MILLIMAN WHITE PAPER

# Modeling the pandemic risk

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In December 2019, a new disease now known as COVID-19, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged in Wuhan, China.

In this paper, we discuss how to model pandemics such as COVID-19 and give an illustration on U.S. data. We also provide an overview of how to model the future effects of the COVID-19 pandemic on mortality and explore a basic vector-borne disease model.

## SARS-CoV-2, not a typical virus

SARS-CoV-2 is the only modern virus that has led most governments across all parts of the globe to confine their populations. The following main characteristics differentiate it from past known viruses:

- **Super-spreaders that are difficult to detect:** 70% of infected individuals do not transmit the virus to any other individual, and 10% to 20% of infected individuals contribute to 80% of contaminations, see [1]. SARS-CoV-2 therefore spreads mainly via "super-spreaders."
- **A virus that is mainly transmitted through the air:** Other viruses are transmitted through secretions (e.g., Ebola) or sexual intercourse (e.g., AIDS), which is much easier to prevent than transmission through the air as SARS-CoV-2.
- **Mutations that promote propagation:** Many variants are appearing, accelerating the dynamics of the pandemic, and calling into question the effectiveness of vaccines. Most viruses mutate into less lethal forms, but SARS-CoV-2 mutations have led to continued infection and deaths.
- **A low case fatality rate:** There are diseases much more lethal than SARS-CoV-2, such as the Ebola virus which kills up to 90% of those infected, see [2]. Likewise, the case fatality rate of SARS in 2003 was estimated at 43% in those over age 60 and 13% in those under 60, against 0.23% to 1.15% for COVID-19, see [3] and [4].
- **Extremely varied symptoms:** Unlike the flu symptoms, which are well known, a very wide variety of symptoms has been observed: gastric disorders, skin manifestations, conjunctivitis, olfactory hallucinations, many organs impacted, e.g., the heart, kidneys, and lung, etc. In addition, more than 30% of patients have one or more features of long-COVID recorded between three and six months after a diagnosis of COVID-19, see [5].

## How to model the pandemic of COVID-19

SIR-type models conventionally model epidemics. They are compartmental models in which each type of population (usually S-Susceptible, I-Infectious, R-Recovered) can interact with each other.

One of the simplest SIR models is presented in Figure 1: because populations $S$ and $I$ are assumed to be connected, each individual from population $I$ infects individuals of population $S$ with rate $\alpha$ (per unit of time); the so-called infection or contamination rate. As such, the number of infected individuals is increased by the number $\alpha SI$ at each time step. Also, infected individuals are "removed," that is, recover or die, with a rate denoted by $\beta$ and usually (ambiguously) named the "recovery" rate. Note that in this simple model, the population $R$ contains both healings and deaths.

**FIGURE 1: SIR MODEL**


As this model cannot distinguish recovered from deaths compartments, a SIRD model will be used in the following.

**FIGURE 2: AN EXAMPLE OF SIR MODEL MODULATION**

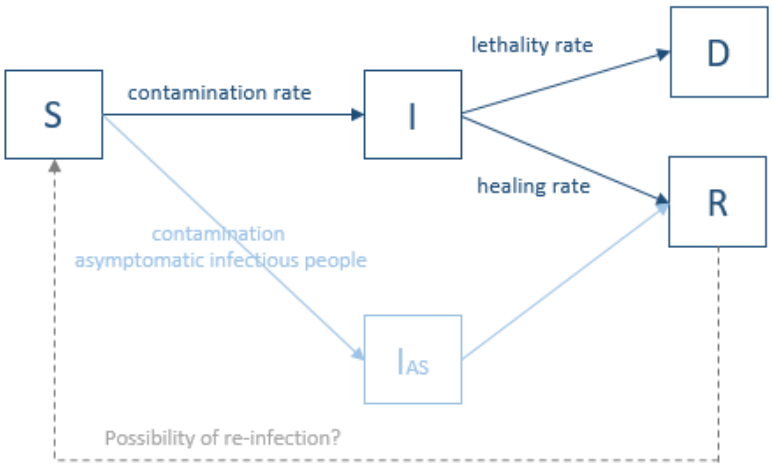



Figure 2 presents a more sophisticated SIR-type model:

- Infectious people are divided into two categories. $I_{AS}$ are asymptomatic infectious people, and $I$ are infectious people who present symptoms of the virus.
- Recoveries are divided into $R$-healings and $D$-deaths.
- It is possible to assume that cured individuals may be infected again, which is represented by the gray dotted arrow.

Under this more complex model, additional parameters need to be estimated:

- The lethality rate, which is commonly called case fatality rate (CFR), represents the risk of death among infected cases. This is of particular interest in the rest of this paper.
- The contamination rate leading to asymptomatic individuals. This is challenging to estimate as the number of infected asymptomatic cases is not fully known. Therefore, assumptions have to be made.
- The rate at which already cured individuals can be put into the $S$ population to be potentially infected again. This is also challenging to estimate, and again, assumptions have to be made.
- The recovery rate (or healing rate). This is difficult to estimate for the asymptomatic population, as there is limited data about this component.

Incubation time, travel, and mitigating actions can also be added to the model. In addition, several modulations of the SIR model have been tested in literature: for instance, the SEIR model (see [6]), where a component for Exposed is added to the model.

For our illustration, we use a parametric SIRD model, based on the following available data variables for the United States: confirmed cases, symptomatic infected people, recoveries, and deaths. We then make death projections and use the real data to check the predictions provided by the model.

The dynamics for each group of individuals are modeled using the following differential equations, which are a mathematical translation of Figure 2 in the simplified case where there are no reinfections and $I_{AS} = 0$:

$$\begin{aligned}
\frac{\mathrm{d}S(t)}{\mathrm{d}t} &= -\frac{\beta(t)}{N} I(t) \cdot S(t) \\
\frac{\mathrm{d}I(t)}{\mathrm{d}t} &= \frac{\beta(t)}{N} I(t) \cdot S(t) - \gamma(t)I(t) - \delta(t)I(t) \\
\frac{\mathrm{d}R(t)}{\mathrm{d}t} &= \gamma(t)I(t) \\
\frac{\mathrm{d}D(t)}{\mathrm{d}t} &= \delta(t)I(t)
\end{aligned}$$

where:

- $\beta(t)$ is the contamination rate
- $\delta(t)$ is the lethality rate
- $\gamma(t)$ is the recovery rate.

Note that the variables $S, I, R, D, \beta, \delta, \gamma$ used in the model are assumed to only depend on time $t$. In particular, they are assumed not to depend on the age and gender of the individuals. Taking these two additional dimensions into account in the model would make it much more complex.

Rather than integrating the age and gender dimensions into the SIRD model, a refinement method is used to derive the excess mortality rate due to COVID-19 by age and gender from the death projections of the SIR model.

### CONTAMINATION RATE

The contamination rate $\boldsymbol{\beta(t)}$ varies over time, and its parametric shape depends on the mitigation measures taken.

The contamination rate is chosen to be exponentially decreasing from the starting date of public mitigation measures. The shape of the contamination rate changes after several mitigation measures come to an end such as indoor dining services and school closings.

### LETHALITY RATE

The lethality rate $\boldsymbol{\delta(t)}$ is assumed to decrease over time, thanks to:

- Containment measures that allow the entire population to be less affected, and therefore also the most fragile who are most likely to die from the disease.
- The progress made by the medical profession in terms of treating the virus and caring for patients.

Note that the lethality rate can be recalibrated in the event of the appearance of a new, more lethal variant of the virus.

### RECOVERY RATE

The recovery rate $\gamma(t)$ is assumed to be constant over time in our case, but it could vary, for example as new medical treatments are discovered.

### $R_0$ PARAMETER

The so-called basic reproduction number $\mathrm{R}_0$ is the average number of people a given infected individual infects in a fully susceptible population. The estimation of this parameter $\mathrm{R}_0$ can change over time with the evolution of the underlying parameters (contamination, recovery, and fatality rates), and due to the implementation of certain mitigation practices. In the modeling example above, the reproduction number can be written as follows:

$$\mathrm{R}_0 = \frac{\beta}{\gamma + \delta}$$

Note, however, that in the case of the SARS-CoV-2 virus the $\mathrm{R}_0$ parameter does not truly reflect the reality of transmission, as 70% of infected individuals do not transmit the virus to any other individuals, and 10% to 20% of infected individuals contribute to 80% of contaminations, see [1].

# Data

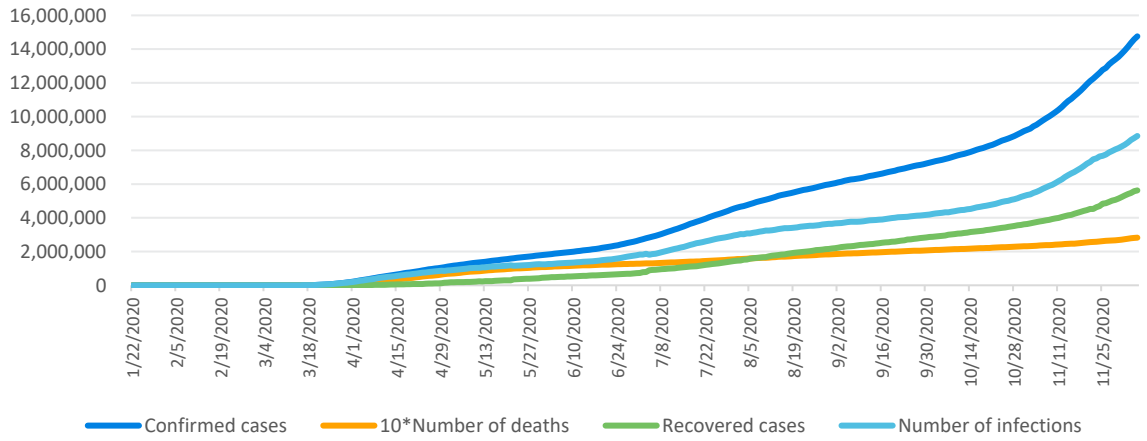
We will focus on U.S. data. Data on the spread of COVID-19 in the United States from January 22, 2020, to December 6, 2020, are shown in Figure 3. We chose to make mortality projections using data up to early December 2020, as the vaccination campaign in the United States started in mid-December 2020 and it is still too early to determine the impact of vaccinations. The data we use are from the Johns Hopkins University and are defined as follows:

- **Confirmed:** Counts include confirmed and probable cases.
- **Deaths:** Counts include confirmed and probable deaths.
- Recovered: Recovered cases are estimates based on local media reports, and state and local reporting when available, and therefore may be substantially lower than the true number.
- **Infected:** the number of infected individuals is deduced from the other variables using the following formula: Infected = Confirmed – Recovered – Deaths. Note that the accuracy of the number of infected is dependent on the accuracy of each of the individual components of the formula.

Note that all estimates may vary by state and that a probable case or death is defined as any one of the following:

- It meets clinical criteria and epidemiological linkage with no confirmatory laboratory testing performed for SARS-CoV-2
- It meets presumptive laboratory evidence
- It meets vital records criteria with no confirmatory laboratory evidence for SARS-CoV-2

**FIGURE 3: EMPIRICAL DATA OF COVID-19 SPREADING IN THE UNITED STATES**




There is a clear progression of the virus from the end of June 2020 (Confirmed curve). This finding is likely to be biased by the fact that more tests may have been performed from that point on.

# Calibrating and forecasting

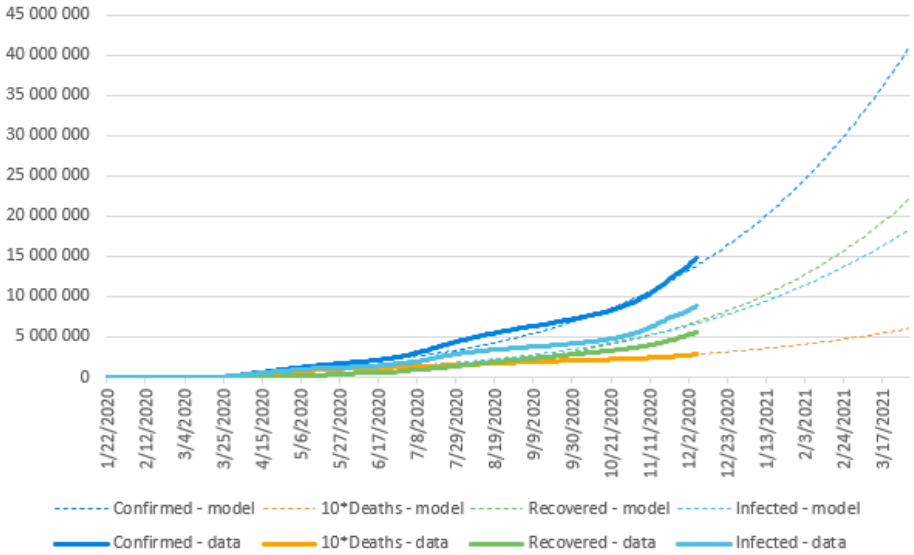
As the mitigation decisions are different for each state, dynamics at the country level is an aggregation of many dynamics. By way of illustration, the dynamics of the states of New York, New Jersey, and Massachusetts are presented, as well as their projections by the model, in Figures 5 to 7.

We start with a country-level projection that uses aggregate data from all U.S. states.

We then consider the states of New York, New Jersey, and Massachusetts, which are respectively the third, seventh, and 12$^{th}$ most affected states in terms of number of COVID-19 deaths as of October 3, 2021.

## COUNTRY-LEVEL DEATH PROJECTIONS

**FIGURE 4: COUNTRY-LEVEL PROJECTIONS**

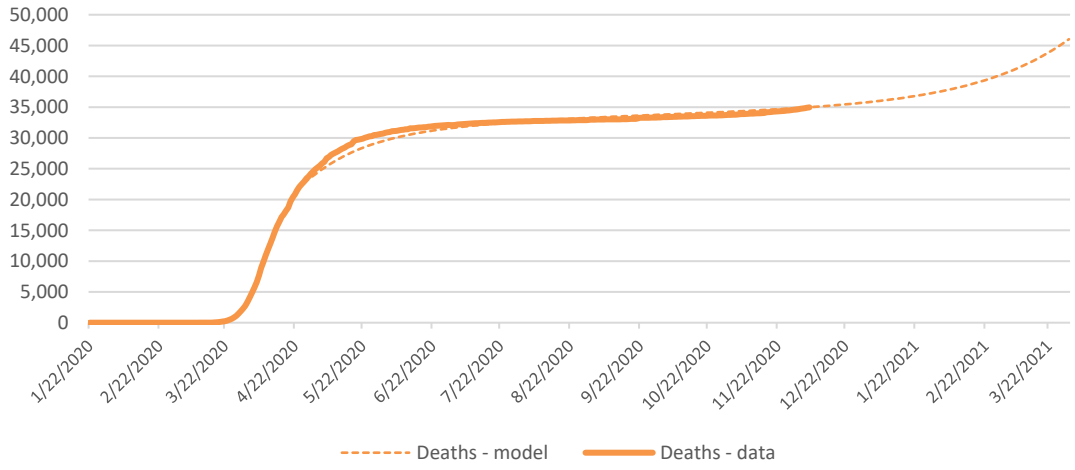



When using aggregated data at the country level, the model predicts a total of 331,000 deaths at the end of the year 2020 and 606,000 deaths at the end of March 31, 2021.

In reality, the number of deaths as of December 31, 2020, is 352,000, and is 552,000 as of March 31, 2021. Thus, our model underestimates the number of deaths projected at the end of the year 2020 and gives pessimistic results at the end of March 31, 2021.

## STATE-LEVEL DEATH PROJECTIONS

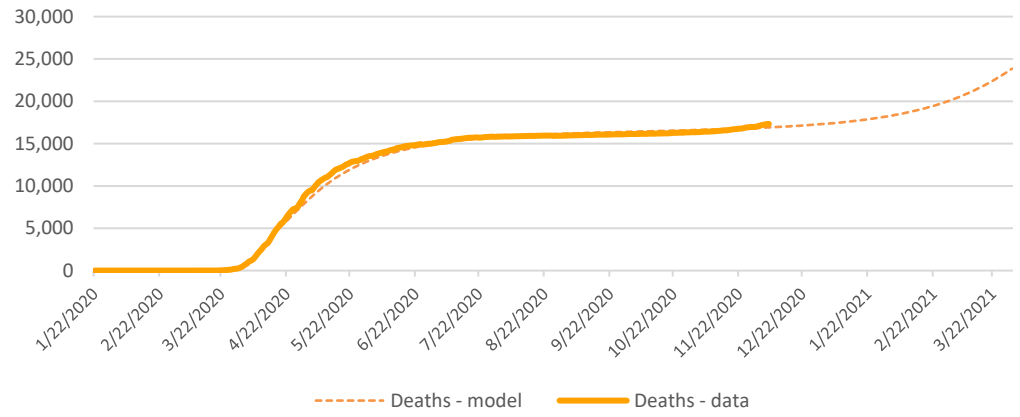
**FIGURE 5: NEW YORK DEATH PROJECTIONS**




When using data at the New York state level, the model predicts a total of 36,000 deaths at the end of the year 2020 and 46,000 deaths at the end of March 31, 2021.

In reality, the number of deaths as of December 31, 2020, is 38,000, and is 50,000 as of March 31, 2021. Thus, our model underestimates the number of deaths projected at the end of the year 2020 and at the end of March 31, 2021.

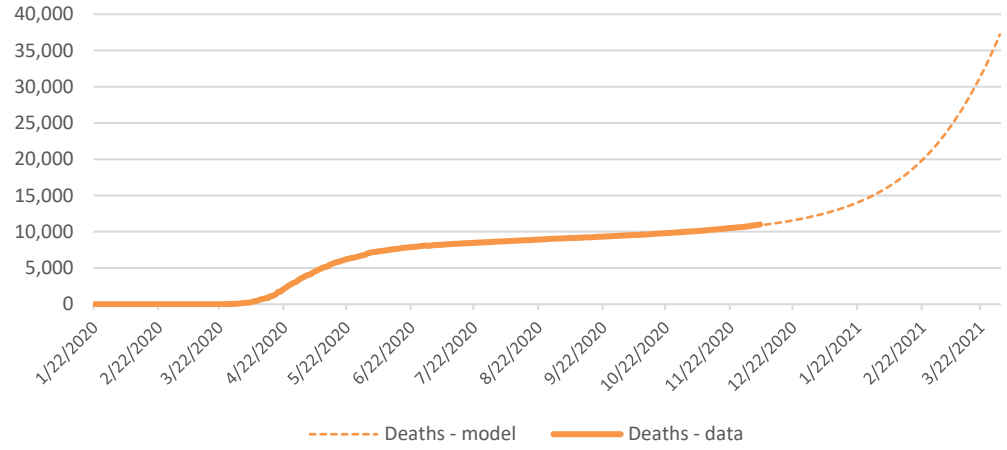
**FIGURE 6: NEW JERSEY DEATH PROJECTIONS**




When using data at the New Jersey state level, the model predicts a total of 17,000 deaths at the end of the year 2020 and 24,000 deaths at the end of March 31, 2021.

In reality, the number of deaths as of December 31, 2020, is 19,000, and is 25,000 as of March 31, 2021. Thus, our model slightly underestimates the number of deaths projected in the short term and the medium term.

**FIGURE 7: MASSACHUSETTS DEATH PROJECTIONS**




When using data at the Massachusetts state level, the model predicts a total of 12,000 deaths at the end of the year 2020 and 37,000 deaths at the end of March 31, 2021.

In reality, the number of deaths as of December 31, 2020, is 12,000, and is 17,000 as of March 31, 2021. Thus, our model prediction is good in the short term but far too pessimistic in the medium term.

We therefore note that, in general, our model gives good predictions of the number of deaths over a short horizon, with an order of magnitude close to that of the real data. Nevertheless, for distant horizons, its predictive capacity is limited because the future trend of the epidemic's spread depends on parameters that are not taken into account in the model and that could vary a lot depending on the state (public mitigation measures, population behavior, appearance of super-spreaders, etc.)

## CONVERSION INTO EXCESS MORTALITY BY AGE GROUP AND GENDER

The objective of this section is to determine a modeled structure of the U.S. excess mortality rate due to COVID-19 by age and gender for the first quarter (Q1) of year 2021. Note that this is an absolute excess mortality rate, i.e., it is additive compared to regular mortality.

For each region (state or the whole country), the aim is to find the age and gender specific mortality rate for the region, called $\mu(a, g)$, where $a$ is the age group and $g$ is the gender.

We look for the age pyramid $E(a, g)$ in the region, on January 1, 2020 (see [7]), and the number of COVID-19 deaths $\widetilde{D}(a, g)$ by age group $a$ and gender $g$ (see [8]).

We denote by $D_{proj}$ the projected total number of COVID-19 deaths at the end of Q1 2021, provided by the SIR-type model. Then we deduce the mortality rate $\mu(a, g)$ by the following relationship:

$$\mu(a, g) = \frac{D_{proj}}{\sum_a \widetilde{D}(a, g)} \cdot \frac{\widetilde{D}(a, g)}{E(a, g)}$$

Figure 8 depicts the U.S. modeled excess mortality rate due to COVID-19 by age group and gender at the country level while Figures 9 to 11 depict the U.S. excess mortality rates due to COVID-19 by age group and gender at the state level for New York, New Jersey, and Massachusetts.

It is observed that the order of magnitude of the modeled excess mortality rate is similar between the country level and the state levels of New York and New Jersey. However, the order of magnitude for the Massachusetts modeled excess mortality rate is much higher. This is a direct consequence of the fact that the SIR-type model predicts a much larger number of deaths for Massachusetts. See the section on state-level death projections for more details.

**FIGURE 8: U.S. MODELED EXCESS MORTALITY RATE BY AGE AND GENDER**

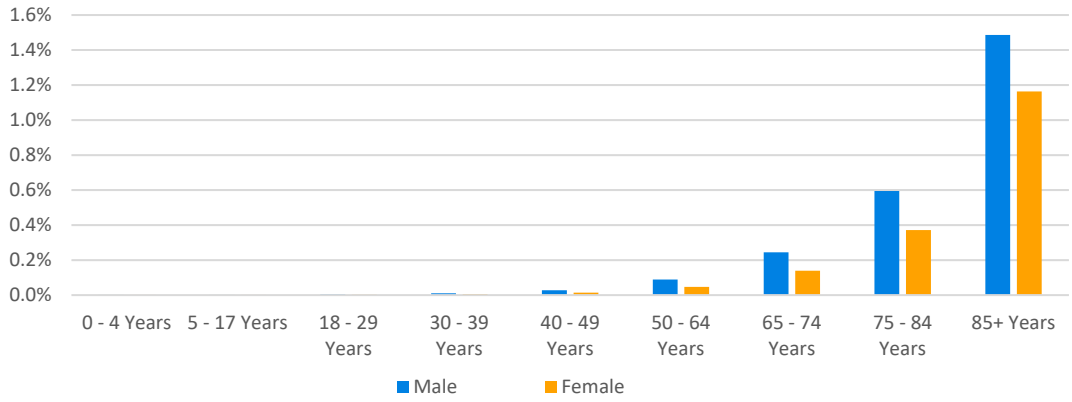



**FIGURE 9: NEW YORK MODELED EXCESS MORTALITY RATE BY AGE AND GENDER**

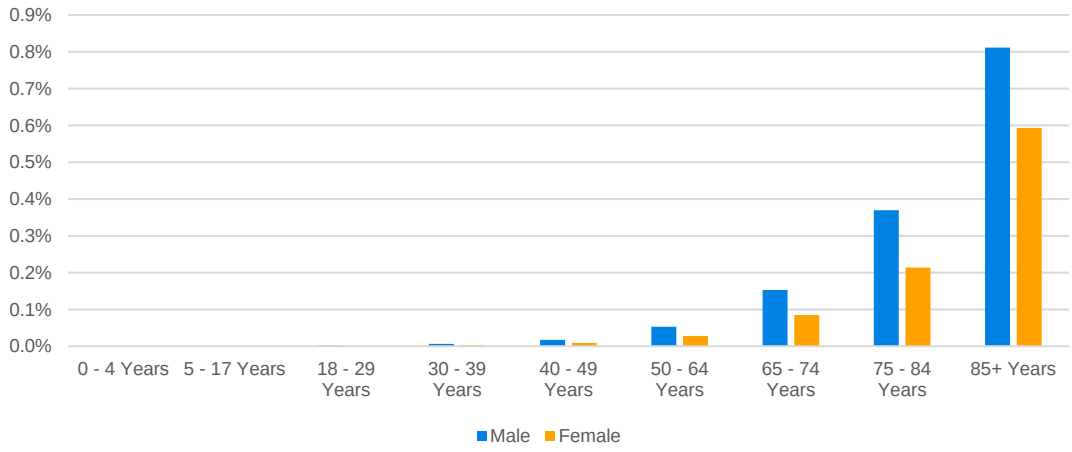


**FIGURE 10: NEW JERSEY MODELED EXCESS MORTALITY RATE BY AGE AND GENDER**

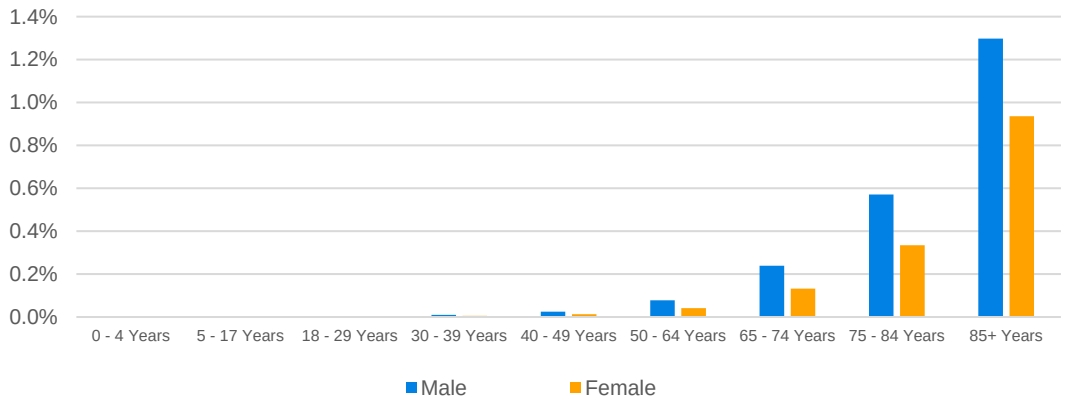
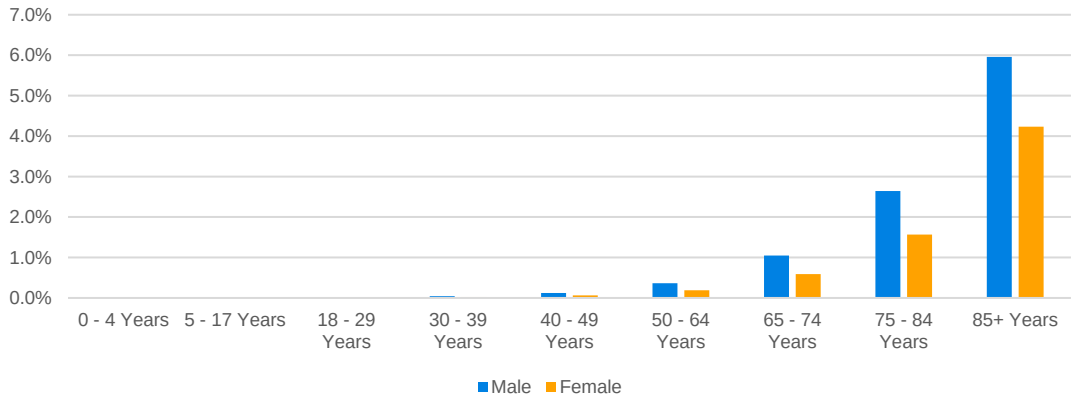



**FIGURE 11: MASSACHUSETTS MODELED EXCESS MORTALITY RATE BY AGE AND GENDER**




## ADJUSTING THE MODEL FOR THE EXISTENCE OF A NEW STRAIN AND VACCINATION OF THE POPULATION

Genetic variants of SARS-CoV-2 have been emerging and circulating around the world throughout the COVID-19 pandemic. According to the Centers for Disease Control and Prevention (CDC, see [9]), a variant of concern is a variant for which there is evidence of an increase in transmissibility, more severe disease (e.g., increased hospitalizations or deaths), significant reduction in neutralization by antibodies generated during previous infection or vaccination, reduced effectiveness of treatments or vaccines, or diagnostic detection failures.

As of August 13, 2021:

- The B.1.1.7 (Alpha), B.1.351 (Beta), B.1.617.2 (Delta), and P.1 (Gamma) variants circulating in the United States are classified as variants of concern.
- 50% of the U.S. population are fully vaccinated against COVID-19 and 8.8% are only partly vaccinated against the disease.

In [10], the authors studied a novel multi-strain SIR-type epidemic model with selective immunity by vaccination. They assumed that the newer strain does not exhibit cross-immunity with the original strain, hence the individuals who are vaccinated and recovered from the original strain become susceptible to the newer strain.

Before the emergence of a new strain, the modified SIR model has four compartments, which are given by:

- Susceptible $S$: Individuals in this compartment are healthy but are susceptible to be infected by the disease because they are not vaccinated.
- Vaccinated $V$: Individuals who were given a vaccine, making them immune to the disease.
- Infected $I_1$: Individuals who are infected by the disease.
- Removed $R$: Individuals who were infected but are now recovered and immune to the disease.

It is assumed that the individuals are vaccinated with a vaccination rate $p$. The dynamics for each group of individuals are modeled using the following differential equations, which are a mathematical translation of Figure 12:

$$\frac{dS}{dt} = (1-p)\mu N - \frac{\beta S I_1}{N} - \mu S$$

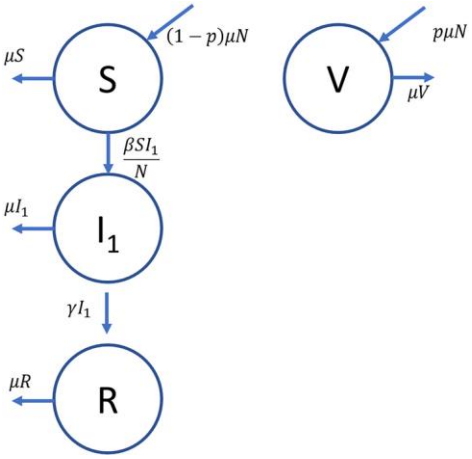
$$\frac{dI_1}{dt} = \frac{\beta S I_1}{N} - (\gamma + \mu) I_1$$

$$\frac{dV}{dt} = p\mu N - \mu V$$

$$R = N - S - I_1 - V$$

where μ is assumed to be the natural birthrate of the population, and consequently the natural deathrate of the population to keep the population size constant, $\beta$ is the contamination rate, and $\gamma$ is the removal rate.

**FIGURE 12: COMPONENT DIAGRAM FOR THE EMERGING DISEASE MODEL BEFORE THE EMERGENCE OF A NEW STRAIN, SEE [10]**




Suppose that a new strain of the disease is introduced to the population. This new strain will have a different contamination rate $\beta'$ and removal rate coefficient $\gamma'$. This results in the existence of another component $I_2$ for those who are infected with the new strain. Moreover, it is assumed that an individual recovered from the newer strain cannot be reinfected by the older strain.

This means that the number of components that need to be monitored will increase from four to six, with the addition or modification of the following compartments:

- $R_1$: Individuals who have recovered from the original strain but are now susceptible to the newer strain.
- $I_2$: Individuals who are infected by the newer strain.
- $R_2$: Individuals who were previously infected by the newer strain but have now been removed due to recovery or treatment.

Thus, the dynamics for each group of individuals are modeled using the following differential equations, which are a mathematical translation of Figure 13:

$$\frac{dS}{dt} = (1-p)\mu N - \frac{\beta S I_1}{N} - \frac{\beta' S I_2}{N} - \mu S$$

$$\frac{dI_1}{dt} = \frac{\beta S I_1}{N} - (\gamma + \mu) I_1$$

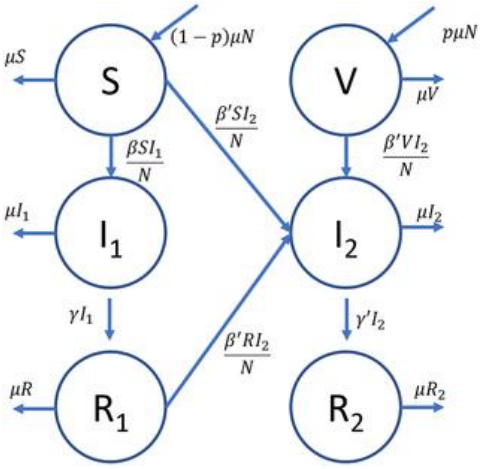
$$\frac{dV}{dt} = p\mu N - \frac{\beta' V I_2}{N} - \mu V$$

$$\frac{dR_1}{dt} = \gamma I_1 - \frac{\beta' R_1 I_2}{N} - \mu R_1$$

$$\frac{dI_2}{dt} = \frac{\beta'(S + V + R_1) I_2}{N} - (\gamma' + \mu) I_2$$

$$R_2 = N - S - I_1 - I_2 - R_1 - V$$

**FIGURE 13: COMPONENT DIAGRAM FOR THE EMERGING DISEASE MODEL AFTER THE EMERGENCE OF A NEW STRAIN, SEE [10]**




# How to model the future effects of the pandemic

### DIRECT LONG-TERM EFFECTS AND INDIRECT MEDIUM-TERM EFFECTS

Typically, people recover from COVID-19 after two to six weeks. For some people, some symptoms may persist or reappear for weeks, months, or even more than a year after the initial recovery, see [11]. Such persistent symptoms can also occur for people with mild illness, even for young adults and children who do not have any underlying chronic illness.

The long-term health consequences of COVID-19 remain largely unclear [12]. The aim of a study on individuals six months post contraction of COVID-19 [13] is to describe the long-term health consequences of patients with COVID-19 who are discharged from hospital and to investigate the associated risk factors, in particular the severity of the disease. The authors use an ambidirectional cohort study of patients with COVID-19 who were released from Jin Yin-tan Hospital (Wuhan, China) between January 7, 2020, and May 29, 2020. Six months after infection, COVID-19 survivors mainly suffered from fatigue or muscle weakness, trouble sleeping, and anxiety or depression. Patients who were more seriously ill during their hospital stay tended to have more severe impaired pulmonary diffusion and abnormal chest imaging manifestations. In addition, 13% of patients who did not appear to have acute kidney injury during hospitalization showed signs of kidney dysfunction. The results of the study also showed that many patients continue to live with some of the effects of the virus after recovery. These symptoms can interfere with daily activities and have a significant negative impact on quality of life.

Another study [14] was done on the long-term effects of Severe Acute Respiratory Syndrome (SARS), the coronavirus that emerged in 2003. The authors showed a persistent and significant impairment in the exercise capacity and health status of SARS survivors over a period of 24 months. Healthcare workers with SARS have suffered an even greater negative impact.

In another study [15], the authors found that 40% of people recovering from SARS still had symptoms of chronic fatigue 3.5 years after diagnosis. Note that SARS has been caused by SARS-CoV, a coronavirus identified in 2003.

In addition to the previous direct effects of COVID-19, indirect medium-term effects must also be considered:

- **Access to vaccination:** The poorest countries with less access to vaccination will likely experience higher excess mortality.
- **Effectiveness of the vaccines:** Will vaccines protect against the virus, including any mutations of the virus?
- **Social and economic conditions following the pandemic:** In a study [16], the authors found that employment changes during and after the 2008 global financial crisis had a strong adverse effect on chronic health for five broad types of health conditions, with the strongest effects for mental health conditions. Quantitatively, they estimated that a 1% fall in employment leads to a 2% increase in the prevalence of chronic illness. To put this in context, if employment were to fall by the same amount as it fell in the 12 months after the 2008 crisis, around 900,000 more people of working age in the UK would be predicted to suffer from a chronic health condition. Nevertheless, they estimated that the shock to employment from the coronavirus pandemic is likely to be much larger than this and so they expect a larger rise in poor health, which would also generate a higher excess mortality.

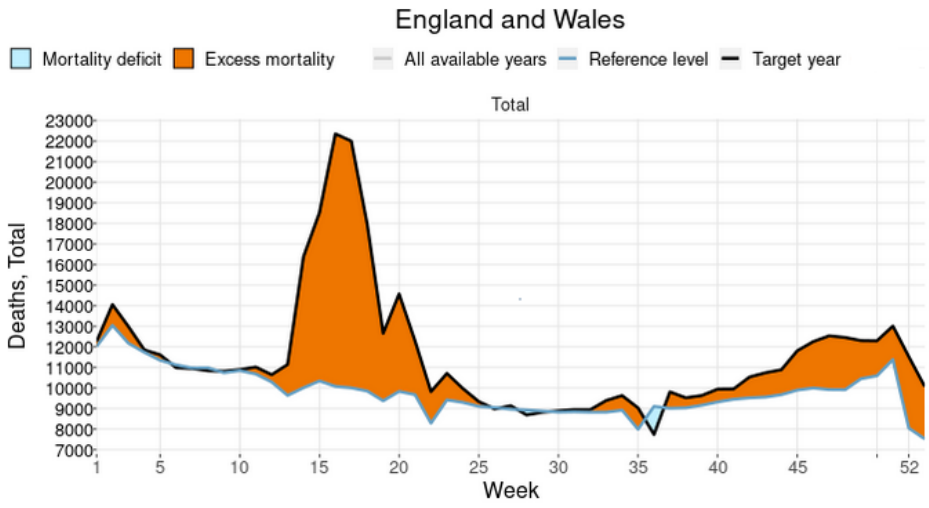
## HOW TO PROJECT FUTURE MORTALITY AFTER A PANDEMIC

The Continuous Mortality Investigation (CMI) Mortality Projections Model is widely used across the insurance and reinsurance industry to project longevity improvements. The model is updated regularly for emerging mortality trends within England and Wales. It is a deterministic model based on the assumption that current rates of mortality improvement converge to a single long-term rate. The latest version of the Model, CMI_2020, was published in March 2021, see [17].

In a typical year, the CMI_2020 model would be calibrated to England and Wales population mortality data up to December 31, 2020 and use data from the previous 40 years to inform the starting point for future mortality improvements.

However, because of the COVID-19 pandemic, mortality in 2020 in England and Wales was significantly higher than in recent years, see Figure 14. The CMI chose to introduce a new parameter in the model that allows users to explicitly adjust the weighting given to the 2020 data. The core value of CMI_2020 places no weight on data for 2020, and 100% weight on all other years.

**FIGURE 14: WEEKLY EXCESS MORTALITY IN ENGLAND AND WALES FOR THE YEAR 2020 COMPARED TO THE PERIOD OF 2010-2019, SEE [18]**



Thus, several calibration methods can be chosen in order to carry out mortality projections using the latest version of the CMI_2020 model:

- **Keep the CMI 2019 model:** This reflects the view that COVID-19 is likely a one-off temporary shock and the 2020 mortality experience is not relevant for projecting future improvements in mortality.
- **Use the CMI_2020 model with no weight to the 2020 data:** This produces a slight reduction in life expectancies, in comparison with the CMI_2019 model. This also reflects that the 2020 mortality experience is not relevant.
- **Use the CMI_2020 model with a nonzero weight to the 2020 data:** This reflects a view that the 2020 mortality experience is relevant as the future mortality rates will be impacted by COVID-19, as discussed in the previous point. In order to choose the most relevant weight, assumptions about the impacts of the pandemic on the future mortality rates should be made.

It should also be mentioned that COVID-19 is continuing to generate excess deaths during the year 2021, and thus the CMI is likely to take a similar approach to its model next year. Care should be taken in choosing the weights to apply to the data for the years 2020 and 2021 as they interact.

In the United States, the most recent 2021 versions of the Retirement Plans Experience Committee (RPEC) mortality improvement model and the Mortality Improvement Model (MIM) allow the actuary to make an adjustment for COVID-19. This adjustment factor would be applied to the projected mortality improvement rates for 2022, 2023, 2024, and 2025 and beyond. This factor can create a smaller or larger set of mortality improvement assumptions.

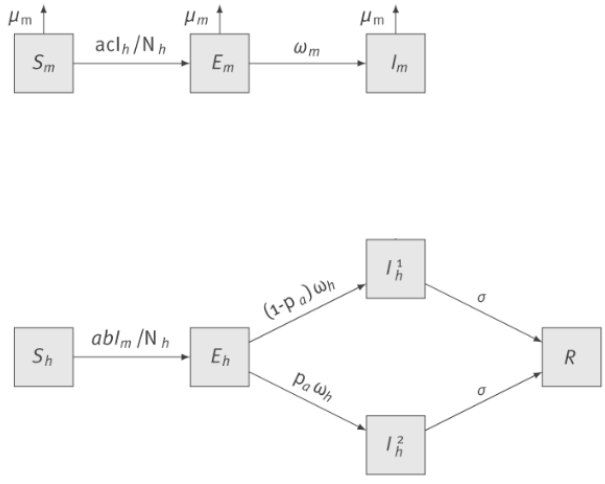
### CASE OF VECTOR-BORNE DISEASES

Finally, another type of epidemic, like vector-borne diseases, could emerge in the future. Unlike COVID-19, which is primarily transmitted from human to human (see [19]), vector-borne diseases are transmitted by insects or animals, for example via mosquitoes.

Following [20], such an epidemic can be modeled by a refined SIR-type model. Unlike classic SIR-type models, two populations have to be modeled: human individuals and mosquito vectors.

- **Human individuals:** They start in a susceptible state $S_h$ and then can be infected through bites of infectious mosquito vectors $I_m$ at rate $a$ and with the probability $b$. When infected, these individuals change to the exposed state $E_h$. They become infectious and symptomatic $(I_h^1)$ with the probability $\omega_h(1 - p_a)$, or asymptomatic $(I_h^2)$ with the probability $\omega_h p_a$. Finally, infectious individuals become recovered $R$ at rate $\sigma$ and cannot be infected again.
- **Mosquitoes:** If a susceptible mosquito vector $S_m$ bites an infectious human at rate $a$, the mosquito can become exposed $(E_m)$ with the probability $c$ and then infectious $(I_m)$ at the rate $\omega_m$, characterized by the inverse of the extrinsic incubation period. Note that the infection probability of a susceptible mosquito exposed to the pathogen $c$ is dependent on both the local temperature and the degree of adaptation of the mosquito to its environment.

**FIGURE 15: COMPONENT DIAGRAM FOR A VECTOR-BORNE DISEASE SIR-TYPE MODEL**



# Conclusion

This paper has described models that can be used to model pandemics such as COVID-19. These models allow for the modeling of both infection and mortality dynamics and, as shown in the report, the back-testing has confirmed that the results produced were generally reasonable.

That said, note that the scenarios are made based on incomplete data and uncertain assumptions, in particular regarding the behavior of the population and political decisions. The trajectories described by the models depend on the assumptions chosen; if the assumptions do not come true, the actual dynamics observed may be different from the projections provided by the models.

In this paper, two types of models have been described:

- SIR-type models are used to project a number of deaths over a short horizon during a pandemic. They can be adjusted to take into account the existence of a new variant of the virus, and the vaccination rate of the population. As demonstrated above, some projections may be inaccurate, based on the relevance of the assumptions of the model.
- Other types of models (e.g., the CMI model) are used to project mortality over a longer horizon. Note that in order to produce more accurate mortality projections, these models can be refined and adjusted by incorporating assumptions about the direct and indirect future effects of the pandemic on mortality.



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# References

[1] Adam, Dillon C., et al. "Clustering and superspreading potential of SARS-CoV-2 infections in Hong Kong." Nature Medicine 26.11 (2020): 1714-1719.

[2] Dixon, M. G., & Schafer, I. J. (2014). Ebola viral disease outbreak—West Africa, 2014. MMWR. Morbidity and mortality weekly report, 63(25), 548.

[3] Brazeau, N. F., Verity, R., & Jenks, S. COVID-19 Infection Fatality Ratio: Estimates from Seroprevalence. 2020. Imperial College London: London

[4] Donnelly, Christl A., et al. "Epidemiological determinants of spread of causal agent of severe acute respiratory syndrome in Hong Kong." The Lancet 361.9371 (2003): 1761-1766.

[5] Taquet, Maxime, et al. "Incidence, co-occurrence, and evolution of long-COVID features: A 6-month retrospective cohort study of 273,618 survivors of COVID-19." PLoS Medicine 18.9 (2021): e1003773.

[6] Adam J Kucharski, Timothy W Russell, Charlie Diamond, Yang Liu, John Edmunds, Sebastian Funk, Rosalind M Eggo, on behalf of the Centre for Mathematical Modelling of Infectious Diseases COVID-19 working group. 2020. Early dynamics of transmission and control of COVID-19: a mathematical modelling study. The Lancet. Available at https://www.thelancet.com/action/showPdf?pii=S1473-3099%2820%2930144-4

[7] US State Population by Characteristics: 2010-2019, United States Census Bureau Available at: https://www.census.gov/data/tables/time-series/demo/popest/2010s-state-detail.html

[8] Demographic Trends of COVID-19 cases and deaths in the US reported to CDC Available at: https://COVID.cdc.gov/COVID-data-tracker/#demographics

[9] CDC SARS-CoV-2 Variant Classifications and Definitions Available at: https://www.cdc.gov/coronavirus/2019-ncov/variants/variant-info.html#Concern

[10] Fudolig, Miguel, and Reka Howard. "The local stability of a modified multi-strain SIR model for emerging viral strains." PloS one 15.12 (2020): e0243408.

[11] Huang, Lixue, et al. "1-year outcomes in hospital survivors with COVID-19: a longitudinal cohort study." The Lancet 398.10302 (2021): 747-758.

[12] Lopez-Leon, S., Wegman-Ostrosky, T., Perelman, C. et al. More than 50 long-term effects of COVID-19: a systematic review and meta-analysis. Sci Rep 11, 16144 (2021). https://doi.org/10.1038/s41598-021-95565-8

[13] Huang, Chaolin, et al. "6-month consequences of COVID-19 in patients discharged from hospital: a cohort study." The Lancet 397.10270 (2021): 220-232.

[14] NGAI, Jenny C., KO, Fanny W., NG, Susanna S., et al. The long-term impact of severe acute respiratory syndrome on pulmonary function, exercise capacity and health status. Respirology, 2010, vol. 15, no 3, p. 543-550.

[15] Lam, Marco Ho-Bun, et al. "Mental morbidities and chronic fatigue in severe acute respiratory syndrome survivors: long-term follow-up." Archives of internal medicine 169.22 (2009): 2142-2147.

[16] Banks, James, Heidi Karjalainen, and Carol Propper. "Recessions and health: The long-term health consequences of responses to the Coronavirus." Fiscal Studies 41.2 (2020): 337-344.

[17] CMI Bureau. CMI Mortality Projections Model: CMI 2020.Mortality Projections Committee Working Paper 147, 2021

[18] Németh, László, Dmitri A. Jdanov, and Vladimir M. Shkolnikov. "An open-sourced, web-based application to analyze weekly excess mortality based on the Short-term Mortality Fluctuations data series." PloS one 16.2 (2021): e0246663.

[19] Animals and COVID-19 Available at: https://www.cdc.gov/coronavirus/2019-ncov/daily-life-coping/animals.html

[20] Sochacki, Thomas, et al. "Imported chikungunya cases in an area newly colonised by Aedes albopictus: mathematical assessment of the best public health strategy." Eurosurveillance 21.18 (2016): 30221.