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NON-PHARMACEUTICAL INTERVENTIONS AND MORTALITY IN U.S. CITIES DURING THE GREAT INFLUENZA PANDEMIC, 1918-1919

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ABSTRACT

A key issue for the ongoing COVID-19 pandemic is whether non-pharmaceutical public-health interventions (NPIs) retard death rates. The best information about these effects likely comes from flu-related excess deaths in large U.S. cities during the second wave of the Great Influenza Pandemic, September 1918-February 1919. NPIs, as measured by Markel, et al. (2007), are in three categories: school closings, prohibitions on public gatherings, and quarantine/isolation. Although an increase in NPIs clearly flattened the curve in the sense of sharply reducing the ratio of peak to average death rates, the estimated effect on overall deaths is small and statistically insignificant. One possibility is that the NPIs were not more successful in curtailing overall mortality because the average duration of NPIs was only around one month. Another possibility is that NPIs mainly delay deaths rather than eliminating them.

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The mortality experienced during the Great Influenza Pandemic of 1918-1920 likely provides the best historical information on the plausible upper found for outcomes under the ongoing coronavirus (COVID-19) pandemic. Barro, Ursúa, and Weng (2020) compiled and discussed the cross-country data on flu-related deaths during the Great Influenza Pandemic. Based on information for 48 countries, that study found that the Pandemic killed around 40 million people, 2.1 percent of the world's population. When applied to current population, the corresponding number of deaths is 150 million.

An important issue is how public-health interventions, such as those being implemented currently in U.S. states and most countries for the ongoing coronavirus pandemic, affect mortality. A difficulty in isolating causal effects of NPIs in the current environment is reverse causality; for example, U.S. state and city governments clearly react to numbers on mortality, hospitalizations, and cases by adjusting the extent of interventions. Specifically, when the extent of disease increases, governments are more likely to close their economies, and vice versa. Although reasonable from the perspective of desirable policy, this endogenous reaction makes it difficult to assess the effects of the NPIs. For scientific purposes, the ideal setting would be controlled experiments, whereby NPIs were adjusted randomly across governmental jurisdictions. This kind of experimentation does not exist today or during the Great Influenza Pandemic. But the nature of the evolution of the Great Influenza across U.S. cities offers the potential to use instrumental variables to assess the causal effects of NPIs on death rates.

I. Non-Pharmaceutical Interventions and Flu-Related Death Rates

Epidemiologists, notably Markel, et al. (2007); Hatchett, Mecher, and Lipsitch (2007); and Bootsma and Ferguson (2007), have studied effects of non-pharmaceutical public-health

interventions (NPIs) on flu-related excess deaths in large U.S. cities over the 24-week period corresponding to the peak of the Great Influenza Pandemic, September 1918-February 1919. Subsequently, I use the label "the epidemiologists" to refer to this trio of authors. The weekly data on flu-related excess death rates come from U.S. Census Bureau, *Weekly Health Index*, reproduced in Collins, et al. (1930, Appendix, Table B). Continuous weekly data over the study period are available for 45 of the 50 largest U.S. cities, where this group of 50 corresponds to a central-city population in the 1910 U.S. Census of at least 100,000.[1] Monthly data on flu-related excess deaths are available for these cities back to 1910 (Collins, Appendix Table A).

The extensive research by Markel, et al. (2007, Table 1 and supplemental figures) involved collection of data on NPIs from September 1918 to February 1919 for 43 of the 45 cities that have full weekly data on flu-related excess mortality. Atlanta and Detroit were excluded, but the current study adds these cities to the sample. The Markel data on NPIs were organized into three broad categories: school closings, prohibitions on public gatherings, and quarantine/isolation.[2] The underlying information comes from articles in two newspapers in each city, along with other sources. The main data were reported as number of days in which NPIs of the various types were in effect, with a focus on a variable that considers the presence of any type of NPI. For example, when school closings and prohibitions on public gatherings prevail on the same day, the variable records two days' worth of NPI.[3] Barry (2007a, 2007b) raises objections to the NPI data collected by Markel, et al. (2007) for New York City, and these

[1]The five missing cities are Bridgeport, Jersey City, Memphis, Paterson, and Scranton.
[2]Hatchett, Mecher, and Lipsitch (2007) consider many more categories of NPIs but analyze only 17 of the 43 cities considered by Markel, et al. (2007). Bootsma and Ferguson (2007) studied the timing of the introduction of a set of NPIs in 16 cities (15 of those considered by Markel plus Atlanta). Measures of NPIs across countries in 2020 are in UBS (2020). Measures of NPIs across U.S. states and localities in 2020 are in Opportunity Insights (2020).
[3]Correia, Luck, and Verner (2020) also use the Markel data on NPIs, focusing on how number and speed of implementation of NPIs impact manufacturing employment and output in U.S. cities. Lilley, Lilley, and Rinaldi (2020) critique the methodology used by Correia, Luck, and Verner.

objections are discussed below. Business closings, emphasized in the context of the ongoing coronavirus pandemic, were not implemented in broad form during the Great Influenza, although staggering of business hours was common.[4] Likely because of the absence of substantial business closings, Markel, et al. (2007) did not focus on this category of NPI. However, the variable for prohibitions of public gatherings includes closings of theatres, bars, entertainment and sporting events, and so on.

The present analysis considers two characteristics of each city's flu-related excess death rates: first, the overall death rate out of the total population over the full 24-week study period, and second, the ratio of the peak weekly death rate during the period to the average of the weekly death rates. (This average equals the overall death rate divided by 24.) A lower overall death rate is a reasonable gauge of the ultimate success of the NPIs. In contrast, a lower relative peak implies a smoother pattern, often described as a "flattening of the curve," which can be desirable from the standpoint of easing burdens on the healthcare system, possibly leading thereby to fewer deaths. However, for a given overall death rate (and a given initial death rate, which is typically close to zero), if an NPI lowers the relative peak, the implication is that the intervention delays deaths but does not ultimately avoid them.

For illustrative purposes, Figure 1 shows the weekly evolution of flu-related excess death rates from September 1918 to February 1919 for 8 of the 45 cities studied: Boston, New York, Philadelphia, Pittsburgh, Chicago, New Orleans, St. Louis, and San Francisco. The typical pattern is that the excess death rate starts around zero, rises to a sharp peak, then falls rapidly and remains positive for several more weeks. For example, Boston and especially Philadelphia have pronounced peaks in early to mid October 1918. Pittsburgh has a smaller peak but high death

[4]Velde (2020, Figure 4) constructed measures of these types of restrictions on business activity for 42 cities (those considered by Markel plus Atlanta and Detroit, with Lowell, Milwaukee, and New Haven omitted).

rates over a longer period around the peak. New York and Chicago have smaller peaks and a milder overall experience. New Orleans and San Francisco show second peaks in early 1919. St. Louis has the mildest outcomes overall without a pronounced peak.

The epidemiologists used standard epidemiological models to study the dynamics of flu-related deaths during the Great Influenza Pandemic, as illustrated by Figure 1. The present analysis focuses instead on two measures of overall outcomes—the overall death rate over the study period (which corresponds to the areas under the curves in Figure 1) and the size of the peak weekly death rate, when measured relative to the average of the weekly death rates.

As mentioned, there is concern that NPIs—measured, say, by length of time in force—and flu-related death rates are simultaneously determined at the level of cities. On the one hand, the basic hypothesis is that more NPIs reduce death rates. On the other hand, NPIs implemented by city governments are likely to respond to death rates in terms of numbers realized or anticipated. The implicit assumption in the statistical analysis by the epidemiologists is that NPIs are determined exogenously; that is, shifts in actual or anticipated death rates do not impact the chosen NPIs. Bootsma and Ferguson (p. 7592) recognize the endogeneity problem—"Causality will never be proven, because, unsurprisingly, control measures were nearly always introduced as case incidence was increasing and removed after it had peaked"—but did not deal with it. The present research attempts to account for the potential endogeneity of NPIs by employing instrumental variables.

Appendix Table A1 shows all of the data used for the 45 large U.S. cities in the sample. The variables include measures of flu-related excess death rates, measures of non-pharmaceutical interventions (NPIs), and other variables. Table 1 has descriptive statistics for the variables detailed in Appendix Table A1.

II. Regression Analysis

In the parts of their analysis that assess overall outcomes for flu-related death rates, the epidemiologists rely on an array of pair-wise correlations involving NPIs, flu-related death rates, and other variables. One shortcoming of this approach is that it does not account for possible correlations with variables outside of each pair—for example, in assessing the connection between NPIs and death rates, there is no consideration of the association of NPIs with other variables that influence mortality. In addition, as already noted, the approach does not even attempt to establish causation between the two variables in each pair—is it NPIs that affect death rates or vice versa or both?

The present analysis deals with these issues by employing the multivariate-regression framework familiar to economists. This analysis includes the use of instrumental variables to possibly isolate causal effects of NPIs on flu-related death rates.

A. First-Stage Regression for NPIs

An important consideration is that the second wave of the Great Influenza began in the United States by late August 1918, likely coming from Europe and then appearing around Boston at the army base Fort Devens and the Navy's Commonwealth Pier facility.[5] Shortly thereafter, sailors leaving Boston on ships spread the flu to Philadelphia and New Orleans, leading subsequently to spread to other places. From this perspective, distance from Boston (shown in Appendix Table A1, column 10) may serve as an exogenous measure of how early the flu epidemic tended to reach each city in the sample. Specifically, cities further from Boston typically had more time to prepare and were, therefore, more likely to react in terms of the

[5]For a discussion, see Barry (2004, pp. 181 ff.).

implementation of NPIs.[6] Empirically, distance from Boston, entered in a quadratic form, has considerable explanatory power for the NPI variable. The pair-wide relationship between distance and overall NPIs, shown in Figure 2, is positive with a simple correlation coefficient of 0.65. (The NPI variable is in Appendix Table A1, column 3.)

Although distance from Boston is clearly exogenous, the distance-from-Boston variable might be problematic for instrumental estimation if this variable affects flu-related deaths directly (or is correlated with other variables that relate to these deaths). That is, the effects of distance on mortality may not work solely through the channel of influencing choices of NPIs. This issue is taken up in subsequent discussion.

Another possibility is that differences across cities in political structure may result in exogenous differences in choices of NPIs. A clear difference in 1918 was between cities with an elected powerful mayor interacting with an elected city council versus the Commission form of government, which was first adopted in 1901 and involved the election of commissioners who had joint executive authority. In these systems, the mayor, either elected or chosen by the commissioners, had little power. In later years, this system was replaced particularly by the city-manager form of municipal government, a structure that also superseded some of the strong-mayor administrations. In 1918, at least 14 of the 45 cities in the sample had a Commission or other form of government that lacked a strong mayor (including Washington DC, which was run by the federal government). The largest cities in the non-mayoral group were Buffalo, Newark, New Orleans, and Washington DC. In general, more densely populated cities were more likely to have mayoral systems in 1918.

[6]Possibly the distance measure could be improved by using the time required to transit from Boston to a particular location, given the transportation technology available in 1918. However, a measure of time required to transit from Boston to each city by train or boat did not improve on the explanatory power of the first-stage regression for explaining NPIs.

The underlying conjecture is that having a mayoral system, which tends to feature a high concentration of political power, is more likely than a system without a powerful mayor to enact NPIs aimed to counter the spread of the Great Influenza. Hence, the expectation is that a dummy variable for the presence of a strong mayor (shown in Appendix Table A1, column 11) would be expected to have a positive coefficient in a first-stage regression for explaining the duration of NPIs implemented by each city.

The first-stage OLS regression for NPIs across 45 cities for September 1918-February 1919 is:

$$\text{(1)}\quad \underset{}{\text{NPI} = 0.068^{*} + \underset{(0.039)}{0.202^{***}}\cdot(\text{distance Boston}) - \underset{(0.053)}{0.035^{**}}\cdot(\text{distance Boston})^{2} + \underset{(0.016)}{0.045}\cdot\text{Mayor},}\quad \underset{(0.031)}{}$$

where $R^2 = 0.50$, F-Statistic = 13.6, mean of dependent variable = 0.24 (years), standard error of regression = 0.093, standard errors of coefficients are in parentheses, and statistical significance of each coefficient is denoted by *** at 1%, ** at 5%, and * at 10%. The p-value for the coefficients of the two distance variables jointly is 0.000. (With the mayoral dummy variable excluded, the F-Statistic is 18.8.)

The overall F-Statistic from equation (1) of 14 (19 if the mayoral variable is omitted) indicates that the distance-from-Boston variable is not a weak instrument. Equation (1) implies that the estimated marginal effect of distance on NPIs is positive for most of the sample and is roughly zero at the furthest away cities on the west coast, for which the distance variable is around 3 (in thousands of miles). The estimated coefficient of the mayoral dummy variable is positive, as expected, but insignificantly different from zero at the 10% level.[7]

[7]If added to the regression for NPIs, population density in 1910 has an estimated coefficient that is close to and insignificantly different from zero. The other coefficients remain similar to those shown in equation (1).

B. Second-State Regressions for Flu-Related Death Rates

1. Effects of NPIs on cumulative and relative peak excess death rates. Table 2 has second-stage regressions applying to the 45 U.S. cities for weeks ending from September 8, 1918 to February 22, 1919. The analysis considers two dependent variables: the overall excess flu-related death rate and the relative peak death rate (shown in Appendix Table A1, columns 1 and 2).

Aside from NPIs, flu-related death rates in each city likely depend on age structure and other demographic characteristics and would depend on the nature of healthcare facilities to the extent that these were effective in curbing mortality. To take account of these effects, the regressions include as an explanatory variable the flu-related gross mortality rate from a prior period[8]—specifically, the median rate for 1910-1916, corresponding to the months, September-February, used for the dependent variable. This variable, shown in Appendix Table A1, column 8, is calculated from information in Collins, et al. (1930, appendix Table A). The idea is that this measure would reflect city characteristics such as demographics and healthcare facilities to the extent that they influence flu-related deaths in general and, therefore, likely also the excess deaths experienced during the 1918-1919 Pandemic. In addition, the regressions include as an explanatory variable a measure of a city's weather, given by heating-degree days (Appendix Table A1, column 9).

The pair-wise association between NPIs and flu-related excess death rates during the study period is in Figure 3. This relationship is negative, as expected (simple correlation coefficient equals -0.28). Regression results in columns 1 and 2 of Table 2 show the estimated effects of NPIs on the overall flu-related excess death rate. For the OLS regression in column 1,

[8]A variable of this type was used by Bootsma and Ferguson (2007, p. 7588).

the estimated coefficient on NPI is negative but insignificantly different from zero at the 10% level. In the two-stage least-squares (TSLS) regression in column 2,[9] the estimated coefficient on NPI is larger in magnitude, as would be expected with positive reverse causation between death rates and choices of NPIs. However, the estimated coefficient on NPI is still insignificantly different from zero at the 10% level. In terms of magnitudes, the point estimate of the NPI coefficient of -0.33 in column 2 means that a one-standard-deviation change in NPI (by 0.13 in Table 1) implies an estimated change by 0.04 in the flu-related overall death rate, compared to the respective mean and standard deviation of 0.52 and 0.15 (as shown in Table 1). Therefore, even if the point estimate of the coefficient on the NPI variable were viewed as reliable, only a minor part of the variations in death rates would be attributed to variations in NPIs.

The estimated coefficient of the prior flu-related gross death rate is positive and statistically significant at the 1% level in columns 1 and 2. As mentioned, these coefficients likely pick up effects from demographics and healthcare facilities. The coefficients of heating-degree days are negative and statistically significant at the 5% level. These results indicate, surprisingly, that colder places (during fall and winter) are estimated to have significantly lower excess flu-related death rates.[10]

The pairwise association between NPIs and the relative peak death rate is in Figure 4. This relationship is negative, as expected, with a simple correlation coefficient of -0.56. Regression results in columns 3 and 4 of Table 2 show the estimated effects of NPIs on the relative peak death rate. The estimated coefficients on the NPI variable in columns 3 (OLS)

[9]Instrumental variables are those shown on the right-hand side of equation (1) along with the gross flu-related mortality rate for the September-February months of 1910-1916 and heating-degree days.
[10]The simple correlation coefficient between heating-degree days and the gross flu-related mortality rate for the September-February months of 1910-1916 is close to zero.

and 4 (TSLS) are negative and highly statistically significant, with the coefficient under TSLS notably larger in magnitude. Again, a higher magnitude under TSLS makes sense because a higher relative peak death rate would encourage the enactment of NPIs with longer duration. Therefore, in the OLS results (column 3), the magnitude of the negative effect on death rates is attenuated.[11] In terms of magnitudes, the point estimate of the coefficient on the NPI variable of -8.6 in column 4 implies that a one-standard-deviation change in NPI (by 0.13 in Table 2) implies a change in the relative peak death rate by 1.1, compared to the respective mean and standard deviation of 4.6 and 1.3 (as shown in Table 2). Hence, the variations in NPI likely account for a substantial part of the observed variations in relative peak death rates.

Overall, there is clear evidence that an increase in NPIs flattens the pattern of excess flu-related death rates, gauged by the drop in the ratio of the peak weekly death rate to the sample average of the weekly death rates. This result indicates that NPIs—specifically as measured by Markel, et al. (2007)—do matter for flu-related mortality. But this influence shows up far more in the relative peak death rate than in the overall death rate.

Columns 5-8 of Table 2 consider the separate roles of the three types of NPIs—school closings, prohibitions of public gatherings, and quarantine/isolation (shown in Appendix Table A1, columns 4-6). There are insufficient instruments to allow for endogeneity of all three types of NPI individually. However, it seems plausible that, while the overall duration of NPIs is endogenous with respect to flu-related death rates, the distribution among the three types may be exogenous. Therefore, the TSLS estimation in columns 6 and 8 includes on the instrument list the variables used before in columns 2 and 4 along with the difference between the durations of

[11]Measurement error in the NPI variable can also be a source of this attenuation.

the various NPIs (school closings minus prohibitions of public gatherings and school closings minus quarantine/isolation).

For the overall excess flu-related death rate, the OLS results are in column 5 and the TSLS results in column 6. The only significant coefficients related to NPIs are the negative ones on prohibitions of public gatherings. The results in columns 5 and 6 accept the hypothesis with p-values of 0.16 and 0.18, respectively, that the coefficients of the three NPI variables are the same. Thus, the results conform with the specification in columns 1 and 2 that the three forms of NPIs can be combined into a single measure that adds up the durations of the three types.

For the relative peak death rate, the OLS results are in column 7 and the TSLS results in column 8. As in columns 3 and 4, the allowance for endogeneity of overall NPIs makes a substantial difference, with the magnitudes of the estimated coefficients notably larger under TSLS (column 8) compared to those under OLS (column 7). The results accept the hypothesis that the coefficients of each form of NPI are equal, with a p-value of 0.74 for OLS and 0.90 for TSLS. Thus, it is again satisfactory to combine the three types of NPIs into a single additive form, as in columns 3 and 4.

2. Effects from public-health response time. Columns 9-12 of Table 2 consider another measure of how NPIs were implemented—the public-health response time or PHRT constructed by Markel, et al. (2007, Table 1) and shown in Appendix Table A1, column 7.[12] A higher PHRT indicates more delay in a city implementing the first intervention intended to retard flu-related deaths. Figure 5 shows from the pair-wise relationship that the PHRT variable is

[12]The Markel definition of PHRT is the days between the date when the flu-related excess death rate reached twice a baseline death rate and the (usually later) date of the first non-pharmaceutical intervention. Their baseline corresponded to the date at which the excess flu-related death rate equaled twice the average gross flu-related death rate for 1910-1916.

negatively but weakly associated with distance from Boston, with a simple correlation coefficient of -0.20. Thus, while being further from Boston clearly raises the number of NPIs employed (Figure 2), it has a weaker connection with acting quickly to install some form of NPI. The PHRT also has positive but small simple correlation coefficients with the overall excess death rate (0.17) and the relative peak death rate (0.10).

The available instruments are insufficient to distinguish the NPI and PHRT variables—moreover, the first-stage regression for PHRT analogous to equation (1) has an F-Statistic of only 1.7. For this reason, the TSLS results shown in Table 2, columns 10 and 12, include as instruments the variables discussed before related to NPI along with the PHRT variable itself.

For the overall excess death rate, when the PHRT variable is included in columns 9 and 10, the only significant coefficient related to NPIs is the positive one on PHRT in the OLS estimation (column 9). A positive value here means that a longer delay in implementing some form of intervention leads to a higher overall death rate. However, in column 9, the coefficient on NPI and the coefficients for NPI and PHRT jointly differ insignificantly from zero at the 10% level. In the instrumental estimation (column 10), none of the NPI related coefficients differ significantly from zero at the 10% level. Thus, the inclusion of the PHRT variable still does not provide much statistical support for the hypothesis that non-pharmaceutical interventions matter for overall mortality rates. These weak statistical findings run counter to the epidemiologists' stress on implementing NPIs quickly to curtail the spread of the disease.[13]

For the relative peak death rate, in columns 11 and 12, the regressions still show significantly negative effects from the NPI variable. The results also show, surprisingly, negative estimated coefficients on the PHRT variable; that is, a longer delay is estimated to

[13]See Markel, et al. (2007, p. 648); Hatchett, Mecher, and Lipsitch (2007, pp. 7583-7584); and Bootsma and Ferguson (2007, p. 7591).

reduce the relative peak death rate. The estimated coefficient is significant at the 10% level in the case of OLS (column 11) and at the 5% level for TSLS (column 12). These results reflect interactions between PHRT and NPI—these two forms of interventions are inversely related (simple correlation coefficient of -0.54), meaning that places with more NPIs tended to respond with a shorter delay. Therefore, the simple correlation (0.10) between PHRT and the relative peak death rate reflects partly a proxying of larger PHRT for lower NPI (which has a substantially positive estimated effect on the relative peak death rate). Once the NPI variable is held fixed, as in columns 11 and 12, the coefficient on PHRT becomes negative.

To interpret these results, imagine that an NPI is put into effect with a duration of 30 days. If PHRT=0, the NPI is in place from a point near the beginning of the epidemic. In contrast, if the start of the NPI is delayed by a week, so that PHRT=0.02 years, the NPI is still in effect for 30 days but begins one week later and lasts one week further into the future. The results indicate that this rise in PHRT reduces the relative peak death rate. This effect likely arises because, with a higher PHRT over some range, the NPIs in place match up better with the highest death rates.

3. Additional effects of distance from Boston. A possible concern with the TSLS results in Table 2 is that greater distance from Boston, by allowing more time to prepare, may impact flu-related deaths directly, not just through influencing choices of NPIs. Since development of medical treatments (including a vaccine that turned out to be ineffective) would not have been relevant, the main possibility here is through preparation of healthcare facilities.[14]

[14]Another possibility is that delay could be useful by raising the chance of spontaneous disappearance of the virus during the relevant timeframe.

However, it is unclear in 1918-1919 that better healthcare facilities mattered a lot for flu-related mortality. Thus, this form of benefit from delaying the onset of disease may be unimportant.

A colleague suggested the hypothesis that cities further from Boston tended to be those that suffered more from the first wave of the Great Influenza—roughly January to April 1918—and, thereby, had more immunity against the second wave.[15] This idea can be checked by using the flu-related excess mortality rate for each city from January to April 1918. This variable, from Collins, et al. (1930, Appendix Table A), is in Appendix Table A1, column 15. The correlation of this excess death rate with distance from Boston turns out to be weakly negative, -0.12, not positive as conjectured. The excess death rate for January to April 1918 can also be added to the regressions in Table 2, columns 1 and 2, for explaining the overall flu-related excess death rate from September 1918 to February 1919. The estimated coefficients on the January-April 1918 variable are 0.40 (s.e.=0.68) for column 1 (OLS) and 0.47 (0.70) for column 2 (TSLS); that is, the coefficients differ insignificantly from zero. Hence, there is no evidence at the level of cities that greater exposure to the flu in the first wave diminished mortality rates in the second wave. (Note that these regressions hold fixed the flu-related gross mortality rate for the September-to-February periods of 1910-1916.)

4. Measured quarantine in New York City. As mentioned before, Barry (2007a, 2007b) has raised objections to the NPI data constructed by Markel, et al. (2007) for New York City.[16] The Markel data (supplement, slide 30) show that New York imposed a mandatory

[15]The first wave of the Great Influenza appeared in the United States at least by the end of February 1918 at an army base in Kansas—see Barry (2004, p. 169).

[16]Barry (2007b) also raises objections about measurements for Chicago, but these objections seem mainly to concern the form of presentation. Markel's (2007, supplement, slide 12) numbers for prohibitions of public gatherings and quarantine/isolation in Chicago are similar to those of Bootsma and Ferguson (2007, appendix, pp. 2-4).

quarantine starting September 26, 1918, effective for 73 days. There were no school closings and no prohibitions of public gatherings. Barry (2007a, 2007b) argues that the quarantine, while announced, was likely never seriously implemented, in which case it would be appropriate to treat New York as having an NPI of zero. However, Aimone's (2010) discussion indicates that some aspects of a quarantine did operate in New York. Given these uncertainties, it seems appropriate to compare the initial results, with New York's NPI reflecting a quarantine, with those modified to have no quarantine operating in New York.

The regressions in Table 2, cols. 1-8, were redone after modifying the NPI variable and the quarantine/isolation component for New York to equal 0, rather than the 0.2 years used before. This modification has minor effects on all of the results. In Table 2, columns 1 and 2, the estimated effects of the NPI variable on cumulative death rates remain negative but close to zero. In columns 3 and 4, the estimated effects of the NPI variable on the relative peak death rate are still negative and highly statistically significant. The estimated coefficient falls in magnitude from -5.9 (s.e.=1.3) to -5.5 (1.3) in column 3 and from -8.6 (2.0) to -8.4 (1.8) in column 4. Similarly, in columns 5-8, the results change only in minor ways with the revised treatment of New York.[17] Thus, the inference is that, even if Barry (2007a, 2007b)'s criticism of the measurement of quarantine in New York City is valid, a modification to account for this objection leaves the main results intact.

III. Concluding Observations

The regressions applied to 45 large U.S. cities during the peak of the Great Influenza Pandemic from September 1918 to February 1919. The results demonstrate that NPIs, as

[17]The regressions in columns 9-12 were not rerun because it was unclear how to define the PHRT variable for New York when it is viewed as having never implemented an NPI.

measured by Markel, et al. (2007), have large and statistically significant negative effects on relative peak flu-related excess death rates; that is, more interventions clearly flattened the curve for mortality. However, the impacts on overall death rates are much weaker and are mostly statistically insignificantly different from zero. Given the clear success in depressing the relative peak death rate, the key question is why the NPIs implemented in 1918-1919 were not more successful in depressing overall deaths.

There are two main possibilities. One is that the NPIs were not maintained long enough to have a substantial negative effect on overall deaths.[18] Table 1 shows that the mean durations of school closings and prohibitions of public gatherings were only 36 days (0.10 years), whereas that for quarantine/isolation was even shorter, 18 days (0.05 years). In this view, NPIs maintained for longer periods—as in the interventions deployed currently in the United States and elsewhere to combat the COVID-19 pandemic—would have had more payoff in terms of avoiding overall deaths.

The second possibility is that NPIs mostly delay deaths (consistent with the clear flattenings of the curves) but do not avoid them (consistent with the weak or null effects on overall deaths). This assessment makes it much less likely that extensive NPIs are a good idea and tends, instead, to favor the approach currently employed in Sweden, whereby only limited NPIs are used to attempt to curtail the spread of COVID-19. It is also possible that this second view was accurate for the Great Influenza Pandemic in 1918-1919 but not for the ongoing COVID-19 Pandemic, where delays in the spread of disease actually allow for improvements in healthcare facilities and medical treatments.

[18]This explanation is favored by Hatchett, Mecher, and Lipsitch (2007, p. 7582) and Bootsma and Ferguson (2007, p. 7588). However, these conclusions were not clearly related to the statistical findings.

The difference between interpretations one and two is of great importance for guiding NPI policies aimed at curbing deaths from the COVID-19 Pandemic and possible future pandemics. One piece of evidence from the Great Influenza that favors the first view comes from Australia. Because of a swift and strict maritime quarantine policy, Australia managed to avoid the Pandemic entirely during 1918 (see Barry [2004, pp. 375-376]). In fact, Australia was the only one of the 48 countries studied in Barro, Ursúa, and Weng (2020, Table 1) that recorded a non-positive flu-related excess death rate in 1918. Most significantly, Australia's avoidance of an outbreak in 1918 did not lead to higher mortality once the flu arrived in early 1919. Australia's overall flu-related death rate for 1918-1920 was only 0.3%, much lower than the average of 1.4% for the 48 countries. Moreover, the presence of Australia in the southern hemisphere does not account for this outcome—New Zealand and South Africa had much higher overall flu-related morality rates in 1918-1920 (0.7% and 3.4%, respectively). Thus, there is an indication that Australia's strong NPI in the form of a strict maritime quarantine did reduce overall flu-related death rates.

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Table 1

Descriptive Statistics

Variable:	**NPI**	**School closings**	**Public gatherings**	**Quarantine**	**PHRT**	**Excess death rate 9/18-2/19**
Units	years	years	years	years	years	percent of population
Mean	0.24	0.10	0.09	0.05	0.02	0.52
Median	0.18	0.08	0.08	0	0.02	0.53
Std dev.	0.13	0.06	0.05	0.07	0.02	0.15
Max	0.47	0.29	0.22	0.26	0.10	0.82
Min	0.08	0	0	0	-0.03	0.22
Variable:	**Relative peak death rate**	**Distance Boston**	**Death rate 1910-1916**	**Heating-degree days**	**POP 1910**	**POP density 1910**
Units	ratio	1000 miles	percent of population	degrees Fahrenheit	1000s	1000s/sq. mile
Mean	4.57	1.01	0.086	11.0	434	8.6
Median	4.43	0.83	0.086	11.9	224	7.5
Std dev.	1.32	0.92	0.023	3.8	760	4.5
Max	7.93	3.10	0.147	16.9	4768	18.6
Min	2.82	0	0.040	1.0	100	1.7

Note: These statistics apply to the variables defined and shown in Appendix Table A1.

Table 2

Effects from Non-Pharmaceutical Interventions (NPIs)

45 U.S. Cities, September 1918-February 1919

Dependent variable:	**Overall Flu-Related Excess Death Rate**		**Relative Peak Death Rate**	
	(1)	**(2)**	**(3)**	**(4)**
Method:	OLS	TSLS	OLS	TSLS
Constant	0.41** (0.14)	0.54** (0.20)	5.96*** (0.36)	6.61*** (0.50)
Non-pharmaceutical interventions, NPI	-0.11 (0.17)	-0.33 (0.30)	-5.9*** (1.3)	-8.6*** (2.0)
Flu-related gross death rate 1910-1916	2.90*** (0.94)	2.24* (1.22)	--	--
Heating-degree days	-0.0105** (0.0049)	-0.0123** (0.0054)	--	--
R-squared	0.37	0.35	0.31	0.25
Standard error of regression	0.119	0.121	1.11	1.16
Number of observations	45	45	45	45

Table 2, continued

Dependent variable:	Excess Death Rate	Excess Death Rate	Relative Peak Death Rate	Relative Peak Death Rate
	(5)	(6)	(7)	(8)
Method:	OLS	TSLS	OLS	TSLS
Constant	0.34**	0.50**	5.86***	6.52***
	(0.14)	(0.20)	(0.39)	(0.54)
School closings	0.51	0.27	-6.7*	-8.3**
	(0.41)	(0.48)	(3.6)	(3.9)
Prohibitions on public gatherings	-0.97**	-1.24**	-3.2	-7.5
	(0.47)	(0.55)	(4.4)	(5.2)
Quarantine/isolation	0.12	-0.16	-7.2***	-9.4***
	(0.28)	(0.39)	(2.3)	(2.8)
p-value, 3 NPIs same coefficients	0.16	0.18	0.74	0.90
p-value, 3 NPI coefficients=0	0.25	0.18	0.001	0.001
Flu-related gross death rate 1910-16	3.33***	2.53**	--	--
	(0.96)	(1.24)		
Heating-degree days	-0.0070	-0.0091	--	--
	(0.0051)	(0.0056)		
R-squared	0.43	0.39	0.32	0.26
Standard error of regression	0.12	0.12	1.13	1.18
Number of observations	45	45	45	45

Table 2, continued

Dependent variable:	**Overall Flu-Related Excess Death Rate**		**Relative Peak Death Rate**	
	(9)	**(10)**	**(11)**	**(12)**
Method:	OLS	TSLS	OLS	TSLS
Constant	0.25 (0.15)	0.36 (0.24)	6.69*** (0.51)	7.46*** (0.72)
Non-pharmaceutical interventions, NPI	0.15 (0.21)	-0.03 (0.38)	-7.46*** (1.52)	-10.0*** (2.3)
Public-health response time, PHRT	2.19** (1.06)	1.61 (1.45)	-17.7* (9.0)	-25.8** (10.7)
p-value, NPI & PHRT jointly	0.11	0.14	0.000	0.000
Flu-related gross death rate 1910-1916	3.65*** (0.98)	3.11** (1.36)	--	--
Heating-degree days	-0.0113** (0.0047)	-0.0120** (0.0049)	--	--
R-squared	0.43	0.42	0.37	0.33
Standard error of regression	0.114	0.116	1.07	1.11
Number of observations	45	45	45	45

***Significant at 1% level.
**Significant at 5% level.
*Significant at 10% level.

Notes to Table 2

The sample applies to 45 large U.S. cities observed from week ending September 14, 1918 to that ending February 22, 1919. The dependent variable in columns 1, 2, 5, 6, 9, and 10 is the overall excess flu-related death rate, given in Appendix Table A1, column 1. The dependent variable in columns 3, 4, 7, 8, 11, and 12 is the relative peak death rate, given in Appendix Table A1, column 2. Standard errors of coefficients are in parentheses. OLS is ordinary least-squares. TSLS is two-stage least-squares. In column 2, the instrumental variables are the distance from Boston and its square, the mayor dummy, the flu-related gross mortality rate for the September-February periods of 1910-1916, and heating-degree days. In column 4, the instrumental variables are the distance from Boston and its square and the mayor dummy. In columns 6 and 8, the instrument lists include also the difference between school closings and prohibitions of public gatherings and between school closings and quarantine. In columns 11 and 12, the instrument lists include also the PHRT variable. All variables are shown in Appendix Table A1.

Figure 1

Evolution of Flu-Related Excess Death Rates in Selected Cities

September 1918-February 1919

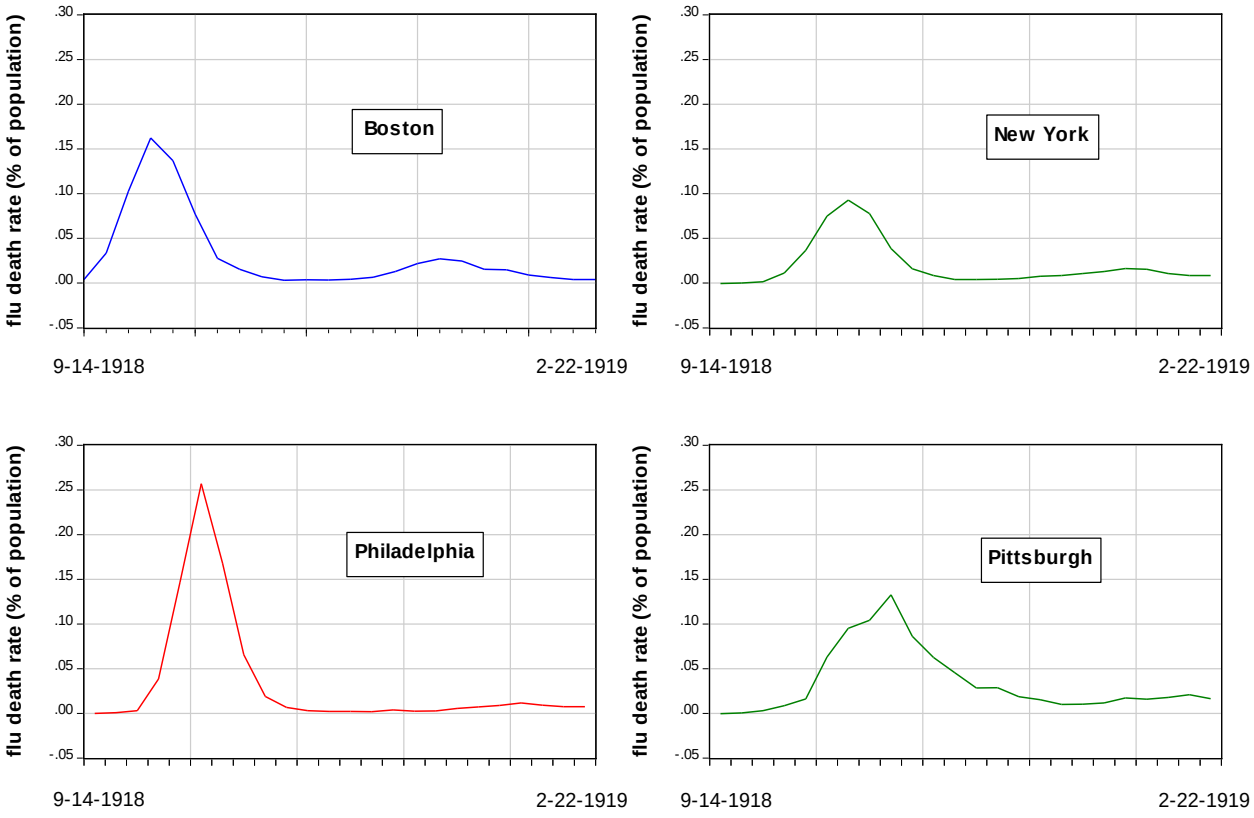
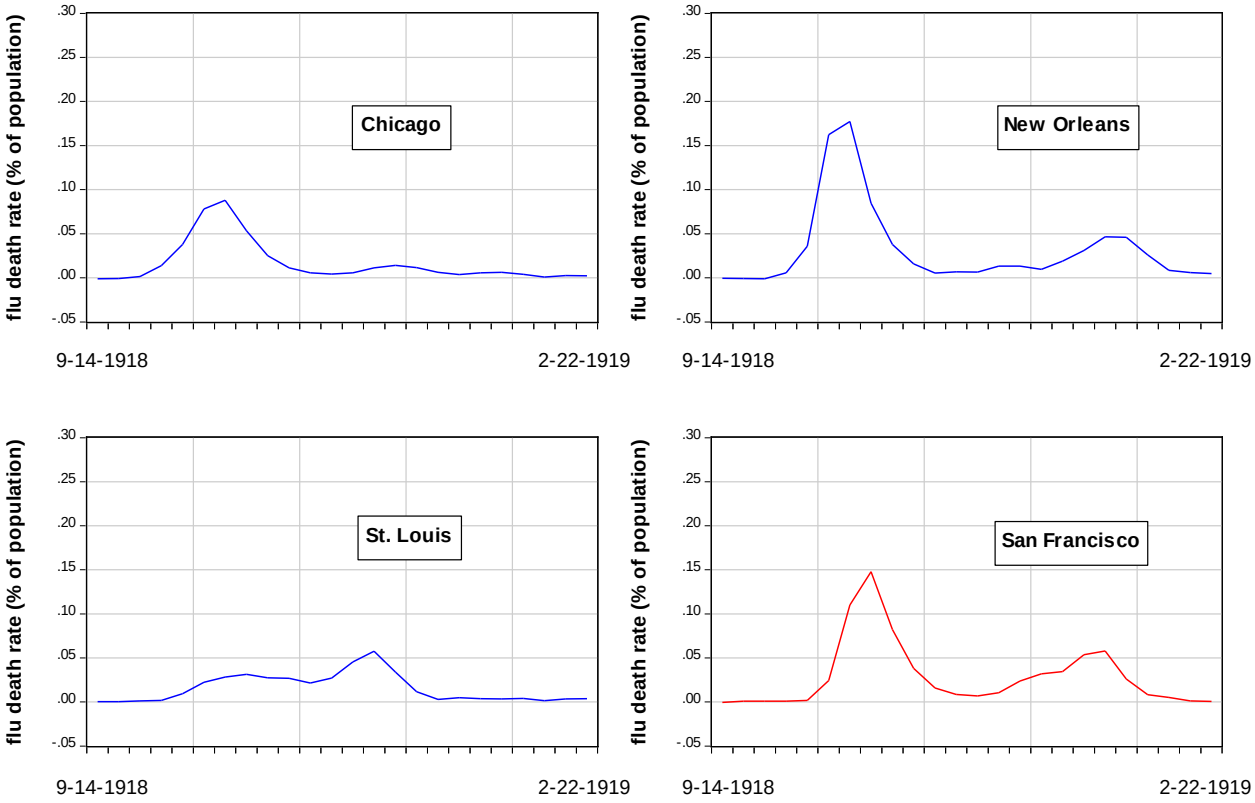


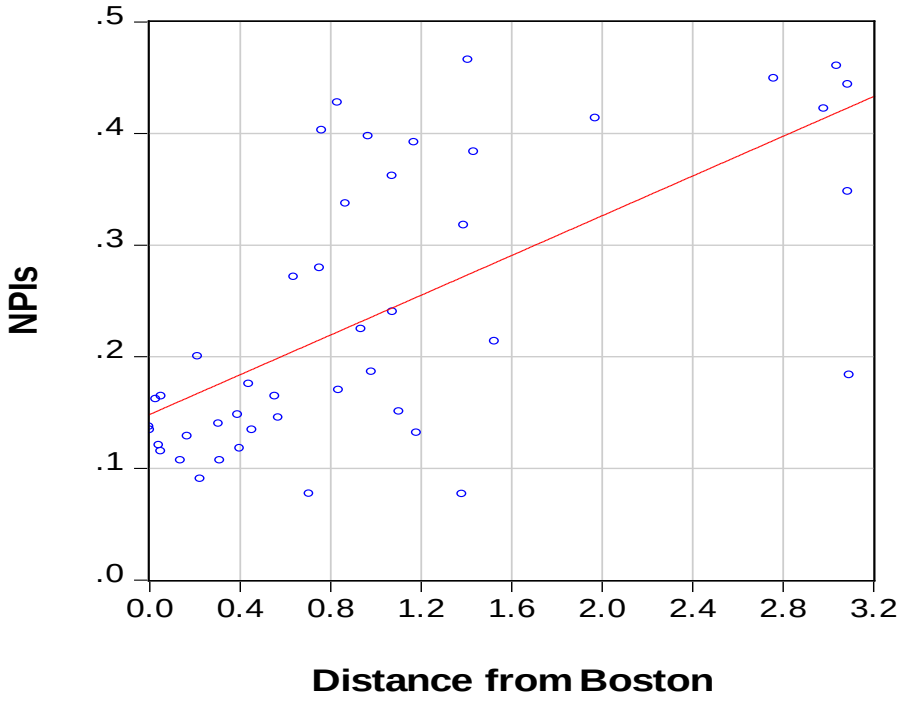
Figure 1, continued



Note: The graphs show the evolution of the weekly flu-related excess death rate (percent of city population) for each city from the week ending September 14, 1918 to that ending February 22, 1919. Data are from Collins, et al. (1930, Appendix Table B).

Figure 2

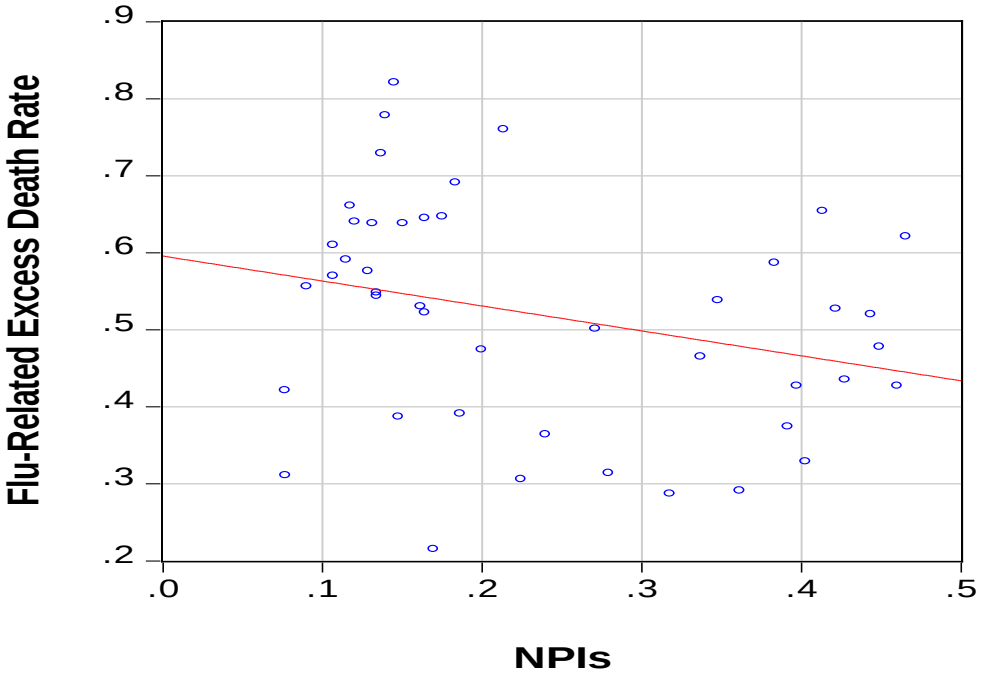
Relationship between Distance from Boston (thousands of miles) and NPIs (years of implementation)



Note: The sample is for 45 U.S. cities. Distance from Boston is the minimum distance shown by *Google Maps*. Non-Pharmaceutical Interventions (NPIs) from September 1918 to February 1919 are from Markel, et al. (2007, Table 1), updated to include Atlanta and Detroit. See Appendix Table A1.

Figure 3

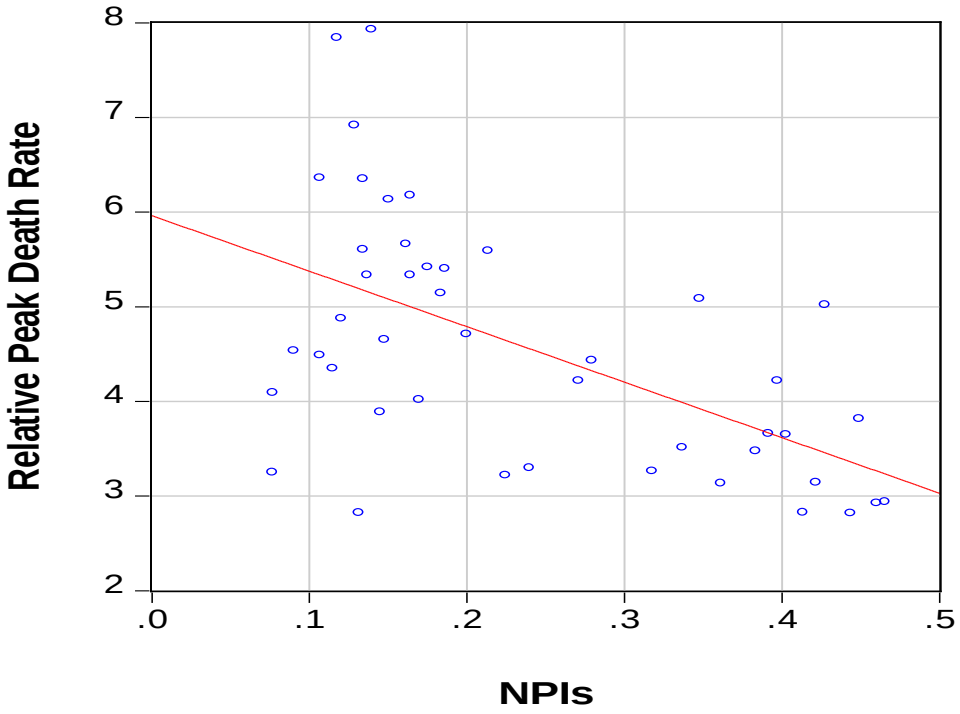
Relationship between NPIs and Excess Flu-Related Death Rate



Note: The sample is for 45 U.S. cities. NPIs from September 1918 to February 1919 is from Markel, et al. (2007, Table 1), updated to include Atlanta and Detroit. Overall flu-related excess death rate for September 1918 to February 1919 is calculated from Collins, et al. (1930, Appendix, Table B). See Appendix Table A1.

Figure 4

Relationship between NPIs and Relative Peak Death Rate

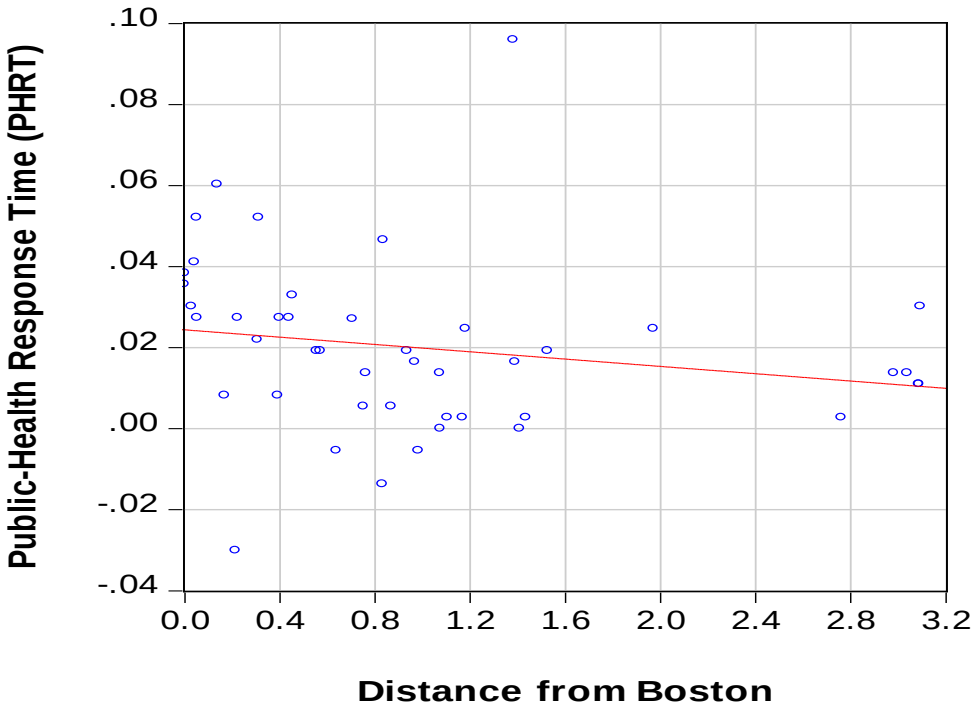


Note: The sample is for 45 U.S. cities. NPIs from September 1918 to February 1919 is from Markel, et al. (2007, Table 1), updated to include Atlanta and Detroit. The relative peak death rate, defined as the ratio of the peak weekly death rate to the average of the weekly death rates, is calculated from Collins, et al. (1930, Appendix, Table B). See Appendix Table A1.

Figure 5

Relationship between Distance from Boston (thousands of miles)

and Public-Health Response Time (PHRT in years)



Note: The sample is for 45 U.S. cities. Distance from Boston is the minimum distance shown by *Google Maps*. The public-health response time or PHRT is from Markel, et al. (2007, Table 1), updated to include Atlanta and Detroit. See Appendix Table A1.

Appendix Table A1 Data Used in Regressions

	(1)	(2)	(3)	(4)	(5)
City	**Excess death rate 9/18-2/19**	**Relative peak death rate**	**NPI**	**School closings**	**Public gatherings**
Albany	0.576	6.917	0.129	0.090	0.038
Atlanta	0.364	3.297	0.240	0.112	0.049
Baltimore	0.661	7.843	0.118	0.071	0.047
Birmingham	0.638	2.821	0.132	0.071	0.060
Boston	0.729	5.333	0.137	0.071	0.066
Buffalo	0.548	6.350	0.134	0.077	0.058
Cambridge	0.544	5.603	0.134	0.071	0.063
Chicago	0.391	5.402	0.186	0.000	0.107
Cincinnati	0.465	3.510	0.337	0.173	0.164
Cleveland	0.501	4.216	0.271	0.063	0.077
Columbus	0.329	3.647	0.403	0.186	0.216
Dayton	0.435	5.021	0.427	0.093	0.222
Denver	0.654	2.826	0.414	0.219	0.093
Detroit	0.311	4.090	0.077	0.030	0.047
Fall River	0.645	6.177	0.164	0.088	0.077
Grand Rapids	0.215	4.019	0.170	0.044	0.077
Indianapolis	0.306	3.216	0.225	0.104	0.066
Kansas City MO	0.621	2.937	0.466	0.205	0.115
Los Angeles	0.527	3.142	0.422	0.290	0.132
Louisville	0.427	4.215	0.397	0.162	0.162
Lowell	0.530	5.660	0.162	0.082	0.079
Milwaukee	0.291	3.134	0.362	0.107	0.148
Minneapolis	0.287	3.261	0.318	0.156	0.148
Nashville	0.638	6.132	0.151	0.071	0.079
New Haven	0.610	4.485	0.107	0.000	0.107
New Orleans	0.760	5.589	0.214	0.112	0.101
New York	0.474	4.709	0.200	0.000	0.000
Newark	0.556	4.532	0.090	0.052	0.038
Oakland	0.538	5.086	0.348	0.110	0.088
Omaha	0.587	3.475	0.384	0.077	0.118
Philadelphia	0.778	7.928	0.140	0.077	0.063
Pittsburgh	0.821	3.888	0.145	0.068	0.077
Portland OR	0.520	2.815	0.444	0.101	0.096
Providence	0.591	4.345	0.115	0.060	0.055
Richmond	0.522	5.333	0.164	0.082	0.082
Rochester	0.387	4.651	0.148	0.074	0.074
San Francisco	0.691	5.140	0.184	0.101	0.082
Seattle	0.427	2.923	0.460	0.099	0.101
Spokane	0.478	3.816	0.449	0.189	0.181
St. Louis	0.374	3.658	0.392	0.200	0.192
St. Paul	0.421	3.249	0.077	0.033	0.044
Syracuse	0.570	6.358	0.107	0.058	0.049
Toledo	0.314	4.433	0.279	0.142	0.137
Washington DC	0.647	5.416	0.175	0.088	0.088
Worcester	0.640	4.875	0.121	0.060	0.060

Table A1, continued

City	(6) Quarantine	(7) Response time, PHRT	(8) Death rate 1910-1916	(9) Heating-degree days	(10) Distance from Boston
Albany	0.000	0.008	0.093	15.2	169
Atlanta	0.079	0.000	0.104	5.6	1076
Baltimore	0.000	0.027	0.115	8.3	401
Birmingham	0.000	0.025	0.110	5.4	1182
Boston	0.000	0.036	0.110	12.2	0
Buffalo	0.000	0.033	0.080	14.7	455
Cambridge	0.000	0.038	0.098	12.2	3
Chicago	0.079	-0.005	0.111	12.3	983
Cincinnati	0.000	0.005	0.091	10.5	869
Cleveland	0.132	-0.005	0.067	11.4	640
Columbus	0.000	0.014	0.074	11.8	763
Dayton	0.112	-0.014	0.078	12.4	833
Denver	0.101	0.025	0.086	13.1	1972
Detroit	0.000	0.027	0.083	13.0	707
Fall River	0.000	0.027	0.108	12.9	53
Grand Rapids	0.049	0.047	0.045	14.5	837
Indianapolis	0.055	0.019	0.076	11.9	936
Kansas City MO	0.145	0.000	0.081	10.3	1410
Los Angeles	0.000	0.014	0.060	1.0	2983
Louisville	0.074	0.016	0.083	8.9	969
Lowell	0.000	0.030	0.097	14.2	30
Milwaukee	0.107	0.014	0.066	14.8	1074
Minneapolis	0.014	0.016	0.067	16.9	1391
Nashville	0.000	0.003	0.122	7.6	1105
New Haven	0.000	0.060	0.119	12.4	138
New Orleans	0.000	0.019	0.114	1.5	1526
New York	0.200	-0.030	0.103	10.2	215
Newark	0.000	0.027	0.088	10.1	225
Oakland	0.151	0.011	0.064	2.6	3089
Omaha	0.189	0.003	0.073	13.1	1435
Philadelphia	0.000	0.022	0.091	10.7	308
Pittsburgh	0.000	0.019	0.147	11.4	572
Portland OR	0.247	0.011	0.045	8.5	3088
Providence	0.000	0.052	0.099	14.4	51
Richmond	0.000	0.019	0.108	8.2	556
Rochester NY	0.000	0.008	0.073	14.6	392
San Francisco	0.000	0.030	0.074	3.7	3095
Seattle	0.260	0.014	0.040	9.4	3039
Spokane	0.079	0.003	0.055	15.6	2762
St. Louis	0.000	0.003	0.098	10.5	1171
St. Paul	0.000	0.096	0.053	16.0	1383
Syracuse	0.000	0.052	0.077	15.1	312
Toledo	0.000	0.005	0.066	13.6	754
Washington DC	0.000	0.027	0.086	8.3	440
Worcester	0.000	0.041	0.096	12.6	43

Table A1, continued

	(11)	(12)	(13)	(14)	(15)
City	**Mayor dummy**	**Population 1910**	**Area**	**Population Density 1910**	**Excess death rate, 1-4/18**
Albany	1	100.3	10.8	9.3	-0.002
Atlanta	1	154.8	25.7	6.0	0.051
Baltimore	1	558.5	30.1	18.6	0.036
Birmingham	0	132.7	48.3	2.7	0.119
Boston	1	670.6	41.1	16.3	0.035
Buffalo	0	423.7	38.7	10.9	0.022
Cambridge	1	104.8	6.3	16.6	0.029
Chicago	1	2185.3	185.1	11.8	-0.019
Cincinnati	1	363.6	49.8	7.3	0.033
Cleveland	1	560.7	45.6	12.3	0.031
Columbus	1	181.5	20.3	8.9	0.019
Dayton	0	116.6	15.7	7.4	0.010
Denver	1	213.4	57.9	3.7	0.022
Detroit	1	465.8	40.8	11.4	0.026
Fall River	1	119.3	33.9	3.5	-0.029
Grand Rapids	0	112.6	16.8	6.7	0.013
Indianapolis	1	233.6	33.0	7.1	0.031
Kansas City MO	1	248.4	58.5	4.2	0.063
Los Angeles	1	319.2	99.2	3.2	-0.005
Louisville	1	223.9	20.7	10.8	0.065
Lowell	0	106.3	13.0	8.2	0.045
Milwaukee	1	373.9	22.8	16.4	0.022
Minneapolis	1	301.4	50.1	6.0	0.016
Nashville	0	110.4	17.1	6.5	0.091
New Haven	1	133.6	17.9	7.5	0.007
New Orleans	0	339.1	196.0	1.7	0.014
New York	1	4767.9	286.8	16.6	0.026
Newark	0	347.5	23.2	15.0	0.036
Oakland	0	150.2	45.7	3.3	0.022
Omaha	0	124.1	24.1	5.1	0.019
Philadelphia	1	1549.0	130.2	11.9	0.050
Pittsburgh	1	533.9	41.4	12.9	0.126
Portland OR	0	207.2	48.4	4.3	0.014
Providence	1	224.3	17.7	12.7	0.020
Richmond	1	127.6	10.0	12.8	-0.003
Rochester NY	1	218.1	20.1	10.9	0.020
San Francisco	1	416.9	46.5	9.0	0.016
Seattle	1	237.2	55.9	4.2	0.014
Spokane	0	104.4	36.8	2.8	0.013
St. Louis	1	687.0	61.4	11.2	0.037
St. Paul	0	214.7	52.2	4.1	0.011
Syracuse	1	137.2	17.3	7.9	0.032
Toledo	1	168.5	25.0	6.7	0.021
Washington DC	0	331.1	60.0	5.5	0.043
Worcester	1	146.0	37.0	3.9	0.023

Notes to Table A1

The sample comprises 45 of the 50 U.S. cities with center-city populations in 1910 above 100,000. These 45 have weekly data on flu-related excess death rates over the second and most deadly wave of the Great Influenza Pandemic: the 24 weeks from week ending September 14, 1918 to week ending February 22, 1919. The remaining 5 large cities lack parts of the weekly data over this period.

Flu-related death rates are calculated as percent of city population. Excess death rate 9/18-2/19 is the overall flu-related excess death rate over the 24-week sample from the week ending September 14, 1918 to the week ending February 22, 1919. These values are calculated from the weekly data given in Collins, et al. (1930, Appendix Table B). (A typo in the data for Pittsburgh for November 23, 1918 was corrected based on the information in Davis [1918].) The excess death rate is the difference between the rate for each city and week and the median of flu-related death rates for the corresponding city and week for 1910-1916. The relative peak death rate is the ratio of the highest weekly death rate in the 24-week sample to the average death rate (the overall value divided by 24). NPI is the duration in years of three forms of non-pharmaceutical interventions—school closings, prohibition of public gatherings, and quarantine/isolation—over the 24-week sample, as estimated by Markel, et al. (2007, Table 1 and supplemental figures). Data on NPIs for Atlanta and Detroit were obtained from information in, respectively, *The Atlanta Constitution* and *The Detroit Free Press*, Public Health Reports for the two cities, and the *Influenza Encyclopedia*, available at influenzaarchive.org. The three forms of NPIs are shown separately, and NPI is the sum of these three. The public-health response time or PHRT, defined in Markel, et al. (2007, Table 1), is the difference in years between the date of the first NPI implementation and the time when the weekly excess flu-related death rate reached twice the median gross death rate applicable to the corresponding month in 1910-1916.

The flu-related gross death rate for 1910-1916 is calculated from the median values shown for the September-February months in Collins, et al. (1930, Appendix Table A). Heating-degree days, based on average daily temperature and a target of 60 degrees Fahrenheit, is from weatherdatadepot.com and applies around the year 2000. The data are for the full calendar year but are expressed per day. Distance from Boston is the minimum distance in thousands of miles from *Google Maps*. Mayor dummy is for the presence of a strong elected mayor in 1918, as opposed to a Commission or other form of city government. The underlying information comes from CQ Researcher (1930) and *Wikipedia*. Population 1910 (in thousands) and area (in square miles) are for central cities from 1910 U.S. Census. Population density is the ratio of population to area. Excess death rate 1-4/18 is the overall flu-related excess death rate for the 4-month period from January 1918 to April 1918 from the monthly data given in Collins, et al. (1930, Appendix Table A).