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# COMPARING AGE-SPECIFIC MORTALITY PATTERNS OF COVID-19 MORTALITY

A thesis submitted in partial satisfaction
of the requirements for the degree of

MASTER OF ARTS

in

ECOLOGY AND EVOLUTIONARY BIOLOGY

by

**Chloe Rickards**

June 2022

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**Abstract**

Comparing Age-Specific Mortality Patterns of COVID-19 Mortality

Chloe Rickards

Mortality patterns between different populations vary because of many factors including genetic variation, environmental conditions, and age. The ongoing COVID-19 pandemic has killed nearly 1 million people in the United States and nearly 6 million globally, and has severely affected older populations. Accurately estimating the age-specific infection fatality rate (IFR) of SARS-CoV-2 for different populations is crucial for assessing and understanding the impact of COVID-19 and for appropriately allocating limited vaccines and treatments. We estimated age-specific IFRs of wild-type SARS-CoV-2 using published seroprevalence data and recorded deaths in New York City (NYC) from March to May 2020. We estimated the probability of death given infection using a Bayesian framework that accounted for delays between infection and seroconversion and infection and death. IFRs increased more than 75-fold with age, from 0.07% in individuals between 18-45 years old to 5.3% in individuals over 75. IFRs in NYC were higher than IFRs in England, Switzerland, Belgium, France, and Spain for individuals younger than 65 years old, but similar for older individuals. These results suggest that the age-specific fatality of COVID-19 differs among developed countries and raises questions about factors underlying these differences.

**Acknowledgments**

The text of this thesis includes a reprint of the following pre-print material: Rickards C, Kilpatrick AM. Age-Stratified SARS-CoV-2 Infection Fatality Rates in New York City estimated from serological data. *medRxiv*; 2020. doi: https://doi.org/10.1101/2020.10.16.20214023 [1]

The co-author listed in this publication directed and supervised the research which forms the basis for the thesis.

Thank you to Javier Perez-Saez and co-authors for making their R code available which greatly facilitated the analyses presented here. Thank you to members of the Kilpatrick lab for feedback.

## Introduction

Organisms are subject to a variety of sources of mortality that can exert selective pressures throughout their lifespan, including predation, starvation due to competition or fluctuating resources, extreme environmental conditions, and infectious diseases. Mortality due to infectious disease depends on several factors, including host characteristics [2,3], pathogen characteristics [4,5], host density [6], population structure [7], external selection pressures [8], and age [6]. Human infectious disease mortality outcomes are also impacted by pre-existing conditions [3], socioeconomic inequality [9], racial inequality [9,10], and immigration status [11].

A detailed understanding of differences in disease mortality between populations requires accounting for as many factors as possible that might influence mortality. For many diseases, age is a key factor influencing mortality, so comparing mortality between populations is best done with age-specific mortality estimates. For example, mortality from HIV/AIDS, which primarily impacts adults aged 15-49 [12], are much higher in sub-Saharan Africa compared to the rest of the world [12]. However, sub-Saharan Africa has, on average, a younger population than the rest of the world [13]. Controlling for a younger population within sub-Saharan Africa may illuminate other HIV/AIDS mortality risk factors, such as differences in healthcare access [7,12].

Age-specific variation in disease mortality is also apparent for many plants and animals. Some diseases affect a specific life stage, such as damping-off diseases in plant seedlings [14], or Ribeiroia in amphibian larvae [15]. Other diseases, such as feline immunodeficiency virus (FIV) in African lions [16], cause mortality in later stages of life. Identifying age-specific disease mortality patterns can often help in targeting management or conservation efforts – for example, Sudden Oak Death (*Phytophthera ramorum*) affects mature oaks with larger diameters [17], so prevention efforts are often concentrated on these types of trees [18].

Sometimes, age-specific mortality patterns due to disease are difficult to quantify due to interactions with other causes of mortality, such as predation. The prion disease Chronic Wasting Disease (CWD) circulates among some populations of North American cervids. CWD takes 1-2 years to kill its host, so mortality due to CWD is typically observed in young adults [19]. Wolves preferentially feed on deer and elk that are slower or impaired (e.g. neonates, adults with CWD), and thus easier to capture [8]. The combination of age-specific predation and disease creates complex dynamics within the host population [19,20]. In areas where predators are reintroduced, prey populations that were initially controlled by infectious disease may begin to be controlled by predators, thus reducing the incidence of disease and altering age-specific mortality patterns [19,21]. The intersection of age-specific predation and age-specific disease mortality and its impacts on population ecology are only just beginning to be explored.

Mortality for some human diseases is higher in older and younger individuals because of lower immune function [22]. However, the relationship between age-specific declines in immune function and age-specific mortality outcomes sometimes creates counterintuitive patterns. For example, pneumonia mortality rates are typically highest in the youngest and oldest age groups, creating a "U-shaped" curve, that is consistent with weaker immune function in young and old individuals [23,24]. However, mortality in healthy adults (i.e. in the 20-40 year-old age group) was surprisingly high during the 1918-1919 influenza pandemic, creating a "W-shaped" curve [24], due to a hyper-reactive immune response characterized by a cytokine storm.

Age-specific disease mortality patterns have also been strikingly apparent for COVID-19 [25]. The risk of dying from this disease increases strongly with age [25], except for very young children (<5) which have an elevated risk of dying compared to children 5-14 years old. However, unlike pneumonia, even very young children less than five years old are at a much-reduced risk of dying, compared to elderly adults [26]. Intriguingly, mortality from COVID-19 appears to vary among populations, even when quantifying age-specific mortality rates. My aim was to quantify the probability of mortality given infection with SARS-CoV-2 (i.e., the infection fatality rate, IFR) in a population that suffered a very large outbreak in spring 2020: New York City, USA.

**Age-Stratified SARS-CoV-2 Infection Fatality Rates in New York City from Serological Data**

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## Introduction

As of February 2022, COVID-19 has killed nearly 1 million people in the US and nearly 6 million globally [27]. The infection fatality rate (IFR) – the chance of dying after becoming infected – is a crucial metric for understanding the disease severity of SARS-CoV-2. Accurate age-specific IFRs are needed to allocate limited supplies of vaccines, respirators, and ICU beds to minimize mortality from COVID-19 [28]. Additionally, the comparative fatality risk of children is needed to make good policy decisions about reopening schools and day care centers, as well as implementing mask and social distancing policies in these and other places. However, IFR estimates can vary widely between different places. For example, there were substantial differences in age-specific IFRs among countries such as Mexico and Sweden – up to a 5-fold difference in the oldest age group [29,30]. This indicates the need for age-specific and population-specific IFR estimates when considering the mortality impacts of COVID-19 on a specific community.

Accurately estimating IFRs requires both 1) quantification of the total number of infections (including undetected cases) [31], and 2) accounting for delays between infection and death. First, the total number of infections includes not only reported cases, but also undetected cases, many of which, in COVID-19, are asymptomatic or mildly symptomatic. So, often, the IFR is underused in comparison to the symptomatic or *case* fatality risk (CFR), which uses reported cases for its fatality estimate. Still, there exist methods to quantify all infections for a pathogen, the most accurate of which are serosurveys. Serosurveys detect the presence of antibodies

within the blood serum of an individual. If serosurveys include a random or unbiased subset of the population, they can be used to estimate the number of infections in a population [32]. For example, serosurveys sourced from blood banks bias a population sample toward individuals who are eligible and willing to donate blood.[31]. Well-designed seroprevalence studies on SARS-CoV-2 are crucial for forming a basis to an accurate IFR estimate.

Second, deaths due to COVID-19 occur, on average, 20.2 days (95% CI 8.0 – 50.0 [33]) after infection. This delay can be broken down into several components [33], including the incubation period [34], the delay between symptom onset and case reporting [35], and the delay between reporting and death [33]. Accurately estimating IFRs requires properly accounting for these delays, as well as the delay between infection and mounting detectable antibodies (seroconversion), which can be challenging when cases are fluctuating substantially (e.g. [33]).

Previous studies of age-specific IFRs for COVID-19 have found a log-linear increase in IFR with age [25,36–41], except for elevated deaths in very young children [42]. Above, 18 years of age, there was a 3- to 4-fold increase in IFR for every 20 years of age % increase in IFR with every five years of age [25,36–41].

We estimated age-specific IFRs using data from a seroprevalence study conducted in New York City shortly after the peak of the spring 2020 epidemic [43], publicly available case and death records [44], and Bayesian inference to account for delays between infection, symptom onset, case ascertainment, death and seroconversion [33].

## Methods

We used data from New York City from a serological survey conducted over a 10-day period (April 19-28, 2020), two-weeks after the peak in cases in Spring 2020 [43]. Survey participants for the serosurvey were recruited at grocery stores without prior advertisement to reduce bias [43]. The serosurvey was conducted through the entire state of New York, but we focused on New York City because of the much higher seroprevalence in New York City, and highly heterogenous seroprevalence across the state [43]. We calculated two sets of IFRs using only *confirmed* COVID-19 deaths or including both confirmed and probable deaths recorded by the New York City Department of Health and Mental Hygiene. Case counts and number of deaths were obtained from the New York City Department of Health and Mental Hygiene archive webpage for the dates 3/9/20 to 5/17/20 [44].

We estimated IFRs using a previously established Bayesian statistical framework [33], which combines seroprevalence estimates (including uncertainty) with time series of cases and deaths. We estimated the number of infections and the fraction dying using log-normal distributions for the delays between infection, symptom onset, becoming a case, seroconversion, and death [33]. This approach estimates the cumulative number of infected people at the time of the seroprevalence study and the fraction of those infected that go on to die.

To link the seroprevalence survey and the deaths reported we had to address mismatches in the age groups for the two datasets. The New York City serosurvey

study reported data in 4 age groups: 18-34, 35-44, 45-54, and 55+ [43]. The New York City Department of Health and Mental Hygiene reported deaths in five age groups: 0-17, 18-44, 45-64, 65-74, and 75+ [44]. We estimate IFR values using to the finer age classes used for deaths by the NYC Dept. of Health and Mental Hygiene, as follows. First, we estimated the IFR for the 0-17 age class. For this youngest age class, there was no seroprevalence estimate for NYC [43], but there were recorded deaths due to COVID-19 in NYC [44]. To estimate the IFR for this age class, we used the seroprevalence ratio between the 0-17 age class and the 18-44 age class using a serosurvey from 2020 in Spain [45]. This ratio was 0.66, and seroprevalence in NYC for the 18-44 age class was 22.3%, resulting in an estimated seroprevalence for 0-17 year-old individuals in NYC of 14.8% (6.89 - 28.2%). Second, we used a single seroprevalence value for individuals over the age of 55 [43] to match reported deaths spanning three different age classes (45-64, 64-75, and 75+) [44]. We used the 55+ seroprevalence value for the 64-75 and 75+ age classes and used a population-weighted average of the 45-54 and 55+ seroprevalences for the 45-64 age class [43,46].

From the serosurvey results [43], the case and death data [44], and the delay distributions [33], we can estimate the IFR and the number of people who were infected within our period of interest. Our methods and formulas follow those of Perez-Saez et al. 2021 [33].

For each age class *a,* we modeled the number of deaths due to COVID-19 using a binomial distribution, based on $I_a$, the age-specific number of people infected

with COVID-19, and $IFR_a$, the age specific probability of dying from COVID-19 infection. From these:

$$Deaths_a \sim binomial(I_a, IFR_a)$$

We use Bayesian inference to estimate the age-specific IFR and apply a Fourier transform of a convolution to infer $I_a$, the number of infected people at risk of dying from COVID-19 within an age class $a$. The number of deaths is known and assumed to be measured without error. In our primary analysis, we only used deaths confirmed as COVID-19 [44].

The number of people infected with COVID-19, $I_a$, is estimated up to the midpoint of the serosurvey (04/23/2020, Figure 1). It is dependent on the number of cases up to the serosurvey midpoint [43], the delay between infection and case reporting (10.2 days; 95% CI 3.7 - 29.2 [33,35,47] ), and the probability that an infection gets reported (α, which follows a distribution inferred from the incubation period and the reporting delay).

The number of infections on a certain day, $I_a(t)$, cannot be observed directly. We estimate $I_a(t)$ using the time series of cases $C_a(t)$. $C_a(t)$ is given as a convolution between $I_a(t)$ and $f_C(t)$, the delay distribution:

$$C_a(t) = \alpha \int_0^\infty I_a(t-\tau) f_C(\tau) d\tau$$

Note that the delay distribution is not age-specific. From there, we apply a Fourier transform, rearrange some terms, and then perform an inverse Fourier transform:

$$I_a^* = \mathcal{F}^{-1}\left\{\frac{\mathcal{F}\{C_a\}}{\mathcal{F}\{f_C\}}\right\}$$

Where the star (*) indicates this is an inferred value and $I_a^* = \alpha I_a$, where α is the probability of infection reporting.

With these relationships, we can estimate the age-specific IFRs using Bayesian inference. We assumed the IFR have a beta distribution with the parameters $\alpha_a$ and $\beta_a$, again where $a$ is age class:

$$IFR_a \sim Beta(\alpha_a, \beta_a)$$

Reparametrizing this prior gives us the following hyperparameters:

$$\gamma_a = \frac{\alpha_a}{\alpha_a + \beta_a}$$

$$\lambda_a = \alpha_a + \beta_a$$

The hyperparameters have the following hyperprior distribution:

$$\gamma_a \sim Beta(1,6.5)$$

$$\lambda_a \sim Pareto(0.1,1.5)$$

To infer $IFR_a$, first we draw values for $\gamma_a$ and $\lambda_a$, then transform the values into $\alpha_a$ and $\beta_a$. Then, we can draw from the priors for $IFR_a \sim Beta(\alpha_a, \beta_a)$.

Now, we can find the likelihood of a given $IFR_a$ value given seroprevalence, deaths, inferred infections, and delay distributions. The log likelihood of a given $IFR_a$ value can be expressed as follows, using a binomial likelihood for deaths:

$$ll \approx -\log(M) + \log\left\{\sum_{m=1}^{M} \binom{I_{a,m}^{sero}(i)\phi_a(i)}{D_a(i)} IFR_a^{D_a(i)}(1 - IFR_a)^{I_{a,m}^{sero}(i)\phi_a(i) - D_a(i)}\right\}$$

In order to account for uncertainty within the seroprevalence estimates, we drew from a truncated normal distribution (bounded between 0 and 1) based on the serosurvey results for each age class [43] . $M$ is the number of posterior draws we are using (we used M = 1000), $m$ is the draw index, $I_a^{sero}$ is the seroconverted population draw ($I_a^{sero} = \theta_a * P_a$, where $\theta_a$ is the seroprevalence draw and $P_a$ is the population for this age class). The number of deaths on day $i$ is $D_a(i)$. $\phi_a$ contains the $I_a^*$ term we derived earlier, such that:

$$\phi_a(i) = \frac{\sum_{j=0}^{T} I_a^*(i-j)p_D(j)}{\sum_{j=0}^{T} I_a^*(i-j)p_{sero}(j)}$$

Where $j$ is the number of days since infection, $p_{sero}$ is the probability of seroconversion during day $j$, and $p_D$ is the probability of death during day $j$.

We compared the age-specific IFR estimates for New York City to nine other age-specific IFR estimates [25,29,30,33,39–41,48,49]. We excluded studies that were not based on population-representative serosurveys or did not properly account for the distribution of delays between infection, seroconversion and death (see study-specific exclusion criteria in Supplementary Table S2). One of the nine estimates showed a crude IFR estimate (without accounting for delay distribution) for the entire state of New York, but we included it to provide a baseline comparison against our New York City estimate [41]. Another provides a global age-specific IFR estimate

based on available serology, case, and death data [25]. Where applicable, we included IFR estimates that include care home resident deaths because New York City also included care home deaths in their reporting.

**Results**

The New York City serosurvey took place in late April, in the latter third of the initial epidemic, when new cases per day had fallen to approximately half of the peak (Fig 1) [43]. The serosurvey estimated that, by late April 2020 in New York City, approximately 1.5 million infections had occurred and 22.7% of the population was seropositive [43]. By approximately mid-May, when the last infections detected in the serosurvey would have occurred, there were nearly 16,000 confirmed COVID-19 deaths, along with nearly 4,500 probable COVID-19 deaths.

We estimated the confirmed-death-only age-specific IFRs for SARS-CoV-2 in New York City to increase logarithmically more than 75-fold from 0.07% in 18-44 to 5.2% in 75+ year-olds. In addition, assuming the ratio of seroprevalences in children and adults in New York City were similar to those in Spain (see Methods), the 10 deaths observed in 0-17 year-olds in New York City suggest an IFR for this age group of 0.0022% (0.00045-0.0057). If we included both confirmed and probable COVID-19 deaths, the IFRs were 24-33% higher across adult age classes (Table S2).

To put the New York City fatality rates in context, we compared our IFR estimates to other studies (Figure 2). IFRs from New York City for the 18-44 and 45-64 age classes were higher (with non-overlapping 95% confidence intervals) than corresponding IFRs for England [39], Switzerland [33], Spain [40], Belgium [49],

Sweden [30], and the global estimate [25]. In contrast, the IFRs for the oldest two age classes, 65-74 and 75+ were lower (with overlapping 95% CIs) than IFRs from England, Spain, and Switzerland but were higher than corresponding IFRs from Belgium (Figs 2, S2). Our age-specific estimates for IFRs in New York City were similar to those in New York State for all age groups except the oldest (75+), which was greater by 1.4-fold in New York State.

**Discussion**

There were several differences between the IFRs we estimated for New York City and those in other countries. IFRs in New York City were higher for two younger age classes (18-44, 45-64) than five other studies based on large-scale serosurveys (England, Switzerland, Spain, Belgium, Sweden) as well as the global baseline and accounted for 26% of the 15,885 deaths by May 16. This may have been due to a higher prevalence of pre-existing conditions, as 79% of COVID-19 deaths in 18-44 year-olds, and 85% of deaths in 45-64 year-olds in New York City had pre-existing conditions. However, IFRs for the two older age classes (55-64 and 75+) were lower than estimates for some other countries despite 79% and 76% of these deaths also having pre-existing conditions.

One shortcoming of our study is that the oldest age class for the seroprevalence study was broad (55+) and combined ages (60-69, 70-79, 80+) in which IFRs differed 10-fold in other studies [25,30,33,39,40,49], whereas we had deaths split into finer age classes (45-64, 65-74, and 75+). The variation in seroprevalence within older age classes in some studies [42] emphasizes the

importance of reporting both seroprevalence and deaths for SARS-CoV-2 for finer age classes to enable a more accurate understanding of the fatality of COVID-19 for these older most vulnerable age groups.

We found that IFRs in New York City showed similar log-linear increases with age as many other studies, but there were substantial differences among countries in IFRs for a given age group (Figure 2). The causes for differences in IFRs among countries are poorly understood [25], and deserve future study. However, the reductions in COVID-19 mortality due to vaccination, better case management and treatments that have been available since late 2020 makes understanding these differences difficult except through retrospective studies.

Retrospective IFR studies from the beginning of the pandemic still hold relevance, even with the emergence of variants such as B.1.617.2 (Delta variant) and B.1.1.529 (Omicron variant). Serosurvey-based IFR estimates for Delta and Omicron are unavailable, likely because of the extreme difficulty in assembling a sufficiently large group of naïve (i.e. unexposed, unvaccinated) individuals. When considering the epidemiology of the variants, the serosurveys and IFR estimates done on wild-type COVID-19 may still illustrate relevant patterns– especially when it comes to age-specificity.

**Figures and Tables**

**Table 1. Infection fatality rate (IFR) estimates in New York City for five age classes, using *confirmed* COVID-19 deaths (excluding probable deaths).**

| Age class | Population [46] | Confirmed COVID-19 Deaths, as of May 17, 2020 [44] | Estimated Infection Prevalence [14] (95% CI) | IFR (95% CI) % |
|---|---|---|---|---|
| 0-17 | 1,783,174 | 10 | 14.8 (6.89-28.2)* | 0.0022 (0.00045-0.0057)* |
| 18-44 | 3,493,918 | 625 | 22.4 (19.7-25.0) | 0.067 (0.054-0.081) |
| 45-64 | 2,112,562 | 3556 | 24.1 (21.8-26.5) | 0.56 (0.46-0.65) |
| 65-74 | 689,816 | 3963 | 21.5 (19.6-23.5) | 2.1 (1.7-2.4) |
| 75+ | 551,853 | 7731 | 21.5 (19.6-23.5) | 5.2 (4.2-6.1) |

***Estimated seroprevalence and IFR estimates for the 0-17 age class are derived from NYC population data and seroprevalence trends observed in Spain** [12]**, as our serosurvey source did not include serology data for this age group**

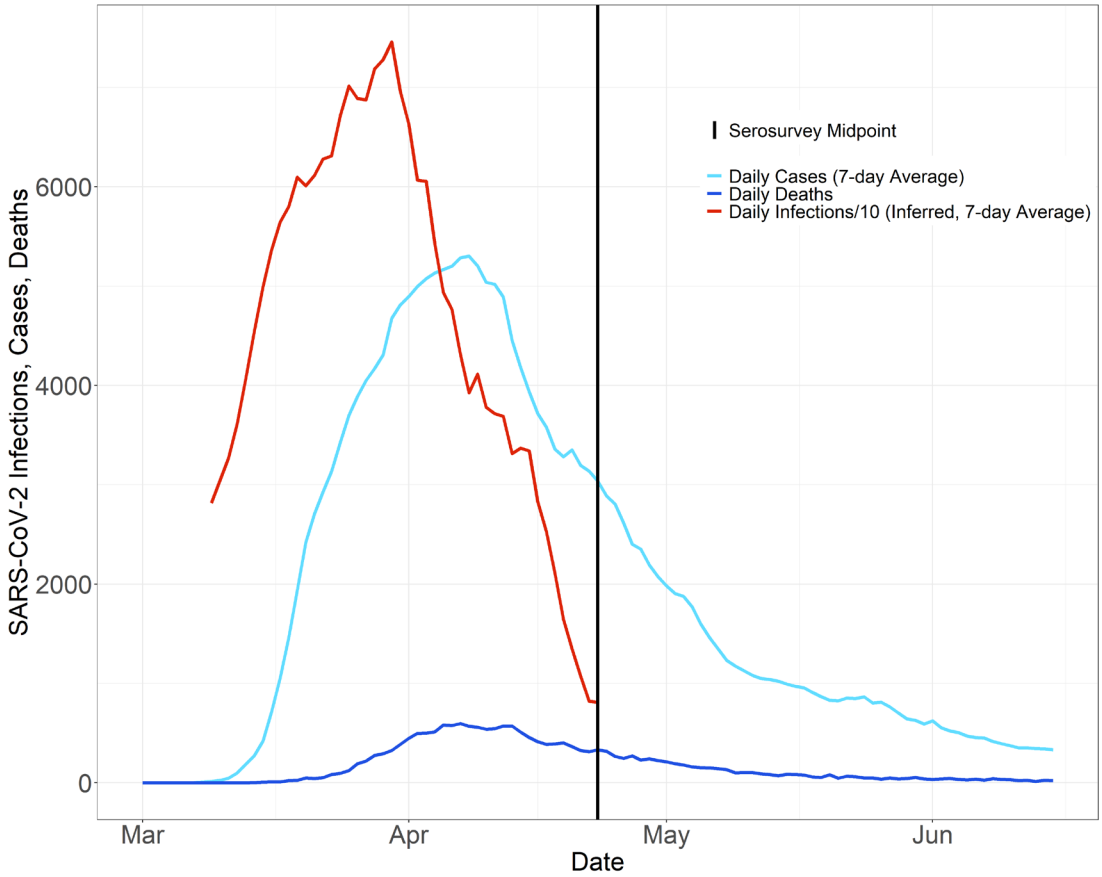


**Figure 1. Timeline of COVID-19 confirmed cases and deaths, and inferred SARS-CoV-2 infections over time in New York City in 2020, and the midpoint date of a serosurvey. To facilitate display, infections were divided by 10.**

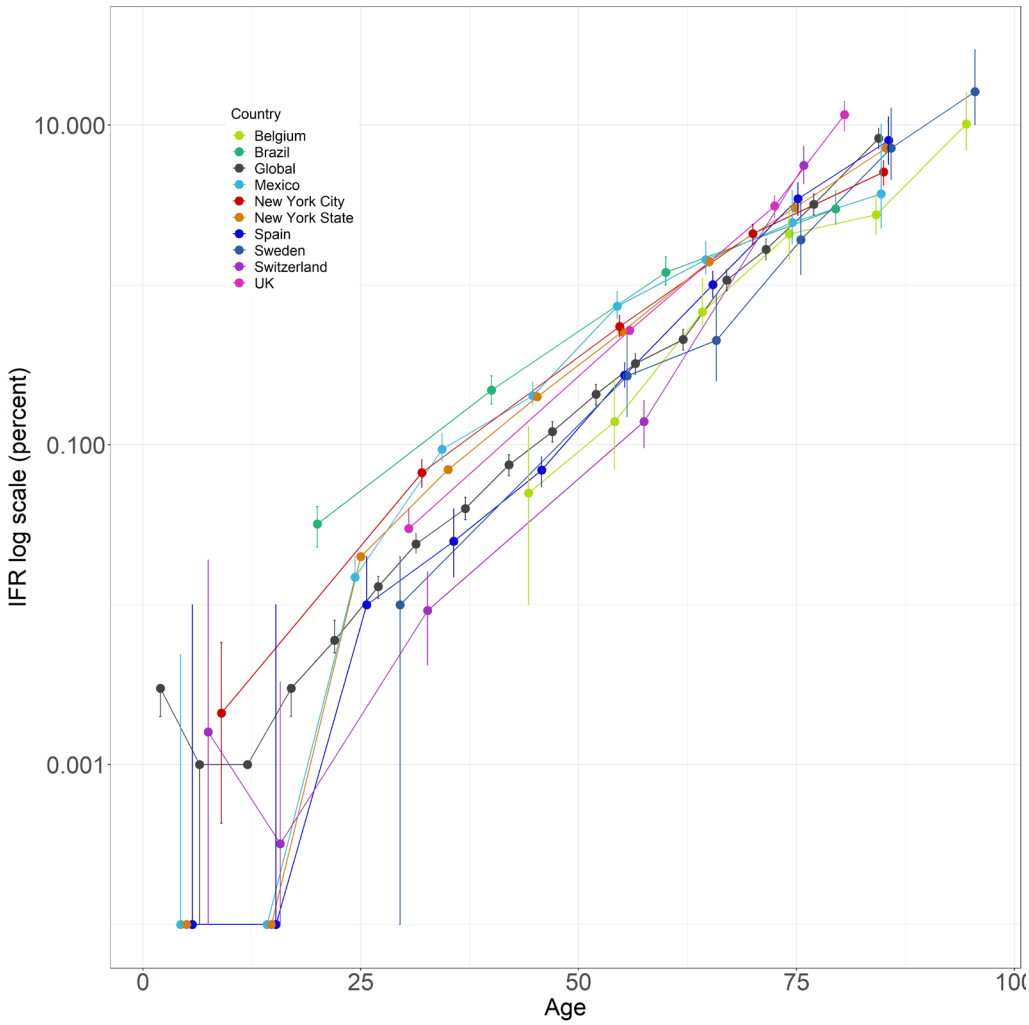


**Figure 2. Global comparison of age-specific infection fatality ratio (IFR) of COVID-19 on a log-scale (mean ± 95% CI) with points plotted at the midpoint of the age-class on the x-axis. Points from different studies are slightly jittered along the x-axis to facilitate presentation. Lines show age-specific IFRs for different populations, using only confirmed COVID-19 deaths where possible. This study is represented as "New York City" in red. IFR means and confidence intervals that estimate a value of 0 are represented as 0.001.**

**Supplemental Tables and Figures**

**Table S1. Infection fatality rate (IFR) estimates in New York City for five age classes, using confirmed and probable COVID-19 deaths. The last column notes the percent increase in the IFR comparing confirmed deaths to confirmed and probable deaths.**

| Age class | Population [46] | Combined (Confirmed + Probable) COVID-19 Deaths, as of May 17, 2020 [44] | Estimated Infection Prevalence (95% CI) [14] | IFR (95% CI) | % increase from IFR estimates using confirmed deaths only |
|---|---|---|---|---|---|
| 0-17 | 1,783,174 | 13 | 14.7 (6.66 – 27.2)* | 0.0038 (0.00099 – 0.0093)* | 72.7%* |
| 18-44 | 3,493,918 | 750 | 22.3 (19.4 – 24.9) | 0.083 (0.068 – 0.1) | 23.9% |
| 45-64 | 2,112,562 | 4490 | 24.1 (21.8 – 26.4) | 0.72 (0.61 – 0.83) | 28.6% |
| 65-74 | 689,816 | 4893 | 21.5 (19.6 – 23.4) | 2.7 (2.3 – 3.1) | 28.6% |
| 75+ | 551,853 | 10105 | 21.5 (19.6 – 23.4) | 6.9 (5.7 – 8.1) | 33% |

***Estimated seroprevalence and IFR estimates for the 0-17 age class use seroprevalence trends observed in Spain [12] to NYC population data** [46]**. See Methods for details.**

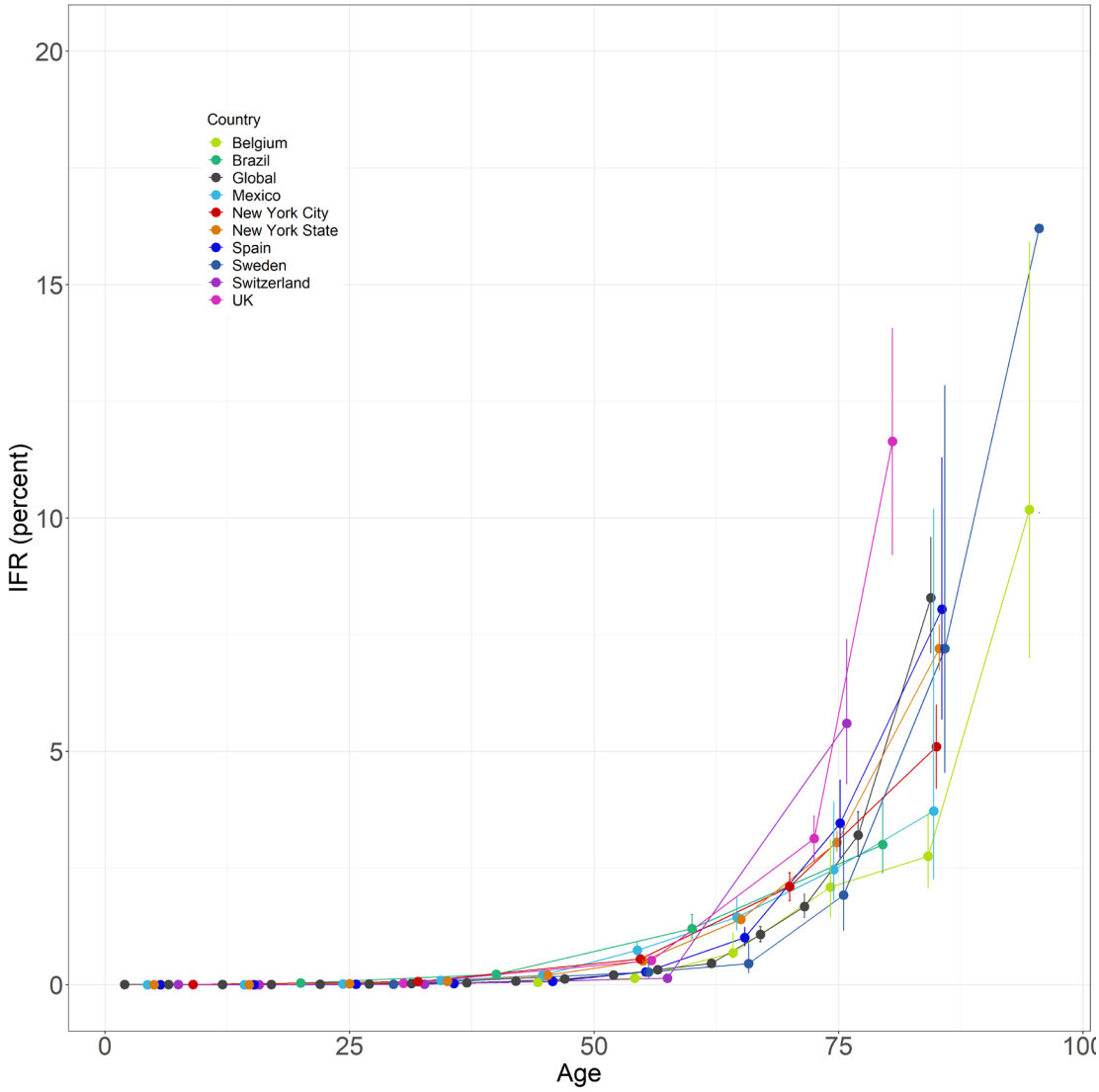


**Figure S1. Global comparison of age-specific infection fatality ratio (IFR) of COVID-19 on an untransformed-scale (mean ± 95% CI) with points plotted at the midpoint of the age-class on the x-axis. Points are slightly jittered along the x-axis facilitate presentation. Lines show age-specific IFRs for different populations, using confirmed-only COVID-19 deaths where possible. This study is represented in red, as "New York City".**

**Table S2. Exclusion criteria for global comparison of age-specific IFRs in Figures 2 and S1. Serosurveys based on blood donors were not considered.**

| Country | Author | Reason for exclusion |
|---|---|---|
| Portugal | Kislaya et al. [50] | Participants were recruited from hospital and laboratory network and likely biased towards people coming into those locations |
| Italy | Poletti et al. [51] | Low sample size of deaths (n < 10 for most age groups) |
| Germany, Canada | Wagner et al. [52], Tang et al. [53] | Did not account for delay between infection and death |
| India | Cai et al. [54] | Inconsistent mortality data |
| Kenya, Denmark | Uyoga et al. [55], Erikstrup et al. [56] | Based on blood donations |
| Global | Levin et al. [37], COVID-19 Forecasting Team [42] | Used a point estimate for delays between infection and death or seroconversion |

## Conclusion

We found that in New York City, COVID-19 IFRs were higher for intermediate age classes (18-44, 45-55) compared to other populations. One potential reason for these differences could be that New York City residents had higher rates of underlying conditions compared to other populations. Other factors, such as hospital capacity, racial inequality, income inequality, and population density may also have also contributed to the differences observed in New York City IFRs [57,58].

Another factor that can impact mortality risk is host genetics, and ongoing research is examining the contribution of genetic variation to SARS-CoV-2 infection and mortality [59–61]. One hypothesis is that different individuals express different levels of angiotension-converting enzyme 2 (ACE2). ACE2 is the receptor protein that binds to the spike protein of SARS-CoV-2 and allows the virus to enter the body [62,63]. Expression of ACE2 is age-specific; children tend to have lower levels of this protein, and elderly individuals tend to have higher levels [26], which is broadly consistent with age-specific IFRs for COVID-19.

Hosts that are infected by multiple pathogens may suffer higher mortality than hosts only infected with a single pathogen [64,65]. In COVID-19 patients, coinfection with HIV/AIDS, bacterial pathogens such as *Staphylococcus aureus*, and fungal pathogens such as *Candida albicans* increase the risk of mortality due to COVID-19 [66,67]. A better understanding of coinfection among population may illuminate differences among populations in COVID-19 IFRs.

Quantifying the mortality risks of a disease can be used to target interventions and reduce disease impacts. In most countries COVID-19 vaccines were first made available to the elderly (as well as health care workers). After controlling for one risk factor such as age, other variables may become more apparent and further inform a public health strategy to prevent loss of life during an epidemic. Differences in COVID-19 mortality risk due to underlying conditions, coinfections, and host genetics are still being explored with relation to COVID-19, but further understanding the reasons for mortality disparities between populations can reduce deaths during the ongoing COVID-19 pandemic and inform our course of action during future pandemics.

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