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May 2020

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19 May 2020

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Important note: This version of the paper was written during the early months of the global pandemic. The research underlying it was based upon the limited data available to us as of 14 May 2020. The situation is very fluid, and our research will be updated and refined as additional data become available.

The paper is available at: http://www.pensions-institute.org/wp-content/uploads/wp2007.pdf

Summary

Covid-19 has predominantly affected mortality at high ages. It kills by inflaming and clogging the air sacs in the lungs, depriving the body of oxygen – inducing hypoxia – which closes down essential organs, in particular the heart, kidneys and liver, and causes blood clots (which can lead to stroke or pulmonary embolism) and neurological malfunction.

Evidence from different countries points to the fact that people who die from Covid-19 are often, but not always, much less healthy than the average for their age group. This is true for England & Wales – the two countries we focus on in this study. The implication is that the years of life lost through early death are less than the average for each age group, with how much less being a source of considerable debate. We argue that many of those who die from coronavirus would have died anyway in the relatively near future due to their existing frailties or co-morbidities. We demonstrate how to capture this link to poorer-than-average health using a model in which individual deaths are 'accelerated' ahead of schedule due to Covid-19. The model structure and its parameterization build on the observation that Covid-19 mortality by age is approximately proportional to all-cause mortality. This, in combination with current predictions of total deaths, results in the important conclusion that, everything else being equal, the impact of Covid-19 on the mortality rates of the surviving population will be very modest. Specifically, the degree of anti-selection is likely to be very small, since the life expectancy of survivors does not increase by a significant amount over pre-pandemic levels.

We also analyze the degree to which Covid-19 mortality varies with socio-economic status. Headline statistics suggest that the most deprived groups have been disproportionately affected by Covid-19. However, once we control for regional differences in mortality rates, Covid-19 deaths in both the most and least deprived groups are also proportional to the all-cause mortality of these groups. However, the groups in between have approximately 10-15% lower Covid-19 deaths compared with their all-cause mortality.

We argue that useful lessons about the potential pattern of accelerated deaths from Covid-19 can be drawn from examining deaths from respiratory diseases, especially at different age ranges. We also argue that it is possible to draw useful lessons about volatility spikes in Covid-19 deaths from examining past seasonal flu epidemics. However, there is an important difference. Whereas the spikes in seasonal flu increase with age, our finding that Covid-19 death rates are approximately proportional to all-cause mortality suggests that any spike in Covid-19 mortality in percentage terms would be similar across all age ranges.

Finally, we discuss some of the indirect consequences for future mortality of the pandemic and the 'lockdown' measures governments have imposed to contain it. For example, there is evidence that some surviving patients at all ages who needed intensive care could end up with a new impairment, such as organ damage, which will reduce their life expectancy. There is also evidence that many people in lockdown did not seek a timely medical assessment for a potential new illness, such as cancer, or deferred seeking treatment for an existing serious illness, with the consequence that non-Covid-19-related mortality rates could increase in future. Self-isolation during lockdown has contributed to an increase in alcohol and drug

consumption by some people which might, in turn, reduce their life expectancy. If another consequence of the pandemic is a recession and/or an acceleration in job automation, resulting in long-term unemployment, then this could lead to so-called 'deaths of despair' in future. Other people, by contrast, might permanently change their social behaviour or seek treatments that delay the impact or onset of age-related diseases, one of the primary factors that make people more susceptible to the virus – both of which could have the effect of increasing their life expectancy. It is, however, too early to quantify these possibilities, although it is conceivable that these indirect consequences could have a bigger impact on future average life expectancy than the direct consequences measured by the accelerated deaths model.

1. Introduction

Covid-19, the new coronavirus that began in Wuhan in China in late 2019 and circumnavigated the Earth in a matter of weeks, creating the worst global pandemic since Spanish Flu in 1918-19, has had the greatest impact on those aged over 50, particularly males – as Figure 1 shows in the case of England & Wales (EW).[1]

Figure 1: Age-specific mortality rates due to Covid-19, per 100,000 people, England & Wales, occurring in March 2020
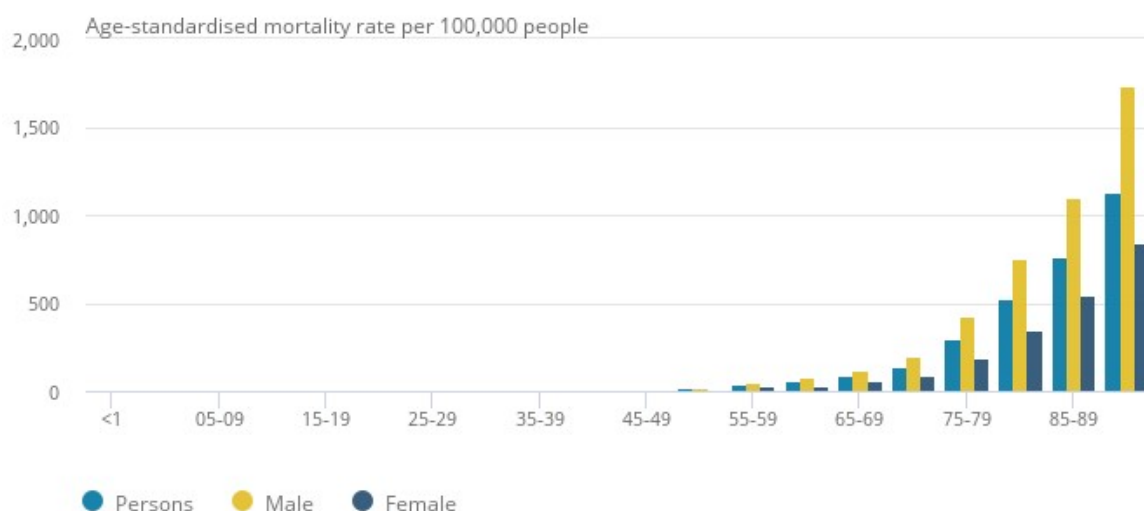
Source: Figure 5 in *Deaths involving COVID-19, England & Wales: deaths occurring in March 2020*, Office for National Statistics (ONS);
https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/bulletins/deaths involvingcovid19englandandwales/deathsoccurringinmarch2020

Covid-19 kills by inflaming and clogging the air sacs in the lungs, depriving the body of oxygen – inducing hypoxia – which closes down the body's other essential organs.[2] The virus can also lead to heart inflammation (e.g., myocarditis), irregular heart rhythms risking cardiac arrest, kidney, liver and intestinal damage, blood clots (which can lead to stroke or pulmonary embolism), and neurological malfunction. This can be exacerbated by a 'cytokine storm', a dangerous overreaction of the immune system which fails to stop when the threat has passed.[3] Around half of patients hospitalized with Covid-19 have blood or protein in their urine, indicating damage to their kidneys. Up to 30% of intensive-care patients lose kidney

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[1] There will be similar figures for other countries.

[2] Lenny Bernstein, Carolyn Y. Johnson, Sarah Kaplan and Laurie McGinley (2020) Coronavirus destroys lungs: But doctors are finding its damage in kidneys, hearts and elsewhere, *Washington Post*, 15 April; https://www.washingtonpost.com/health/coronavirus-destroys-lungs-but-doctors-are-finding-its-damage-in-kidneys-hearts-and-elsewhere/2020/04/14/7ff71ee0-7db1-11ea-a3ee-13e1ae0a3571_story.html

[3] When the body first recognizes a virus or a bacterium, cytokine proteins warn the body's cells to trigger an immune response. However, the response can be overly aggressive and fail to self-regulate or immunomodulate and return the immune system to a state of homeostasis. See: Apoorva Mandavilli (2020) The Coronavirus Patients Betrayed by Their Own Immune Systems, *New York Times,* 1 April; https://www.nytimes.com/2020/04/01/health/coronavirus-cytokine-storm-immune-system.html

function and require a form of dialysis called continuous renal replacement therapy.[4] It is likely that many Covid-19 patients with existing illnesses related to lungs, kidneys, etc who needed to go into intensive care failed to survive. However, some surviving intensive care patients may develop a new life shortening impairment (e.g., related to organ damage) that they did not have before.

The aim of this paper is to develop scenarios for the potential impact of Covid-19 on future higher-age mortality, once the pandemic has run its course.[5] To do this, a number of questions need to be addressed:

- How many of the deaths caused by Covid-19 would have happened in the relatively near future in any event because those infected by the virus were frail and had serious existing illnesses (or co-morbidities) which meant that they had a shorter life expectancy relative to their peers even if they had not caught the virus? In other words, how many deaths have been marginally 'accelerated' by the virus compared with expected (or 'scheduled') deaths? This is likely to be a function of the age, gender, and socio-economic status of infected individuals, as well as the infection rate in their vicinity. And, as a consequence, what is the impact on the average life expectancy of the general population after the pandemic?

- How many people who recovered from Covid-19 developed an impairment, such as organ damage, that they did not have before and which shortened their life expectancy? This is likely to be related to the number of infected people who needed intensive care in hospital.

- How many people who self-isolated during the government 'lockdown'[6] did not then seek timely medical diagnosis or treatment for other conditions during the pandemic, e.g., cancer, and developed more serious cases as a result, which could reduce their life expectancy?

- How many people will have their life expectancy reduced – and in extreme cases 'die of despair' – because of an increase in alcohol/drug consumption and suicide, as a consequence of self-isolation or long-term unemployment if the economy remains in recession for an extended period and/or many more jobs are automated in response to the pandemic?

- How many people will permanently change their social behaviour or seek treatments that delay the impact or onset of age-related diseases, one of the primary factors that make people more susceptible to the virus – both of which could have the effect of increasing their life expectancy?

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[4] Lenny Bernstein et al (op cit) and Paul Basilio (2020) COVID-19: Damage found in multiple organ systems, *MDLinx*, 15 April; https://www.mdlinx.com/internal-medicine/article/6870

[5] By run its course, we mean the state in which the population has achieved sufficient herd immunity naturally or by means of effective treatment or a vaccine that further seasonal outbreaks do not lead to the virus spreading exponentially through the population (i.e., the reproduction factor $R_0 < 1$). However, we are not trying to predict the outcome of the current pandemic or claim that we know best about how to control it.

[6] In order to stop the spread of the infection, most governments throughout the world locked down their economies and made all but essential workers stay at home (self-isolate). While people were allowed out to buy food and medicine and to exercise, they had to keep two metres apart from anyone else (social distancing).

To address these questions, we need to make assumptions about models, scenarios and data. Given the uncertainties involved, we choose to use a standard model of mortality, the Gompertz model, and develop scenarios using a simple model for accelerated deaths. The left-hand panel of Figure 2 shows, for a particular age cohort, the impact on the cohort deaths curve if Covid-19 deaths are randomly distributed across the age range, while the right-hand panel shows the impact if the Covid-19 deaths are front-loaded and affect more those who are currently less healthy (and have more co-morbidities) than the average for this age cohort. Since more than 97% of deaths occur above the age of 50,[7] we concentrate on this age range – which also happens to be the age range over which the Gompertz model fits well. In terms of country, we use data from EW and comment briefly on an application of the model to US data – although the model is general enough to be adapted for use in all countries.

Figure 2: The accelerated deaths model – random vs front-loading of Covid-19 deaths
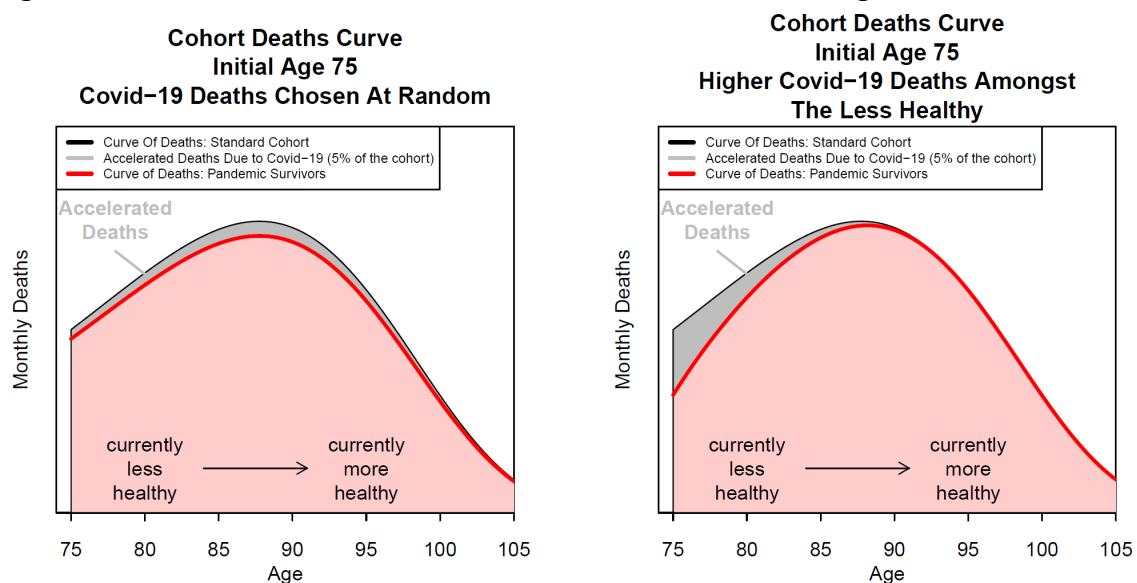
Specific questions for assessing the longer-term impact include:

- What will be the total number of deaths over the course of the pandemic?
- What will be the age distribution of the deaths?
- What will the gender split be?
- What will be the split between socio-economic groups?
- How much of the differences between these sub-groups can be explained by differences in all-cause mortality?
- What proportion of Covid-19 survivors are carrying forward some form of impairment (e.g., organ damage) and how long will these impairments last?
- What is the impact of delays in the diagnosis and treatment of other illnesses?
- What are the likely behavioural responses to the pandemic?

At the time of writing, there is information available that allows us to address some of these questions. We estimate that the pandemic will produce a total of around 75,000 to 85,000

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[7] In England & Wales.

deaths in EW if social distancing measures are maintained,[8] and we have reasonable data on the gender split and the age distribution. On the other hand, we have relatively little information so far on socio-economic differences, health impairments and behavioural responses.

We therefore concentrate in this paper on the relationship between existing co-morbidities (frailties) and Covid-19 deaths. Professor David Spiegelhalter (see Figure 3) and other commentators have highlighted a parallel relationship between the log of non-Covid-19 death rates and age and the log of Covid-19 death rates and age (in both cases above age 30), implying that Covid-19 mortality seems to be proportional to all-cause mortality at adult ages. In other words, Covid-19 appears to increase a cohort's short-term mortality rate by a common multiplicative factor, whatever the cohort's current baseline mortality rate.

Figure 3: The age gradient of Covid-19 deaths in England & Wales compared with non-Covid-19 deaths from all causes
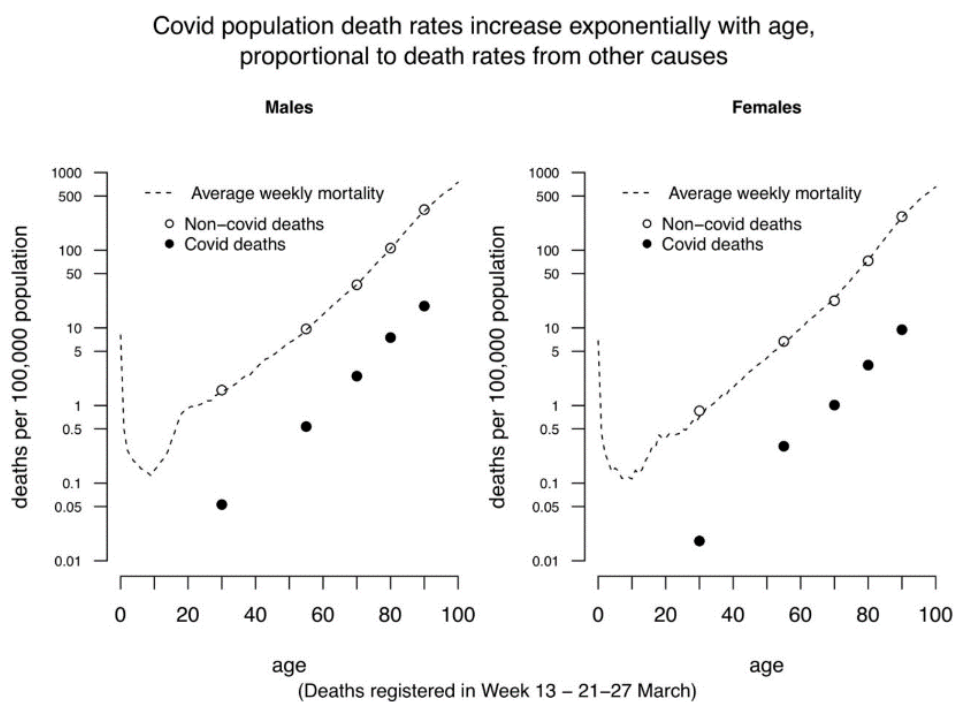
Note: All-cause death rates for England & Wales versus death rates from Covid-19 for males and females.
Source: David Spiegelhalter blog, Winton Centre, 11 April 2020; https://medium.com/wintoncentre

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[8] IPE reports that deaths during the first wave of the pandemic were around 60,000 based on Club Vita estimates as of 12 May 2020 (https://www.ipe.com/news/uk-covid-19-toll-understated-but-long-term-factors-crucial-say-consultants/10045494.article). Official data from the UK Office for National Statistics (ONS) states that as of 1 May 2020, there have been 35,000 Covid-19 deaths (https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/bulletins/death sregisteredweeklyinenglandandwalesprovisional/weekending1may2020). Based on the date of peak deaths (4-8 April 2020, depending on region), the amplitude of the peak, and the rate of decline in deaths since the peak, we project that there will be another 15,000 deaths in the first wave. This gives a total of around 50,000 Covid-19 'official' deaths. If we add 50-70% for under-reporting, we project 75,000-85,000 'true' Covid-19 deaths in the first wave. See also https://www.bbc.co.uk/news/health-52623141. This assumes that there are no further significant flare-ups of the virus in the first wave.

2. A Model of the Direct Impact of Covid-19 on Future Higher-Age Mortality

We will use the term 'frail' to refer to people with co-morbidities that would raise their risk of death significantly relative to healthy individuals of the same age (i.e., with no significant co-morbidities). We use the relationship in Figure 3 to investigate the link between Covid-19 and frailty by age as measured by all-cause death rates: older people are more `frail' and so are more likely to die in the next year.[9]

Figure 3 suggests a possible way of linking Covid-19 mortality with frailty at age x:

Covid mortality rate(x) = All-cause mortality rate(x) $\times$ Infection rate(x)

$$\times \text{ Relative frailty}(x) \tag{1}$$

where the all-cause mortality rate should be interpreted as all-cause mortality in the absence of the pandemic. Figure 3 suggests that Infection rate(x) $\times$ Relative frailty(x) does not depend much on age, but has some dependence on sex. For a more rigorous discussion, see the Appendix.

The evidence indicates that a significant proportion of people who die from Covid-19 are in a frail state. Figure 4 for EW, for example, shows that those who died had significant co-morbidities, in particular, ischaemic heart diseases, dementia and Alzheimer's disease, chronic lower respiratory diseases,[10] influenza (flu) and pneumonia, and diabetes. Only a small percentage (Figure 4: 7-12% depending on subgroup) had no pre-existing conditions. Data for New York State are consistent with this: only 11% of those who died had no co-morbidities.[11] Figure 5 shows that the four main causes of death during March 2020, apart from Covid-19, are dementia and Alzheimer's disease, ischaemic heart diseases,[12] chronic lower respiratory diseases, and cerebrovascular disease.[13] A possible inference of Figures 4 and 5 is that Covid-19 deaths can be broken down into three groups:

- deaths of people who have one or more co-morbidities and would have died from these in the relatively near future, had they not contracted Covid-19;[14]
- deaths of people who have one or more co-morbidities, but would have been expected to live beyond 2020 had they not contracted Covid-19 (perhaps for many years); and

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[9] The two types of frailty (age and co-morbidity) are, of course, strongly connected. As a cohort ages, the proportion who have one or more significant co-morbidities will increase steadily, and this, in turn, pushes up all-cause mortality.

[10] For example, asthma, chronic obstructive pulmonary disease (COPD), bronchitis and emphysema.

[11] Source: https://covid19tracker.health.ny.gov/views/NYS-COVID19-Tracker/NYSDOHCOVID-19Tracker-Fatalities (accessed 5/5/2020)

[12] Diseases that narrow the heart (coronary) arteries that supply blood to the heart muscle.

[13] Diseases that affect the blood vessels and blood supply to the brain, leading to strokes.

[14] Figure 5 shows that the mortality rates for the top four non-Covid-19 causes of death is below the 5-year average by varying amounts, especially for ischaemic heart diseases. This suggests that some of the deaths recorded as Covid-19 deaths could equally well have been recorded as one of the top four non-Covid-19 causes.

Figure 4: Percentage of deaths involving Covid-19 by main pre-existing condition, sex and age, England & Wales, occurring in March 2020
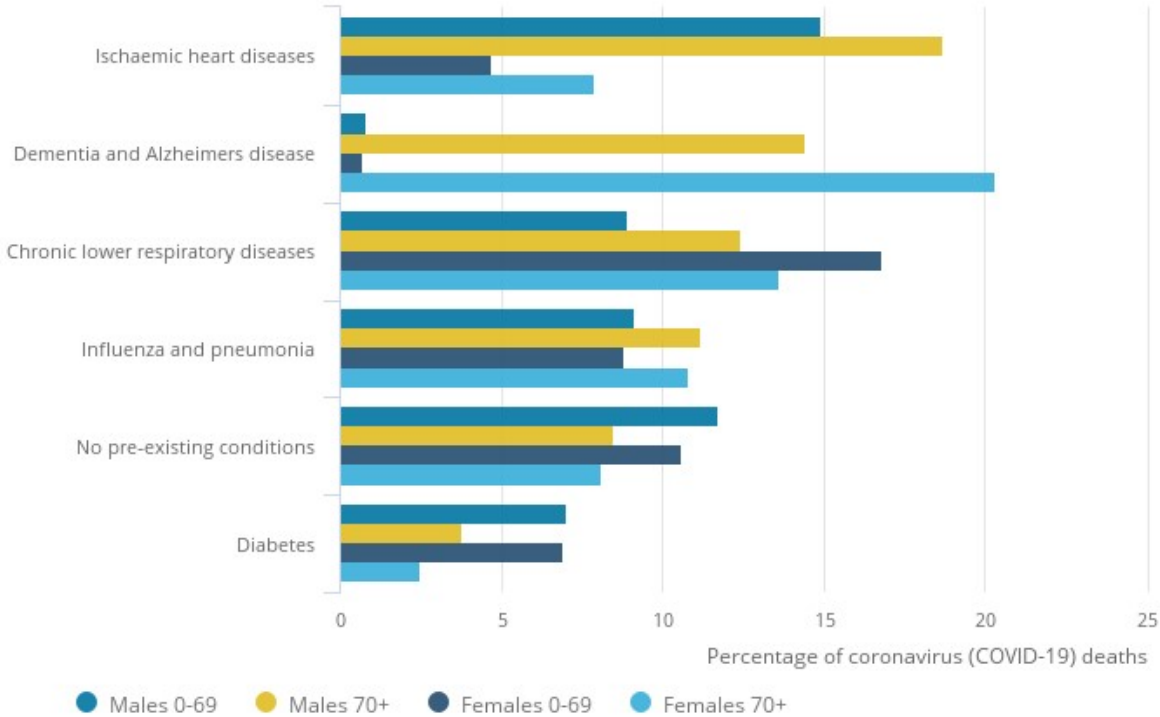
Figure 5: Age-standardized mortality rate for the five leading causes of death, per 100,000 people, England & Wales, occurring in March 2020
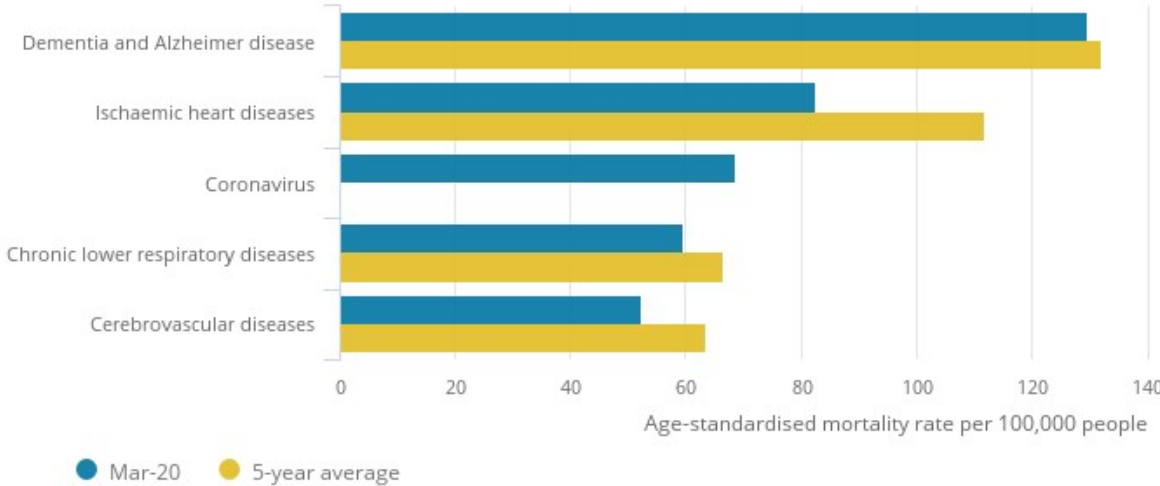
- deaths of people (between 7-12% according to Figure 4) who had no co-morbidities and who would otherwise have had a longer life expectancy, perhaps consistent with the average for their cohort.

The data in Figures 4 and 5, therefore, lend weight the view that a reasonable proportion of those who died from Covid-19 would have died this year anyway. Sadly, a number would have lived for rather longer, with between 7-12% potentially living significantly longer had they not contracted the virus.

Further evidence comes from Docherty et al (2020) who report that, on the basis of the ISARIC WHO Clinical Characterisation Protocol, 53% out of their sample of 16,749 UK people hospitalized due to Covid-19 had significant co-morbidities. This is an overlapping but not identical pool of patients, but the difference between co-morbidities at admission and death indicates a strong link between prior health and ability to survive hospitalization.

We therefore propose the following hypothesis: *Many of those who die from coronavirus would have died anyway in the relatively near future due to these co-morbidities*. Within a given cohort, Covid-19 deaths will, therefore, be more prevalent amongst people who already had a shorter expected lifetime (due to these co-morbidities) compared to the average for the cohort.

The model that we propose below is speculative and highly stylized. Its purpose is to explore the possible impacts of the pandemic. Once additional mortality experience data become available, the model can be refined, including detailed calibration to different populations. Further, we note that we are not attempting to model the path of the epidemic itself, just the aftermath. The simple model is also limited to ages 50 and above.

2.1 *Model A: Baseline Model*

We calibrate our baseline model for mortality (in the absence of the pandemic) to EW. We assume that the period life table for death rates follows a Gompertz function with a growth rate 0.105 and a death rate of 0.01 at age 65. The assumed long-term mortality improvement rate is 1.5% per annum at all ages.

Projected deaths are calculated on a monthly basis. We will therefore write $d_A(t, x) =$ cohort deaths in month t for a cohort with initial size 100,000 and initial age x. As a function of t, $d_A(t, x)$ defines what we refer to as the baseline cohort deaths' curve.

2.2 *Model B: Accelerated Deaths Due to Covid-19*

We now adjust Model A to model those lives within the cohort that are likely to die of Covid-19. For each age cohort, deaths are divided into two groups: those who die from Covid-19 ('accelerated' deaths); and those who either survive the infection or are not infected at all. Specifically, we assume that out of the $d_A(t, x)$ deaths expected or `scheduled' to die in month t, a proportion, $\pi(t, x)$, will die from Covid-19 (and, since we are not concerned with the timing of Covid-19 deaths, we assume, for simplicity, that these deaths occur in month 1). The shape of $\pi(t, x)$, as a function of t, is chosen to reflect the observation that most people

who die from Covid-19 have existing significant co-morbidities. To keep things simple, we choose a decreasing exponential form for $\pi(t,x)$.[15]

$$\pi(t,x) = \frac{\alpha(x)}{\rho(x)}\exp[-t/(12\rho(x))]. \tag{2}$$

Although this functional form is subjective, it captures the idea that people who are expected to die sooner rather than later are more likely also to die from Covid-19 if they have contracted the virus. Other forms for $\pi(t,x)$ could equally be plausible, but the exponential form is simple and easy to understand.

$\alpha(x)$ defines what we call the *amplitude* of the effect at age x. As an approximation, $\alpha(x)$ measures the number of Covid-19 deaths as a *proportion* of expected deaths due to all causes at age x (i.e., in the next year).[16]

$\rho(x)$ is what we call the *reach* of the accelerated deaths. As an approximation, the reach measures the expected remaining years of life lost (i.e., remaining life expectancy) by those who die immediately from Covid-19.[17] As a group, they would have otherwise lived, approximately, for an extra $\rho(x)$ years on average. A higher $\rho(x)$ means that the distribution of accelerated deaths is spread over a wider range of previously `scheduled' years of death. In the extreme case of $\rho(x) \to \infty$ (and, simultaneously, $\alpha(x)/\rho(x)$ staying constant), $\pi(t,x)$ is constant (for each cohort) meaning that there is no relationship between frailty (existing significant co-morbidities) and deaths from Covid-19.

The amplitude can be observed with reasonable accuracy after the pandemic is over (at least if Covid-19 deaths are counted accurately).[18] The reach is much more difficult to observe, but might be amenable to estimation if, for example, data become available on the various co-morbidities being carried by those who died.

Covid-19 deaths in month 1 for the cohort currently aged x (per 100,000) are thus $d_C(x) = \sum_t \pi(t,x)d_A(t,x)$, and the curve of deaths for survivors of the pandemic is $d_S(t,x) = \left(1 - \pi(t,x)\right)d_A(t,x)$ for $t = 1,2,\dots$.

To demonstrate how the model works and the impacts on life expectancies, we begin with an extreme mortality scenario in which we assume 500,000 deaths when we adjust cohort sizes to match the approximate 20 million people in the age range 50-100 in EW. This corresponds to the worst-case scenario predicted by the Imperial College Covid-19 Response Team assuming no government intervention to slow down the spread of the pandemic.[19]

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[15] $\alpha(x) > 0, \ \rho(x) > 0.$

[16] The accuracy of the approximation depends of the shape of the curve of deaths in the first few years. If the curve is flat, then the approximation is accurate. If the curve is rising (at ages up to about 85), $\alpha(x)$ underestimates the true proportion of Covid-19 deaths. If the curve is falling (at ages above 90), $\alpha(x)$ overestimates the true proportion of Covid-19 deaths.

[17] Remaining life expectancy (years of life lost, YLL) as an approximation depends on the shape of the cohort deaths' curve. If the deaths' curve is rising (ages up to, say, 85), then YLL will be greater than the reach. If the deaths' curve is falling (above age 90, say) then YLL will be less than the reach.

[18] Or if excess mortality during the pandemic can be accurately estimated.

[19] Ferguson et al (2020).

Figure 6 illustrates the accelerated deaths model in the worst-case extreme-mortality scenario. The black curve represents expected monthly deaths (for an initial cohort size of 100,000, all aged 75) in the absence of Covid-19.[20] The grey triangular area represents the people who die from Covid-19 (3,060 per 100,000 in this example). The shape of $\pi(t,x)$, multiplied by $d_A(t,x)$ dictates the shape of the grey region. The red bar on the left counts regular deaths plus Covid-19 deaths in month 1, and the red area illustrates the cohort deaths' curve for those who survive the pandemic (either because they were not infected or they recovered). The modified red area and the associated mortality curve results in a form of *anti-selection*.[21] The shape of the red curve implies that survivors are, on average, healthier than the average for the cohort before the pandemic and so have lower average mortality, but their future mortality experience gradually reverts over time (depending on $\rho(x)$) to standard mortality.

Figure 6: Impact of accelerated deaths on a cohort deaths' curve for the worst-case extreme-mortality scenario
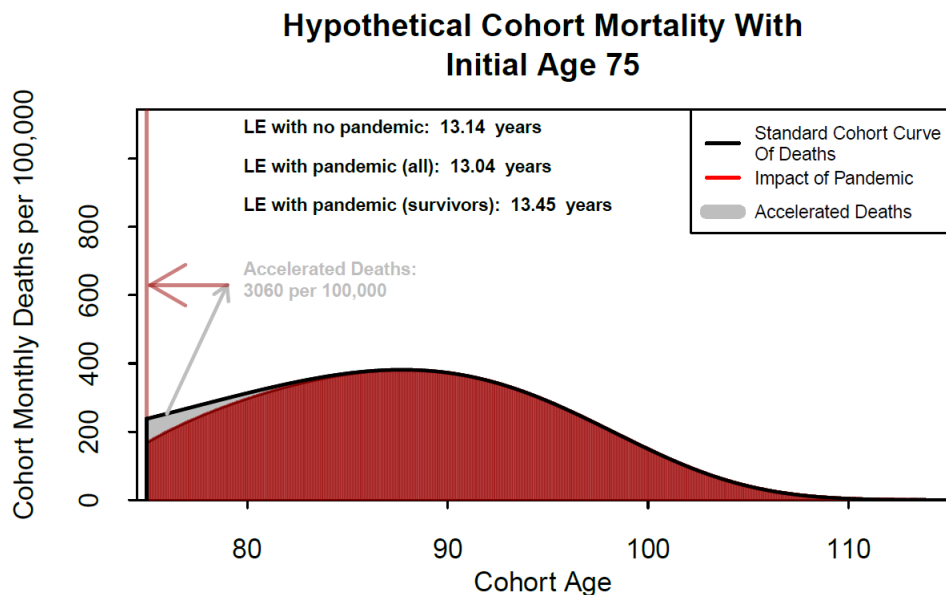
Note: The black line shows the cohort's projected curve of deaths in the absence of the pandemic. The red area shows the curve of deaths for the survivors. With the pandemic and our assumed model for accelerated deaths, the grey triangle highlights the numbers of accelerated deaths. The vertical red line on the left shows the same number of deaths as the grey triangle.

Some survivors, most likely those who needed intensive care, could end up with a new impairment, such as organ damage, which will reduce their life expectancy. However, for survivors as a whole, we conjecture that their life expectancy has increased relative their age cohort before the outbreak of the pandemic. In our example, the life expectancy of survivors increases from 13.14 to 13.45, while the life expectancy for the whole cohort (because of

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[20] We choose age 75 purely for illustration and also because the grey triangle in Figure 6 would be smaller and harder to visualize had we used a lower age.

[21] The term anti-selection (or adverse selection) is widely used for this type of effect. However, it is important to note that the use of the term does not imply that individuals (at the population level) are exercising any choices.

premature Covid-19 deaths) falls to 13.04 from 13.14. It is important to note that these differences – which measure the magnitude of the anti-selection effect – are very small.

The impact on accelerated deaths of a change in the amplitude, $\alpha(x)$, is straightforward. For example, doubling $\alpha(x)$ doubles the depth of the grey region in Figure 6 and doubles the number of accelerated deaths.

Figure 7 shows the effect of a change in the reach, while keeping the amplitude fixed. The left and right-hand plots show the impact of extending the reach from 2 to 4 years. The grey area with the accelerated deaths narrows at the left-hand end, but stretches out to the right. The total number of deaths remains approximately the same (since the amplitude is fixed), but the expected years of life lost by the Covid-19 victims approximately doubles. Additionally, increasing the reach reduces the magnitude of the anti-selection in a given year, although the time to revert to `standard' mortality is increased.

Figure 7: Sensitivity of cohort deaths' curves to changes in the reach for the worst-case extreme-mortality scenario
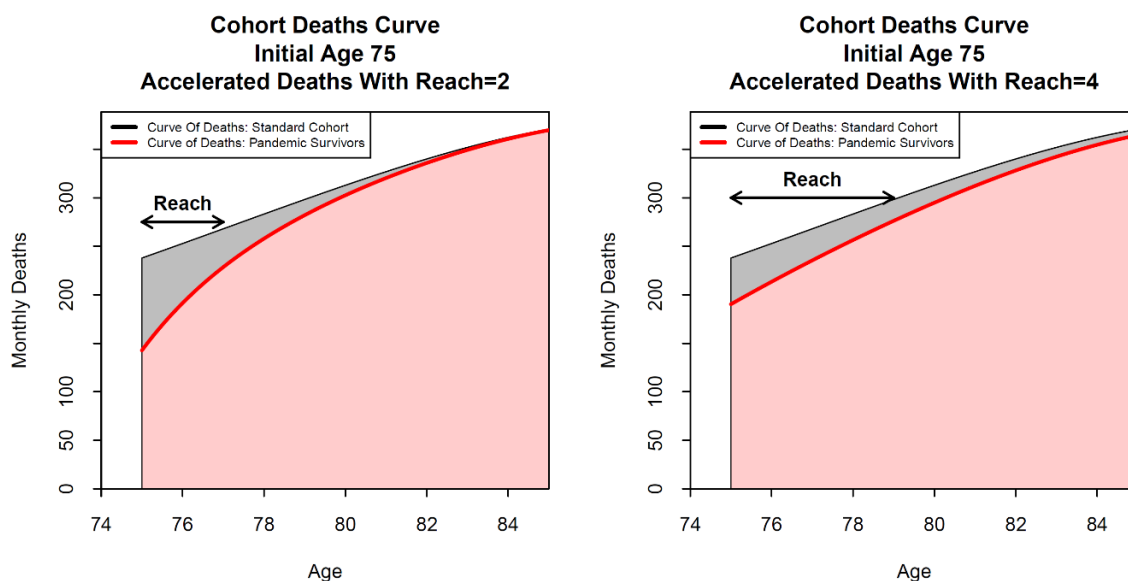
Note: Left and right: amplitude is fixed. Left: reach is 2 years. Right: reach is 4 years.

In Figure 8, for the same extreme mortality scenario as Figure 6, we look at the impact on life expectancies.[22] In the top plot, the small difference between the black and red lines is only just visible. The bottom plot shows the same relationship expressed as a ratio which makes the consequence of the pandemic clearer: the anti-selection effect means that survivors have an increase in life expectancy of between 1% at age 65 rising to over 6% at age 90.

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[22] Looking at the impact on life expectancies can be used as a proxy for the impact on lifetime annuity values, particularly in a world of very low interest rates.

Figure 8: Impact on life expectancy of accelerated deaths for the worst-case extreme-mortality scenario
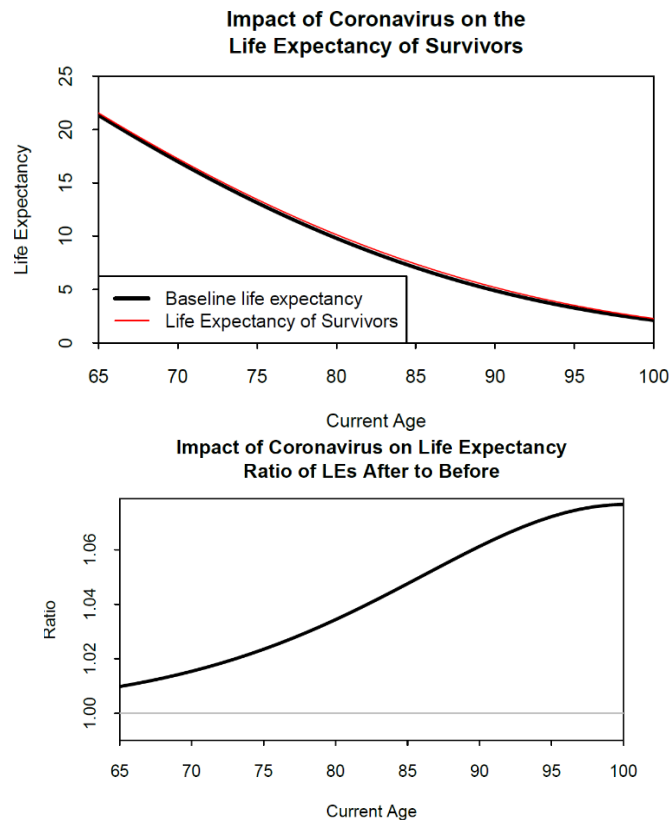
Note: Scenario as in Figure 6. Top: life expectancies before the pandemic by age (black line) and for survivors (red line). Bottom: the ratio of life expectancy for survivors to the life expectancy before the pandemic.

3. Plausible Covid-19 Scenarios

In the light of the UK government's lockdown policy and assuming social distancing measures are maintained after the lockdown is lifted, our best estimate is around 75,000 to 85,000 Covid-19 deaths for EW, less than 20% of the number of deaths in the worst-case scenario. In the scenarios that follow, we will modify the amplitude and reach functions so that the model generates approximately 80,000 accelerated deaths. In due course, we will have a better idea of the final number, although it might never be known how many deaths due to Covid-19 were incorrectly reported.[23]

In our new baseline case (Scenario A – see Table 1), we will assume that $\alpha(x) = 0.07272$ and $\rho(x) = 4$ for all x. This implies that, at all ages, approximately 7% of all deaths over the next year are assumed to be due to Covid-19, while those who die of Covid-19 lose approximately 4 years of life. We consider three additional scenarios:

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[23] Such deaths may need to be estimated by comparing actual to expected mortality during the pandemic event.

- Scenario B sets $\alpha(x) = 0.21816$ and $\rho(x) = 4$, for all x.
- Scenario C1 sets $\alpha(x) = 0.14228$ and $\rho(x) = 2$, for all x.
- Scenario C2 sets $\alpha(x) = 0.15152$ and $\rho(x) = 8$, for all x. [24]

Table 1: Scenarios considered

Scenario	Accelerated deaths function, $\pi(t,x)$ (equation (2))		Age-related amplitude function, $\alpha(x)$ (equation (3))				Comment
	$\alpha(x)\,\forall\,x$	$\rho(x)\,\forall\,x$	x_0	λ	α_0	α_1	
A	0.14544	4	–	–	–	–	Baseline scenario of of 80,000 deaths with reach of 4 years
B	0.21816	4	–	–	–	–	Amplitude (Covid-19 deaths as a *proportion* of all-cause deaths) up by 50%
C1	0.14228	2	–	–	–	–	Reach (years of life lost) halved
C2	0.15152	8	–	–	–	–	Reach (years of life lost) doubled
D	–	4	83	5	0.1032	0.2064	Distribution of deaths shifted to higher ages, assuming 80,000 deaths
E	–	4	70	5	0.2304	0.1152	Distribution of deaths shifted to lower ages, assuming 80,000 deaths

The impact on life expectancies under the four scenarios A, B and C1 and C2 is illustrated in Figure 9. For the baseline Scenario A, we can see that, unsurprisingly, the impact of anti-selection on life expectancies is much smaller than in Figure 8. At younger ages, life expectancies rise by only about 0.2%, compared with 1% in Figure 8. When we double the amplitude (Scenario B), the impact on life expectancies doubles at all ages. When we halve the reach (Scenario C1), the shape of the curve in Figure 9 changes, but the impact on survivors' life expectancy remains very small. When we double the reach (Scenario C2), the curve moves closer to that of scenario A. [25]

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[24] Note that for both C1 and C2, $\alpha(x)$ needed to be adjusted to ensure the model generated 80,000 deaths.
[25] If we increase the reach to infinity then anti-selection disappears completely and the ratios plotted in Figure 9 would drop back to exactly 1 at all ages.

Figure 9: Ratios of the life expectancies of survivors under scenarios A, B and C to life expectancies before the pandemic
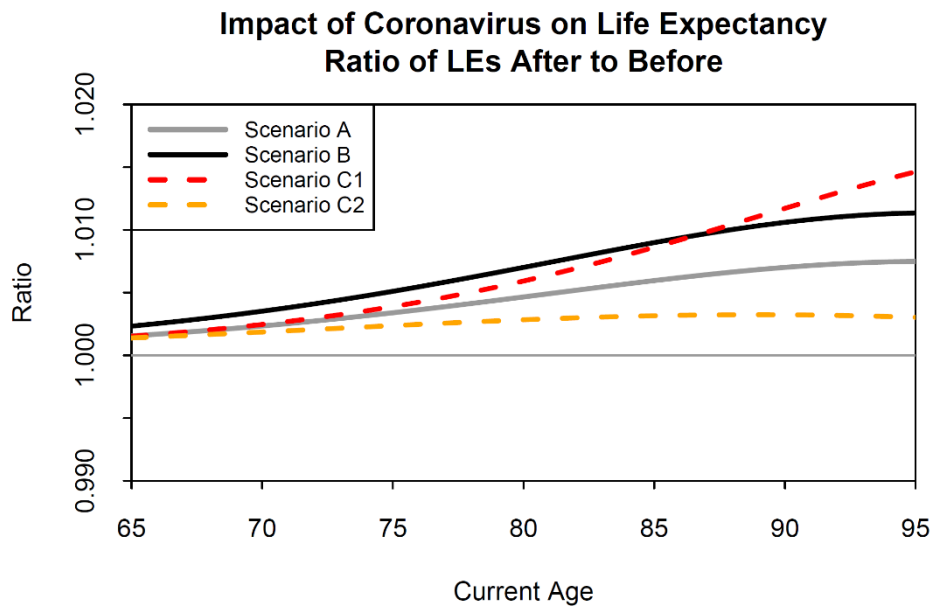
Note: See Table 1 for descriptions of scenarios

A key conclusion is that, even with 80,000 Covid-19 deaths in England & Wales, the impact on the life expectancy of survivors is likely to be very modest.

Another conclusion is that both amplitude and reach are important parameters for us to estimate (amplitude being, arguably, much easier as it is closely linked to actual deaths at each age).

4. Age Dependency in the Amplitude Function

We next consider to what extent the amplitude, $\alpha(x)$, might vary by age and, to motivate this, we compare registered deaths in EW and the US. Figure 10 shows the age profile of Covid-19-registered deaths in EW versus the US. It can be seen that the shapes of the EW and US profiles are quite different with, in relative terms, a much higher proportion in the US of deaths in the 50s and 60s – a difference that cannot be explained by differences in the age profiles of the two populations. This might be due to differences in the degree of social distancing at different ages in the two countries. Another possible reason relates to differences in the healthcare systems. In the US, infection rates might be the same as the UK, but poor access to high-quality healthcare for the more deprived in the US population might push up Covid-19 death rates. After age 65, access to Medicare in the US might mitigate this, but health challenges persist for those who were underinsured or uninsured during their working lifetimes and a gap persists in the quality of healthcare between those reliant on Medicare versus those with supplementary private healthcare in retirement.

Figure 10: Age profile of Covid-19 deaths in England & Wales and the US by age group as a percentage of all deaths above age 45
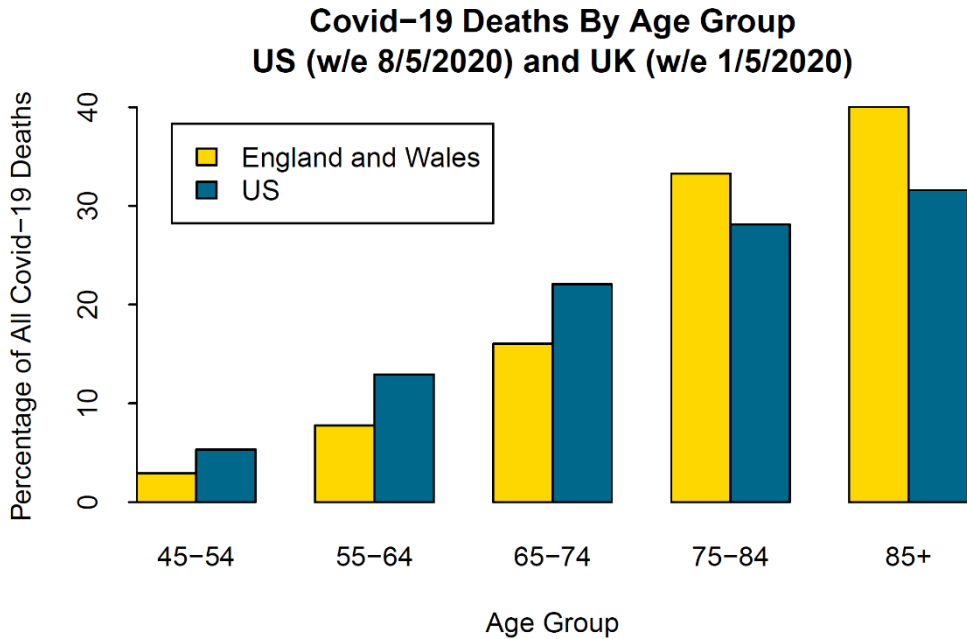
Note: EW– death registrations up to week ending 1 May. US – registrations up to week ending 8 May.

Sources: EW –
https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/datasets/weekly
provisionalfiguresondeathsregisteredinenglandandwales

US – https://www.cdc.gov/nchs/nvss/vsrr/covid_weekly/

For our model to produce a reasonable match to both EW and the US, we need to allow the amplitude to vary with age. We, therefore, introduce two new scenarios, D and E, where, instead of being constant, $\alpha(x)$ follows a logistic function of age x:[26]

$$\alpha(x) = \alpha_1 + (\alpha_0 - \alpha_1)/\left(1 + \exp\left(\frac{x-x_0}{\lambda}\right)\right). \tag{3}$$

The function starts with an amplitude equal to α_0 at low ages, transitioning smoothly up (if $\alpha_0 > \alpha_1$) or down (if $\alpha_0 < \alpha_1$) to a new level of α_1 at high ages. The transition from α_0 to α_1 is centred around x_0, and the speed of transition depends on λ. For low ages, $\alpha(x) \approx \alpha_0$, and for high ages, $\alpha(x) \approx \alpha_1$.

The two new scenarios are (see Table 1):

- Scenario D sets $x_0 = 83$, $\lambda = 5$, $\alpha_0 = 0.1032$ and $\alpha_1 = 0.2064$, with $\rho(x) = 4$ for all x: a low amplitude at low ages gradually shifts to a higher amplitude at high ages, relative to Scenario A.

[26] The logistic function provides a simple mechanism to move smoothly from a high amplitude at one end to a lower amplitude at the other. For a given reach function, $\rho(x)$, we can calibrate the four parameters of the logistic function, to match observed death counts in each age group. Subsequently, we can test for goodness of fit to see if the form of the logistic function could be improved.

- Scenario E sets $x_0 = 70$, $\lambda = 5$, $\alpha_0 = 0.2304$ and $\alpha_1 = 0.1152$, with $\rho(x) = 4$ for all x:[27] a high amplitude at low ages gradually shifts to a lower amplitude at high ages, relative to Scenario A.

Compared to Scenario A, Scenario D results in fewer deaths at the younger ages and more deaths at the high ages (see Figure 11, grey and black lines). Figure 11 also shows the age profile for registered deaths[28] in EW. We can see a significant difference between Scenarios A and D, with Scenario D (black line) providing a much better match to the EW data (gold dots). Scenario E (not plotted) shifts the balance in the other direction from Scenario A towards younger ages rather than old. Scenario E happens to fit US data well up to the 75-84 age group, but underestimates deaths in the 85+ age group, indicating that the form of $\alpha(x)$ in equation (2) needs to be modified to fit the data of other countries better.

A key conclusion is that our model provides an extremely good fit with actual deaths across a wide range of ages (at least for EW), and that the model can be adapted to explain different patterns of COVID-19 mortality emerging in different countries.[29]

Figure 11: Comparison of modelled deaths with actual deaths by age in the England & Wales
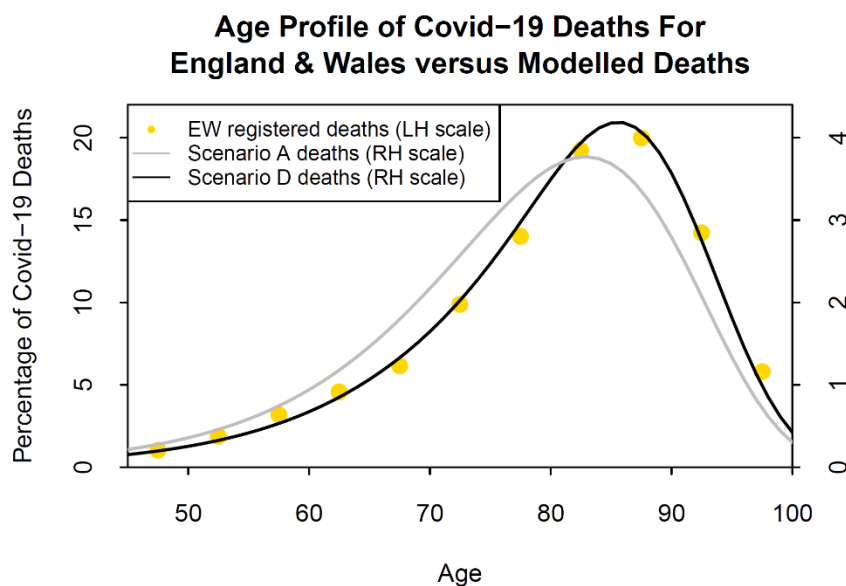
Note: EW deaths: males and females combined, in 5-year age groups (40-44, ..., 85-59, 90+; 90+ split 71:29 to create 90-94 and 95+). See Table 1 for details of Scenarios A and D.

Source: ONS weekly Covid-19 death registrations up to week ending 1 May, accessed 12/5/2020.

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[27] The choice of parameters in Scenario D was the result of some trial and error with the objective of getting a good fit to the EW Covid-19 deaths curve.

[28] Registered deaths lag behind occurrences, but the *distribution* by age will be approximately the same.

[29] However, we should stress that the Scenario A, D and E death curves are based on a stylized model with a hypothetical mortality curve and population profile, only approximately calibrated to EW data.

5. Potential Socio-Economic Differences in Mortality

We now explore a different facet of the data that has recently been published by the UK Office for National Statistics (ONS) concerning the distribution of deaths across different socio-economic groups. Media reports have suggested that members of lower socio-economic groups are more likely to contract Covid-19 than those in higher groups. This is because the former live in more crowded dwellings in poorer neighbourhoods and are more likely to have to leave home and travel on crowded public transport in order to go to work (especially `essential workers', such as those working in the health and care systems), while the latter are more likely to be able to work from home and exercise in private gardens or spacious parks, thereby reducing the risks of catching the virus. If the infections resulted in relatively more deaths, this could widen socio-economic divisions.[30] Figure 3 suggests a link between Covid-19 and all-cause mortalities, while Figures 4 and 5 suggest a link between Covid-19 deaths and particular co-morbidities that can, in turn, be associated with social deprivation.

Figure 12: Age-standardized mortality rates, all deaths and deaths involving Covid-19, Index of Multiple Deprivation, England, deaths occurring between 1 March and 17 April 2020
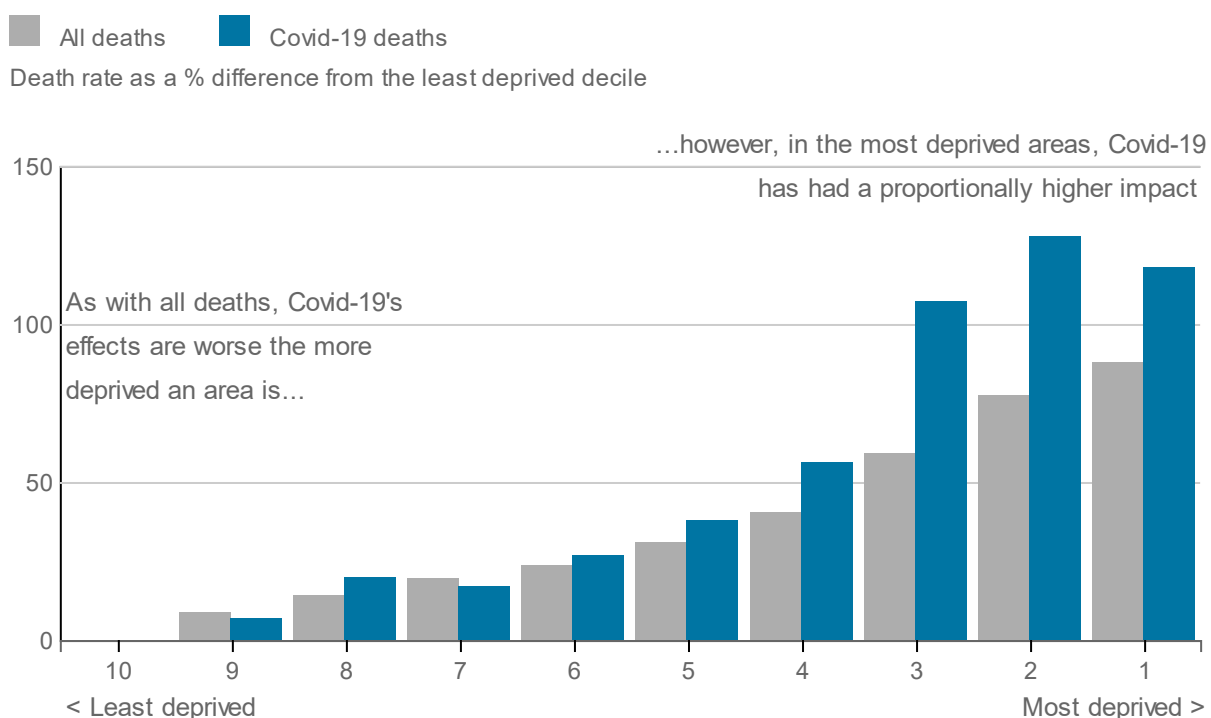
Source: Office for National Statistics (1 May 2020) *Deaths involving COVID-19 by local area and socioeconomic deprivation: deaths occurring between 1 March and 17 April 2020*.
https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/bulletins/deaths involvingcovid19bylocalareasanddeprivation/deathsoccurringbetween1marchand17april

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[30] Max Fisher and Emma Bubola (2020) As coronavirus deepens inequality, inequality worsens its spread, *New York Times*, 15 March; https://www.nytimes.com/2020/03/15/world/europe/coronavirus-inequality.html

ONS data for England, using the Index of Multiple Deprivation (IMD),[31] appear to support the view that there are significant socio-economic differences in Covid-19 mortality rates: the percentage difference between mortality of the most and least deprived deciles is much larger than that for all-cause mortality. See Figure 12 which shows mortality *relative to* the least deprived decile 10.[32]

However, the source article for this data also indicates that infection and death rates in London are more than twice those of other regions. London also has a disproportionate number of deprived neighbourhoods (especially IMD deciles 2 and 3),[33] relative to the country as a whole, and, if this is taken into account, then the impact of Covid-19 on deprived neighbourhoods is somewhat different from what the raw data in Figure 12 implies.

This is illustrated in Figure 13. The grey and blue bars are as in Figure 12. The gold bars adjust the blue bars for regional differences in Covid-19 mortality, and, in particular, the 'London effect'.[34] The grey bars (all-cause mortality[35]) and gold bars (Covid-19 mortality) can be directly compared against each other. We now see that Covid-19 mortality is very similar to all-cause mortality for deciles 1, 2 and 3 and 10, the three most deprived and the least deprived areas, respectively. However, Covid-19 mortality in deciles 4 to 9 is about 10 to 15% lower than we would anticipate if Covid-19 mortality was proportional to all-cause mortality. In other words, once we control for regional differences in mortality rates, Covid-19 deaths in both the most and least deprived groups are in proportion to the all-cause mortality of these groups – in line with the findings in Figure 3 for the population as a whole by age. However, the groups in between have lower Covid-19 deaths compared with their all-cause mortality. The reason for this is not clear, although it might be, as suggested above, because they were better able to adapt to lockdown and maintain more effective social distancing than the other groups.

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[31] Index of Multiple Deprivation. See:
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/464430/English_Index_of_Multiple_Deprivation_2015_-_Guidance.pdf

[32] Office for National Statistics (1 May 2020) *Deaths involving COVID-19 by local area and socioeconomic deprivation: deaths occurring between 1 March and 17 April 2020.*
https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/bulletins/deathsinvolvingcovid19bylocalareasanddeprivation/deathsoccurringbetween1marchand17april

[33] Index of Multiple Deprivation. See:
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/464430/English_Index_of_Multiple_Deprivation_2015_-_Guidance.pdf

[34] A simple multiplicative model is used for age-standardized mortality rates (ASMRs) by region, i and IMD decile, j: i.e., $ASMR(i,j) = r_i d_j$. Values for r_i and d_j are derived by matching modelled regional and decile ASMRs to those in Figure 12, taking account of the number of Lower Layer Super Output Areas (LSOAs) by region and IMD decile.

[35] Note that all-cause mortality has not been adjusted for deprivation. The explanation is that it does not tend to vary much by region unlike Covid-19 mortality. In the context of the model used here (see previous footnote), the r_i in $r_i d_j$ are all equal for all-cause mortality.

Figure 13: Age standardized mortality rates by IMD decile relative to the least deprived group 10
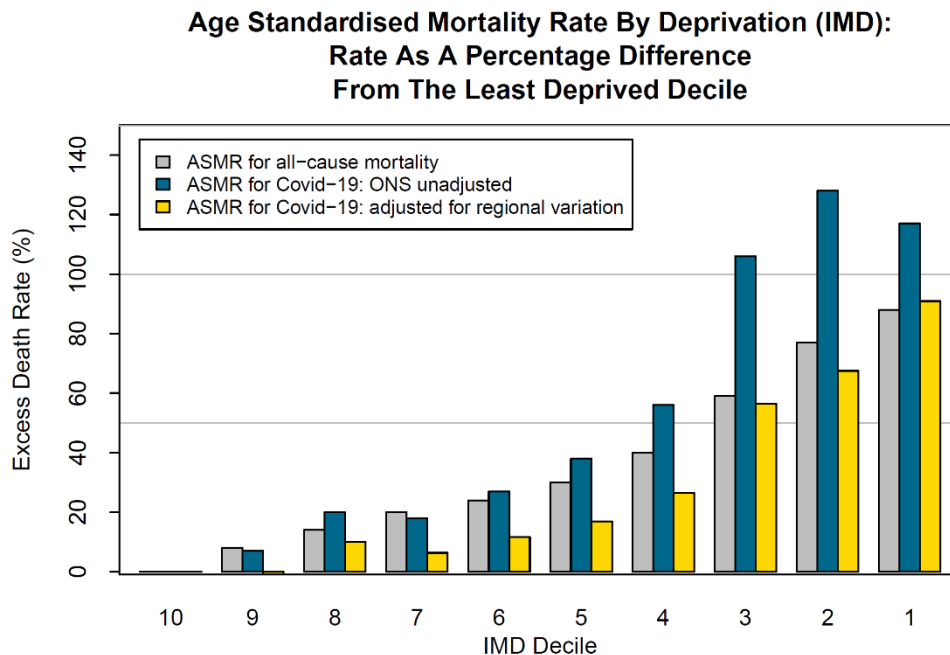
Note: Grey bars are based on all-cause mortality from 1 March to 17 April. Blue bars use age-standardized mortality rates (ASMRs) for Covid-19 based on unadjusted ONS figures. Gold bars show the ASMR's after adjustment for regional differences in death rates from Covid-19.

In line with equation (1), the socio-economic analysis suggests a possible way of linking Covid-19 mortality with frailty for IMD group i at age x:

Covid mortality rate(i, x) = All-cause mortality rate(i, x) × Infection rate(i, x)

$$\times \text{ Relative frailty}(i, x) \qquad (4)$$

This relationship could also be extended down to the individual level, i, and reflect individual co-morbidities. Equation (4) could then be used to improve the accelerated deaths model.

6. Observable Consequences of Accelerated Deaths – Lessons from the Pattern of Deaths from Respiratory Diseases

We will clearly see higher death rates in 2020 than expected at the beginning of the year, but what are the prospects for 2021 and beyond in terms of national death rates? Everything else being equal, the accelerated deaths model will predict low death rates in 2021 due to anti-selection, gradually reverting to previously predicted levels of mortality. The peak in 2020 will depend primarily on the amplitude at various ages, while the size of the dip in 2021 and rate at which rates revert to `normal' will depend more on the reach. Identifying these effects in the data will, though, be complicated by the fact that each year in the future will have its own randomness including the possibility of further waves of Covid-19, as well as seasonal influenza etc, which might mask any dip in 2021 due to Covid-19.

Helpfully, we can identify exactly this type of pattern in past mortality rates. Figure 14 shows death rates for respiratory diseases only (i.e., not all-cause mortality) by gender, age group and deprivation over the period 2001-2016.

Figure 14: Death rates (logarithmic scale) from respiratory diseases in England by gender, age group, year and deprivation
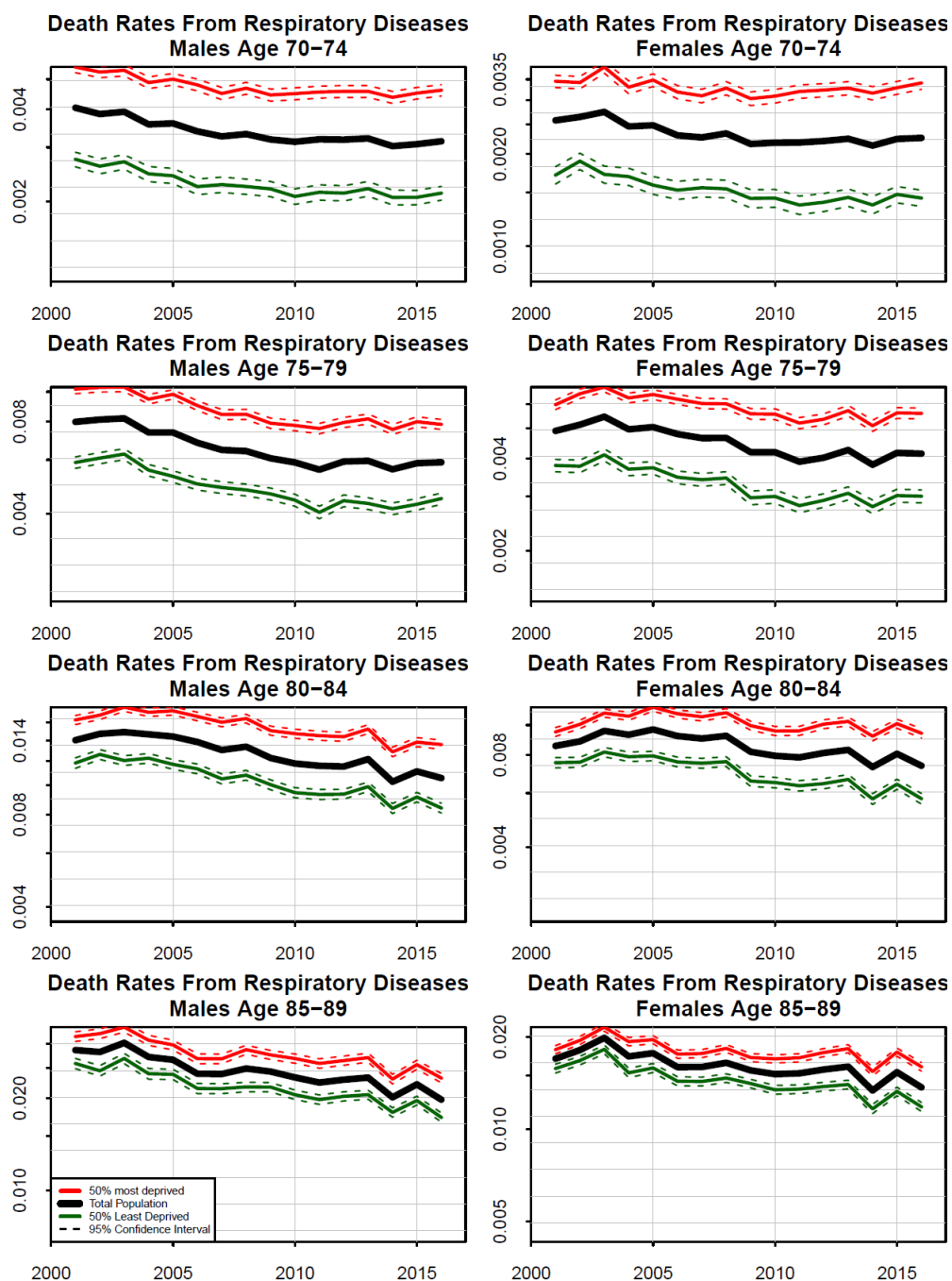
Note: The 50% most deprived are based on neighbourhoods in England that have the highest levels of *income deprivation* (Cairns et al, 2019). Horizontal grey lines: moving from bottom to top, each grey line represents 20% higher mortality (on a log scale) than the line below. (Source: data adapted from Cairns et al, 2019.)

We can see clearly synchronized patterns over the years 2013, 2014 and 2015: a peak followed by a dip followed by another peak. This pattern is consistent[36] with the 2012-13 influenza epidemic *accelerating* the deaths of people with chronic respiratory disease in 2013. This was followed by a dip in 2014 when there were fewer people with chronic respiratory diseases than normal, in combination with it being a quiet year for influenza. The next year, 2015, had a further peak potentially linked to another severe influenza season.

Also notable in the data is the fact that the pattern is strongest in the 85-89 age group, of similar magnitude for males and females, and weakens as we move to younger age groups. There are two potential reasons for the weakening signal at younger ages. First, it might simply be that there were many fewer accelerated deaths at younger ages. Second, the *reach* of the acceleration might vary with age, with a longer reach at young ages and a short reach (e.g. $\rho(x) = 1$, say) at high ages, with many people dying in the same year.[37]

We conclude that useful lessons about the potential pattern of accelerated deaths from Covid-19 can be drawn from examining deaths from respiratory diseases, especially at different age ranges.

7. Lessons from Past Seasonal Influenza Epidemics

Even before the Covid-19 pandemic, UK actuaries had been paying a good deal of attention to weekly mortality registrations from the ONS and their potential impacts on mortality projections.[38] These weekly returns are characterized by predictable seasonal fluctuations (with generally higher death rates in winter) and unpredictable influenza epidemics. Flu epidemics typically begin to emerge in December but their precise timing is uncertain and their magnitude even more so. The ONS regularly publish weekly data by gender and age group,[39] and, periodically, further subdivisions by IMD decile.[40] A snapshot of this data for the 75-84 age group is presented in Figure 15. Deaths per 100,000 have been plotted on a log scale to emphasize the fact that the magnitudes of both predictable annual seasonal fluctuations and unpredictable flu epidemics do not depend significantly on either deprivation or gender. For example, if a flu epidemic pushes weekly death counts up by 20% amongst the most deprived males, then there will approximately be a 20% spike in weekly deaths for females and for the least deprived. Plots for other ages suggest that influenza spikes tend to get *smaller* (in percentage terms) at *younger* age groups. This contrasts with Covid-19, where

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[36] There might, of course, be other reasons for this synchronization.

[37] Note that if the reach is much less than 1 year, then our ability to identify the effect in annual data actually diminishes again, as it is only deaths that would have occurred in the same calendar year that are accelerated. Further, the anti-selection effect would be very short lived.

[38] See, e.g., Continuous Mortality Investigation (2019).

[39] See
https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/datasets/weekly provisionalfiguresondeathsregisteredinenglandandwales

[40] ONS (2019) *Weekly deaths registrations by IMD, sex and age group, England & Wales, 2005 to 2018*
https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/adhocs/10929w eeklydeathsregistrationsbyimdsexandagegroupenglandandwales2005to2018

death rates are approximately *proportional* to all-cause mortality suggesting that any spike in weekly mortality in percentage terms would be *similar* across all age groups.[41]

Figure 15: Weekly death registrations per 100,000 by deprivation and gender over the period 2005 to 2018 for the age group 75 to 84

Weekly Deaths Per 100,000 Population
Males Aged 75–84

Weekly Deaths Per 100,000 Population
Females Aged 75–84
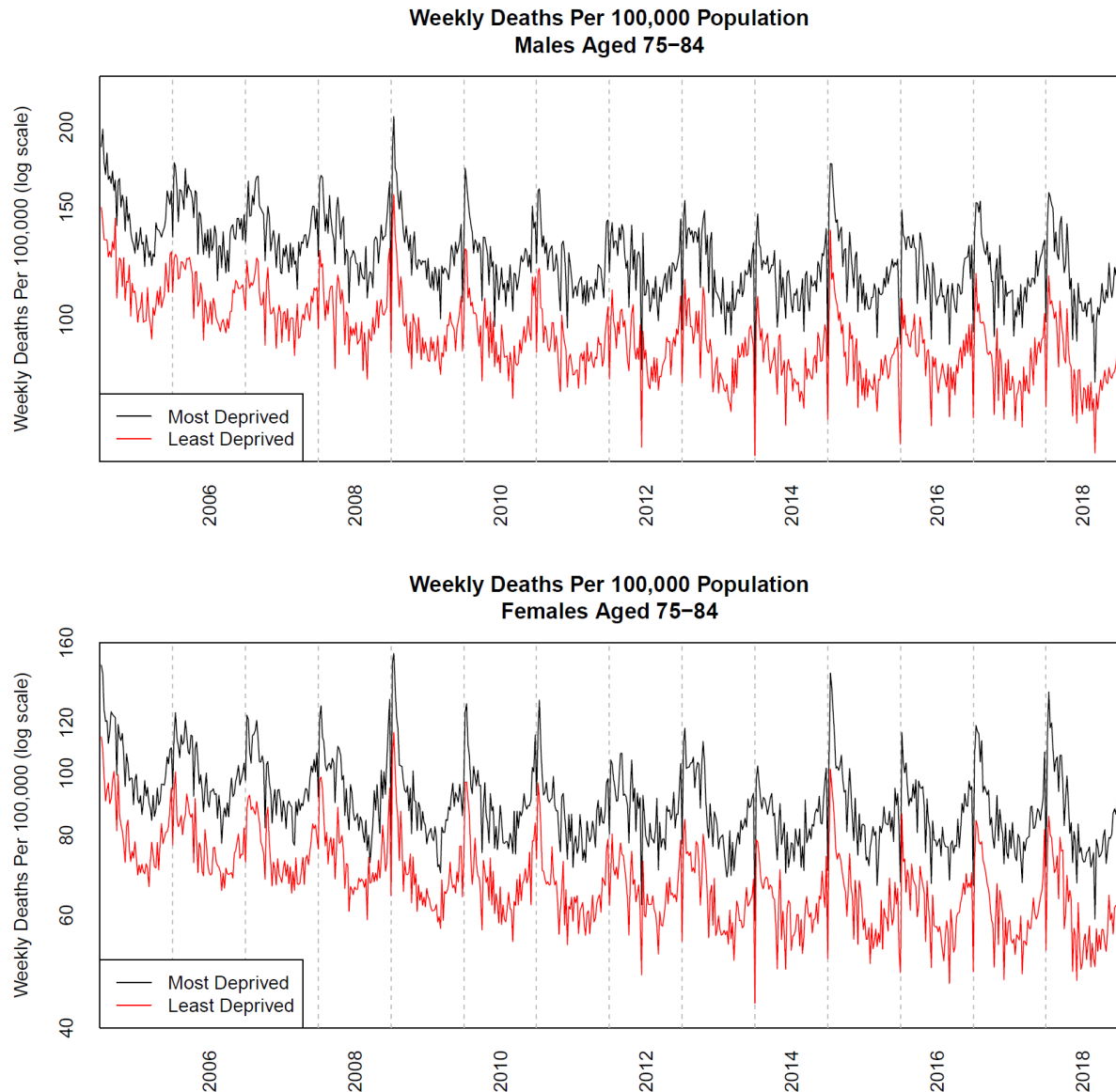
Note: Most deprived: IMD deciles 1 to 5. Least deprived: IMD deciles 6 to 10. Death counts have been standardized to account for the different population counts in the most and least deprived areas.[42]

The weekly deaths plots and the similarity of the size of spikes suggest that influenza mortality has some similarity with Covid-19, at least within a given age group: specifically, influenza death rates appear to be proportional to underlying frailty (after taking account of the fact

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[41] It is important to note at this stage that we do know whether Covid-19 will have a seasonal pattern.
[42] The 50% most deprived areas have significantly less than 50% of the population at high ages. A plot of raw death counts would distort comparison of the most and least deprived.

that older males are more frail than females, and that more deprived people are more frail than less deprived people at a given age).

It is possible that the proportionality argument could be more subtle, however. If influenza deaths are more closely linked to specific co-morbidities rather than overall frailty (i.e., all-cause mortality) then the weekly-death spike sizes might be linked to those co-morbidities.[43] The fact that this is not obvious, suggests that influenza deaths are linked to a range of co-morbidities, which, in turn, have similar proportions by gender and deprivation to all-cause mortality.

It is important, also, to note what the differences might be between influenza and Covid-19. First, they are different viruses that will act in different ways.[44] Second, it is 'business as usual' during an outbreak of influenza, contrasting with a 'lockdown' during the Covid-19 pandemic. But lockdown affects different socio-economic groups in different ways. As we discussed earlier, a greater proportion of more-deprived people still have to go out to work making them more vulnerable to infection. In due course, it will be important, therefore, to analyze weekly data by deprivation both during and after the pandemic to assess what the differences might be and to ascertain whether post pandemic mortality rates in the most deprived groups are disproportionately impacted.

We conclude that it is possible to draw useful lessons about volatility spikes in Covid-19 deaths from examining past seasonal flu epidemics. However, there is an important difference. Whereas the spikes in seasonal flu increase with age, our finding that Covid-19 death rates are approximately proportional to all-cause mortality suggests that any spike in Covid-19 mortality in percentage terms would be similar across all age ranges.

8. Indirect Impacts of the Covid-19 Pandemic

The previous sections, and the model itself, assume that mortality rates for survivors revert to previous levels of mortality with no change to the annual mortality improvement rate. There are a number of reasons why this might not turn out to be the case, although, at present, none can be easily quantified:

- Some of those who have recovered from Covid-19 might carry forward some degree of impairment, possibly temporary, but also possibly long term. For example, a post-mortem study on patients who succumbed to the virus found that many experienced extensive kidney failure, while hospitals in Wuhan and New York City are treating many surviving Covid-19 patients for kidney failure.[45] Similarly, liver function has

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[43] As a stylized example, suppose influenza deaths were linked only to and in proportion to chronic obstructive pulmonary disease (COPD), which, in turn, is heavily dependent on smoking habits. If 100% of males and 0% of females were smokers, then we would see a significant influenza spike in weekly deaths for males but almost none for females.

[44] Unlike Covid-19, influenza is not completely new to our immune systems or to our health care systems. It is possible that over time, as more of us become exposed to Covid-19 and begin to develop anti-bodies and as more effective treatments and vaccines become available, Covid-19 will behave more like influenza.

[45] Yang et al (2020).

failed to return to normal with many Covid-19 patients.[46] Other examples include Covid-19 survivors developing sepsis[47] or experiencing strokes, despite some being young and having only mild symptoms.[48] Hard data on the numbers of impaired survivors is scarce at present. However, if impairments are more widespread (covering many more people than just intensive care survivors) and these do shorten life expectancies, then our model will need to be modified to treat those with impairments differently from other survivors (i.e., split the red region in Figure 6 into two subsections). The discovery, in April 2020, that the drug remdesivir shortens recovery times in serious Coronavirus illness may mitigate these impairments going forward.[49]

- Delays in the treatment of non-Covid-19 patients because the health system cut back its non-essential services in order to redirect resources to deal with the pandemic and because 'coronaphobia' has led people to delay getting a diagnosis for other potentially serious illnesses. For example, thousands of cancers are failing to be diagnosed every week because patients are not going to their doctor. Cancer Research UK reports that referrals by doctors for urgent hospital appointments had fallen by 75% – equivalent to 2,300 cases per week. Another 400 cancers a week were being missed because 200,000 weekly screenings for breast, cervical, lung and bowel cancer was suspended during the lockdown.[50,51] A study from University College London (UCL) and the Health Data Research Hub for Cancer (DATA-CAN) predicted that up to 18,000 more people could die from cancer over the next year in England because of the impact of Covid-19.[52]

- Self-isolation during lockdown and the economic recession that followed the ending of lockdown might have consequences for medium and long-term mortality:
  - Some of those who have found themselves furloughed as a result of the lockdown increased their alcohol and drug consumption.[53]

[46] Wu et al (2020).

[47] Zhou et al (2020).

[48] Ariana Eunjung Cha (2020) Young and middle-aged people, barely sick with covid-19, are dying of strokes - Doctors sound alarm about patients in their 30s and 40s left debilitated or dead, *Washington Post*, 25 April; https://www.washingtonpost.com/health/2020/04/24/strokes-coronavirus-young-patients/

[49] Heidi Ledford (2020) Coronavirus drug remdesivir shortens recovery, but is not a magic bullet - Despite conflicting data, the highly anticipated results will make the treatment a standard of care in the United States, *Scientific American*, 30 April; https://www.scientificamerican.com/article/coronavirus-drug-remdesivir-shortens-recovery-but-is-not-a-magic-bullet/

[50] Sophie Borland (2020) 2,700 cancers MISSED every week: Coronavirus crisis causes urgent GP hospital referrals to plummet as patients are reluctant to visit their doctor, *Daily Mail*, 21 April; https://www.dailymail.co.uk/news/article-8242789/2-700-cancers-MISSED-week-Coronavirus-crisis-causes-urgent-GP-hospital-referrals-plummet.htmls

[51] Screenings in the US are also almost down to zero. See EPIC Health Research Network (1/5/2020) *Preventive Cancer Screenings during COVID-19 Pandemic,* https://ehrn.org/wp-content/uploads/Preventive-Cancer-Screenings-during-COVID-19-Pandemic.pdf

[52] Laura Bundock (2020) Coronavirus epidemic could lead to 18,000 more cancer deaths in a year, study suggests, *Sky News*, 29 April; https://news.sky.com/story/coronavirus-epidemic-could-lead-to-18-000-more-cancer-deaths-in-a-year-study-suggests-11980287

[53] See, e.g., (1) Melinda Fulmer (2020) Quarantinis are a meme of this crisis - but relying on alcohol could lead to serious problems, *Washington Post*, 28 April; https://www.washingtonpost.com/lifestyle/wellness/drinking-problem-home-coronavirus-lockdown/2020/04/27/69c45984-865e-11ea-a3eb-e9fc93160703_story.html. (2)

- If the economy falls into recession for an extended period leading to long-term unemployment and/or more jobs are automated in response to the pandemic,[54] this can result in higher death rates, including 'deaths of despair'.[55]
- The long-term impact of the economic downturn might reduce spending on medical and pharmaceutical research, causing a reduction in long-term future mortality improvements.

- The pandemic might cause general medical advances to stall for a number of years as resources are redirected to finding a vaccine and treatments for Covid-19.

- On the positive side, the behavioural changes required by lockdown – such as social distancing, wearing face masks in public, and reduced automobile usage – might have health benefits if these changes are adopted long term.

- Similarly, the Covid-19 pandemic might also speed up the search for treatments that delay ageing, one of the primary factors that make people more susceptible to the virus. This is because if ageing can be slowed at the cellular level, then the diseases that afflict older people in particular – cancer, heart disease, dementia, and now Covid-19 – can be prevented or their effects ameliorated. Two drugs in particular are attracting interest. The first is metformin which has already been shown to have a positive effect in mitigating or delaying diabetes, cardiovascular diseases, cancer, and dementia. For example, elderly diabetics on metformin experience 20% lower mortality than age-matched subjects without diabetes. The second is a new category of drugs called rapalogues which have been shown to extend health and lifespan in animal experiments and to increase resistance to flu and reduce respiratory tract infections in older human adults.[56]

David Batty (2020) Coronavirus crisis could increase users' drug habits – addiction expert says lockdown will be 'tipping crisis' for some recreational users, *Guardian*, 3 May; https://www.theguardian.com/society/2020/may/03/coronavirus-crisis-could-increase-users-drug-habits-report

[54] The Brookings Institute predicts that up to 50% of all jobs could be replaced by robots following the pandemic. See Mark Muro, Robert Maxim, and Jacob Whiton (2020) The robots are ready as the COVID-19 recession spreads, *Brookings blog*, 24 March; https://www.brookings.edu/blog/the-avenue/2020/03/24/the-robots-are-ready-as-the-covid-19-recession-spreads/

[55] Long-term unemployment is associated with an increase in opioid use and suicide. See, e.g., (1) Anna Mikhailova and Charles Hymas (2020) Ministers braced for the economy to slump by more than 14 per cent in the next three months - Concerns that tens of thousands of Britons may die prematurely from the worst recession in history, *Daily Telegraph*, 10 April; https://www.telegraph.co.uk/politics/2020/04/10/ministers-braced-economy-slump-14-per-cent-next-three-months. (2) Olga Khazan (2020)the true cause of the opioid epidemic - New research supports the idea that economic distress led to an increase in opioid abuse; but some say the origins of the epidemic are far more complicated, *The Atlantic*, 2 January; https://www.theatlantic.com/health/archive/2020/01/what-caused-opioid-epidemic/604330. (3) Office for National Statistics (2019) Middle-aged generation most likely to die by suicide and drug poisoning, 13 August; https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/healthandwellbeing/articles/middleagedgenerationmostlikelytodiebysuicideanddrugpoisoning/2019-08-13. (4) Stuckler et al (2009). (5) Mäki and Martikainen (2012). (6) Case and Deaton (2015, 2017). (7) Reeves et al (2015). (8) Krueger (2017).

[56] Jamie Metzl and Nir Barzilai (2020) Drugs that could slow aging may hold promise for protecting the elderly from Covid-19, *Leaps Mag*, 23 April; https://leapsmag.com/drugs-that-could-slow-aging-may-hold-promise-for-protecting-the-elderly-from-covid-19/

These potential impacts lead us to consider two further scenarios, which are simple modifications to the parameterization of the baseline Gompertz model (see Table 1):

- Scenario F involves a permanent 5% upwards shock in future mortality rates affecting selected cohorts;
- Scenario G involves a permanent reduction of 0.1 percentage point in the annual mortality improvement rate relative to the assumed pre-pandemic improvement rate (i.e. 1.5% falls to 1.4% per annum).

Table 2 shows the impact on life expectancy at selected ages. In Scenario F, life expectancies would fall, for example, by 1.4% at age 50 and 2.8% at age 80. In Scenario G, life expectancies would fall by 0.8% at age 50 and 0.4% at age 80. In other words, these scenarios have a substantially bigger impact on the life expectancy of survivors than Scenarios A, B, C1 and C2 (see, for example, Figure 9). *It is therefore possible that these indirect consequences could have a much bigger impact on average life expectancy than the direct consequences measured by the accelerated deaths model.*

Table 2: The long-term impact on life expectancy of the Covid-19 pandemic

Scenario	Impact on life expectancy at age:			
	50	60	70	80
Baseline: No Covid-19 pandemic	36.62	26.26	17.12	9.86
A	36.63 (+0.02%)	26.27 (+0.05%)	17.14 (+0.12%)	9.88 (+0.23%)
F	36.12 (-1.4%)	25.80 (-1.7%)	16.73 (-2.2%)	9.58 (-2.8%)
G	36.33 (-0.8%)	26.07 (-0.7%)	17.01 (-0.6%)	9.81 (-0.4%)

We should also take into account the possibility that whether or not a country introduced a lockdown made little difference to that country's long-term mortality rate from Covid-19. Dr John Lee, a retired professor of pathology, argues that, although a lockdown can slow down the spread of the virus compared with a country where a lockdown is not imposed, it could eventually catch up. He points to Sweden which did not lock down to the same extent as the UK, but had a very similar Covid-19 mortality curve to the UK. He also points out that the economic damage in the UK (and elsewhere) from the lockdown and the longer-term consequences for non-Covid-19 mortality that we discussed above will be enormous in the UK compared with Sweden or South Korea which did not lock down at all and also had many fewer deaths as a result of a highly effective 'test, trace and isolate' policy.[57]

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[57] (1) John Lee (2020) There's no direct evidence that the lockdowns are working, *Spiked-online*, 17 April; https://www.spiked-online.com/2020/04/17/theres-no-direct-evidence-that-the-lockdowns-are-working/

(2) John Lee (2020) The Grand Delusion: Bail-out billions shield us from the reality - our economy is in tatters. Boris MUST stop us sinking into a new depression, *Daily Mail*, 8 May; https://www.dailymail.co.uk/news/article-8302055/Bail-billions-anaesthetise-reality-economy-tatters.html

9. Conclusions

To investigate the impact of Covid-19 on higher-age mortality, we developed a simple model of accelerated deaths (compared with scheduled deaths) that depends on two parameters: an *amplitude* parameter which measures the number of Covid-19 deaths as a *proportion* of expected deaths in the next year due to all causes at the same age; and a *reach* parameter which measures the expected remaining years of life lost (i.e., remaining life expectancy) by those who die immediately from Covid-19.

We showed that, for plausible values of these two parameters, many of those who died from coronavirus would have died anyway in the relatively near future due to the presence of certain co-morbidities that Covid-19 fatally impacts, in particular, ischaemic heart diseases, dementia and Alzheimer's disease, chronic lower respiratory diseases, influenza and pneumonia, and diabetes. We also showed that the increase in life expectancy of survivors is likely to be very modest, around 0.2% at age 65. This implies that the impact of anti-selection on future life expectancies is negligible.

We noted that the model could be improved if we allowed amplitude to be a logistic function of age instead of being constant at all ages. With an appropriate parametrization of the logistic function, the model provided an excellent fit for Covid-19 deaths for England & Wales (EW).

In terms of socio-economic differences in mortality, we argued that we needed to be careful about interpreting the data. Raw data from EW appears to suggest that members of lower socio-economic groups are more likely to contract and die from Covid-19 than those in higher groups. However, the raw data is biased by regional variations in Covid-19 mortality, in particular London which has much higher Covid-19 mortality and also a disproportionate number of deprived neighbourhoods relative to the rest of the EW. Once this is taken into account, IMD (Index of Multiple Deprivation) data indicated that Covid-19 mortality is proportional to all-cause mortality for both the least deprived and most deprived groups. But it is 10 to 15% lower than we would anticipate for the middle-ranking deprivation groups, suggesting that these groups were better able to reduce their chances of contracting Covid-19 by, for example, working from home during the height of the pandemic.

The accelerated deaths model could be used to predict that death rates will be low in 2021 due to anti-selection, before gradually reverting to previously predicted levels of mortality. The peak in 2020 will depend primarily on the amplitude at various ages, while the size of the dip in 2021 and the rate at which mortality reverts to `normal' will depend more on the reach. Identifying these effects in the data will, though, be complicated by the fact that each year in the future will have its own randomness including the possibility of further waves of Covid-19, as well as seasonal influenza.

We argued that the particular pattern of death rates for respiratory diseases by gender, age group and deprivation over the period 2001-2016 could provide useful lessons for further analysis of the Covid-19 outbreak. For example, in EW, there were synchronized patterns of mortality from respiratory diseases over the years 2013, 2014 and 2015: a peak followed by

a dip followed by another peak. This pattern is consistent with the 2012-13 influenza epidemic *accelerating* the deaths of people with chronic respiratory disease in 2013. This was followed by a dip in 2014 when there were fewer people with chronic respiratory diseases than normal, in combination with it being a quiet year for influenza. We predict that Covid-19 will follow a similar pattern over the next few years.

However, we need to be careful about drawing lessons from previous influenza outbreaks themselves. In the case of EW, the magnitude of deaths from both predictable annual seasonal fluctuations and unpredictable flu epidemics do not depend significantly on either deprivation or gender – although the mortality volatility spikes tend to get smaller (in percentage terms) at younger ages. With Covid-19, by contrast, death rates are approximately proportional to all-cause mortality by age, so that mortality spikes in percentage terms would be expected to be similar across all age groups.

It is also important to consider the indirect consequences of the pandemic. While our simple model predicts that mortality rates for survivors and annual mortality improvement rates return to pre-pandemic levels, this might not be the case for a number of reasons. Recoverees might have acquired a new long-term impairment, such as kidney damage, which reduces their life expectancy. Some people might have delayed getting a diagnosis for other potentially serious illnesses, such as cancer: the latter potentially causing an increase in cancer death rates in 2021. Life expectancy might be reduced for people who responded to the self-isolation of the government-imposed lockdown by increasing alcohol and drug consumption. If the pandemic leads to a global recession or the widespread automation of jobs, levels of long-term unemployment might rise significantly with an associated increase in mortality for those affected, including deaths of despair. On the other hand, people might change their social and workplace behaviour or seek anti-ageing treatments that together reduce their chances of catching covid-19 and this helps to increase their life expectancy. Overall, the impact on the life expectancy of survivors might be much larger than the direct impact as measured using the accelerated deaths model.

Despite being very early days in the course of the worst pandemic to hit the human race in a century, we believe we have found a very simple model for predicting post-pandemic mortality, especially at higher ages. The model is built on the observation that Covid-19 mortality in adults appears to be proportional to all-cause mortality. This is then extended to account for the observation that with respiratory diseases, there can be spikes in mortality in one year which are reversed in the following year, so that if Covid-19 behaves in a similar way, deaths are accelerated over scheduled deaths for a short period before returning to normal. We chose parameters for the model based on little more than educated guesswork, but these can be easily modified in the light of experience. The model can also be modified once we get clearer evidence on any indirect consequences of the pandemic or discover that relaxing the lockdown leads to an immediate surge in infections requiring the re-imposition of the lockdown – and this continues until a vaccine is introduced (if ever).

Finally, our study provides a useful framework for addressing three key challenges in the post-pandemic estimation of the life expectancy of particular groups of lives, such as those in pension schemes and insured annuity blocks. First, it shows a way of adjusting the experience

data from pension schemes and insured annuity blocks collected during the pandemic period in order to avoid mis-estimating future mortality rates. Since Covid-19 appears to increase a cohort's short-term mortality rate by a common multiplicative factor, whatever their current baseline mortality rate, this factor can be used to normalize experience data collected during the pandemic period. Second, it shows how to assess anti-selection risk, if any, in the surviving population in a flexible way that can be applied under a wide range of Covid-19 outcomes and across different countries. Third, it offers an approach to estimating the volatility that may arise in immediate post-pandemic mortality through an analysis of past seasonal influenza epidemics.

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Data

- England & Wales, ONS: Deaths registered weekly in England and Wales, provisional https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/datasets/weeklyprovisionalfiguresondeathsregisteredinenglandandwales/2020
- US, CDC: Weekly Updates by Select Demographic and Geographic characteristics https://www.cdc.gov/nchs/nvss/vsrr/covid_weekly/

Appendix

We describe here a formal version of equation (1). We consider a single individual aged x who has been infected at time 0, and then scale the cumulative Covid-19 death rate by the proportion of the age cohort infected. Define

- $\mu_A(x) =$ death rate at age x from all causes in the absence of Covid-19
- $\mu_C(s, x) =$ death rate at time s and at age x from Covid-19 (assuming that the individual has been infected at time 0).

For simplicity in this Appendix, we will assume that $\mu_A(x)$ is constant over the next year (although in reality it is slowly rising), while, in contrast, $\mu_C(s, x)$ takes the shape of a short-lived, bell-shaped curve: see Figure 16.

Figure 16: Comparison of all-cause death rates and an individual Covid-19 death rate curve from the date of initial infection
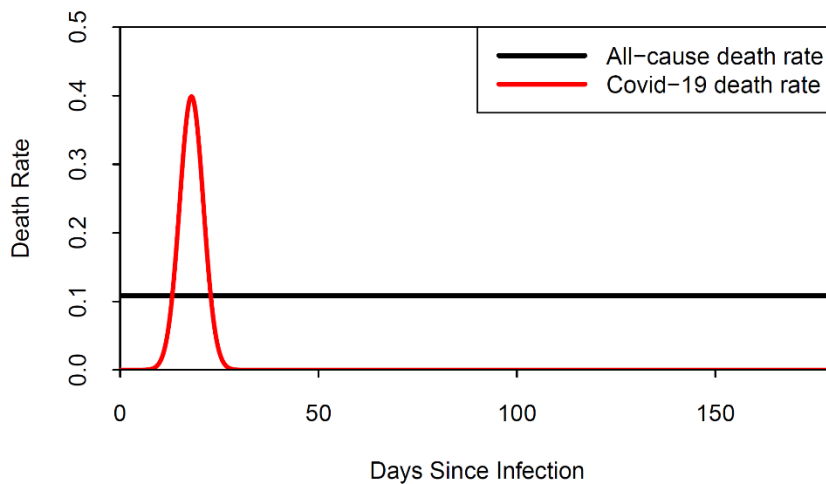
Note: The all-cause death rate is the underlying rate in the absence of Covid-19. The two curves are intended to be illustrative only and are not drawn to scale.

Over the time interval $[0, T]$, the cumulative death rates from the two sources are $\mu_A(x)T$ and $\int_0^T \mu_C(s, x)ds$. Because the risk of dying from Covid-19 is short lived, the latter tends to a constant $M_C(x)$ as T tends to infinity, with convergence after only 4 or 5 weeks, say. Thus, over a given time period T (greater than 5 weeks, say), we can define

$$\text{Relative frailty, } \omega(x) = M_C(x)/(\mu_A(x)T).$$

Note that this does mean that the value of Relative frailty depends on the time horizon T.

Finally, for the full age cohort, x, we reintroduce the Infection rate$(x) = v(x)$. The proportion that *survives* the pandemic (excluding all-cause deaths) is then

$$\exp(-M_C(x)v(x)) = \exp(-\mu_A(x)T \times v(x) \times \omega(x)),$$

and the expressions in the brackets correspond to the left and right hand sides of equation (1), respectively.