

**Finance and Economics Discussion Series  
Divisions of Research & Statistics and Monetary Affairs  
Federal Reserve Board, Washington, D.C.**

**Supply-Side Effects of Pandemic Mortality: Insights from an  
Overlapping-Generations Model**

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**2020-060**

Please cite this paper as:

Gagnon, Etienne, Benjamin Johannsen, and David López-Salido (2020). "Supply-Side Effects of Pandemic Mortality: Insights from an Overlapping-Generations Model," Finance and Economics Discussion Series 2020-060. Washington: Board of Governors of the Federal Reserve System, <https://doi.org/10.17016/FEDS.2020.060>.

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# Supply-Side Effects of Pandemic Mortality: Insights from an Overlapping-Generations Model

Etienne Gagnon, Benjamin Johannsen, and David López-Salido\*

July 7, 2020

## Abstract

We use an overlapping-generations model to explore the implications of mortality during pandemics for the economy's productive capacity. Under current epidemiological projections for the progression of COVID-19, our model suggests that mortality will have, in itself, at most small effects on output and factor prices. The reason is that projected mortality is small in proportion to the population and skewed toward individuals who are retired from the labor force. That said, we show that if the spread of COVID-19 is not contained, or if the ongoing pandemic were to follow a mortality pattern similar to the 1918–1920 Great Influenza pandemic, then the effects on the productive capacity would be economically significant and persist for decades.

**JEL Codes:** E21, E27, E43.

**Keywords:** Pandemics, potential output, real wage, equilibrium real interest rate, demographics.

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# 1 Introduction

As of this writing, there are over 2.5 million confirmed cases and 125,000 deaths attributed to COVID-19 in the United States, with both counts rising steadily.<sup>1</sup> The Institute for Health Metrics and Evaluation (IHME) at the University of Washington projects 200,000 pandemic-related deaths by the fall. Although several communities have reported progress in slowing the progression of the virus, many others have not, with several states registering records numbers of daily new cases. Health experts warn that the number of deaths and cases will continue to rise in the foreseeable future because of the absence of a vaccine, effective antiviral drugs, and mass testing. On the economic front, the mandatory and voluntary social distancing measures enacted to contain the pandemic have led to a deep recession, with the unemployment rate soaring a record-shattering 10 percentage points in April 2020, to about 15 percent. Although most observers expect economic activity to rebound in the second half of this year, many jobs and businesses are unlikely to survive the downturn, especially if the public lacks confidence to resume normal activities.

In this paper, we use a general-equilibrium, overlapping-generations model to explore some implications of COVID-19 mortality for the economy’s productive capacity. Our analysis is motivated by economic research suggesting that past pandemics had significant macroeconomic effects—notably by depressing output, lowering interest rates, and raising real wages—that sometimes persisted for decades.<sup>2</sup> Our model and its calibration are the same as in Gagnon et al. (2016). This model allows us to target mortality rates by age that are consistent with epidemiological estimates showing heavy mortality from COVID-19 among older infected individuals. The model also accounts for the fact that employment varies by age and birth cohort, allowing us to track the effects of mortality on the aggregate labor supply. Furthermore, the model’s life-cycle elements allow us to investigate how the stock of capital endogenously adjusts, through individual consumption/saving decisions, when worker mortality causes an unexpected fall in the labor supply. In the decentralized equilibrium of our model, this adjustment operates through a rise in real wages and a fall in interest rates, as suggested in empirical studies of past pandemics. It bears emphasis that our model only speaks to the direct effects of pandemic-related mortality on the economy’s productive capacity and, thus, is not designed to address the cyclical downturn brought by the pandemic or the effectiveness of the economic policy response.<sup>3</sup>

Overall, our model simulations suggest that, under current epidemiological projections, the direct effects of COVID-19 mortality on the economy’s productive capacity are small. This conclusion

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<sup>1</sup>These statistics are from the COVID-19 Case Tracker web site maintained by the Center for Systems Science and Engineering at the John Hopkins University, which provides real-time tracking of various official statistics on COVID-19. The statistics were retrieved on June 23, 2020 from the web site <https://coronavirus.jhu.edu/>.

<sup>2</sup>See Jordà et al. (2020) for time-series evidence of long-lasting effects based on a multi-country panel of 12 past pandemics. See also Clark (2007) for evidence of persistently higher real wages in the decades that followed the 14th century Black Plague.

<sup>3</sup>The literature on the cyclical and policy aspects of the pandemic-driven recession is growing rapidly. See, among several contributors, Guerrieri et al. (2020) and Goolsbee and Syverson (2020) on the nature of the economic shock; Eichenbaum et al. (2020) and Bodenstein et al. (2020) on the macroeconomic effects of social distancing; Coibion et al. (2020) and Cajner et al. (2020) on the labor market slump; Carroll et al. (2020) and Chetty et al. (2020) on consumption behavior; and Elenev et al. (2020) and Brunnermeier and Krishnamurthy (2020) on the policy response.

reflects both the limited number of projected fatalities in proportion to the U.S. population and their concentration among senior citizens who have already exited the labor force and are past their reproductive years. Our finding that the effects on the productive capacity of the economy are small may be useful to modelers who study the cyclical implications of COVID-19 because it implies that the longer-run values of economic variables around which models are often linearized may be largely unaffected by the pandemic. We want to emphasize that our analysis in no way implies that the economic and social effects of COVID-19 are small. The human toll that COVID-19 has already taken is heartbreaking. Additionally, the cyclical economic effects of COVID-19 have already caused dramatic rises in unemployment and declines in consumer spending and output.

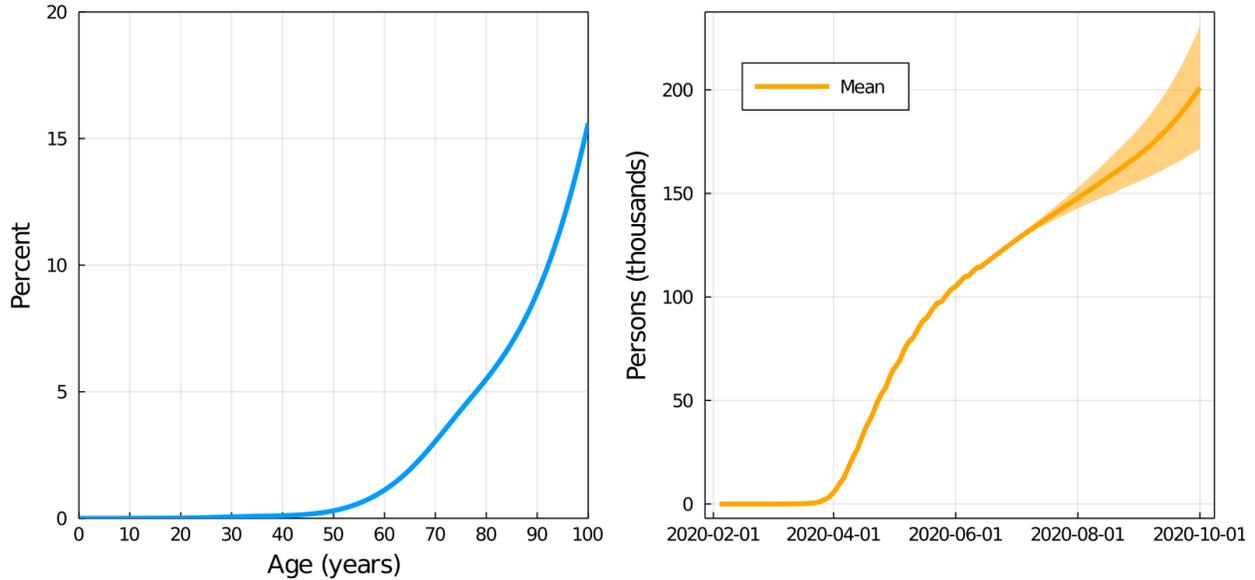
For comparison, and to highlight the economic transmission channels of pandemic mortality, we simulate the model under two alternative scenarios that lead to significantly greater mortality overall. In the first alternative scenario, we assume that COVID-19 infections soar until the number of cases is sufficient to ensure herd immunity, which many experts estimate to be around 70 percent of the population. In the second alternative scenario, we assume that the overall deadliness of the virus and the age distribution of fatalities are consistent with mortality during the Great Influenza pandemic of 1918 to 1920. In contrast to COVID-19, the Great Influenza was most lethal for young adults. Both alternative scenarios lead to economically significant and persistent declines in output in the model, along with increases in real wages and declines in real interest rates. Under the COVID-19 alternative scenario leading to herd immunity, the size of the population and labor supply eventually return to their pre-pandemic trends because the fatalities occur almost entirely among older individuals who are past their reproductive period. By contrast, under the Great Influenza alternative scenario, for which mortality affects young adults during their reproductive years, the model predicts that output would be on a permanently lower trajectory and that the adjustment of factors prices would drag out beyond the next couple of decades.

The paper is organized as follows. Section 2 reviews how we constructed mortality rates by age for COVID-19 and the Great Influenza pandemics. Section 3 discusses the key transmission channels in our model, presents our main simulation results, and discusses some limitations. Section 4 summarizes our key findings and conclusions.

## 2 Mortality assumptions

The key driving factor in our model simulation is the excess mortality associated with pandemics and how it is distributed across age groups. The unconditional probability that a person will die because of a pandemic (henceforth the “excess mortality rate”) can be expressed as the product of the probability that this person will be infected by the virus (known as the “attack rate” in epidemiological studies) and the deadliness of the virus conditional on being infected (known as the “infection fatality rate”). Because of data and testing limitations, estimates of the attack rate and infection fatality rate of COVID-19 remain imprecise even though millions of cases have been identified in the United States and abroad. Nonetheless, it is apparent that the virus is much

Figure 1: Baseline mortality assumptions for COVID-19 pandemic  
 Infection fatality rate                      Total projected deaths



**Source:** Authors’ interpolation of estimates from Verity et al. (2020); Institute for Health Metrics and Evaluation (IHME) – University of Washington.

**Notes:** In the right panel, the shaded area around the mean represents the projection’s 95-percent uncertainty band. The IHME projection for total U.S. deaths attributable to COVID-19 is dated June 13, 2020, and uses smoothed data.

more lethal for older individuals than for younger ones, in contrast with mortality during the Great Influenza, which, as we show below, was highest among young adults.

## 2.1 Mortality from COVID-19

The left panel of Figure 1 shows the baseline infection fatality rate, by age, that we use in our simulations. This rate is an interpolation of estimates by age deciles from Verity et al. (2020), who control for under-reporting (the possibility that mild cases are often not diagnosed), censoring (the possibility that infections and deaths are reported with a lag), and how these factors may vary with age.<sup>4</sup> The infection fatality rate is almost nil for children and young adults, but then rises rapidly with age as individuals reach their 50s and beyond. Among individuals aged 80 years or more, the overall infection fatality rate estimated by Verity et al. (2020) is 7.8 percent.

In our simulations, we posit that all individuals face the same risk of becoming infected, which Verity et al. (2020) argue is consistent with past studies showing that respiratory infections do not vary substantially by age. We target an attack rate that is consistent with COVID-19 causing 200,000 deaths across all age groups. As the right panel of Figure 1 shows, this target mortality is consistent with the mean total mortality predicted by the IHME through October 1, 2020 (the final period in the IHME projection). This predicted mortality is conditioned on an assumed infection

<sup>4</sup>Our interpolation uses cubic splines.

fatality rate by age that is similar to that estimated by Verity et al. (2020), along with assumptions about social distancing measures, other health policies, and their effects on the transmission of the virus and mortality. The attack rate consistent with 200,000 fatalities is about 5.5 percent of the U.S. population.

Whether the latter assumption understates or overstates the ultimate number of casualties is highly uncertain at this stage. On the one hand, many U.S. states (such as New York and New Jersey) and countries (such as China, South Korea, and Western European countries) that faced initially large caseloads have greatly reduced the number of new infections. Moreover, health authorities and the public have adapted their policies and behavior in ways that slow the propagation of the virus. Even so, continued U.S. and global transmission, resistance by part of public to remediation measures, and the lack of effective vaccines and therapeutics suggest a material possibility that the number of infections could rise substantially. Indeed, our assumed attack rate of 5.9 percent is well below the range of immunization rates (that is, the fraction of the population protected through past infection or vaccination, when available) that experts believe is required to ensure herd immunity. Because of this potential for greater mortality than current projected, and to illustrate the economic mechanisms of pandemics, we will consider the alternative assumption that 70 percent of the U.S. population ultimately contracts the virus. This proportion is a common estimate of the immunization rate needed to ensure herd immunity.<sup>5</sup> Finally, we note that little is known regarding the longer-run consequences of COVID-19 infection for survivors at this stage because the virus is so new. However, evidence is mounting that a small proportion of those infected may subsequently suffer from various chronic ailments.<sup>6</sup> In our simulations, we assume that survivors enjoy the same labor supply and mortality risk over the remainder of their lives as those of the same birth cohort who did not become infected. To the extent that long-term complications emerge, then our model may understate the negative effects of COVID-19 on the labor supply for a given mortality rate.

## 2.2 Mortality from the Great Influenza

The Great Influenza afflicted the U.S. population in waves between the spring of 1918 and the spring of 1920, with the wave in the fall of 1918 being the deadliest and most widely spread geographically.<sup>7</sup> Retrospective estimates of this pandemic’s attack rate and infection fatality rate are highly imprecise due to the absence of testing methods at the time. However, information on the number of deaths related to the pandemic is sufficiently detailed to yield a reasonable approximation of the associated excess mortality by age, which is the key ingredient in our simulations. In particular, we estimate the excess mortality rate of the Great Influenza by assembling a dataset of annual mortality statistics, by age bin and cause of death, using the annual mortality tables published by the U.S. Bureau of the Census. We calculate the number of deaths attributed to influenza as well as to all forms of

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<sup>5</sup>For example, among the health experts surveyed by the Science Media Center, estimates of the immunization rate needed to ensure herd immunity from COVID-19 ranged between 60 to 90 percent of the population .

<sup>6</sup>For a survey and discussion, see Parshley (2020).

<sup>7</sup>See Crosby (2003), Kolata (1999), and Barry (2004) for accounts of this pandemic.

pneumonia and bronchopneumonia, which were frequent complications of the flu.<sup>8</sup> We use cubic splines to interpolate the statistics by age bin to the model’s quarterly frequency. We then calculate the mortality rates due to these causes that are in excess of the corresponding average mortality rate for the 1913–1917 reference period. Our Appendix A and replication materials contain the data sources and methodology.

The left panel of Figure 2 displays the fraction of the population that died from influenza and related pulmonary causes in 1918, 1919, and 1920. In 1918 alone, almost one percent of individuals in their late 20s and early 30s died from these causes—an order of magnitude more than had been observed, on average, in the previous five years. Mortality was also unusually elevated among other individuals aged 70 years or less, with significant excess mortality among newborns and young children. By contrast, the mortality of individuals aged more than 70 years in 1918, while large in comparison to that of most other age groups, was less than had been observed in the previous five years.<sup>9</sup> Why the Great Influenza affected young adults most severely, and why it spared the elderly to some degree, remains debated. Researchers have notably suggested that high mortality rates among young adults might reflect overactive immune responses, that the elderly had acquired protective immunity from past exposures to similar virus strains, or that exposure to other disease, such as tuberculosis and the 1889–1890 “Russian Flu” pandemic, could have led to health complications among young adults.<sup>10</sup> As we discuss below, regardless of its causes, the fact that the Great Influenza affected young adults most severely meant that it led to a significant and permanent reduction in the aggregate labor supply.

The right panel of Figure 2 shows the excess mortality rate, by age, that we use in our counterfactual simulations of a pandemic matching the mortality risk observed during the Great Influenza. The excess mortality rate sums the mortality rates from influenza and related pulmonary causes between 1918 and 1920 that are in excess of what was observed, on average, during the 1913–1917 period. Thus, if all waves of the pandemic had occurred at once, it would have added as much as  $1\frac{1}{4}$  percentage points to the mortality rate of individuals in their late 20s and early 30s, whereas individuals aged more than 70 years would have benefited from reduced mortality risk.

### 3 Model Simulations

To explore the possible supply-side effects of pandemic mortality, we simulate the general-equilibrium, overlapping-generations model from our paper Gagnon et al. (2016).<sup>11</sup> We consider three mortality scenarios: a “baseline” COVID-19 scenario in which 200,000 individuals die; a “herd immunity”

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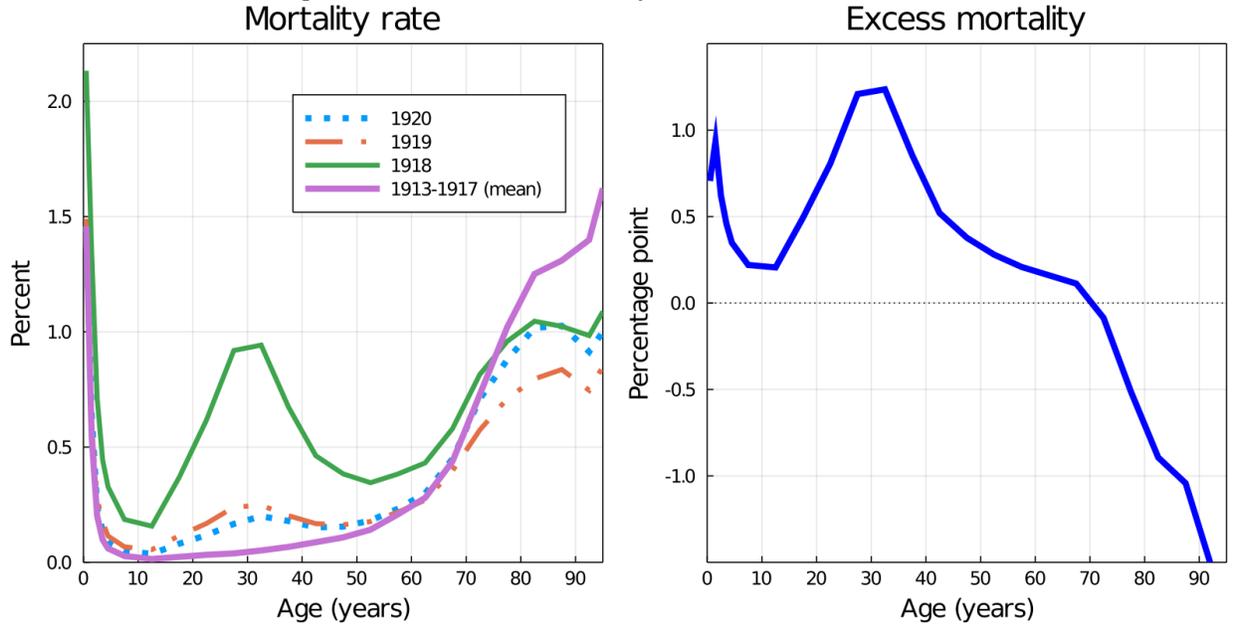
<sup>8</sup>For discussion of the role of secondary pulmonary infections in exacerbating mortality from the Great Influenza, see Morens and Fauci (2007) and Brundage and Shanks (2007).

<sup>9</sup>Our mortality rate estimates based on official mortality data broadly accord with the age pattern of deaths reported by other authors using alternative data sources, such as Simonsen et al. (1998).

<sup>10</sup>See Morens and Fauci (2007), Gagnon et al. (2013), and the references therein for discussions of the possible explanations for the pattern of mortality by age during the Great Influenza.

<sup>11</sup>See Gagnon et al. (2016) and the accompanying replication materials for a full description of the model and its baseline calibration.

Figure 2: Estimated mortality from the Great Influenza



**Source:** Authors’ calculations using data from the annual publication “Mortality Statistics” of the U.S. Bureau of the Census for the years 1913 to 1920.

**Notes:** The mortality rates and excess mortality rates encompass all death categorized under “influenza” and related pulmonary causes. See Appendix A for details.

COVID-19 scenario in which 70 percent of the population is infected; and a “Great Influenza” scenario in which the excess mortality rate by age matches our estimates of excess mortality for all waves of the Great Influenza.

### 3.1 Summary of the model

The production side of the model is neoclassical, with a representative firm operating a Cobb-Douglas production function. Households are representative of their birth cohorts in terms of demographic characteristics. They must allocate their period income between consumption and savings, a decision that is made with knowledge that households will rely primarily on savings to sustain consumption late in life. Household’s labor endowment varies according to the observed trend in employment rates by age and birth cohort. By aggregating across households’ life-cycle decisions, the model generates predictions for the paths of the aggregate stock of capital and the aggregate labor supply, whose ratio plays a central role in our analysis.

The model is calibrated to observed and projected trends in mortality risk, labor supply, and fertility choices of American households. Therefore, the model can be used to explore the macroeconomic implications of changes in cohort sizes over time, including changes caused by excess mortality due to pandemics. Of note, the model is not designed to address near- to medium-term disruptions to economic activity caused by factors such as social distancing measures, business bankruptcies, policy interventions, the effects of uncertainty on economic decisions, or demand-driven cyclical

disturbances more generally. Thus, the results should be interpreted as an exploration of the direct effects of pandemic mortality on the productive capacity of the economy.

We simulate pandemic mortality as a single wave of fatalities that hits in the second quarter of 2020—that is, in the period when mortality associated with COVID-19 soared in the United States. Obviously, pandemic mortality can occur over several quarters or even years—indeed, past pandemics have generally featured more than one wave of infection. That said, given our interest in the consequences over extended periods, the lumping of fatalities into a single quarterly period is largely inconsequential for our key conclusions.

### 3.2 The importance of the aggregate capital-labor ratio

The aggregate capital-labor ratio plays a central role in our simulation results, and thus warrants some prior discussion. The representative firm’s production function can be expressed as

$$Y_t = A_t (K_t)^\alpha (L_t)^{1-\alpha} = A_t K_t \left( \frac{K_t}{L_t} \right)^{\alpha-1},$$

where  $Y_t$  is real output,  $A_t$  is total factor productivity,  $K_t$  is the aggregate capital stock,  $L_t$  is aggregate labor, and  $\alpha \in (0, 1)$  is a parameter. Because the aggregate stock of capital is fixed within the period, current output depends solely on, and inversely to, the capital-labor ratio (equivalently, current output depends solely on, and positively to, aggregate labor).

We assume that the firm rents its capital and labor inputs in competitive markets, so that the real wage,  $W_t$ , and the real (gross) rental rate of capital,  $R_t$ , can also be expressed as functions of the aggregate capital-labor ratio,

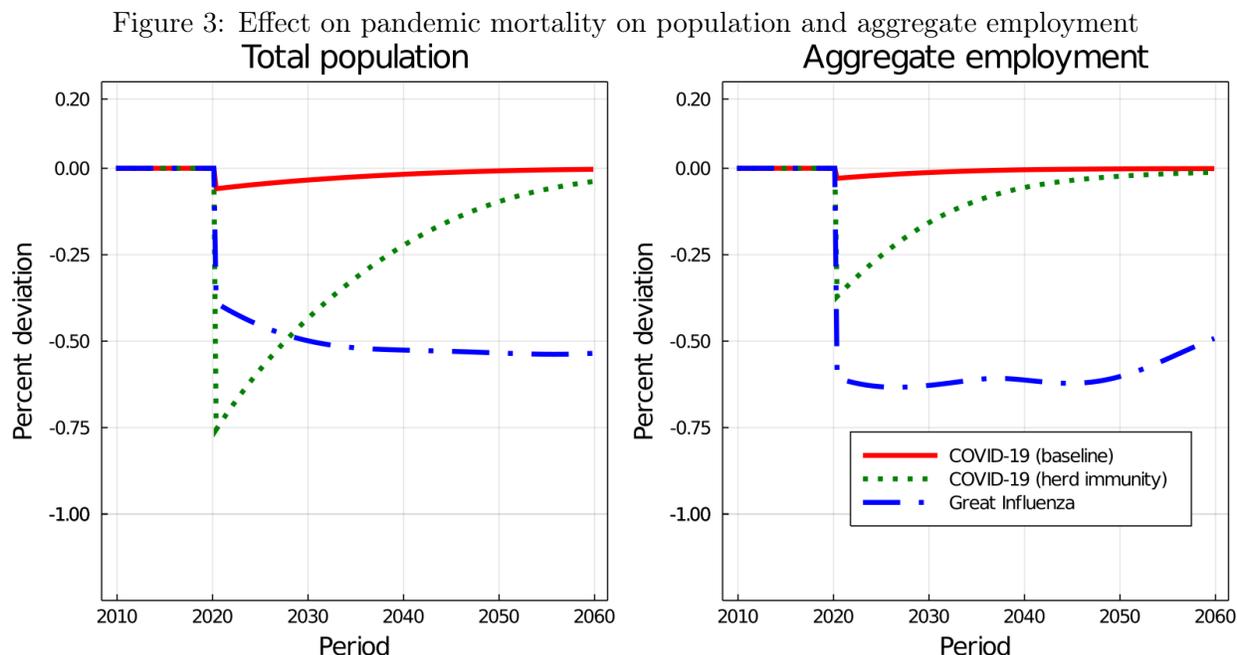
$$W_t = (1 - \alpha) A_t \left( \frac{K_t}{L_t} \right)^\alpha ; R_t = \alpha A_t \left( \frac{K_t}{L_t} \right)^{\alpha-1}.$$

Under these assumptions, sudden mortality among workers causes output to fall by depressing the labor input. The increased scarcity of labor relative to capital, in turn, causes real wages to rise and the rental rate of capital to fall. The relative scarcity of labor more generally causes interest rates to fall, with the real short-term interest rate in the model being expressed as  $r_t = R_t - \delta$ , where  $\delta$  is the depreciation rate of capital. Because the capital stock adjusts slowly through individual consumption/saving decisions—and because the path of the aggregate labor supply is determined by exogenous demographic variables—the effects of pandemic-related mortality on the capital-labor ratio and on other variables can persist for a long while. Jordà et al. (2020) highlight these channels to explain their findings that real wages rose and interest rates declined in the wake of several past pandemics.

### 3.3 Implications for the population and labor supply

Figure 3 shows, for each of the three scenarios, the effects of pandemic mortality on the population and aggregate labor supply (both expressed in percent deviations from their no-pandemic trends).

For COVID-19, the death of 200,000 persons in the baseline scenario reduces the population by 0.06 percent (left panel). Under an infection rate consistent with achieving herd immunity, a little over 2.5 million persons would die, which is equivalent to 0.75 percent of the U.S. population. For both COVID-19 scenarios, the model predicts that the effects of pandemic mortality on the size of the population will be essentially undone in the coming few decades because almost all deaths occur among individuals who are past their reproductive period, leaving population growth through births essentially unchanged.<sup>12</sup>



**Source:** Authors’ calculations.

**Notes:** The simulations are conducted under the assumption that all fatalities occur in 2020:Q2. The attack rate is calibrated to yield 200,000 total deaths in the “COVID-19 (baseline)” simulation and set to 70 percent of the population in the “COVID-19 (herd immunity)” simulation. The “Great Influenza” simulation is calibrated to our estimates of excess mortality rates during the 1918–1920 flu pandemic.

The initial reductions in aggregate employment in the two COVID-19 scenarios (right panel) are half as small as the corresponding reductions in the population. The relatively small initial employment declines reflect the fact that COVID-19 mortality is strongly skewed toward the elderly, many of whom had exited the labor force. The effect on aggregate employment also dissipates faster than the effect on the population because the workers who die are closer to retirement than to death. As with the decline in the population, the decline in aggregate employment ultimately proves transitory because COVID-19 mortality has essentially no influence on fertility.

<sup>12</sup>For simplicity, we hold constant international migration in our simulations. U.S. migration flows fell sharply in response to COVID-19 as global travel sank and as U.S. authorities implemented travel and immigration restrictions. Deaths abroad are also skewed toward older individuals who are past their reproductive period. What effect, if any, these aspects of the pandemic will ultimately have on U.S. net migration and its composition is highly uncertain at this stage.

Figure 3 also reports responses under our counterfactual “Great Influenza” scenario. When the mortality risk matches our estimates for the 1918–1920 flu pandemic, the number of fatalities totals 1.3 million (0.39 percent of the population), which is many times larger than under our baseline COVID-19 scenario and roughly half as much as as under the “herd immunity” scenario. The initial fall in aggregate employment, at 0.61 percent, is a bit larger than the effect on the population, reflecting that fact that young workers were unusually likely to die from the virus. Importantly, and in contrast to the two COVID-19 scenarios, the effects of Great-Influenza-like mortality are largely permanent because the death of young adults leads to fewer births. In fact, the model predicts that the decline in the population relative to its pre-pandemic trend should accentuate over the remainder of the decade, reflecting the assumption that elderly mortality diminishes thanks to some protection to the specific virus strain.

In sum, the simulations illustrate that both the total mortality rate and the age distribution of fatalities are important determinants of the evolution of the population and workforce.

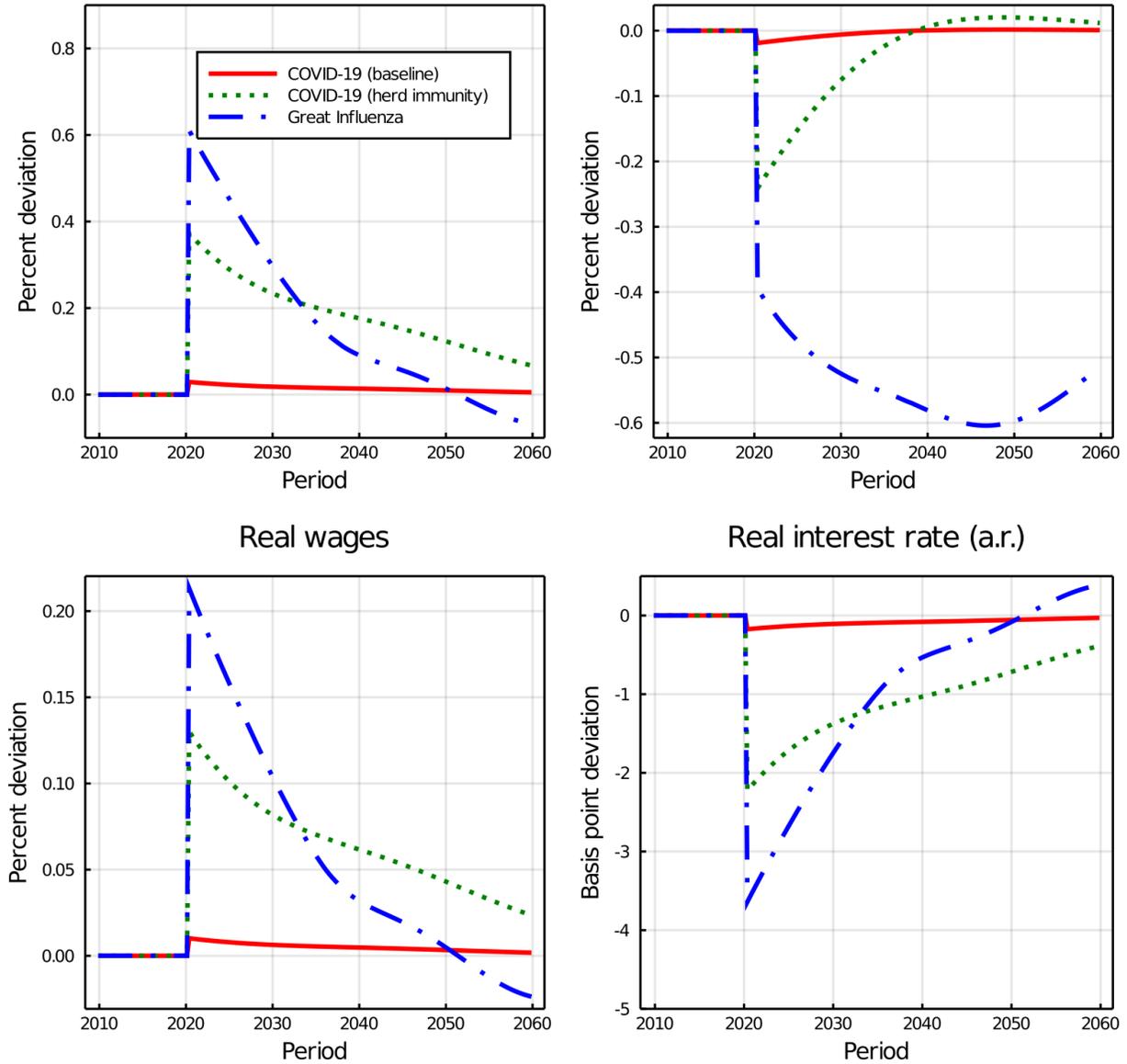
### 3.4 Implications for output, real wages, and interest rates

Figure 4 shows the implications of pandemic mortality for some key macroeconomic variables. Because of the sudden and persistent drop in labor supply, along with the slow adjustment of the capital stock, all three scenarios feature a sudden and persistent rise in the capital-labor ratio (top-left panel). Pandemic mortality also leads to a reduction in output (top-right panel); under our assumptions about the production function, this initial drop equals the contraction in the labor supply adjusted by the labor share in production. For the baseline COVID-19 scenario, this initial drop in employment is small, leading to a correspondingly small decline in output. By contrast, under the herd immunity scenario and the Great Influenza scenario, the output drops due to increased mortality are economically significant, at 0.24 percentage point and 0.39 percentage point, respectively.

The subsequent evolution of output largely depends on whether the drop in employment is temporary or permanent. Under the two COVID-19 scenarios, aggregate employment ultimately recovers, which helps output return to its trend level within a couple of decades. Under the Great Influenza scenario, output never returns to its original trend because of the permanent step down in aggregate employment. Moreover, in that scenario, the model predicts that output would slide farther below its pre-pandemic trend this decade and next. The reason is that sudden mortality leaves the capital stock above its new normal level (as evidenced by the jump in the capital-labor ratio). Over time, households increase their capital holdings by less than they would otherwise, thus contributing to a further fall in aggregate output. The slow decumulation of capital also cushions the fall in output under the two COVID-19 scenarios, which can be seen from the fact that output returns to its trend level sooner than either aggregate employment.

The bottom panels show the effects of pandemic mortality on real wages and the real short-term interest rate. Again, the effects in the baseline COVID-19 scenario are tiny, reflecting the correspondingly tiny rise in the capital-labor ratio. By comparison, mortality in the heard immunity

Figure 4: Macroeconomic implications of pandemic mortality  
 Capital-labor ratio                      Real output



**Source:** Authors' calculations.

**Notes:** The simulations are conducted under the assumption that all fatalities occur in 2020:Q2. The attack rate is calibrated to 200,000 total deaths in the “COVID-19 (baseline)” simulation and set to 70 percent of the population in “COVID-19 (herd immunity).” The “Great Influenza” simulation is calibrated to our estimates of excess mortality rates during the 1918–1920 flu pandemic.

and Great Influenza scenarios cause real wages to rise 0.13 percent and 0.21 percent, respectively, relative to the no pandemic case. The corresponding effect on the (annualized) real short-term interest rate is always small, however, peaking at less than 4 basis points in the Great Influenza scenario. Intuitively, the change in the interest rate corresponds to the change in the *level* of the real rental rate of capital ( $R_t$ ). Because the value of  $R_t$  is small to begin with, the level of  $R_t$  does not fall much even when  $R_t$  moves by a significant amount in percentage terms.

### 3.5 Discussion

Our simulations suggest that, unless mortality from COVID-19 turns out much larger than currently projected, the economy’s productive capacity will be largely intact. In that sense, past pandemics may be misleading guides for the possible longer-run effects of COVID-19. This conclusion and our quantitative results are subject to several considerations and caveats.

Importantly, by design, the model does not capture cyclical influences and thus cannot address, say, how social distancing measures, policy stimulus, and business bankruptcies affect the economy over the near to medium terms. In addition, the model abstracts from channels other than pandemic mortality that could affect the economy’s productive capacity. For example, the pandemic-induced recession could lead to a permanently lower output trajectory by hindering investment (see Barlevy (2007)). Higher public and private borrowing as fiscal authorities, businesses, and households seek to lessen the blow from the crisis could apply upward pressure on equilibrium interest rates and persistently crowd out some investment (see Engen and Hubbard (2005) and Laubach (2009) for evidence of such an effect). Similarly, the pandemic could alter the behavior of consumers and firms persistently, for example by limiting participation in group activities or altering global supply chains.

In addition, the direct macroeconomic effects of pandemic mortality could differ from those predicted by the model for a number of reasons. For instance, the effects could be smaller than we estimate because, for a given age, individuals with pre-existing health conditions are both more likely to die from COVID-19 and tend to have lower labor force participation than healthy individuals. Similarly, because many COVID-19 casualties had relatively low wealth, their deaths could leave the capital-labor ratio closer to its normal level than our model suggests, in which case a smaller rise in real wages and fall in real interest rates would be observed. Other non-modeled elements could instead magnify some of the macroeconomic effects. Because labor supply is exogenous in our model, the normalization of the capital-labor ratio following the death of workers operates through a reduction in household savings over time. Under an endogenous labor supply, bequests from deceased individuals might create an income effect that leads to a reduction in the labor supply of survivors, thus accentuating the fall in output and swings in factor prices.

Finally, even under our most extreme mortality scenarios, our model predicts that the direct effect of COVID-19 mortality on the level of interest rates is small. This conclusion contrasts with the empirical findings of Barro et al. (2020) and Jordà et al. (2020), who report that the Great Influenza and other historic pandemics led economically significant declines in real interest rates.

The multi-country regression of Barro et al. (2020) notably suggests that a mortality rate of the kind of observed in the United States during the Great Influenza lowered the real return on government bills by about 3.5 percentage points. They trace back this decline to both a fall in the expected real safe interest rate and an increase in inflation at the time. The extent to which the predicted decline reflects cyclical aspects not present in our paper, the concurrent effects of World War I (which the authors attempt to capture), other factors, or statistical noise is unclear.<sup>13</sup> Using multi-country panel data going back centuries, Jordà et al. (2020) estimate that pandemics are associated with a peak decline in the real interest rate of nearly 2 percent. However, the structure of the economy has changed dramatically—even in the past century—and the deadliness of pandemics such as the Black Plague dwarfed that of COVID-19.<sup>14</sup> Under our assumptions about the production function, roughly one quarter of the workforce would need to die to lower the real interest rate by 2 percentage points.

## 4 Conclusion

Our simulations suggest that, under current epidemiological projections, mortality from COVID-19 may have, in itself, at most small effects on the productive capacity of the U.S. economy and production factor prices. This conclusion contrasts with evidence that past pandemics had longer-lasting macroeconomic effects, and speaks to the fact that mortality from COVID-19 is heavily concentrated among older individuals. Of course, this finding should not be understood as diminishing in any way the pandemic’s heavy human cost and economic disruptions over the short to medium terms, aspects that our model does not address by design. In addition, if the spread of COVID-19 is not contained, then the effects on the productive capacity of the U.S. economy could be significant and felt for decades.

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<sup>13</sup>Because of elevated statistical noise, one cannot reject the polar hypotheses that the decline in the real interest rate caused by the Great Influenza was either permanent or fully reversed within a year or two.

<sup>14</sup>We also note that Jordà et al. (2020) find no statistically significant decline in the real interest in the first decade that follows past pandemics, with the peak effect being registered about 20 years after the pandemic.

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## A Estimating the Mortality Rates during the Great Influenza

This appendix derives estimates of mortality rates and excess mortality rates, by age, attributable to the Great Influenza of 1918 to 1920. The main source of information is a series of annual reports titled *Mortality Statistics* published by the Bureau of the Census on the number of deaths and their main causes. Copies of the reports in a PDF format are accessible through the Center for Disease Control at the following link: [https://www.cdc.gov/nchs/products/vsus/vsus\\_1890\\_1938.htm](https://www.cdc.gov/nchs/products/vsus/vsus_1890_1938.htm).

### A.1 Mortality Rates

Let  $MR_{a,t}^{US}$  be the mortality rate of individuals aged  $a$  in period  $t$  in the United States that is due to influenza. We measure the mortality rate as

$$MR_{a,t}^{US} = 100 \times \text{Number of civilian deaths due to influenza}_{a,t}^{US} \times \text{Population}_{a,t}^{US},$$

where  $\text{Population}_{a,t}^{US}$  is the U.S. civilian non-institutional population. The mortality rates in excess of normal yearly mortality due to influenza, which we use in the model simulations, are discussed below.

Our focus on the civilian population is due to the fact that the Bureau of the Census reports deaths for the civilian population and the military population separately, with the later population being much smaller and the information much less detailed. Moreover, the Great Influenza began when a large number of persons served in uniform because of World War I; these persons faced mortality risk that arguably were not necessarily representative of the population. That said, many of those mobilized were young adults, a category that was hit severely by the disease.

There are a few methodological challenges in translating death and population data from the Bureau of the Census into mortality rate statistics. One challenge is that only a subset of states reported mortality statistics. As the table below shows, the number of reporting areas grew steadily throughout the 1910s as new states joined the United States and some existing states began to report data. Hawaii joined the United States as a territory in 1917 but the Bureau opted to report its deaths separately from those of other states “because of the distant location of Hawaii and the peculiar constitution of its population.”

Estimates of the size of the U.S. and registration area populations are subject to some uncertainty, especially during intercensal periods. The population estimates produced late in the 1910s were unusually uncertain because WWI led to significant movements of population. For these reasons, and to ensure consistency across mortality and population sources, the population statistics reported in the table above all come from the report *Mortality Statistics 1920*, which includes information from the 1920 census. In contrast to population estimates, death counts and death causes are generally not revised from one report to the next, even after a new census information is published. Mortality statistics are published for 5-year age bins starting with the bin “0 to 4 years” and ending with the bin “95 to 99 years.” The yearly reports also include statistics per year of age through age 4 years (to zoom in on early childhood mortality) and for 100 years and over. Our

Table 1: Estimates of the civilian populations in the United States and reporting areas

Year	$Population (1922)_t^{US}$	$Population (1922)_t^{RA}$	Coverage (percent)
1913	96,512,407	63,200,625	65.5
1914	97,927,516	65,813,315	67.2
1915	99,342,625	67,096,681	67.5
1916	100,757,735	71,349,162	70.8
1917	102,172,845	74,984,498	73.4
1918	103,587,955	81,333,675	78.5
1919	105,003,065	85,166,043	81.1
1920	106,418,175	87,486,713	82.2

**Source:** Bureau of the Census, *Mortality Statistics 1920* (published in 1922); authors' calculations.

**Notes:** The variables  $Population (1922)_t^{US}$  and  $Population (1922)_t^{RA}$  represent the total population in the United States and the states and territories reporting mortality statistics at the time, respectively. The reporting area population excludes Hawaii. Population statistics are as of July 1 of each year.

interpolation to a quarterly age frequency uses the most disaggregated information by age where possible.

In our analysis, we treat deaths from non-reporting states and Hawaii as missing at random, that is, we assume the people in reporting and non-reporting areas face the same mortality risk. Accordingly, we approximate the population-wide mortality rates using the ones for registration areas (which use “RA” superscripts),

$$MR_{a,t}^{US} \approx MR_{a,t}^{RA} = 100 \times \frac{\text{Number of civilian deaths due to influenza}_{a,t}^{RA}}{Population_{a,t}^{RA}}.$$

Another complication is that we do not have population statistics by years of age in the registration areas. We posit that the age composition in registration areas is the same as for the overall U.S. population. Population counts by years of age are available for most age groups in the Bureau’s 2016 vintage of historical data. One exception is for people aged 75 years or more, which are aggregated into a single age category. As an alternative, we use the mid-year population estimates generated by our model; these estimates use population, life cycle, and migration information to populate all age periods and are consistent with the 2016 vintage. See the technical appendix to Gagnon et al. (2016) for our methodology. The 2016 historical total population estimates differ a little--by about 1 percent or less--from the 1922 total population estimates shown in the table above, in part because of small differences in the coverage of states and territories. We adjust the population estimates to account for these small differences. The imputed population by age in registration areas is

$$Population_{a,t}^{RA} \approx Population (1922)_t^{RA} \times \frac{Population (2016)_{a,t}^{US}}{Population (2016)_t^{US}}.$$

As a check on the mortality rates, we can look at estimates produced by Davis and Mitchell

(1920) in a special Bureau report on the death counts in the final four months of 1918 for Indiana, Kansas, and Philadelphia, PA. These estimates use unpublished population counts by age in these areas. The mortality rates for Philadelphia, at around 4 percent for young adults, were among the highest recorded at the time and thus cannot be assumed to be representative of the rates for the U.S. population. That said, the broader age pattern of mortality is consistent with our estimates for all reporting areas.

## A.2 Excess mortality rates

Influenza is a cause of death each year; therefore, its typical effects are incorporated into life-cycle mortality tables. For this reason, our modeling work focuses on the excess mortality rate is the difference between the mortality rates registered during the pandemic and the counterfactual mortality rate that would have been registered if the pandemic had not happened. To obtain a counterfactual, we compute the average mortality rate by years of age in the five-year period that preceded the Great Influenza (that is, for the 1913–1917 period). That is, we calculate the excess mortality rates as

$$EMR_{a,t}^{US} = MR_{a,t}^{US} - MR_{a,1913-1917}^{US}.$$

As we shall see, the death counts suggest that older generations enjoyed some protection from the virus strain that caused the Great Influenza (for a discussion, see, for example, Gagnon et al. (2013)). Accordingly, our excess mortality rate estimates for older generations are negative.

A final complication is that many deaths related to the Great Influenza are miscategorized. Although the yearly reports contain death counts specific to influenza, the Bureau warns that, “in studying the effects of the pandemic of influenza, it is not believed to be best to study separately influenza and the various forms of pneumonia, bronchitis, and the respiratory diseases, for doubtless many cases were returned as influenza when the deaths were caused by pneumonia, and vice versa. The best method, therefore, seems to be to study as one group deaths from influenza and pneumonia (all forms), disregarding deaths from the other respiratory diseases, which were comparatively few.” (Source: Bureau of the Census, *Mortality Statistics 1919*, page 28.) For this reason, we measure excess mortality due to the Great Influenza in terms of excess mortality in the categories “10: influenza,” “91: Bronchopneumonia,” and “92: Pneumonia (Lobarpneumonia and pneumonia (undefined)).”

In our implementation, we excess mortality rates by age due to the Great Influenza separately for the years 1918, 1919, and 1920. We then calculate cumulative excess mortality rates during the Great Influenza epidemic by summing up the excess mortality rates for those three years, as if the virus had hit in a single wave. We make no adjustment for the fact that people had different ages during the three waves; doing so would have at most a tiny effect on the total death counts in proportion to the population and accompanying macroeconomic effects.