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COVID-19 mortalities in England and Wales and the Peltzman offsetting effect

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ABSTRACT
There are two approaches to measuring COVID-19 deaths – 'COVID associated deaths' and 'excess deaths'. An excess deaths framework is preferable, as there is measurement error in COVID associated deaths, due to issues relating to imperfect information about deaths that are directly attributable to COVID-19. The standard measure of excess deaths (comparison of deaths to a 5-year average) is subject to an omitted variables problem, as it attributes the entirety of the variation in mortality to COVID-19. We propose a method to estimate a refined measure of COVID-19 excess deaths in England and Wales that addresses the omitted impact of the first blanket lockdown. Using the counterfactual, we obtain a first stage estimate of excess deaths. In the second stage, this is decomposed into estimates of a refined measure of COVID-19 excess deaths and the excess mortality impact of lockdown. Our results suggest: (i) a refined estimate of mean weekly COVID-19 excess deaths that is 63% of standard excess deaths; and (ii) a positive net excess mortality impact of the lockdown. We make a case that (ii) is due to the Peltzman offsetting effect, i.e. the intended mortality impact of the lockdown was more than offset by the unintended impact.

Introduction

There is uncertainty regarding the number of deaths that are due to COVID-19 worldwide. This is due to known limitations with the two (direct and indirect) main measurement approaches.

In England and Wales there are two direct metrics.[5] Public Health England (PHE) report deaths within 28 days of a positive test; and the Office of National Statistics (ONS) reports deaths where COVID is mentioned on the death certificate. The main drawbacks of these direct metrics are that (i) there are accepted limitations in the extent to which they establish mortality causality[6]; and (ii) because the precise application of direct methods varies across countries, they are less suited to comparative analysis.

The primary indirect metric is 'excess deaths'. This compares total weekly all-cause mortality to a 5-year average (in England and Wales the ONS reports on this basis).[7] The excess deaths metric is more comparable (over time and across countries); and there is somewhat of a consensus regarding its advantages over direct metrics.[8] However, it has two drawbacks: (i) due to the very high concentration of COVID-19 deaths in the elderly (Table 1) it seems likely that some of these deaths would have occurred in any case, and have thus been somewhat 'brought-forward' by the pandemic, rather than being truly excess; and (ii) it does not specifically relate to COVID-19 deaths, due to the approach omitting factors other than COVID-19 that might cause total mortalities to vary. The first issue (mortality displacement) cannot be addressed until more time has passed. In this paper we therefore focus on, and propose a solution to, the omitted variables problem relating to England and Wales, using statistical techniques.[9]

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[4]None of the authors received any external funding for this research.
[5]Often referred to as 'COVID associated' or 'COVID related'.
[6]This limitation is more applicable to the PHE data. However, due to the high rates of comorbidity in relation to COVID-19 deaths, it is also a relevant issue vis-à-vis the ONS data.
[7]As described further by the ONS (2020a).
[8]For example, the Health Foundation (2020) has stated that: 'excess deaths is a better measure than COVID-19 deaths of the pandemic's total mortality [because it] does not depend on how COVID-19 deaths are recorded.' In addition, excess deaths has been advocated by Professor Chris Whitty, the Chief Medical Officer for the UK.
[9]Such methods are commonly used when measuring influenza mortality. For example, Simonsen et al. (1997) apply this approach to identify cyclical deaths relating to influenza epidemics.

Table 1. Deaths involving COVID-19 in England and Wales: as of week ending 28 August 2020.

Age group	Number of COVID-19 deaths	% of COVID-19 deaths accounted for
<10	3	0.0%
10–19	12	0.0%
20–29	74	0.1%
30–39	221	0.4%
40–49	748	1.4%
50–59	2,391	4.6%
60–69	5,047	9.6%
70–79	11,757	22.5%
80–89	20,681	39.5%
90+	11,382	21.8%
Total	**52,316**	**100%**

A range of variables may cause total mortalities to vary, relative to historical averages. Other than COVID-19 itself, policies in the form of non-pharmaceutical interventions (NPIs) are likely to be important factors in explaining mortality variation during the pandemic. These must therefore be parameterized for any refined measure of excess deaths to be robust. Accordingly, we test a range of plausible variables, including modelling the impact of the first lockdown in England and Wales (23 March 2020–13 May 2020).[10]

We adopt a two-stage time series approach.[11] In line with the 5-year excess deaths framework, in the first stage we estimate Poisson models for all-cause mortalities for the 5 years that precede the COVID-19 period (week ending 23 January 2015 – week ending 24 January 2020).[12] We then use each fitted model to predict out-of-sample counterfactual mortalities over the COVID-19 period, which runs through to the end of our study time-horizon: week ending 28 August 2020. The weekly differences between observed all-cause deaths and the predicted counterfactual are the first stage weekly estimates of excess deaths.[13] The first stage estimates, however, omit the impact of other variables; notably, the first national lockdown. Accordingly, second stage regressions are then used to yield estimates that distinguish between the refined measure of COVID-19 excess deaths (above) and the excess mortality impact of the lockdown. There is, of course, a time-lag between infection (i.e. what the lockdown could have *directly* affected) and death (the outcome being measured), which we account for in the second stage regressions.[14]

Three main findings from our second stage results are as follows. First, under our preferred baseline model (which assumes a 4-week lag between infection and death) we find that (i) COVID-19 excess deaths amount to 63% of the standard 5-year average excess deaths; and (ii) in net terms, the lockdown increased all-cause mortalities (associated with an incremental 2,601 excess mortalities per week). Our results are sensitive to the time-lag between infection and death; so a range of lags are analysed. The evidence in the literature is most consistent with a 3 to 4-week lag; and a 4-week lag (as applied in our preferred baseline model) is well supported. Second, although it has been widely reported that COVID-19 deaths are concentrated in the elderly, we find that such deaths have been concentrated in the *very* elderly (75–84 and 85+). Third, we do find that lockdown reduced mortality in net terms in some individual weeks.

We consider the net mortality increasing effect of the first national lockdown may reflect the fact that any intended mortality decreasing impact on COVID-19 deaths was comparatively smaller than, and thus more than offset by, the wider (unintended) mortality increasing impacts. The unintended mortality consequences are consistent with people choosing not to access healthcare for non-COVID-19 illnesses. We therefore posit that we have uncovered an important new case of the well-known Peltzman (1975) offsetting effect, which proposes that people adjust their behaviour to changes in the perceived level of risk (of which there are a number of well-known examples, relating to safety/health policies).

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[10]May 13th was when the most strict lockdown measures were lifted.

[11]Since England and Wales imposed a national lockdown, quasi-natural experimental approaches are not possible, as the relevant counterfactual cannot be observed.

[12]The week ending 24 January 2020 represents our assumed final week of the 'pre-pandemic' period, based on the first known cases occurring on January 30th; see BBC (2020).

[13]This first stage approach is standard and has been widely applied. For example, it was used to estimate excess deaths due to COVID-19 in the U.S., 48 states (excluding North Carolina and Connecticut), the District of Columbia and New York City (Weinberger et al. 2020).

[14]We discuss the evidence on the length of this time-lag in subsection 2.3.

The plausibility of our finding is supported by existing evidence regarding both the (limited) efficacy of the lockdown in achieving its primary aim, and the potential for spillover harms. In light of the evidence, and the well-established possibility of a Peltzman offsetting effect, our finding that the first lockdown in England and Wales had a net mortality increasing impact should not come as a great surprise (although individual policies should be evaluated case-by-case).

The remainder of this paper is structured as follows. Section 2 summarizes explanatory variables that affect mortality. Section 3 presents the general form of our simple two-stage modelling framework. Section 4 describes the data and empirical model specifications. In section 5, we set out our models and discuss the results. Section 6 provides concluding remarks.

Variables that may impact all-cause mortality

Brief overview of the determinants of mortality in the literature

Public health, virology, epidemiology and economics studies point to a number of variables that may impact all-cause mortality; as follows:

- *Environmental/seasonal patterns*. Data shows higher mortality in the winter/poor weather; and lower mortality in the summer/better weather; see Nogueira et al. (2009). Temperature can impact mortality, but the relationship may be non-linear (mortality falls with increases in temperature, but then rises as temperatures become very hot). See Cech et al. (1979); Huynen et al. (2001). Air pollution is positively associated with mortality; Willers et al. (2016).
- *Demographics*. Mortality rates increase with the proportion of elderly people; Chaix et al. (2006).
- *Socioeconomic factors*. Poverty/income is cited as a contributory factor to mortality. The empirical evidence appears mixed; Rodgers (1979); Cutler, Deaton, and Lleras-Muney (2006).

- *Population density*. Studies have shown a positive association between population density and mortality. This is relevant in the context of virus mortality, as transmission is increased where populations are more densely located; Meijer et al. (2012).
- *Healthcare expenditure, resources and public health*. Mortality may fall with investment in healthcare and/or where healthcare resources are increased. In relation to public health, obesity, smoking, etc. may cause mortality to vary; Cutler, Deaton, and Lleras-Muney (2006).

The existing evidence is thus consistent with considering variables within the above categories.

The role of UK policy in explaining COVID-19 mortality

Government COVID-19 policy (and within our estimation period, the first lockdown) also likely impacted mortality. This policy impact must therefore be incorporated, to avoid the omitted variables problem we aim to address.[15] There are two broad types of potential lockdown policy impacts.

First, the direct (intended) impact would typically be to <u>spread out</u> COVID-19 mortalities. The rationale for the first blanket lockdown was framed around the concept of mitigation (slowing, but not reducing in totality, epidemic spread and related deaths). The purpose of this would be to lower peak healthcare demand, to protect the National Health Service (NHS). Lowering the infection (and thus mortality) peak *could* also <u>reduce</u> mortalities in totality, relative to the counterfactual, if said policy prevented the NHS being overwhelmed. Second, the indirect (unintended) consequences of policy responses could be increasing non-COVID-19 mortality. This would be the case if the policy response interrupted healthcare provision and/or affected people's behaviour vis-à-vis broader healthcare. We address the evidence regarding both impacts subsequently. At this juncture, we merely note that the null hypothesis regarding the

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[15]The omission of a lockdown policy variable would result in the risk that the policy impact is misattributed elsewhere within the second stage model; most obviously, to COVID-19 itself.

net overall mortality effect of a blanket lockdown is ambiguous.

Evidence on the time lag between infections, symptoms and deaths

Given the need to account for the lockdown in our modelling, one must do so in a way that reflects the fact that said policy (if effective) directly impacts COVID-19 *infections*, not *deaths*. Thus, impacts on COVID-19 deaths need to take into account the 'lag' from infection to death. The evidence on time lags remains uncertain. However, the World Health Organization (2020) estimated the mean incubation period (infection to symptomatic) at 5–6 days, with a maximum of 14 days (consistent with UK Government quarantine advice). Lauer et al. (2020) also suggest a 5-day incubation.

Verity et al. (2020) calculate the average lag between symptom and outcome (death or recovery). The study was based on case level data for patients that died from COVID-19 in Hubei, China. They found the mean duration from symptom to death was 17.8 days.[16] The World Health Organization (2020) estimated symptom to death being between 2 and 8 weeks. Hawryluk et al. (2020), using more recent data from Brazil, estimated a shorter lag from symptom to death of 15.2 days.

Summarizing the above, the Verity *et al.* study implies a total time lag between infection and mortality of 23 days (5 days to symptomatic; plus 18 days to death). However, the WHO figures imply a longer overall period of 40 days (5 days to symptomatic; plus the mid-point of their 2 to 8 week range above – 35 days). The more recent Hawryluk *et al.* analysis implies a total period of 20 days. Combined, our review of the evidence therefore suggests the average total elapsed time from infection to death likely lies between >3 and <6 weeks. Equally weighting the evidence, mean elapsed time from infection to death is 4 weeks.[17]

Although, on balance, we take the evidence to point to a 4-week lag between infection and death, we recognize the uncertainty around this estimate. We therefore consider the sensitivity of our results to lags of 2, 3 and 5 weeks.

III. General form of the two-stage modelling framework

First stage modelling

The estimation period for the first stage time series models is the pre-COVID period (week ending 23 January 2015–week ending 24 January 2020), where for this sample the time periods are indexed $t \in 1, \ldots, T$. In the first stage, for each all-cause deaths variable (in totality, by age category, etc.) that we analyse, we assume that the observed number of deaths in period t, y_t, is Poisson distributed.[18]

$$y_t \sim Poisson[\mu(m)],$$

where the density of y_t is determined by the conditional mean $\mu(m) \equiv E(y_t|m)$ and m is explained by a set of determinants. The general form of our first stage models is as follows.

$$\mu(m) = \exp(\beta' x_\mathbf{t}) + \varepsilon_t \qquad (1)$$

where $x_\mathbf{t}$ is a vector of observations in period t for the independent variables that explain m, β' denotes a vector of parameters to be estimated comprising parameters pertaining to $x_\mathbf{t}$ and the intercept parameter, and ε_t denotes the idiosyncratic disturbance.

Estimating each of the first stage models involves specifying the Poisson family of distributions using the log link function as this is canonical to this family. As a result, the estimates of the $x_\mathbf{t}$ parameters are coefficients (not marginal effects),

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[16]With a 95% confidence interval of 16.9 to 19.2 days.

[17]This is also the time period from a positive COVID-19 test to mortality that the ONS use as one of the measures of associated deaths. The more recent empirical studies are arguably supportive of a 3-week lag. Hence, one might say the evidence is most consistent with a 3–4 week lag 'in the round'.

[18]We estimate each of the first stage models using maximum likelihood estimation of a generalized linear model (GLM) Poisson regression. For each model we use robust standard errors which we estimate using the Huber / White / Sandwich linearized estimator of the variance. Silva and Tenreyro (2006) demonstrate that an important advantage of the Poisson regression with robust standard errors is that it does not assume that the conditional mean of the dependent variable is equal to its conditional variance. Moreover, turning to whether the general form of the first stage models should instead be a negative binomial model, O'Hara and Kotze (2010) found from their simulations that Poisson and negative binomial models yield identical parameter estimates. However, negative binomial and quasi-Poisson models account for overdispersed data, but by drawing on the result in Silva and Tenreyro (2006) that a Poisson regression with robust standard errors does not assume equidispersion, our model can cater for overdispersed data.

but this has no implications for the out-of-sample counterfactual.

Having estimated Eq. 1 over the pre-COVID period, this fitted model is used to predict weekly counterfactual all-cause deaths over the out-of-sample period (week ending 31 January 2020 – week ending 28 August 2020). This provides estimates of the weekly all-cause deaths if the trend over the estimation period continued. We then calculate the weekly difference between observed all-cause deaths and the predicted counterfactual. These weekly differences are the first stage weekly estimates of excess deaths.

Second stage modelling

In the second stage time series analysis, each estimated measure of excess deaths from the first stage is decomposed into an estimate of the refined measure of COVID-19 excess deaths and an estimate of the excess mortality impact of the lockdown.

Two model specifications are estimate in the second stage via maximum likelihood. From the first, we obtain a mean weekly estimate of the excess mortality impact of the lockdown, and, from the second, we obtain individual weekly estimates of this impact. Both model specifications are estimated using weekly time series data for T_c out-of-sample counterfactual (c) periods, which are indexed $t_c \in 1, \ldots, T_c$. The first model specification serves as the baseline and is as follows.

$$\hat{\rho}(n) = \gamma COVID_{t_c-q} + \delta Lock_{t_c-q} + \varepsilon_{t_c} \qquad (2)$$

where $\hat{\rho}(n)$ is the conditional mean of the estimate of excess deaths from the first stage, and n is explained by lagged COVID and Lock dummy variables. These dummies take values of 1 from the beginning of the COVID and lockdown mortality periods (and zero otherwise), and q denotes the assumed lag between infection and death (see subsection 4.2).

In this first (and also second) of the model specifications in the second stage, we explain the estimate of excess deaths from the first stage using only variables that represent the COVID and lockdown mortality periods. This is for three reasons. First, variables used to explain deaths in the pre-COVID period (i.e. the first stage), such as urbanization, are omitted from the second stage models. This is because such variables are accounted for within the second stage as they are used to estimate the counterfactual deaths for the COVID period, which, in turn, are used to compute the estimate of excess deaths for the COVID period that we explain in the second stage.[19] Second, we omit as a determinant in the second stage models a variable that measures people movement because, although it will impact the estimate of the refined measure of COVID-19 excess deaths and the estimate of the excess mortality impact of the lockdown, its effect will be captured by the COVID and Lock dummies. Third, the rise in working from home during the pandemic is consistent with a reduction in road traffic fatalities, but we do not include a road usage variable in the second stage models to take account of this. This is because the available daily road usage data that was recently introduced and covers only the pandemic is only for Great Britain and not England and Wales.[20] This will though introduce very little (if any) bias into the estimates of the refined measure of COVID-19 excess deaths and the estimates of the excess mortality impact of the lockdown. This is because the COVID and Lock dummies are lagged by 2–5 weeks (with a preference for 4 weeks and with some consideration of 3 weeks) to reflect the time lag between infection and death (for details on this see subsection 4.2), while road traffic fatalities typically occur at the scene of the accident or very soon after, rather than 3 or 4 weeks after. As a result, the reduction in road traffic fatalities associated with the rise in working from home will typically be part of the error terms in the second stage models.[21]

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[19]Relatedly, a further practical reason for this approach is that our measure of urbanization is annual which we assume applies to each week in a year (see subsection 4.1 for more details on this). By including urbanization in the first stage models there is variation in this variable across weeks in different years. Given our second stage samples only relate to part of 2020 there would be no variation in this variable in these samples. Hence the inclusion of this variable in the first stage models and its omission from the second stage.

[20]Also, this data for Great Britain is only available from 1 March 2020 and does not therefore cover all of our second stage sample as the first week of this sample is the week ending 31 January 2020.

[21]We thank an anonymous reviewer for recommending that we provide clear clarification on why in our second stage models we only include determinants that represent the COVID and lockdown mortality periods.

Given excess deaths can be negative or positive, for the models in the second stage, we specify the Gaussian family of distributions and use the identity link function as this is canonical to this family. Therefore, the parameters on the variables that represent the COVID and lockdown mortality periods in the fitted second stage models are marginal effects. From an estimate of Eq. 2, the estimates of γ and δ are mean weekly estimates of our refined measure of COVID-19 excess deaths and the excess mortality impact of the lockdown. This interpretation is because each estimate is relative to the relevant base period in the second stage sample of no COVID/no lockdown. Note that in Eq. 2 (and in the second model specification in the second stage that we turn to next), we omit the intercept to prevent it from capturing any of the COVID and lockdown mortality components of the second stage dependent variable.

Denote the set of out-of-sample counterfactual time periods and the sets of COVID and lockdown mortality time periods $\mathbf{T_c}$, $\mathbf{J}$ and $\mathbf{K}$, respectively. $\mathbf{K} \square \mathbf{J} \square \mathbf{T_c}$ and the periods in $\mathbf{J}$ and $\mathbf{K}$ are indexed $j \in 1, \ldots, J$ and $k \in 1, \ldots, K$. The second model specification for $\hat{\rho}(n)$ is as follows.

$$\hat{\rho}(n) = \gamma COVID_{t_c-q} + \eta_1 Lock1_{t_c-q} + \ldots \\ + \eta_K LockK_{t_c-q} + \varepsilon_{t_c} \quad (3)$$

where we decompose the *Lock* dummy from Eq. 2 into K lockdown indicator variables, $Lock1 + \ldots + LockK$, and use as regressors their q period lags. A lockdown indicator variable takes a value of 1 in the relevant week in the lockdown mortality period (and zero otherwise), and $\eta_1, \ldots, \eta_K$ are parameters to be estimated and are relative to the base period of no lockdown. The mean of the estimates of $\eta_1, \ldots, \eta_K$ will approximate the estimate of the mean weekly excess mortality impact of the lockdown ($\hat{\delta}$) from the fitted Eq. 2. The estimates of $\eta_1, \ldots, \eta_K$ are therefore the weekly deviations about the estimate of the mean weekly excess mortality impact of the lockdown.[22]

IV. Data and details of the empirical model specifications

First stage

Using weekly time series data for the 5 years that precede the first confirmed cases of COVID-19 in York on 31 January 2020 (week ending 23 January 2015–24 January 2020), we estimate 10 first stage models. Each has a different all-cause deaths dependent variable: *Total Deaths*; deaths by age category (<1 year; 1–14; 15–44; 45–64; 65–74; 75–84; and 85+, denoted *Deaths(<1), Deaths(1–14)*, etc.); *Male Deaths*; and *Female Deaths*.[23]

In the first stage models, key explanatory variables are weekly dummies corresponding to calendar week numbers, where, as is standard, one is omitted. These dummies have a lot of explanatory power and would therefore appear to be good proxies for explanatory variables that were not included due to a lack of data (e.g. road usage).[24] In the literature, these dummies are not cited as being key, so we strike a balance between retaining a small number of variables in the models that are regarded as key in the literature and where collectively there is the most evidence of significance. Such variables were significant in a number (but not all) of the models and led

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[22]For our second stage sample, we could not use the same type of approach as in Eq. 3 to obtain estimates of the weekly deviations of our refined measure of COVID-19 excess deaths about the mean estimate from Eq. 2. This would involve estimating a model that decomposes the *COVID* dummy in Eq. 2 into *J COVID* indicator variables. Alternatively, to preserve degrees of freedom, one could obtain estimates of the weekly deviations of our refined measure using a piecewise approach. This would involve estimating multiple models where the models include different subsets of the *COVID* indicator variables that collectively represent the full set. Each model would also include a dummy that collectively accounts for the *COVID* indicator variables that do not enter the model individually. Neither approach was possible in this study due to the relatively small out-of-sample period. For a sufficiently long out-of-sample period, one could use at least the second of the above approaches to estimate the weekly deviations of our refined measure.

[23]Our deaths data are for England and Wales collectively and not for England, Wales, Scotland and Northern Ireland individually. This is because richer data is available for England and Wales. For our study period, weekly data on total all-cause deaths and all-cause deaths by gender and age category is only available for England and Wales and not for the four countries. At the time of our analysis, the available data for the four countries was a long way from being comparable to that for England and Wales. There has since been some improvement in the availability of data for the four countries (e.g. weekly all-cause deaths data by gender and age category is now available for Scotland from January 2020), but this is not comparable to that for England and Wales collectively / individually (e.g. weekly all-cause deaths data by gender and age category is not available for England and Wales individually).

[24]As we use weekly dummies, we are accounting for the weekly trends across the years in our first stage samples, and thus the differences between these trends for different calendar weeks.

us to include temperature (*Temp*) and *Urban*.[25] The available temperature data for England and Wales are mean monthly measures. To construct *Temp* we assume that the mean monthly temperature applies to each week in the month.[26][27] *Urban* is the percentage of the population that live in cities with a population >300,000. To calculate this variable, we use annual mid-year estimates of the populations of cities and assume that the resulting annual *Urban* percentage applies to each week in the year.[28] In terms of further explanatory variables such a population shares in the nine models for disaggregated all-cause mortalities, one could only rationalize including the corresponding disaggregated population share. Given *Temp* and *Urban* represent our sample of determinants that are cited as key in the literature, we retain only those shares that are significant (*Pop Share(<1); Pop Share(15–44)*; and *Pop Share(Female)*). Relevant descriptive statistics are provided in table 2.[29]

Second stage

In stage 2, each significant weekly out-of-sample estimate of excess deaths from stage 1 is decomposed into a refined estimate of COVID-19 excess deaths, and an estimate of the excess mortality impact of the lockdown. We do so by regressing the first stage excess deaths variable (comprising each out-of-sample observation minus the corresponding counterfactual prediction) on variables that reflect the COVID-19 and lockdown mortality periods. The dates of the COVID-19 and lockdown mortality periods in the models are based on variables that assume a time lag between infection and death. Based on the evidence described previously, we focus on a 4-week lag.

In each baseline second stage model, we use two dummy variables to obtain mean weekly estimates of our refined measure of COVID-19 excess deaths and the excess mortality impact of the lockdown. In the first such model, we use the *COVID4Week* dummy, which takes a value of 1 after 4 weeks since the first reported cases in York through to the end of our study period (week ending 6 March 2020 – week ending 28 August 2020); and zero for the first 5 weeks of our second stage sample period (week ending 31 January 2020– 28 February 2020).[30] In the same model, we include the *Lock4Week* dummy, which takes a value of 1 from when the first blanket lockdown was active for 4 weeks from its start on Monday 23 March 2020, through to 4 weeks after the end of the lockdown on 13 May 2020[31] (week ending 24 April 2020–12 June 2020); zero otherwise. We examine the sensitivity of our results to the infection-mortality lag by estimating further second stage models using dummies based on 2, 3 or 5 week lags (denoted *COVID2Week, Lock2Week*, etc.)

The baseline second stage models yield mean estimates of the refined measure of COVID-19 excess deaths and mean estimates of the excess mortality impact of the lockdown. To obtain

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[25]This led to the number of claimants of unemployment related benefit (an indicator of economic activity) being dropped from all the models (not significant or counterintuitive negative sign). Also, all the age category population shares were dropped from the *Total Deaths* model (reasons include insignificance and Stata dropping due to multicollinearity).

[26]We also explored accounting for the possibility of a non-linear 'U-shaped' relationship between temperature and each of our all-cause deaths variables by including $Temp^2$ in each model. The variance inflation factor for $Temp^2$ was always way in excess of 10, which is the 'rule of thumb' threshold that Curto and Pinto (2011) use as an indicator of multicollinearity. We therefore drop $Temp^2$ from all the models.

[27]Very high or very low temperatures can increase mortalities. Data on the maximum and minimum temperatures in a month in England and Wales is available from the Met Office. However, we omit such variables because we do not know which week in a month the maximum (minimum) applies to. This is less of a problem when we construct the *Temp* variable by assuming that the mean monthly temperature applies to each week in the month. This is because the temperature in each week will form part of the calculation of the mean monthly temperature. Also, as *Temp* comprises mean measures it will, to some extent, capture very high and very low temperatures.

[28]At the time, population data for cities and England and Wales was not available for 2020. We therefore construct the *Urban* variable by assuming that the percentage value of this variable for all the weeks in 2019 also applies to the weeks in 2020 that are part of our study period.

[29]Note, for the estimation period, the means of *Male* and *Female Deaths* in Table 2 do not sum exactly to the mean of *Total Deaths*. Over our estimation period, the range of the difference between *Total Deaths* and the sum of *Male* and *Female Deaths* is 0–25. As the ONS note, the male and female death counts may not sum to total deaths because of the recording of male and female deaths by age category; namely, the age category data does not include deaths where age is either missing or not yet fully coded.

[30]The error will pick up the excess deaths in these first 5 weeks. This is not an issue because it is assumed that COVID-19 will not be a cause of death in these weeks and it is COVID-19 deaths that we are interested in.

[31]We take the end of lockdown to be May 13th, as this was the date when the most strict lockdown measures were lifted. This allowed people to make unlimited trips outside of their home per day and to meet another person outside their household, while non-key workers were also urged to return to their workplace.

Table 2. Descriptive statistics.

Variable	Estimation period: week ending Jan 232,015–week ending Jan 242,020				Out-of-sample period: week ending Jan 312,020–week ending Aug 282,020			
	Mean	Std. Dev.	Min	Max	Mean	Std. Dev.	Min	Max
Total Deaths	10,194.46	1324.59	7131	15,050	11,804.45	3765.88	8690	22,351
Deaths(<1)	51.50	8.52	22	73	46.45	7.12	28	58
Deaths(1–14)	18.53	4.48	7	32	15.32	3.52	10	22
Deaths(15–44)	285.32	33.76	146	368	293.26	38.10	219	404
Deaths(45–64)	1207.03	117.13	773	1561	1398.90	333.20	1108	2294
Deaths(65–74)	1684.73	177.60	1185	2321	1913.23	512.81	1481	3380
Deaths(75–84)	2886.62	367.06	2013	4155	3408.77	1153.05	2506	6657
Deaths(85+)	4059.17	681.22	2922	6621	4728.42	1759.90	3178	9601
Male Deaths	5027.45	599.04	3461	7117	5971.55	1978.55	4365	11,445
Female Deaths	5165.44	737.70	3670	7933	5832.81	1799.68	4280	10,906
Temp	10.19	4.42	2.60	18.60	11.72	4.14	6.10	17.20
Urban (%)	25.12	0.06	25.01	25.17	25.17	0.00	25.17	25.17
Pop Share(<1)	0.012	0.0004	0.011	0.012	0.011	0.000	0.011	0.011
Pop Share(15–44)	0.383	0.0041	0.378	0.389	0.378	0.000	0.378	0.378
Pop Share(Female)	0.506	0.0004	0.506	0.507	0.506	0.000	0.506	0.506

Note: All the data is obtained from the ONS, with the exception of the *Temp* variable, which was obtained from the Met Office. There are 262 observations in the estimation period and 31 in the out-of-sample period.

(individual) weekly lockdown mortality estimates, we estimate further second stage regressions. This involves replacing each of the *Lock2Week – Lock5Week* dummy variables in the four baseline second stage models with their decomposition into weekly indicator variables, e.g. we replace *Lock4Week* with *LockApr24, LockMay1, . . ., LockJune12.*

V. Empirical results and discussion

Estimated first stage models and predicted counterfactuals

Table 3 presents, for the 5-year period that precedes the pandemic, the estimates of the Poisson model in Eq. 1 for *Total Deaths, Male Deaths* and *Female Deaths.* For the same period, Table 4 presents the estimates of the corresponding model for deaths in seven age categories.,[32][33] For all the models in these tables and all the second stage models, we use robust standard errors to account for serial correlation (Brännäs and Johansson 1994).

From Tables 3 and 4, we note that a number of the reported coefficients on the weekly dummies are significant, which provides some insight into the substantial collective explanatory power of these 51 weekly variables. The coefficients on *Temp* and

Table 3. Total, male and female first stage models (week ending 23 January 2015 – week ending 24 January 2020).

Variable	Total Deaths	Male Deaths	Female Deaths
Temp	−0.011***	−0.009***	−0.013***
Urban	0.112*	0.255***	1.321**
Pop Share(Female)			223.639**
Week 13	−0.162***	−0.135***	−0.189***
Week 14	−0.124**	−0.095*	−0.153***
Week 15	−0.103	−0.069	−0.137*
Week 16	−0.106*	−0.073	−0.139**
Week 17	−0.109**	−0.074*	−0.144***
Week 18	−0.124**	−0.085	−0.164***
Week 19	−0.161**	−0.127*	−0.197***
Week 20	−0.099**	−0.062	−0.137***
Log-likelihood	−4706.9	−2775.8	−3315.1

Notes: *, ** and *** denote statistical significance at the 5%, 1% and 0.1% levels, respectively. For brevity we only report parameters for selected weekly dummy variables in the year.

Urban are significant in a number of models in these tables. The expected sign of a coefficient on *Temp* is ambiguous and, in line with this, we observe positive and negative coefficients. All but one of the significant coefficients on *Temp* is negative, which indicates that where the relationship is significant, we often find that a decrease in *Temp* is associated with a rise in mortalities. All but one of the significant coefficients on *Urban* is positive, where these positive estimates may be pointing to higher mortalities in urban areas as they are more densely populated. Alternatively, it is conceivable that a significant coefficient on *Urban* is negative, which is what we observe from the *Deaths(1–14)*

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[32]For conciseness, the tables report the parameters for selected weekly dummy variables in the year.

[33]In terms of the diagnostic checks of the models in Tables 3 and 4, we use a Cook distance plot based on the corresponding OLS model specification as well as QQ-plots. For all the observations of the 10 dependent variables, the Cook distance is below a threshold of 1 (and for a small number of observations this distance marginally exceeds an alternative lower threshold of 4/262), which suggests that there are no clear outliers. The QQ-plots show that there are slight deviations from normality in the upper and lower tails of the error distributions from the Poisson models. These deviations are relatively minor and do not suggest that we need to transform any of the dependent variables. The plots are available from the corresponding author on request.

Table 4. Age category first stage models (week ending 23 January 2015 – week ending 24 January 2020).

	Deaths by Age Category						
Variable	<1	1–14	15–44	45–64	65–74	75–84	85+
Temp	0.016*	0.013	0.001	0.00039	−0.004	−0.013***	−0.017***
Urban	0.029	−0.474*	0.591*	0.204***	0.260***	0.004	0.121
Pop Share(<1)	75.541*						
Pop Share(15–44)			8.826*				
Week 13	−0.059	0.061	0.219**	−0.084	−0.143***	−0.159***	−0.213***
Week 14	−0.093	−0.018	0.177	−0.069	−0.112*	−0.132**	−0.150**
Week 15	−0.113	0.149	0.241**	−0.045	−0.073	−0.115	−0.139*
Week 16	0.044	0.177**	0.262**	−0.019	−0.094	−0.110*	−0.151**
Week 17	−0.037	0.026	0.288***	−0.01	−0.088*	−0.114**	−0.162***
Week 18	−0.125	0.077	0.275**	−0.067	−0.120*	−0.118*	−0.167***
Week 19	−0.217*	0.057	0.181	−0.11	−0.146*	−0.160**	−0.198***
Week 20	−0.15	0.036	0.297***	−0.013	−0.078	−0.106**	−0.147***
Log-likelihood	−886.0	−726.7	−1191.7	−1533.7	−1670.4	−2206.7	−3556.4

Notes: *, ** and *** denote statistical significance at the 5%, 1% and 0.1% levels, respectively. For brevity we only report parameters for selected weekly dummy variables in the year.

Table 5. Predicted out-of-sample first stage excess deaths.

	Observed W/E 31 January 2020– W/E 28 August 2020		Counterfactual W/E 31 January 2020 – W/E 28 August 2020		First stage excess deaths (observed-counterfactual)	
Variable	Average	SE	Average	SE	Average	SE
Total Deaths	11,804.5	3765.9	10,042.0	1014.9	1762.4	700.5
Male Deaths	5971.5	1978.6	5002.1	454.8	969.5	355.4
Female Deaths	5832.8	1799.7	4972.4	553.9	860.4	323.2
<1	46.5	7.1	49.0	3.7	−2.6	1.4
1–14	15.3	3.5	18.1	2.2	−2.7	0.7
15–44	293.3	38.1	281.3	18.1	11.9	7.6
45–64	1398.9	333.2	1212.6	86.8	186.3	61.8
65–74	1913.2	512.8	1685.5	120.9	227.7	94.6
75–84	3408.8	1153.1	2825.9	275.6	582.9	212.9
85+	4728.4	1759.9	3963.4	525.5	765.0	329.9

Note: SE denotes standard error.

model and may be due to better access to paediatric healthcare in urban areas.

In Table 5, we present for the 10 all-cause models: average weekly observed deaths over the out-of-sample period; average weekly predicted counterfactual deaths over the same period; and average first stage excess deaths. We find that, on average,

the following weekly first stage excess deaths are significantly greater than zero at the 2.5% level: *Total Deaths; Male Deaths; Female Deaths; Deaths (45–64); Deaths(65–74); Deaths(75–84);* and *Deaths(85+).* In each of these cases this indicates that the observed deaths are, on average, significantly greater than the counterfactual deaths.

Figure 1 shows a time series of observed total deaths over the estimation period; and the observed and predicted counterfactual total deaths over the out-of-sample period. A visual comparison of the relevant portions of this figure suggests that our predicted counterfactual deaths do not look out of line with observed deaths for the same periods in previous years.

Estimated second stage models

Tables 6–9 present (for the out-of-sample period) the estimates of the second stage baseline and lockdown indicator models (Eqs. 2 and 3) for the first

Figure 1. First stage observed and counterfactual total deaths.

Table 6. Second stage models for excess *Total Deaths* (first stage).

	Dependent variable: excess total deaths from the first stage							
	Baseline models				Lockdown indicator models			
Variable	2 weeks	3 weeks	4 weeks	5 weeks	2 weeks	3 weeks	4 weeks	5 weeks
COVID2Week	276.2				276.2			
Lock2Week	6097.68***							
COVID3Week		712.79				712.79		
Lock3Week		4732.71***						
COVID4Week			1411.89*				1411.89*	
Lock4Week			2601.11*					
COVID5Week				2225.88**				2225.88**
Lock5Week				316.74				
Lock10Apr					7835.80***			
Lock17Apr					11,697.80***	11,261.21***		
Lock24Apr					11,377.80***	10,941.21***	10,242.11***	
Lock1May					7721.80***	7285.21***	6586.11***	5772.12***
Lock8May					2791.80***	2355.21***	1656.11**	842.12
Lock15May					4091.80***	3655.21***	2956.11***	2142.12**
Lock22May					2054.80***	1618.21***	919.11	105.12
Lock29May					1209.80***	773.21	74.11	−739.88
Lock5June						−27.79	−726.89	−1540.88
Lock12June							−897.89	−1711.88*
Lock19June								−2334.88**
Log-likelihood	−283.87	−290.03	−294.55	−295.86	−263.22	−276.32	−288.3	−293.55

Note: *, ** and *** denote statistical significance at the 10%, 5% and 1% levels, respectively.

Table 7. Second stage models for excess *Male Deaths* (first stage).

	Dependent variable: excess male deaths from the first stage							
	Baseline models				Lockdown indicator models			
Variable	2 weeks	3 weeks	4 weeks	5 weeks	2 weeks	3 weeks	4 weeks	5 weeks
COVID2Week	223.35				223.35			
Lock2Week	3064.40***							
COVID3Week		475.26				475.26		
Lock3Week		2256.86***						
COVID4Week			841.94*				841.94*	
Lock4Week			1131.18					
COVID5Week				1263.94**				1263.94**
Lock5Week				−50.94				
Lock10Apr					4545.65***			
Lock17Apr					6059.65***	5807.74***		
Lock24Apr					5850.65***	5598.74***	5232.06***	
Lock1May					3629.65***	3377.74***	3011.06***	2589.06***
Lock8May					1185.65***	933.74***	567.06	145.06
Lock15May					1900.65***	1648.74***	1282.06***	860.06
Lock22May					877.65***	625.74**	259.06	−162.94
Lock29May					465.65**	213.74	−152.94	−574.94
Lock5June						−151.26	−517.94	−939.94*
Lock12June							−630.94	−1052.94*
Lock19June								−1270.94**
Log-likelihood	−265.63	−271.56	−275.3	−276.1	−246.49	−259.55	−269.59	−274.2

Note: *, ** and *** denote statistical significance at the 10%, 5% and 1% levels, respectively.

stage excess measures of *Total Deaths, Male Deaths, Female Deaths* and *Deaths(85+)*. Tables A1-A3 (see Appendix) report the estimates of the corresponding models for the first stage excess measures of *Deaths(45–64), Deaths(65–74)* and *Deaths(75–84)*. Note, the coefficients in these tables represent excess mortalities.

The three key findings from the results in Tables 6–9 are as follows. First, from our preferred baseline model (4-week infection-death lag) we find that: (i) COVID-19 excess deaths amount to 63% of the corresponding standard 5-year average excess deaths; and (ii) in net terms, the lockdown increased all-cause mortalities (associated with an incremental 2,601 excess mortalities per week; see coefficient on *Lock4Week*). For completeness, the aforementioned tables also show the results for our sensitivity analysis using lags ranging from 2 to 5 weeks. From this we see that, as the assumed infection-death lag increases, there is a marked decline in the magnitude of the average weekly estimate of the excess mortality impact of the lockdown; accompanied by this

Table 8. Second stage models for excess *Female Deaths* (first stage).

Variable	Baseline models				Lockdown indicator models			
	2 weeks	3 weeks	4 weeks	5 weeks	2 weeks	3 weeks	4 weeks	5 weeks
COVID2Week	118.45				118.45			
Lock2Week	3033.43***							
COVID3Week		302.42				302.42		
Lock3Week		2476.45***						
COVID4Week			634.50*				634.50*	
Lock4Week			1469.88**					
COVID5Week				1026.12**				1026.12**
Lock5Week				367.13				
Lock10Apr					3293.55***			
Lock17Apr					5641.55***	5457.58***		
Lock24Apr					5529.55***	5345.58***	5013.50***	
Lock1May					4094.55***	3910.58***	3578.50***	3186.88***
Lock8May					1603.55***	1419.58***	1087.50***	695.88
Lock15May					2191.55***	2007.58***	1675.50***	1283.88***
Lock22May					1179.55***	995.58***	663.50*	271.88
Lock29May					733.55***	549.58**	217.5	−174.12
Lock5June						125.58	−206.5	−598.12
Lock12June							−270.5	−662.12
Lock19June								−1067.12**
Log-likelihood	−259.3	−265.44	−270.87	−272.78	−236.62	−249.48	−264	−269.96

Note: *, ** and *** denote statistical significance at the 10%, 5% and 1% levels, respectively.

impact becoming generally less significant (and vice-versa).[34] Second, although it has been widely reported that COVID-19 deaths have been concentrated in the elderly, we find that such deaths have been focused in the *very* elderly (75–84 and 85+). Third, from the lockdown indicator models,[35] we can see that there are a small number of cases where an indicator parameter is negative and significant. Specifically, we can see from these models that the *Lock19June* parameters, and a number of those pertaining to *Lock12June* and *Lock5June*, are negative and significant This suggests that in net terms the lockdown only began to save lives 10–12 weeks after the policy was implemented on 23 March 2020 (or, alternatively, 3–5 weeks after the policy ended on 13 May 2020).

The implication of our results is that, at best, lockdown had no significant impact on net

mortalities; and under our preferred 4-week lag model (and 3-week lag model, also well supported by the lag evidence) is associated with a significant net increase in mortalities. As the net impact of the lockdown is a function of both: (i) its efficacy regarding its intended effects; and (ii) the possibility of offsetting effects, in the following we consider other evidence relating to these effects.

In relation to the efficacy of the lockdown, firstly, we note that existing studies provide mixed evidence on the impact of NPIs (and lockdowns specifically) on mortalities. Whilst some report lockdowns reducing mortalities,[36] others find no such impact.[37] Secondly, our data findings are highly consistent with the first lockdown in England and Wales occurring *after* the point at which it could have mitigated peak COVID-19 infections (and thus deaths).[38] This is therefore inconsistent with a no-lockdown counterfactual under which COVID-19 deaths would have

[34]For example, the coefficient on *Lock5Week* is not significant in all the relevant models in Tables 6–9 and A1-A3.
[35]Which in each case yield, as they should, indicator parameters that when averaged equal the coefficient on the lockdown dummy in the corresponding baseline model.
[36]Cho (2020) and Born, Dietrich, and Müller (2020) suggest that a blanket lockdown in Sweden would have reduced excess mortality. Conyon, He, and Thomsen (2020) find that the stringent lockdowns in Denmark and Norway, vis-à-vis Sweden, led to a reduction in mortality rates. Ciminelli and Garcia-Mandicó (2020) find that the shutdown of service sector activities in Italian municipalities was effective in reducing mortalities.
[37]Chaudhry et al. (2020) conduct a cross-country analysis of the 50 countries with the most recorded COVID-19 cases. They find that full lockdowns are not associated with significant reductions in critical cases or overall mortality. Gibson (2020) finds that lockdowns in New Zealand did not reduce COVID-19 mortalities, while Li et al. (2020) find mixed results on the impact of eight NPIs on the R (where two of the NPIs are stay at home orders and restrictions on internal movement). For a sample of 131 countries, Li *et al.* find that only relaxation of school closures and bans on public events and gatherings of more than ten people have a significant impact on the R.
[38]Specifically, COVID-19 deaths by date of occurrence peaked on 8 April 2020, only 16 days after the commencement of the lockdown. Thus, even if a shorter lag of 3 weeks between infection and death is assumed, it would not be possible for the lockdown to have mitigated this peak.

Table 9. Second stage models for excess *Deaths(85+)* (first stage).

	Dependent variable: excess deaths (85+ years) from the first stage							
	Baseline models				Lockdown indicator models			
Variable	2 weeks	3 weeks	4 weeks	5 weeks	2 weeks	3 weeks	4 weeks	5 weeks
COVID2Week	20.3				20.3			
Lock2Week	3008.70***							
COVID3Week		200.26				200.26		
Lock3Week		2445.99***						
COVID4Week			519.11				519.11	
Lock4Week			1460.14**					
COVID5Week				901.76*				901.76*
Lock5Week				378.36				
Lock10Apr					3300.70***			
Lock17Apr					5489.70***	5309.74***		
Lock24Apr					5426.70***	5246.74***	4927.89***	
Lock1May					4004.70***	3824.74***	3505.89***	3123.24***
Lock8May					1659.70***	1479.74***	1160.89***	778.24
Lock15May					2230.70***	2050.74***	1731.89***	1349.24***
Lock22May					1206.70***	1026.74***	707.89*	325.24
Lock29May					750.70***	570.74***	251.89	−130.76
Lock5June						58.74	−260.11	−642.76
Lock12June							−345.11	−727.76
Lock19June								−1047.76**
Log-likelihood	−258.07	−264.74	−270.32	−272.3	−235.13	−249.03	−263.37	−269.4

Note: *, ** and *** denote statistical significance at the 10%, 5% and 1% levels, respectively.

continued to rise.[39] This finding is supported by Wood (2021). Thirdly, COVID-19 deaths and infections are highly concentrated in certain locations. Up to the start of 2021, 27% of COVID-19 deaths in England and Wales occurred in care homes or hospices.[40] It is intuitively doubtful therefore that blanket restrictions on the movement of the wider population could have materially affected COVID-19 infections/mortalities in these settings (meaning that a substantial proportion of COVID-19 deaths may be largely invariant to blanket lockdowns).[41] Collectively, these points provide grounds to suppose that the efficacy of the first national lockdown in England and Wales in reducing COVID-19 deaths was limited, and should not be presumed.

Turning to offsetting increases in other mortalities, a range of evidence supports this arising in practice. Figure 2 contains ONS data showing non-COVID deaths relative to the historical 5-year average (i.e. non-COVID-excess deaths). This reveals large and rapid increases in said deaths that only become materially excess after the beginning of lockdown (week ending 27 March 2020).

Relatedly, Griffin (2020) reports that further ONS data shows that up to the week ending 1 May 2020 (where deaths were registered up to May 9th), only a third of excess deaths in the community (i.e. other than in hospitals) were due to COVID-19. In support of this, for the period 28 December 2019–11 September 2020, the ONS (2020b) report that most of the 27,096 excess deaths in private homes in England and Wales did not involve COVID-19.

There is a possibility that some of the non-COVID excess deaths (above) may be under-diagnosed COVID-19 deaths. However, other research suggests this is unlikely. Pell et al. (2020) provide interesting evidence from coronial autopsies at the John Radcliffe Hospital (Oxford) over the first two months of the lockdown (i.e. from 23 March 2020). Of the 67 autopsies performed at the hospital pertaining to sudden natural deaths, the autopsy reports indicate that only 2 deaths (3%) were undiagnosed COVID-19. Reduced access to healthcare systems associated with the lockdown was recorded as a probable contributory factor to 6 deaths (9%), and a possible contributory factor to a further 8 deaths (12%). These 14

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[39]This is important, because lockdowns only 'spread out' infections (and thus deaths). Hence, the primary means through which they can reduce mortality is by 'flattening the curve' to prevent healthcare services being overwhelmed. For that hypothetical counterfactual to be plausible, the evidence would need to show that infections were rising rapidly at the time of the policy intervention. More generally, given the importance of the timing of infection peaks for policy intervention, see Li and Linton (2021) for predictions of these peaks for the 30 countries with the largest number of cases (excluding China).

[40]ONS weekly data from 28 December 2019 to 1 January 2021.

[41]Specifically, mortality rates in care homes are more likely determined by care home policy including: visitations; staff and resident testing; and PPE equipment provision. Relatedly, it should be noted that many care homes unilaterally banned visits prior to the lockdown (BBC, 2021).
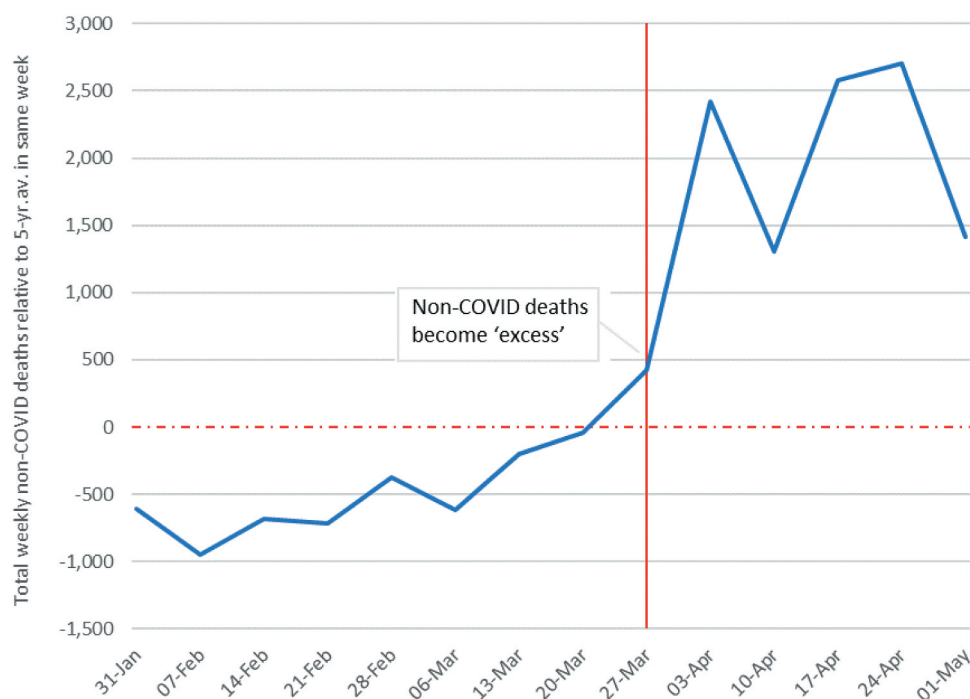
Figure 2. ONS non-COVID excess deaths data.

cases included preventable out-of-hospital deaths, such as acute myocardial infarction and diabetic ketoacidosis, where patients contacted the health services by telephone and were advised to self-isolate at home, rather than attending hospital. The authors also note that there was an increase in deaths from drug and alcohol misuse during the lockdown period.

In light of the above, as the 4 week lag baseline model for excess *Total Deaths* we favour indicates that the lockdown is associated with a net increase in excess mortalities, we posit that we have uncovered an important new case of the well-known Peltzman (1975) offsetting effect. This effect arises because people adjust their behaviour to changes in the perceived level of risk. This leads to the intended effect of a preventative intervention to reduce risk being (more than) offset by the unintended consequences of peoples' (over)compensating riskier behaviour. In this case, this conceivably arises because lockdown may have distorted people's perception of COVID-19 risk, relative to other health conditions, potentially leading them: not to seek help (or not as urgently), causing additional mortalities. Such 'behavioural change' in peoples' decisions on seeking healthcare for non-COVID-19 illnesses is consistent with what we observe from Figure 3, which shows monthly time series of A&E attendance and emergency admissions

in England. This figure reveals a notable change in peoples' choices about seeking healthcare during the lockdown period, as A&E attendance and emergency admissions collapsed despite A&E remaining open, with the sum of the two for April 2020 being 53% lower than that for the same month in 2019.

Peltzman offsetting effects have been observed in relation to previous safety/health interventions, including (i) New automobile safety regulations (e.g. mandatory installation of front seat belts) in the U.S. leading to riskier driving, meaning they had no overall impact on fatalities (Peltzman 1975). (ii) Air bags in cars in the U.S. also leading to riskier driving (Peterson, Hoffer, and Millner 1995). (iii) Higher state cigarette taxes in the U.S. leading to greater health risks through a rise in the consumption of cigarettes with higher tar and nicotine content (Evans and Farrelly 1998). (iv) Higher state soft drink taxes in the U.S. leading to moderate reductions in soda consumption by children, offset by increases in their consumption of other high-calorie drinks (Fletcher, Frisvold, and Tefft 2010). In light of this, our finding that lockdown had offsetting impacts should not be regarded as being in any way unusual.
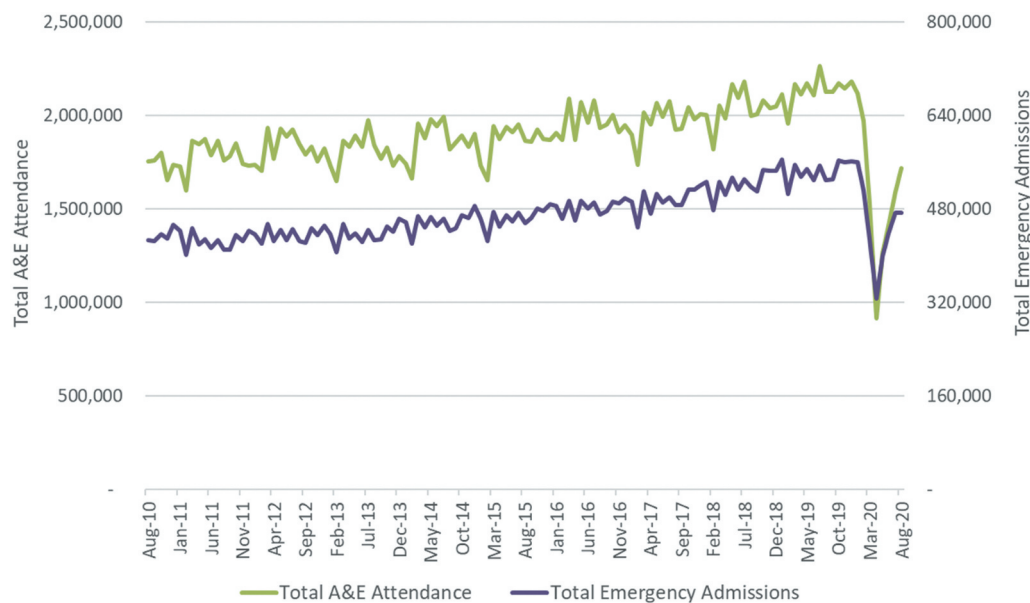
Figure 3. A&E attendance and emergency admissions in England.

VI. Concluding remarks

The two-stage method proposed here focuses on addressing an omitted variables problem with a standard excess deaths approach; namely, a failure to account for policy, and other, impacts.

Our preferred baseline model indicates that the first national lockdown in England and Wales had a net mortality increasing effect. We postulate that this is likely a function of both low efficacy in the lockdown meeting its intended aim, combined with the presence of the Peltzman offsetting effect. Wider evidence supports this proposition.

Our empirical results should only be used to draw inferences about the excess mortality impact of the first blanket lockdown in England and Wales. Impacts of individual policy interventions should be evaluated case-by-case.

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Appendix

Table A1: Second stage models for excess *Deaths(45–64)* (first stage)

| | Dependent variable: excess deaths (45–64 years) from the first stage | | | | | | | |
| | Baseline models | | | | Lockdown indicator models | | | |
Variable	2 weeks	3 weeks	4 weeks	5 weeks	2 weeks	3 weeks	4 weeks	5 weeks
COVID2Week	65.15*				65.15*			
Lock2Week	505.35***							
COVID3Week		113.58**				113.58**		
Lock3Week		357.80**						
COVID4Week			174.33**				174.33**	
Lock4Week			177.54					
COVID5Week				245.71***				245.71***
Lock5Week				−15.71				
Lock10Apr					812.85***			
Lock17Apr					964.85***	916.42***		
Lock24Apr					941.85***	893.42***	832.67***	
Lock1May					624.85***	576.42***	515.67***	444.29***
Lock8May					149.85***	101.42*	40.67	−30.71
Lock15May					303.85***	255.42***	194.67**	123.29
Lock22May					188.85***	140.42**	79.67	8.29
Lock29May					55.85	7.42	−53.33	−124.71
Lock5June						−28.58	−89.33	−160.71*
Lock12June							−100.33	−171.71*
Lock19June								−213.71**
Log-likelihood	−210.57	−216.46	−219.67	−220.18	−192.92	−205.88	−214.17	−218.16

Note: *, ** and *** denote statistical significance at the 10%, 5% and 1% levels, respectively.

Table A2: Second stage models for excess *Deaths(65–74)* (first stage)

| | Dependent variable: excess deaths (65–74 years) from the first stage | | | | | | | |
| | Baseline models | | | | Lockdown indicator models | | | |
Variable	2 weeks	3 weeks	4 weeks	5 weeks	2 weeks	3 weeks	4 weeks	5 weeks
COVID2Week	46.35				46.35			
Lock2Week	742.15***							
COVID3Week		110				110		
Lock3Week		542.50**						
COVID4Week			205.39*				205.39*	
Lock4Week			249.74					
COVID5Week				310.76**				310.76**
Lock5Week				−41.01				
Lock10Apr					1135.65***			
Lock17Apr					1606.65***	1543.00***		
Lock24Apr					1452.65***	1389.00***	1293.61***	
Lock1May					886.65***	823.00***	727.61***	622.24***
Lock8May					263.65***	200.00**	104.61	−0.76
Lock15May					402.65***	339.00***	243.61**	138.24
Lock22May					115.65**	52	−43.39	−148.76
Lock29May					73.65	10	−85.39	−190.76
Lock5June						−16	−111.39	−216.76
Lock12June							−131.39	−236.76
Lock19June								−294.76**
Log-likelihood	−225.45	−230.29	−233.59	−234.05	−207.1	−218.17	−228.39	−232.45

Note: *, ** and *** denote statistical significance at the 10%, 5% and 1% levels, respectively.

Table A3: Second stage models for excess *Deaths(75–84)* (first stage)

| | Dependent variable: excess deaths (75–84 years) from the first stage | | | | | | | |
| | Baseline models | | | | Lockdown indicator models | | | |
Variable	2 weeks	3 weeks	4 weeks	5 weeks	2 weeks	3 weeks	4 weeks	5 weeks
COVID2Week	144.95				144.95			
Lock2Week	1821.55***							
COVID3Week		287.74				287.74		
Lock3Week		1376.89***						
COVID4Week			511.06*				511.06*	
Lock4Week			710.94					
COVID5Week				759.29**				759.29**
Lock5Week				15.58				
Lock10Apr					2559.05***			
Lock17Apr					3589.05***	3446.26***		
Lock24Apr					3457.05***	3314.26***	3090.94***	
Lock1May					2174.05***	2031.26***	1807.94***	1559.71***
Lock8May					774.05***	631.26***	407.94	159.71
Lock15May					1163.05***	1020.26***	796.94***	548.71*
Lock22May					523.05***	380.26**	156.94	−91.29
Lock29May					333.05***	190.26	−33.06	−281.29
Lock5June						1.26	−222.06	−470.29
Lock12June							−318.06	−566.29*
Lock19June								−734.29**
Log-likelihood	−248.58	−254.39	−258.35	−259.33	−229.34	−241.95	−252.59	−257.39

Note: *, ** and *** denote statistical significance at the 10%, 5% and 1% levels, respectively.