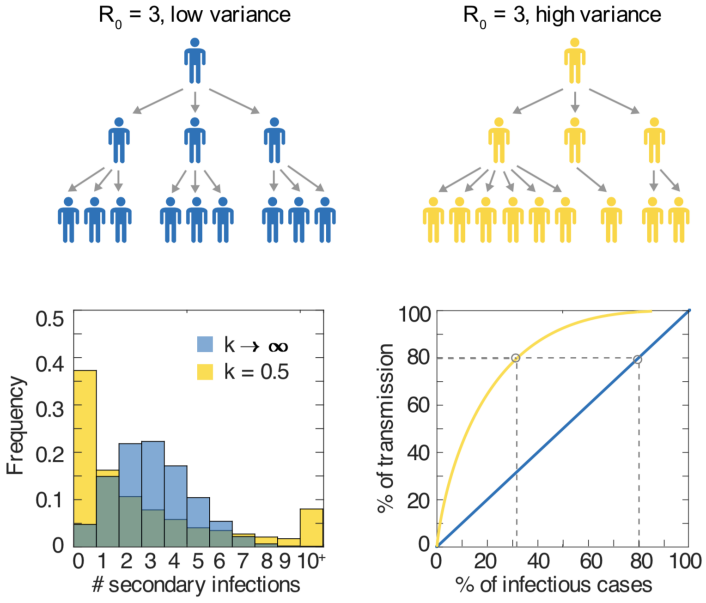


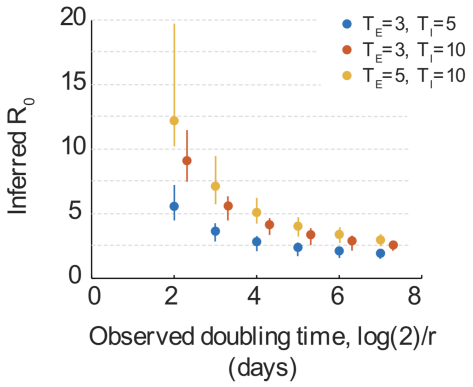
Figure 2: Timescales of epidemic spread. The kinetics of infection spread depend on the timing of disease progression within infected individuals. The *incubation period* and the *symptomatic period* depend on the timing of symptom onset and resolution (smiley/frowny faces). In contrast, the *latent period* and *infectious period* are determined by the window of time in which an infected individual can transmit to someone else (up arrow/red viruses), and may or may not overlap with the timing of symptoms. Three possible scenarios are shown. Transmission pairs, which are sets of individuals for whom we know one infected the other, are often used to estimate these timescales, by measuring either the *generation interval* or *serial interval*.

********** **Box 1: Quantifying transmission dynamics** **********

The basic reproduction number, R_0, describes the average number of secondary infections generated by a single infected individual introduced into an otherwise susceptible population. An epidemic will tend to slow if a fraction $f > 1 - 1/R_0$ of the population is protected from infection.



The variance in secondary infections can be large, leading to a propensity for "superspreading" events [6]. The number of secondary infections is often summarized by a negative binomial distribution with mean R_0 and "dispersion" parameter k (probability distribution function $P(x;R_0, k) = \Gamma(k+x)/\Gamma(k)\Gamma(x)\ p^k\ (1-p)^x$ with $p = (1+R_0/k)^{-1}$). If all individuals have the same intrinsic infectiousness, then the number of secondary infections is expected to be Poisson distributed ($k \to \infty$, blue curves), whereas if there is more heterogeneity, the distribution is more overdispersed (low k value). The variance is also affected by behavior and public health interventions. For COVID-19, a few studies have estimated $k \sim 0.5$ (yellow curve), although with much uncertainty (range 0.1 - 1). Overdispersion implies that a small number of individuals are responsible for a large percent of secondary infections (dotted lines) while most others infect no one, causing infection chains to go extinct.



Estimating R_0 directly is very difficult; instead it is usually inferred indirectly from the rate of exponential growth early in the epidemic (r) combined with knowledge of the timescale of infection (Figure 2) [7]. For example, if we know the average duration of the latent (T_E) and infectious periods (T_I) and assume they are exponentially distributed, then the formula $R_0 = (1+rT_E)(1+rT_I)$ applies (large dots on graph). Different estimates for the distribution shape lead to different estimates for R_0 (error bars). Epidemic growth rates in the range r = 0.1 - 0.4/day have been observed at the country level for COVID-19, corresponding to doubling times between ~2 to 8 days. Estimates of R_0 have generally been between 2 and 3, though sometimes much higher, depending on the setting in which epidemic growth was observed and the assumptions about the transmission intervals.

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Calculating how deadly a disease is using the Case Fatality Risk

So far we've introduced some of the key metrics used to characterize an epidemic: the basic reproductive ratio R_0 summarizes the transmission potential of each infected person, the exponential growth rate r tells us how fast the epidemic is growing in a given setting, and the incubation period, infectious period, and serial/generation interval tell us how the course of disease within an individual patient determines the timescale of new rounds of infection at the population level. But these metrics miss a key feature of disease: how deadly it is.

The lethality of an infectious disease is typically defined as the probability that an infected individual will eventually die of the disease, and is commonly reported as the *case fatality risk*, or *CFR* (Figure 3). For COVID-19, this value has been one of the most hotly debated metrics, and while scientists have generally converged on an estimate of around 1%, it continues to be subject to scrutiny among researchers, as well as by the popular press and general public. Some continue to insist that COVID-19 is "just another flu", whereas others present evidence for total excess deaths far exceeding official reports. To understand these debates, it is important to understand the complications in estimating CFR.

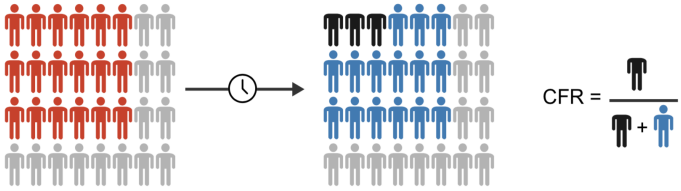
A very common mistake in trying to estimate the CFR is to simply divide the cumulative number of deaths occurring up to a certain day by the cumulative number of cases diagnosed up to that same day. **Box 2** uses simple models to show why this ratio is a biased estimate of the likelihood of death given infection, especially during a rapidly growing epidemic. To correctly get the risk of death, we ideally want a *cohort study*, where we select a group of individuals near the time they are infected, and then follow them over time until they all either recover or die. Since such studies are generally difficult in the context of an ongoing outbreak, there are ways to adjust measures of the simple ratio of deaths to cases to account for epidemic growth and time to death.

Other complications arise when estimating the CFR. Who is counted as a “case” in the denominator in the CFR? More precise definitions of CFR in the epidemiology literature make it clear that a “case” means someone who is diagnosed with infection, either by a specific test or at minimum based on symptoms. But this presents a problem for infections like COVID-19, since we know there is underreporting of true cases due to testing limitations and completely asymptomatic infections. If we truly want to estimate the probability of death given infection - whether or not that infection is symptomatic or diagnosed - then we need to correct for this undercounting, and then the quantity we are estimating is more correctly termed the “infection fatality risk”, or IFR. For COVID-19, attempts to estimate the IFR try to estimate the degree of undercounting by either a) looking to settings with near universal testing, which occurred in travelers returning to their home countries on flights from Wuhan, on cruise ships with large outbreaks, or in countries with extremely rigorous contact tracing programs, or b) conducting random population-level testing to estimate the prevalence of current or past infection.

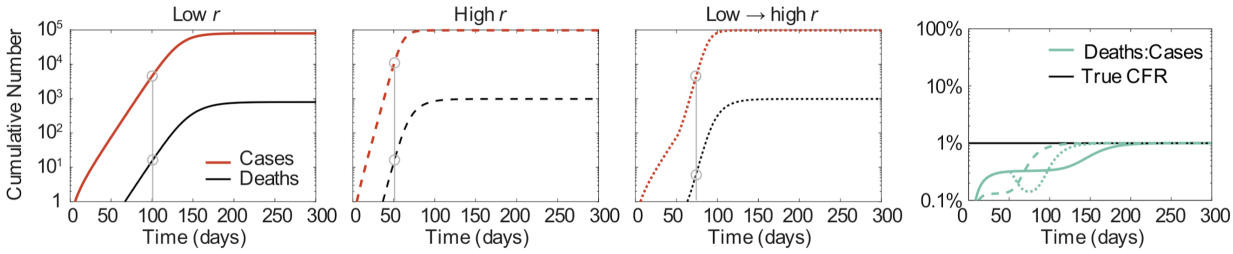
There are other challenges in estimating and interpreting CFR and IFR values for COVID-19 and other infections, such as correctly attributing causes of death in patients with multiple diseases or who die at home without a specific diagnosis. But perhaps most importantly, metrics like the CFR only count deaths, ignoring the many other harms associated with infections among survivors. The long-term complications of COVID-19 and the care required for serious cases (e.g. mechanical ventilation) are still an active area of investigation, and there likely won’t be simple metrics to summarize these effects.

**********Box 2: Estimating the risk of dying from an infection during an epidemic **********

Epidemiologists use the CFR (“case fatality risk”) to describe the percent of individuals confirmed to be infected (“cases”, red) who will eventually die (black) vs recover (blue). The true CFR can only be accurately estimated by following a cohort of infected individuals until their final outcome is observed.



The ratio of the number of deaths observed up to a certain time to the number of cases reported up to that same day (gray circles) can give a very biased estimate of the true risk of death. The bias is especially bad when the epidemic is growing quickly (high *r*), when the growth rate changes rapidly, and when there is a long delay between infection and death. This is because the true pool of cases from which the currently observed deaths were drawn occurred well into the past, when the epidemic was much smaller.



In this simple infection model, infected individuals are only infectious for ~5 days but then take a further ~2 weeks to die. The true CFR is 1% but is dramatically underestimated when taking the ratio of deaths: cases early in the epidemic, or when the growth accelerates. In real data, this ratio can further be confounded by underreporting or delays in reporting cases or deaths.

A note on terminology in epidemiology: The acronym "CFR" is confusingly referred to as the case fatality *rate*, case fatality *ratio*, and case fatality *risk* in different outlets, but in epidemiology, rate, ratio, and risk all have precise meanings. A "rate" generally implies a unit of time; for example, the term "mortality rate" generally means the number of deaths occurring per capita per year, and is rarely used to describe a short term infection which only affects a portion of the population. Only a "risk" metric describes a proportion or percentage, where the individuals counted in the numerator are a subset of those in the denominator, and this is what we need to measure the chance or "risk" of death given infection.
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Translating epidemiological metrics into predictive models

Simple metrics like the basic reproduction number (R_0), the epidemic growth rate (r), and the case fatality risk (CFR) are very helpful for classifying and comparing infections and for quickly communicating quickly about risk, but they are limited in their ability to help us predict the full burden of an epidemic. For example, the number of people who will be killed by an infection and the timescale over which this occurs depends not only on the CFR, but on how many people eventually get infected, which itself depends on how effectively the infection is transmitted, what fraction of the population is susceptible, and the efficacy of control measures. The number of new infections generated each day depends on the number of people currently infected and how long ago they were infected, which determines how many of them have already entered their infectious period. To put all these ideas together and make informed predictions, we need mathematical models.

Most dynamics models used to track the spread of infections in a population fall into the category of "compartmental models", meaning that individuals are classified into a few discrete states based on their infection status, like "susceptible", "infectious", or "recovered" (**Figure 3**) [8,9]. The model then tracks the changes over time in the number of individuals in each state, usually using differential equations or discrete or continuous stochastic processes. These equations are inherently nonlinear since it is the pairwise interactions between susceptible and infectious individuals that lead to the generation of new infections. Some of the simple standard epidemic models will be familiar to physicists from introductory dynamical systems courses and are recognizable by the acronyms which describe the "compartments" they include. For example, the "SIS" model describes infections without lasting immunity, like many sexually-transmitted diseases: susceptible (S) individuals can become infectious (I), but then return to the susceptible (S) state when they recover. In contrast, in the "SIR" model recovered (R) individuals are assumed to be permanently immune, a good approximation for many short-term viral infections like measles or yellow fever.

Just like physicists, infectious disease researchers must balance the desire to create parsimonious models that can be fully analyzed and understood, with the goal of being realistic

enough to generate useful predictions. Compartmental (SIR-type) models are always over-simplifications, because in reality the infection process within one person's body is a continuum of states, as the microbe multiples and migrates between tissues, as the immune system mounts a response, and as symptoms develop in reaction to infection. The process of disease transmission can be much more complicated than the simple "reaction rate" terms used in many equations, as it depends on close personal contacts governed by human behavior and the highly structured nature of our social networks (**Box 3**).

The level of detail needed for a model to be useful depends on the goal of the particular modeling task. For example, some researchers modeling COVID-19 are interested in understanding the potential burden on our health care system, and so they extend "SIR" type models to include advanced stages of infection that require hospitalization or ICU-level care, and also track the portion of individuals who die, as opposed to recovering (**Figure 3**). In studies that made policy recommendations about different social distancing strategies, modelers simulate infection over detailed networks that described the interactions individuals had at home, school, work, and among friends. To understand how effective symptom-based isolation of cases would be with or without additional quarantining of contacts, scientists augment basic models to track the varying level of infectiousness over the whole disease course, from pre-symptomatic infectiousness through peak infectivity to eventual waning.

Scientists continuously debate the relative merits and caveats of different modeling approaches for COVID-19, refine models as our understanding of the disease changes, and try to determine how to best communicate the uncertainty inherent in model predictions to the public (see David Kramer's June 2020 article in Physics Today).

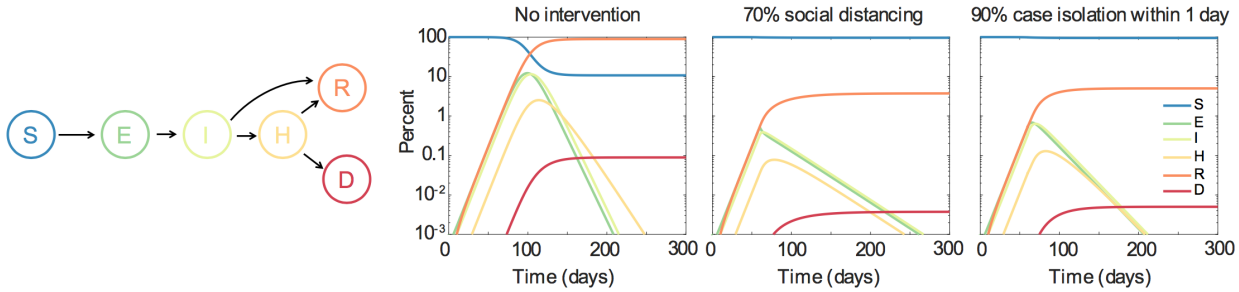


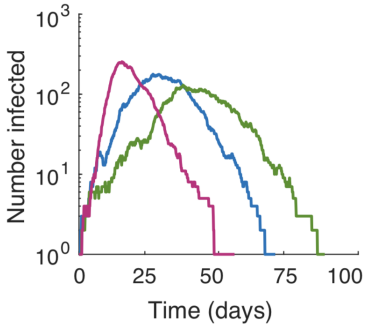
Figure 3: A mathematical model of COVID-19 spread and clinical progression, with and without interventions. This "compartmental" model classifies individuals as susceptible (S), exposed (E), infectious (I), hospitalized (H), recovered (R), or dead (D). The model is simulated as a system of ordinary differential equations. The average duration of the latent period ("exposed" stage) and infectious period are 5 days. 10% of individuals progress to hospitalization, of average length 2 weeks. 1% of all individuals (10% of those hospitalized) eventually die. At baseline, R_0 = 2.5. One of two different interventions is implemented at day 60: either a general "social distancing" policy which reduces all contacts between infected and susceptible individuals by 70%, and another case-based policy in which 90% of infectious individuals will be isolated (preventing onwards transmission) on average 1 day after they become infectious.

**********Box 3: Simulating the spread of infections on networks**********

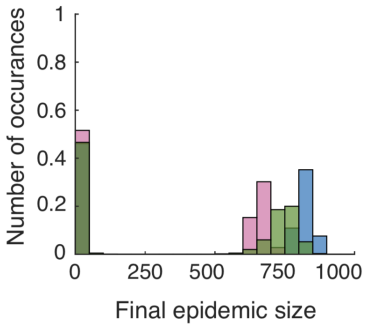
Human contacts are not random or uniform. They can be described by a *contact network*, and this network determines which transmission paths are possible if an infection is introduced into the population. The structure of this network can have a big influence on the extent of disease spread [10].

Uniform random network | Small-world network | Heterogeneous network

To demonstrate these ideas, a simple susceptible-infected-recovered (SIR) model was simulated stochastically on three idealized networks of size N=1000 (images shown for N=100): a uniform random network, in which each individual is connected to exactly 10 randomly-chosen others in the population; a highly-clustered "small-world network", using the Watts-Strogatz algorithm, in which individuals are preferentially connected to exactly 10 neighbors and then 10% of connections are randomly rewired to others in the population, and a heterogeneous network, in which the number of neighbors for each individual is drawn from a gamma distribution with mean and standard deviation of 10. All epidemics started with 1 infected individual.



Epidemic growth is fastest in the heterogeneous networks - boosted by highly-connected superspreaders - and slowest in the small-world network, where high degree of interconnectedness limits the susceptible contacts seen by an infected individual. The final epidemic size (% of recovered individuals when the infection eventually dies out) varies across simulations due to stochastic effects. The average final size is highest in the uniform network and lowest in the heterogeneous network.



More details on the physics of networks can be found in Mark Newman's Nov 2008 article in Physics Today.

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Conclusion

Mathematical analysis and modeling are key tools in the study of infectious diseases, and have played a critical role in our response to the COVID-19 pandemic. Even estimating seemingly simple metrics - like the basic reproduction number, the incubation and infectious periods, and the case fatality risk - requires strict attention to nuances of data and careful formulation of mathematical relationships. When designing more complex models of epidemic dynamics, infectious disease modelers - like physicists - must make tradeoffs between keeping things simple enough to facilitate deep understanding and realistic enough to make accurate forecasts.

Getting the numbers right is always a priority for scientists, but in the middle of a public health crisis, the stakes are higher than ever.

Source code: All the code used to generate the results shown in this paper is publicly available at https://github.com/alsnhll/PhysicsTodayCOVID19, along with more details about the methods used. An online simulation tool for understanding COVID-19 transmission is also available at https://alhill.shinyapps.io/COVID19seir/

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