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

Abstract

Non-pharmaceutical 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 were in three categories: school closings, prohibitions on public gatherings, and quarantine/isolation. An increase in NPIs sharply reduced the ratio of peak to average deaths, with a larger effect when NPIs were treated as endogenous. However, the estimated effect on overall deaths was small and statistically insignificant. The likely reason that the NPIs were not more successful in curtailing mortality is that the interventions had a mean duration of only around one month.

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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) 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. Specifically, Markel, et al. (2007) studied effects of non-pharmaceutical 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 the collection of data on NPIs for 43 of the 45 cities covered by the weekly Census data on 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

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

quarantine/isolation.[2] The underlying information comes from two newspaper reports per 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.

The present analysis considers two characteristics of each city's flu-related 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 seems to be 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, 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.

There is some concern that NPIs (measured, say, by length of time in force) and flu 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

[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).

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 in Boston, starting either at the army base Fort Devens or at the Navy's Commonwealth Pier facility.[3] 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.[4] Empirically, distance from Boston, entered in a quadratic form, has considerable explanatory power for the NPI variable, which is described below in Tables 1 and 2. The pair-wide 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.

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 the first part of Table 1 considers two dependent variables: cumulative flu death rate and relative peak death rate.

Columns 1 and 2 of the first part of Table 1 show estimated effects of NPIs on the cumulative death rate. The pair-wise association is in Figure 2. This relationship is negative, as expected (simple correlation coefficient equals -0.34). However, the estimated coefficients on

[3]For a discussion, see Barry (2004, pp. 183 ff.).

[4]Possibly 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.

NPIs in columns 1 and 2 are negative but insignificantly different from zero even at the 10% level. Results from OLS and two-stage least-squares are similar, indicating that treating NPIs as exogenous—as in Markel, et al. (2007), Hatchett, et al. (2007), and Bootsma and Ferguson (2007)—may be satisfactory for this part of the analysis.[5]

Columns 1 and 2 of Table 1 also include as an explanatory variable the all-cause mortality rate from a prior year, 1910.[6] This variable captures aspects of healthcare and age structure that matter for mortality in general and are, therefore, likely also to influence flu death rates. The estimated coefficients of this variable are positive, significant at the 5% level in column 1 and the 10% level in column 2. The other explanatory variable included is the log of heating-degree days. This variable measures a city's average temperature in the fall-winter period associated with the sample. Surprisingly, colder places, with more heating-degree days, are estimated to have significantly lower flu death rates.

Columns 3 and 4 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.[7] Overall, there is clear evidence that an increase in NPIs flattens out the pattern of

[5]The instrumental variables aside from distance to Boston and its square are the all-cause mortality rate in 1910 and the log of heating-degree days. The F-Statistic for the first-stage equation for NPIs is 10.6. Most of the explanatory power here comes from the two distance variables.
[6]A similar variable was used by Bootsma and Ferguson (2007, p. 7588).
[7]Measurement error in the NPI variable can also be a source of this attenuation.

flu death rates, gauged by the drop in the ratio of the peak weekly death rate to the sample average of the weekly death rates.

Columns 1 and 2 of the second part 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. In this case, only OLS results are presented because there are insufficient instruments to distinguish among multiple endogenous measures of NPIs. However, the previous findings suggest that the OLS results are likely to be informative.

For the cumulative flu death rate in column 1, the only significant coefficient related to NPIs is the negative one on prohibitions of public gatherings. However, the results accept the hypothesis at a p-value of 0.13 that the coefficients of the three NPI variables are the same. For the relative peak death rate in column 2, the estimated coefficient on quarantine/isolation is negative and highly significant, whereas that on school closings is negative and marginally significant. In this case, the results accept the hypothesis at a p-value of 0.86 that the coefficients of the three forms of NPIs are the same. Overall, the inference from the results in columns 1 and 2 is that it was satisfactory to combine the three types of interventions into a single form, as done in the first part of the table.

Columns 3 and 4 of the second part 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).[8] 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

[8]Their 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.

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 death rate (0.16) and the relative peak death rate (0.08).

Column 3, for the cumulative death rate, shows that the estimated regression coefficient on PHRT is essentially zero. Column 4, 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 reflects an interaction between PHRT and NPIs—these two forms of interventions are inversely related (simple correlation coefficient of -0.54), meaning that places with more NPIs tend also 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 4, 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.

Consider now what the results in the first part of 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 -9.1 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 -0.0026 from column 2, a rise in the NPI variable by 0.13 lowers the cumulative death rate by 0.0003, compared with the sample mean of 0.0052 (Table 2). Thus, the smoothing out of flu deaths did not accomplish much in terms of lowering overall mortality.

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 results accord in part with the conclusions reached by Hatchett, et al. (2007) and Bootsma and Ferguson (2007).[9] The lesson

[9]According 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." Our results accord with the effects related to the duration of NPIs but not with the those from delay, which we 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, at least in the range observed within the sample, significantly reduced cumulative or relative peak death rates.

for the ongoing coronavirus pandemic in 2020 is that, to curtail overall deaths, the NPIs used have to be maintained for substantially longer than a few weeks.

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Table 1

Effects from Non-Pharmaceutical Interventions (NPIs)

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

Dependent variable:	**Cumulative Flu Death Rate**		**Relative Peak Death Rate**	
	(1)	**(2)**	**(3)**	**(4)**
Method:	OLS	TSLS	OLS	TSLS
Constant	0.0042** (0.0019)	0.0044 (0.0027)	6.11*** (0.36)	6.81*** (0.50)
Non-pharmaceutical interventions (NPIs)	-0.0023 (0.0017)	-0.0026 (0.0030)	-6.2*** (1.3)	-9.1*** (1.9)
All-cause mortality rate, 1910	0.203** (0.083)	0.195* (0.104)	--	--
Log(heating-degree days)	-0.00071** (0.00032)	-0.00073** (0.00034)	--	--
R-squared	0.36	0.36	0.35	0.27
Standard error of regression	0.0012	0.0012	1.09	1.16
Number of observations	43	43	43	43

Table 1, continued

Dependent variable:	Cumulative Flu Death Rate	Relative Peak Death Rate	Cumulative Flu Death Rate	Relative Peak Death Rate
	(1)	(2)	(3)	(4)
Method:	OLS	OLS	OLS	OLS
Constant	0.0035* (0.0019)	6.03*** (0.40)	0.0035 (0.0022)	7.00*** (0.51)
Non-pharmaceutical interventions (NPIs)	--	--	-0.0014 (0.0022)	-8.14*** (1.50)
School closings	0.0015 (0.0041)	-6.6* (3.6)	--	--
Prohibitions on public gatherings	-0.0101** (0.0049)	-4.4 (4.5)	--	--
Quarantine/isolation	0.0015 (0.0029)	-7.2*** (2.3)	--	--
p-value, 3 NPIs have same coefficients	0.13	0.86	--	--
Public-health response time (PHRT)	--	--	0.008 (0.011)	-21.0** (8.9)
All-cause mortality rate, 1910	0.242*** (0.085)	--	0.222** (0.087)	--
Log(heating-degree days)	-0.00058* (0.00033)	--	-0.00071** (0.00032)	--
R-squared	0.42	0.35	0.36	0.43
Standard error of regression	0.0012	1.12	0.0012	1.04
Number of observations	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, using as instrumental variables the distance from Boston and its square, the all-cause mortality rate, and the log of heating-degree days.

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. Cumulative flu death rate and peak weekly death rate are from Collins, et al. (1930, Appendix Table B). Relative peak death rate is the ratio of the peak weekly death rate to the average of the weekly death rates. All-cause mortality rate is from U.S. Census 1910. Heating-degree days, 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

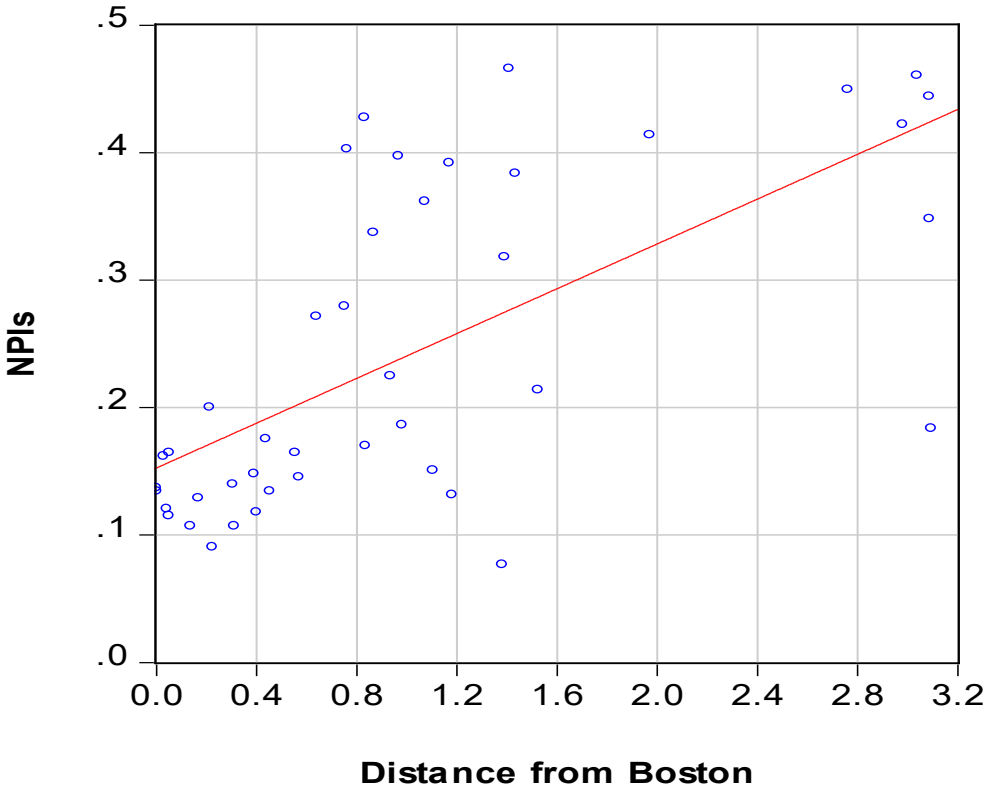
Descriptive Statistics

Variable:	**NPIs**	**School Closings**	**Prohibitions on Public Gatherings**	**Quarantine/ Isolation**	**PHRT**	**Cumulative 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)**	**Mortality Rate 1910**	**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	0.016	2.28	434	8.6
Std dev.	1.32	0.92	0.003	0.59	760	4.5
Max	7.93	3.10	0.023	2.83	4768	18.6
Min	2.82	0	0.010	0.00	100	1.7

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

Figure 1

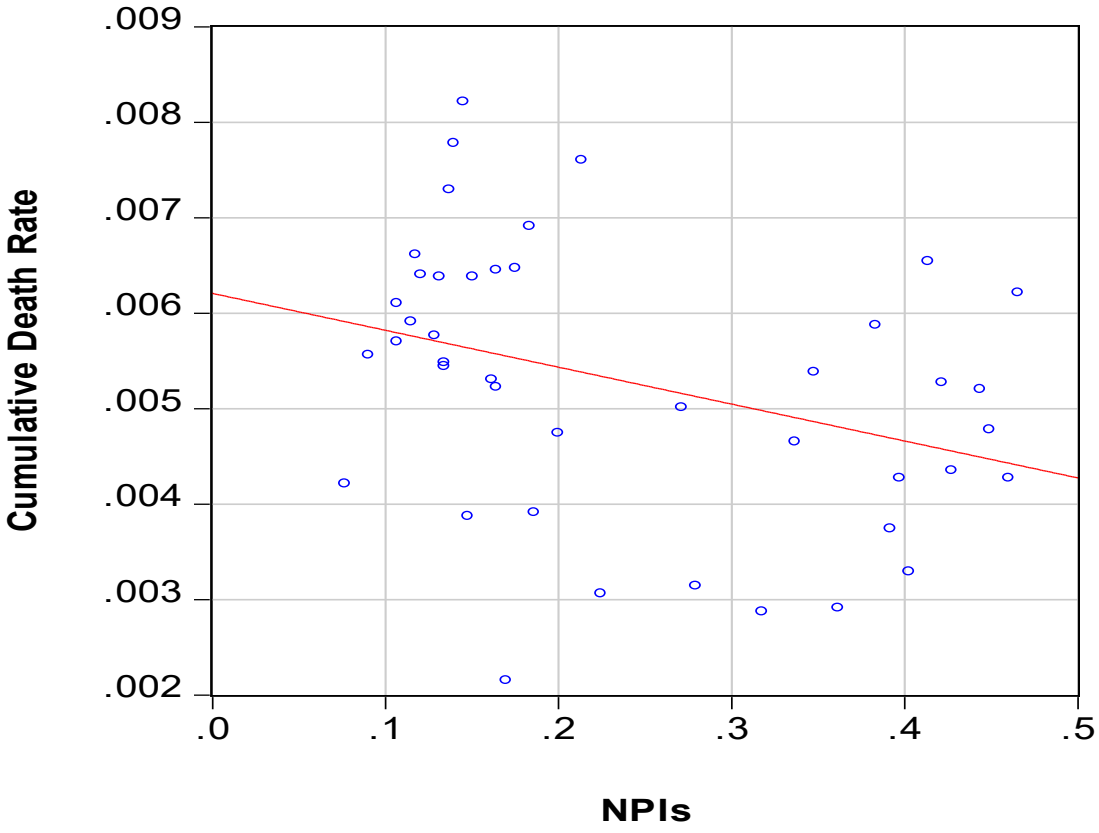
Relationship between Distance from Boston (thousands of miles) and NPIs (years of implementation)



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

Figure 2

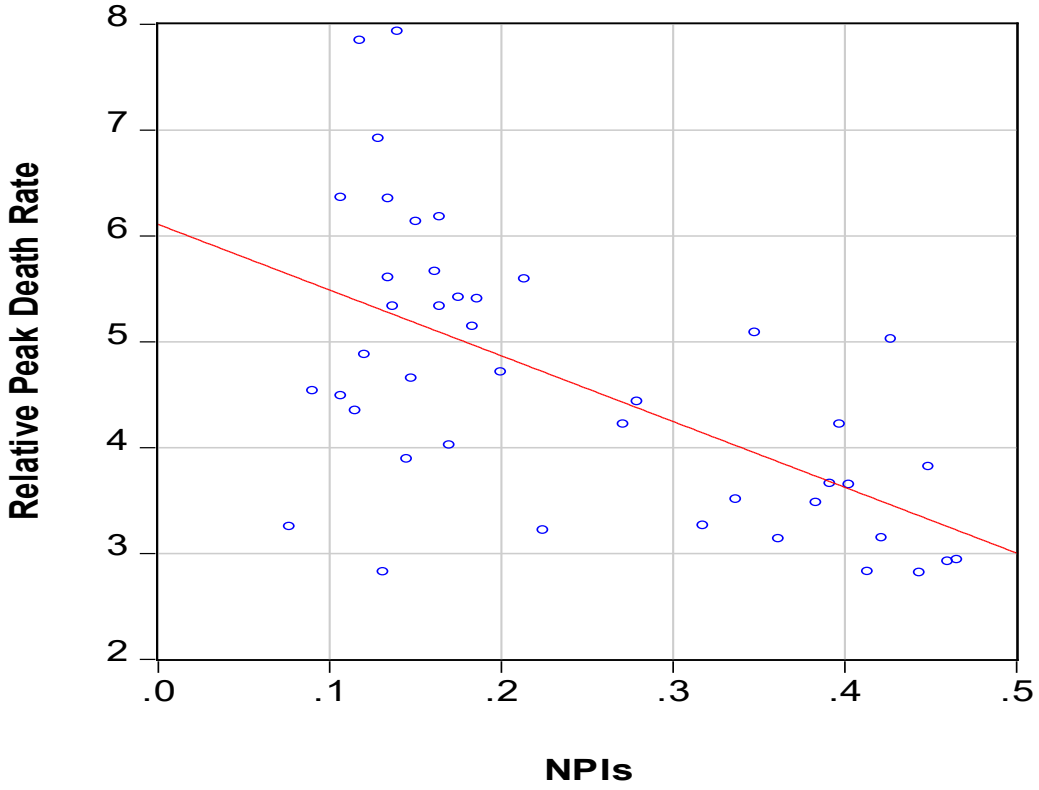
Relationship between NPIs and Cumulative Death Rate



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

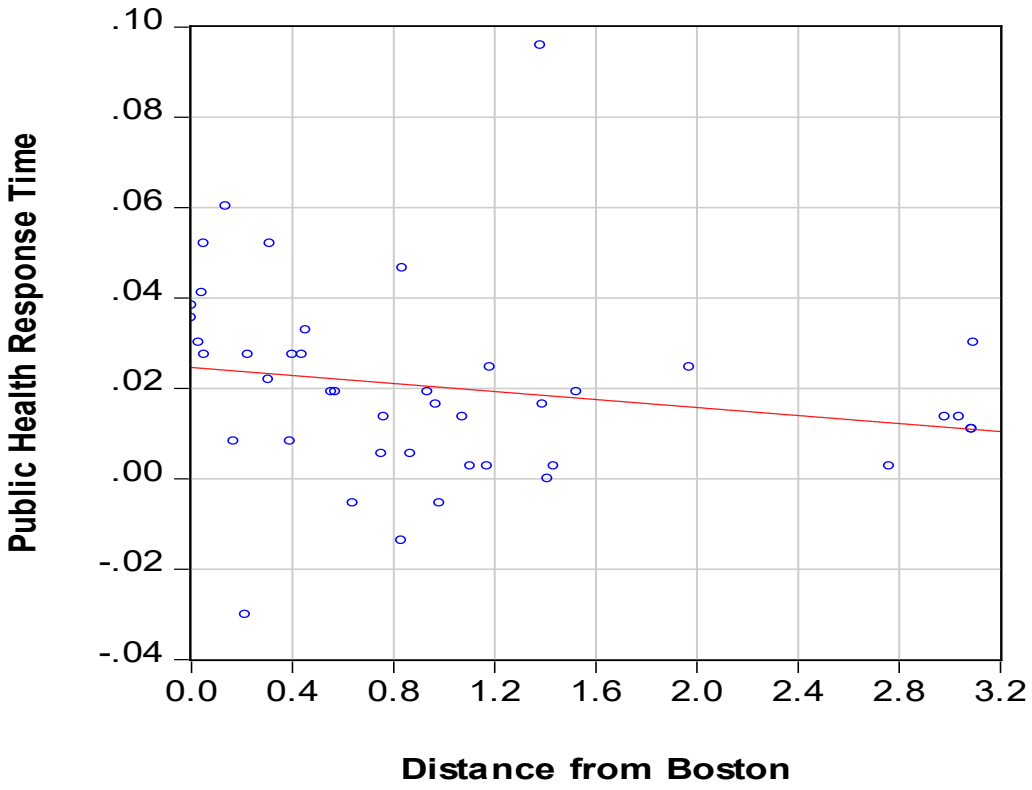


Note: NPIs is from Markel, et al. (2007, Table 1). The relative peak death rate, defined as the ratio of the peak weekly death rate to the average weekly death rate, is calculated from Collins, et al. (1930, Appendix, Table B).

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).