

NBER WORKING PAPER SERIES

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

Robert J. Barro

Working Paper 27049
<http://www.nber.org/papers/w27049>

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
April 2020

I have benefited from comments by Martin Cetron, Sergio Correia, Ed Glaeser, and Chris Meissner. The views expressed herein are those of the author and do not necessarily reflect the views of the National Bureau of Economic Research.

NBER working papers are circulated for discussion and comment purposes. They have not been peer-reviewed or been subject to the review by the NBER Board of Directors that accompanies official NBER publications.

© 2020 by Robert J. Barro. All rights reserved. Short sections of text, not to exceed two paragraphs, may be quoted without explicit permission provided that full credit, including © notice, is given to the source.

Non-Pharmaceutical Interventions and Mortality in U.S. Cities during the Great Influenza Pandemic, 1918-1919

Robert J. Barro

NBER Working Paper No. 27049

April 2020, Revised May 2020

JEL No. I1,I15

ABSTRACT

Non-pharmaceutical public-health interventions (NPIs) were measured by Markel, et al. (2007) for U.S. cities during the second wave of the Great Influenza Pandemic, September 1918-February 1919. The NPIs are in three categories: school closings, prohibitions on public gatherings, and quarantine/isolation. Although an increase in NPIs flattened the curve in the sense of reducing the ratio of peak to average deaths, the estimated effect on overall deaths is small and statistically insignificant. The likely reason that the NPIs were not more successful in curtailing mortality is that the interventions had an average duration of only around one month.

Robert J. Barro

Department of Economics

Littauer Center 218

Harvard University

Cambridge, MA 02138

and NBER

rbarro@harvard.edu

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

An important issue is how public-health interventions, such as those being implemented currently in most countries for the ongoing coronavirus pandemic, affect mortality. For the Great Influenza Pandemic, information of this type is unavailable across countries but does exist for U.S. cities. The present study assesses the effects of these interventions on death rates.

Non-Pharmaceutical Interventions and Flu-Related Death Rates

Markel, et al. (2007) studied effects of non-pharmaceutical public-health interventions (NPIs) on flu-related deaths in large U.S. cities over the 24-week period corresponding to the peak of the epidemic, September 1918-February 1919. The weekly data on flu-related excess death rates in this period come from the 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 cities, corresponding to those for which the central-city population in the 1910 U.S. Census exceeded 100,000.¹

The extensive research by Markel, et al. (2007, Table 1 and appendix figures) included collection of data on NPIs for 43 of the 45 cities covered by the weekly Census data on

¹The five missing cities are Bridgeport, Jersey City, Memphis, Paterson, and Scranton.

mortality. (Atlanta and Detroit were excluded because of missing information.) The 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 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 I discuss these objections below.

The present analysis considers two characteristics of each city's flu-related excess death rates: first, the cumulative 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 cumulative death rate divided by 24.) A lower cumulative 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 cumulative 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.

²Hatchett, 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). Velde (2020, Figure 4) constructed measures of business closings for 42 cities (those considered by Markel plus Atlanta and Detroit, with Lowell, Milwaukee, and New Haven omitted). Measures of NPIs across countries in 2020 are in UBS (2020).

³Correia, 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 cities. Lilley, Lilley, and Rinaldi (2020) critique the methodology used by Correia, Luck, and Verner.

There is some concern that NPIs (measured, say, by length of time in force) and flu-related death rates are simultaneously determined. 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 Markel, et al. (2007), Hatchett, et al. (2007), and Bootsma and Ferguson (2007) is that NPIs are determined exogenously; that is, shifts in actual or anticipated death rates do not impact the chosen NPIs. The present research attempts to take account of the potential endogeneity of NPIs.

An important consideration is that the second wave of the U.S. flu epidemic began 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.⁴ 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, the distance from Boston may serve as an exogenous measure of how early the flu epidemic tended to reach the various cities 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 implementation of NPIs.⁵ Empirically, distance from Boston, entered in a quadratic form, has considerable explanatory power for the NPI variable. The pair-wise relationship between distance and NPIs, shown in Figure 1, is positive with a simple correlation coefficient of 0.65. Because of this strong relationship, the distance measures are good candidates as instrumental variables related to NPIs.

⁴For a discussion, see Barry (2004, pp. 181 ff.).

⁵Possibly the distance measure could be improved by calculating the time required to transit from Boston to a particular location, given the transportation technology available in 1918. For example, physical distance would overstate the time required to reach places, such as New Orleans, that were accessible from Boston by boat.

Regression Results

Table 1 has regression results for the 43 U.S. cities in the 24-week period from September 8, 1918 to February 22, 1919. Descriptive statistics for the variables used are in Table 2. The analysis in Table 1 considers two dependent variables: the cumulative excess flu-related death rate and the relative peak death rate.

Flu-related death rates in each city likely depend on healthcare facilities, age structure, and other demographic characteristics. To take account of these effects, the analysis includes as an explanatory variable the flu-related gross mortality rate from a prior period⁶—specifically, the median rate for 1910-1916, corresponding to the months, September-February, used for the dependent variable. This variable is calculated from the information given in Collins, et al. (1930, appendix, Table A). The idea is that this measure would reflect city characteristics such as healthcare facilities and demographics to the extent that they influence flu-related deaths in general and, therefore, likely the excess deaths experienced during the 1918-1919 pandemic. The analysis also includes as an additional explanatory variable a measure of a city's weather, given by the log of heating-degree days.

Columns 1 and 2 of Table 1 show estimated effects of NPIs on the cumulative flu-related excess death rate. The pair-wise association is in Figure 2. This relationship is negative, as expected (simple correlation coefficient equals -0.34). The estimated coefficients on NPIs in columns 1 and 2 are negative but insignificantly different from zero. Results from OLS and two-stage least-squares are similar,⁷ indicating that treating NPIs as exogenous—as in Markel, et al.

⁶A variable of this type was used by Bootsma and Ferguson (2007, p. 7588).

⁷A possible concern with the TSLS results is that greater distance, by allowing more time to prepare, may have impacted flu-related deaths directly, not just through influencing NPIs. Since development of medical treatments would not have been relevant, the main possibility here is likely to be through preparation of healthcare facilities. In any event, the estimated effects of NPIs on cumulative death rates turned out to be minor.

(2007), Hatchett, et al. (2007), and Bootsma and Ferguson (2007)—may be satisfactory for this part of the analysis.⁸

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. The coefficient of the log of heating-degree days is negative and statistically significant at the 5% level. This result indicates, surprisingly, that colder places (during fall and winter) are estimated to have significantly lower excess flu-related death rates.⁹

Columns 3 and 4 of Table 1 consider the estimated effects of NPIs on the relative peak death rate. The pairwise association is in Figure 3. This relationship is negative, as expected, with a simple correlation coefficient of -0.59. The estimated coefficients on the NPI variable in columns 3 (OLS) and 4 (TSLs) are negative and highly statistically significant, with the coefficient under TSLs notably larger in magnitude. The higher magnitude under TSLs likely arises because a higher relative peak death rate encourages 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.¹⁰ 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—as measured by Markel, et al. (2007)—do matter. But this influence shows up far more in the relative peak death rate than in the cumulative death rate.

⁸The instrumental variables aside from distance to Boston and its square are the gross flu-related death rate for 1910-16 and the log of heating-degree days. The F-Statistic for the first-stage equation for NPIs is 10.8. Most of the explanatory power here comes from the two distance variables.

⁹The simple correlation coefficient between the log of heating-degree days and the gross flu-related mortality rate for 1910-1916 is positive but close to zero.

¹⁰Measurement error in the NPI variable can also be a source of this attenuation.

Columns 5-8 of Table 1 consider the separate roles of the three types of NPIs considered by Markel, et al. (2007)—school closings, prohibitions of public gatherings, and quarantine/isolation. There are insufficient instruments to allow for endogeneity of all three types of NPIs 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 5 and 8 includes on the instrument list the variables used before in columns 2 and 4 along with the difference between the duration of the various NPIs (school closings minus prohibitions of public gatherings and school closings minus quarantine/isolation).

For the cumulative 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 reject the hypothesis at a p-value of 0.03 that the coefficients of the three NPI variables are the same. The results reject the hypothesis that the coefficients of all three NPI variables equal zero with a p-value of 0.04 in column 5 (OLS) and 0.06 in column 6 (TSLS). The estimated coefficients are similar from OLS and TSLS, indicating, as in columns 1 and 2, that it may be satisfactory to treat overall NPIs as exogenous with respect to the cumulative flu death rate. There is also some evidence that restrictions on public gatherings retarded cumulative excess flu death rates.

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 higher under TSLS (column 8) compared to that under OLS (column 7). The TSLS results (column 8) show that the magnitudes of the effects of the three types of NPIs are similar, with the hypothesis of

equal coefficients accepted with a p-value of 0.95. The three coefficients are jointly significantly different from zero, with a p-value of 0.001. An inference from the results is that, for the analysis of relative peak death rates, it is satisfactory to combine the three types of interventions into a single form, as done in the first part of the table (column 4).

Public-Health Response Time

Columns 9 and 10 of Table 1 consider another measure of how NPIs were implemented—the public-health response time or PHRT constructed by Markel, et al. (2007, Table 1).¹¹ A higher PHRT indicates more delay in a city introducing the first intervention intended to retard flu deaths. Figure 4 shows from the pair-wise relationship that the PHRT variable is negatively but weakly associated with distance from Boston, with a simple correlation coefficient of -0.20. Thus, while being further away from Boston clearly raises the number of NPIs employed (Figure 1), 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 cumulative excess death rate (0.16) and the relative peak death rate (0.08).

Because the available instruments are insufficient to distinguish the NPI and PHRT variables, columns 9 and 10 of Table 1 present only OLS results. Column 9, for the cumulative flu death rate, shows that the estimated regression coefficient on PHRT is essentially zero. Column 10, for the relative peak death rate, shows, surprisingly, a significantly negative estimated coefficient on PHRT. This result applies even though the simple correlation between PHRT and the relative peak death rate is weakly positive (Figure 4). The regression result in column 10 reflects an interaction between PHRT and NPIs—these two forms of interventions are

¹¹Their definition of PHRT is the days between the date when the flu death rate reached twice a baseline death rate and the (usually later) date of the first non-pharmaceutical intervention.

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 between PHRT and the relative peak death rate (weakly positive) reflects partly a proxying of shorter PHRT for higher NPI (which has a substantially negative estimated effect on the relative peak death rate). Once the NPI variable is held fixed, as in column 10, the coefficient on PHRT, which reflects the effect on the relative peak death rate conditional on NPI, 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 start of the epidemic (where, according to Markel, et al. [2007, Table 1], the weekly death rate equals twice a baseline rate). 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 starts one week later and lasts one week further into the future. The results suggest that, in the relevant range, the rise in the PHRT does not matter much for cumulative deaths but does reduce the relative peak death rate. The latter effect likely arises because, with a higher PHRT, the NPIs in place match up better with the highest death rates.

Interpreting the Results

Consider what the results in Table 1 imply quantitatively about the effects of NPIs on flu-related death rates. There is clear evidence that an increase in NPIs (measured here by time in effect) smooths out deaths in the sense of reducing the relative peak death rate. Using the estimated coefficient of -8.9 on NPIs from the two-stage least-squares estimation in column 4, a one-standard-deviation rise in the NPI variable (by 0.13 in Table 2) lowers the relative peak death rate by 1.2, compared with the sample mean of 4.6 (Table 2).

In contrast, the effects on cumulative flu death rates are much weaker. Using the (statistically insignificant) point estimate of the coefficient on NPIs of -0.0010 from column 2, a rise in the NPI variable by 0.13 lowers the cumulative death rate by 0.0001, compared with the sample mean of 0.0052 (Table 2). Thus, the smoothing out of flu deaths may have accomplished nothing in terms of lowering overall mortality.

Measured Quarantine in New York City

As noted before, Barry (2007a, 2007b) has raised objections to the NPI data constructed by Markel, et al. (2007) for New York City.¹² The Markel data (appendix, slide 30) show that New York imposed a mandatory 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 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 the quarantine, with those modified to have no quarantine operating in New York.

The regressions for Table 1, 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 1, cols. 1 and 2, the estimated effects of the NPI variable on cumulative death rates remain negative but close to zero. In cols. 3 and 4, the estimated effects of the NPI variable on the relative peak death rate are still

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

negative and highly statistically significant. The estimated coefficient falls in magnitude from -6.2 (s.e.=1.3) to -5.8 (1.3) in col. 3 and from -8.9 (1.9) to -8.4 (1.8) in col. 4. Similarly, in cols. 5-8, the results change only in minor ways with the revised treatment of New York.¹³ Thus, the inference is that, while Barry (2007a, 2007b)'s criticism of the measurement of quarantine in New York City may be valid, a modification to account for this objection leaves the main results intact.

Concluding observations

Given the clear success in depressing the relative peak death rate, the likely reason that the NPIs implemented in 1918-1919 were ultimately not very successful in depressing overall deaths is that the NPIs were not maintained long enough. Table 2 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 18 days (0.05 years).

These conclusions accord in part with those reached by Hatchett, et al. (2007) and Bootsma and Ferguson (2007).¹⁴ The lesson for the ongoing coronavirus pandemic in 2020 is

¹³The regressions in cols. 9 and 10 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.

¹⁴According to Hatchett, et al. (2007, p. 7582): "... cities in which multiple interventions were implemented at an early phase of the epidemic had peak death rates [approximately] 50% lower than those that did not ... Cities in which multiple interventions were implemented at an early phase of the epidemic also showed a trend toward lower cumulative excess mortality, but the difference was smaller ([approximately] 20%) and less statistically significant than that for peak death rates. This finding was not unexpected, given that few cities maintained NPIs longer than six weeks in 1918." Similarly, Bootsma and Ferguson (2007, p. 7588) reported: "... we found the time-limited interventions used reduced total mortality only moderately (perhaps 10-30%), and that the impact was often very limited because of interventions being introduced too late and lifted too early." The present results accord with these conclusions related to the duration of NPIs but not with the those from delay, which were gauged by the public-health response time (PHRT). Given the NPI variable—that is, how long NPIs were employed—the results did not show that earlier action, in the range observed within the sample, significantly reduced cumulative or relative peak death rates.

that, to curtail overall deaths, the NPIs used have to be maintained for substantially longer than a few weeks. Most likely, 12 weeks work much better than 4-6 weeks.

References

- Aimone, Francesco (2010). “The 1918 Influenza Epidemic in New York City: A Review of the Public Health Response.” *Public Health Reports* 125 (Supplement 3), April: 71-79.
- Barro, Robert J., José F. Ursúa, and Joanna Weng (2020). “The Coronavirus and the Great Influenza Pandemic—Lessons from the ‘Spanish Flu’ for the Coronavirus’s Potential Effects on Mortality and Economic Activity.” National Bureau of Economic Research, working paper no. 26866, March.
- Barry, John M. (2004). *The Great Influenza*, New York, Penguin Group.
- Barry, John M. (2007a). “Nonpharmaceutical Interventions Implemented During the 1918-1919 Influenza Pandemic, Letter to the Editor.” *Journal of the American Medical Association*, November 11, 2260-2261.
- Barry, John M. (2007b). “Commentary,” in Michael T. Osterholm, “Commentary: Little Evidence for New York City Quarantine in 1918 Pandemic.” *CIDRAP News*, November 27, available at cidrap.umn.edu.
- Bootsma, Martin C.J. and Neil M. Ferguson (2007). “Public Health Interventions and Epidemic Intensity during the 1918 Influenza Pandemic.” *Proceedings of the National Academy of Sciences* 104 (18): 7588-7593.
- Collins, Selwyn D., Wade H. Frost, Mary Gover, and Edgar Sydenstricker (1930). “Mortality from Influenza and Pneumonia in 50 Large Cities of the United States, 1910-1929.” *Public Health Reports* 45 (39): 2277-2328, September 26.
- Correia, Sergio, Stephan Luck, and Emil Verner (2020). “Pandemics Depress the Economy, Public Health Interventions Do Not: Evidence from the 1918 Flu.” Unpublished, March 26.
- Davis, William H. (1918). “The Influenza Epidemic as Shown in the Weekly Health Index,” meeting of American Public Health Association, Chicago, December 9, available at ajph.aphapublications.org.
- Hatchett, Richard J., Carter E. Mecher, and Marc Lipsitch (2007). “Public Health Interventions and Epidemic Intensity during the 1918 Influenza Pandemic.” *Proceedings of the National Academy of Sciences* 104 (18): 7582-7587.
- Lilley, Andrew, Matthew Lilley, and Gianluca Rinaldi (2020). “Pandemics, Public Health and Economic Growth: Revisiting the Spanish Flu Evidence.” Unpublished, Harvard University, April.

Markel, Howard, Harvey B. Lipman, J. Alexander Navarro, Alexandra Sloan, Joseph R. Michalsen, Alexandra Minna Stern, and Martin S. Cetron. (2007). “Nonpharmaceutical Interventions Implemented by US Cities During the 1918-1919 Influenza Pandemic.” *Journal of the American Medical Association* 298 (6): 644-654.

UBS (2020). *Global Economic Perspectives*. March 26.

Velde, Francois R. (2020). “What Happened to the US Economy During the 1918 Influenza Pandemic? A View Through High-Frequency Data.” Federal Reserve Bank of Chicago, working paper 2020-11, April 17.

Table 1
Effects from Non-Pharmaceutical Interventions (NPIs)
43 U.S. Cities, September 1918-February 1919

Dependent variable:	Cumulative Flu Excess Death Rate		Relative Peak Death Rate	
	(1)	(2)	(3)	(4)
Method:	OLS	TOLS	OLS	TOLS
Constant	0.0046** (0.0013)	0.0044** (0.0019)	6.11*** (0.36)	6.75*** (0.49)
Non-pharmaceutical interventions (NPIs)	-0.0014 (0.0016)	-0.0010 (0.0028)	-6.2*** (1.3)	-8.9*** (1.9)
Flu-related gross death rate 1910-1916	3.18*** (0.86)	3.29*** (1.08)	--	--
Log(heating-degree days)	-0.00075** (0.00029)	-0.00074** (0.00031)	--	--
R-squared	0.45	0.45	0.35	0.28
Standard error of regression	0.0011	0.0011	1.09	1.15
Number of observations	43	43	43	43

Table 1, continued

Dependent variable:	Cumulative Flu Death Rate	Cumulative Flu Death Rate	Relative Peak Death Rate	Relative Peak Death Rate	Cumulative Flu Death Rate	Relative Peak Death Rate
	(5)	(6)	(7)	(8)	(9)	(10)
Method:	OLS	TSLS	OLS	TSLS	OLS	OLS
Constant	0.0038*** (0.0013)	0.0036** (0.0017)	6.03*** (0.40)	6.71*** (0.54)	0.0034** (0.0015)	7.00*** (0.51)
Non-pharmaceutical interventions, NPIs	--	--	--	--	0.0005 (0.0020)	-8.14*** (1.50)
School closings	0.0055 (0.0038)	0.0060 (0.0043)	-6.6* (3.6)	-8.3** (3.9)	--	--
Prohibitions on public gatherings	-0.0123*** (0.0043)	-0.0118** (0.0048)	-4.4 (4.5)	-8.7* (5.2)	--	--
Quarantine/isolation	0.0023 (0.0025)	0.0028 (0.0033)	-7.2*** (2.3)	-9.5*** (2.7)	--	--
p-value, 3 NPIs same coefficients	0.026	0.027	0.86	0.95	--	--
p-value, 3 NPI coefficients=0	0.043	0.059	0.001	0.001		
Public-health response time, PHRT	--	--	--	--	0.015 (0.010)	-21.0** (8.9)
Flu-related gross death rate 1910-16	3.74*** (0.83)	3.87*** (1.03)	--	--	3.73*** (0.93)	--
Log(heating-degree days)	-0.00054* (0.00030)	-0.00052* (0.00030)	--	--	-0.00075** (0.00029)	--
R-squared	0.55	0.55	0.35	0.29	0.48	0.43
Standard error of regression	0.0010	0.0010	1.12	1.17	0.0011	1.04
Number of observations	43	43	43	43	43	43

***Significant at 1% level.

**Significant at 5% level.

*Significant at 10% level.

Notes to Table 1:

Standard errors of coefficients are in parentheses. OLS is ordinary least-squares. TSLS is two-stage least-squares. In columns 2 & 4, the instrumental variables are the distance from Boston and its square, the flu-related gross mortality rate 1910-1916, and the log of heating-degree days. In columns 6 & 8, the instrument list includes also the difference between school closings and prohibitions of public gatherings and between school closings and quarantine/isolation.

The sample comprises 43 U.S. cities with center-city populations in 1910 above 100,000, observed for the 24 weeks from week ending September 14, 1918 to week ending February 22, 1919. NPIs is the time in effect of three forms of non-pharmaceutical interventions: school closings, prohibitions on public gatherings, and quarantine/isolation, from Markel, et al. (2007, Table 1 and supplemental figures). This source also reports the public health response time or PHRT, which is the delay from an early point of the epidemic (defined as the time when the weekly death rate reaches twice a baseline death rate) to the implementation of the first NPI. The cumulative excess flu-related death rate is calculated from data on excess flu-related deaths given in Collins, et al. (1930, Appendix Table B). (An apparent typo in their data for Pittsburgh for November 23, 1918, was corrected based on the information in Davis [1918].) Their excess death rate is the difference between the flu-related death rate for each city and week and the median of estimated flu-related death rates for the corresponding city and week for 1910-1916. The relative peak death rate over the 24-week sample is the ratio of the peak weekly excess flu-related death rate to the average of these weekly excess death rates. 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 for the full calendar year, observed around 2000, is from *weatherdatadepot.com*. Distance from Boston is the minimum distance from each city to Boston according to *Google Maps*.

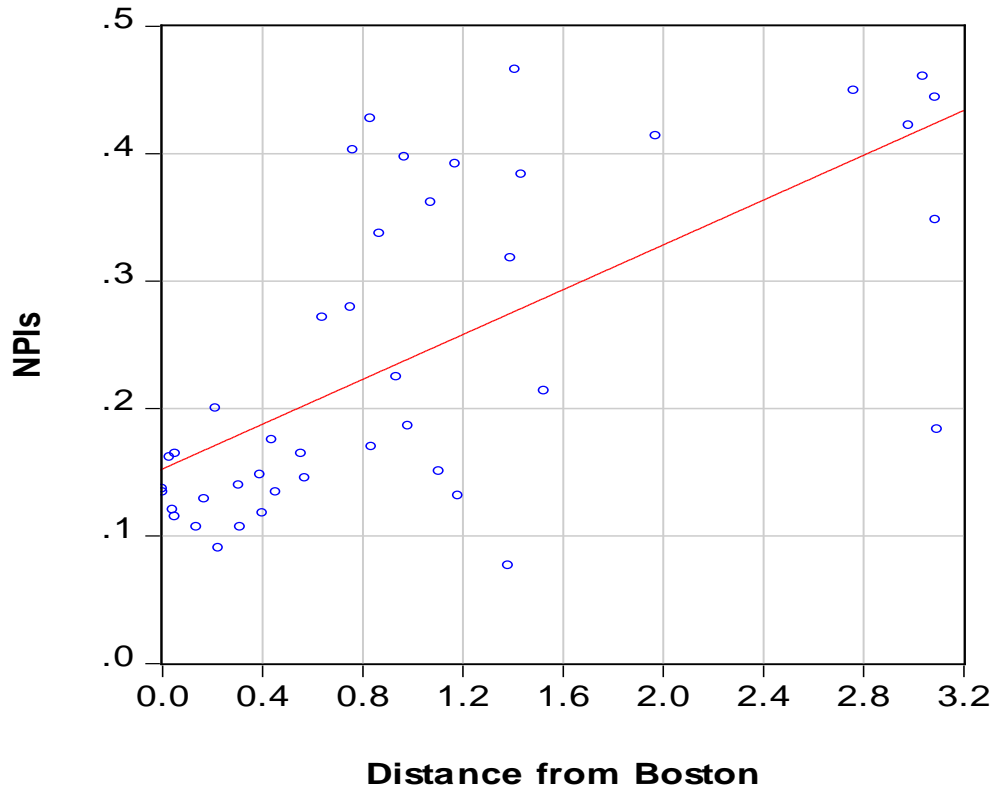
The 43 cities covered are Albany, Baltimore, Birmingham, Boston, Buffalo, Cambridge, Chicago, Cincinnati, Cleveland, Columbus, Dayton, Denver, Fall River, Grand Rapids, Indianapolis, Kansas City MO, Los Angeles, Louisville, Lowell, Milwaukee, Minneapolis, Nashville, New Haven, New Orleans, New York City, Newark, Oakland, Omaha, Philadelphia, Pittsburgh, Portland OR, Providence, Richmond, Rochester, St. Louis, San Francisco, Seattle, Spokane, Toledo, Washington DC, and Worcester.

Table 2
Descriptive Statistics

Variable:	NPIs	School closings	Prohibitions on public gatherings	Quarantine/ Isolation	PHRT	Cumulative excess flu-related death rate
Units	years	years	years	years	years	fraction of population
Mean	0.24	0.10	0.10	0.05	0.02	0.0052
Std dev.	0.13	0.06	0.05	0.07	0.02	0.0015
Max	0.47	0.29	0.22	0.26	0.10	0.0082
Min	0.08	0	0	0	-0.03	0.0022
Variable:	Relative peak death rate	Distance (Boston)	Gross flu-related death rate 1910-16	Log(heating-degree days)	POP 1910	POP density 1910
Units	ratio	1000 miles	fraction per year	log(degrees Fahr-years)	1000s	1000s/sq. mile
Mean	4.57	1.01	0.00086	2.28	434	8.6
Std dev.	1.32	0.92	0.00023	0.59	760	4.5
Max	7.93	3.10	0.00147	2.83	4768	18.6
Min	2.82	0	0.00040	0.00	100	1.7

Note: These statistics apply to variables used in Table 1.

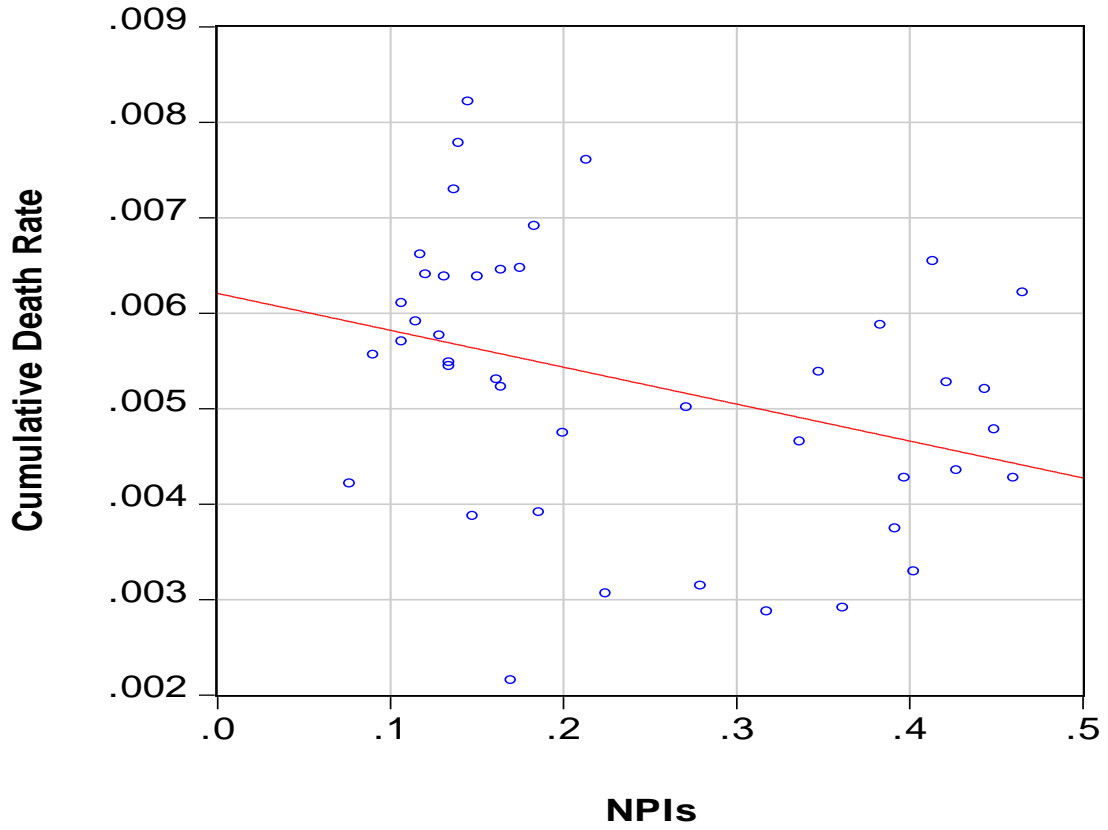
Figure 1
Relationship between Distance from Boston (thousands of miles)
and NPIs (years of implementation)



Note: Distance from Boston is the minimum distance shown by *Google Maps*. NPIs is from Markel, et al. (2007, Table 1).

Figure 2

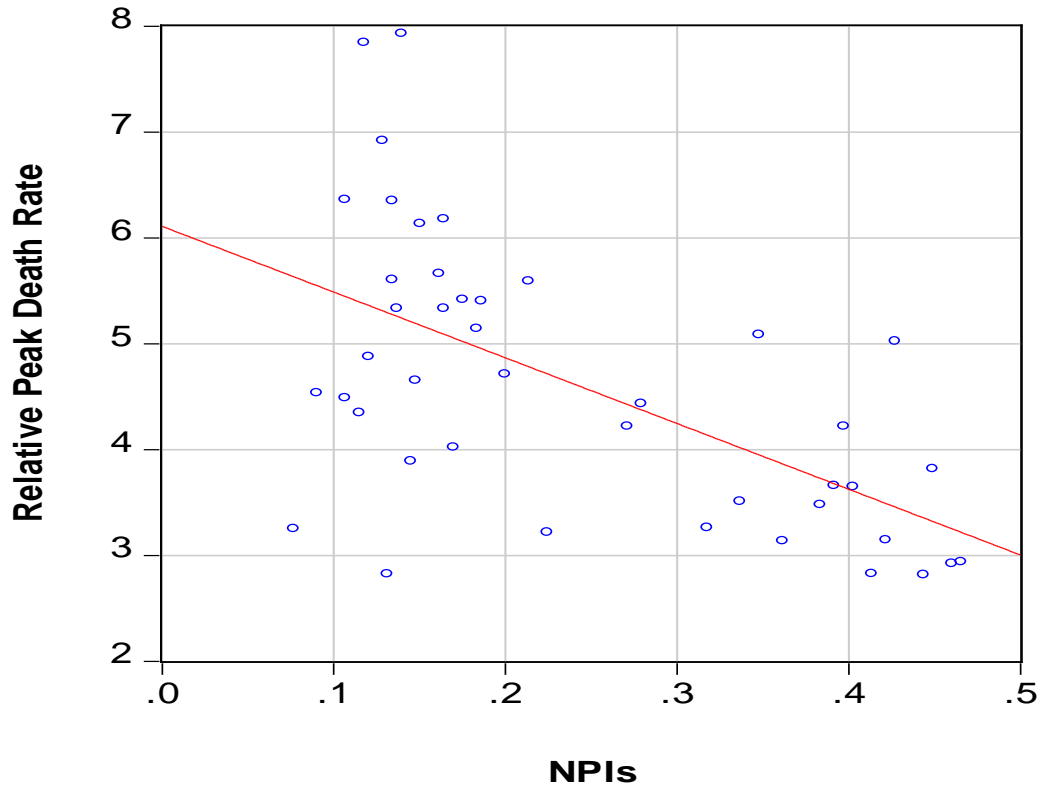
Relationship between NPIs and Cumulative Death Rate



Note: NPIs is from Markel, et al. (2007, Table 1). Cumulative death rate is calculated from Collins, et al. (1930, Appendix, Table B).

Figure 3

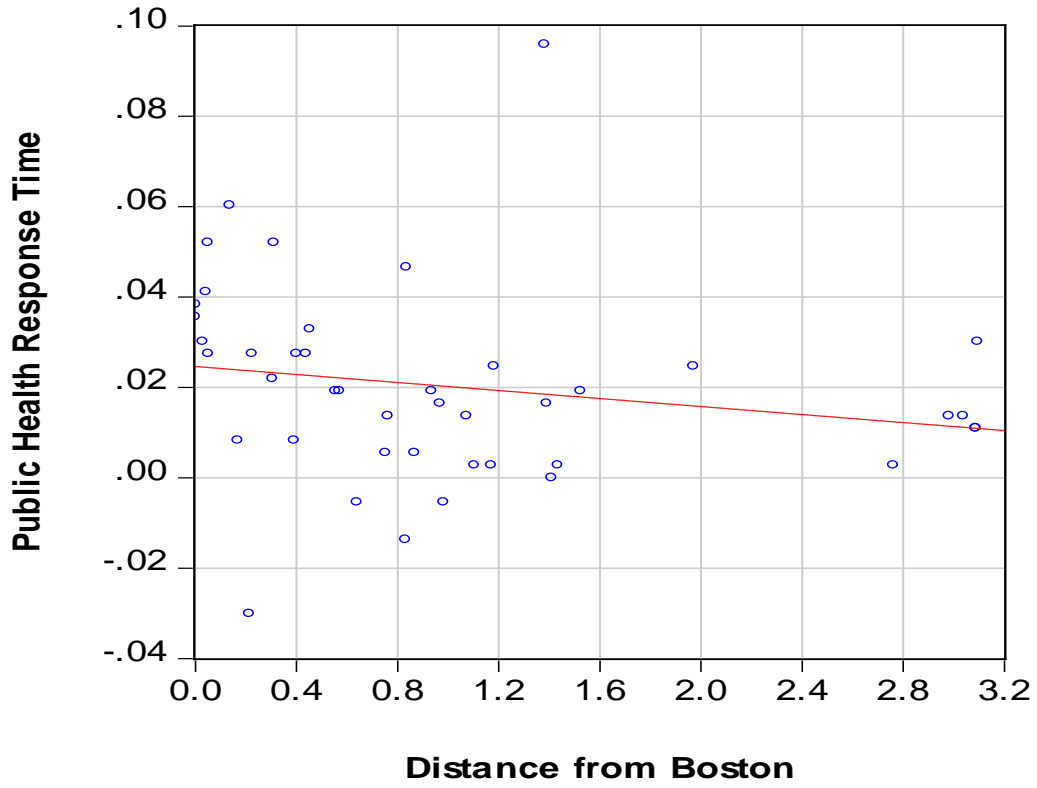
Relationship between NPIs and Relative Peak Death Rate



Note: NPIs is from Markel, et al. (2007, Table 1). The relative peak death rate, defined as the ratio of the peak weekly death rate to the average weekly death rate, is calculated from Collins, et al. (1930, Appendix, Table B).

Figure 4

**Relationship between Distance from Boston (thousands of miles)
and Public Health Response Time (PHRT in years)**



Note: 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).