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

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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

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 were 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 was 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.

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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 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. Specifically, 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 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

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

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

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, 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-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

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.

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, which is described below in Tables 1 and 2. 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.

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: the cumulative excess flu-related death rate and the relative peak death rate.

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.

Flu-related death rates in each city likely depend on city characteristics such as healthcare facilities and age structure. 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 that matter for flu-related deaths in general and, therefore, likely for the 1918-19 period. The analysis also includes as an explanatory variable a measure of a city's weather, given by the log of heating-degree days.

Columns 1 and 2 of the first part 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. (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. Surprisingly, colder places,

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.

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.

with more heating-degree days, are estimated to have significantly lower excess flu-related death rates.⁹

Columns 3 and 4 of the first part 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 out 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 implemented and 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.

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 excess flu death rate in column 1, the only significant coefficient related to NPIs is the negative one on prohibitions of public gatherings. The results reject the

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.

hypothesis at a p-value of 0.03 that the coefficients of the three NPI variables are the same (and with a p-value of 0.04 that all three coefficients are zero). Thus, there is some evidence that restrictions on public gatherings did retard excess flu death rates. 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. These results indicate that, for the analysis of relative peak death rates, 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).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).

Column 3, for the cumulative excess 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

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.

(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 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 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 -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.

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).¹² The lesson 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. Most likely, 12 weeks work much better than 4-6 weeks.

¹²According 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.

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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 Excess Death Rate	Relative Peak Death Rate	Cumulative Flu Excess Death Rate	Relative Peak Death Rate
	(1)	(2)	(3)	(4)
Method:	OLS	OLS	OLS	OLS
Constant	0.0038*** (0.0013)	6.03*** (0.40)	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)	-6.6* (3.6)	--	--
Prohibitions on public gatherings	-0.0123*** (0.0043)	-4.4 (4.5)	--	--
Quarantine/isolation	0.0023 (0.0025)	-7.2*** (2.3)	--	--
p-value, 3 NPIs have same coefficients	0.026	0.86	--	--
p-value, 3 NPI coefficients equal zero	0.043	0.001		
Public-health response time (PHRT)	--	--	0.015 (0.010)	-21.0** (8.9)
Flu-related gross death rate 1910-1916	3.74*** (0.83)	--	3.73*** (0.93)	--
Log(heating-degree days)	-0.00054* (0.00030)	--	-0.00075** (0.00029)	--
R-squared	0.55	0.35	0.48	0.43
Standard error of regression	0.0010	1.12	0.0011	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 flu-related gross mortality rate 1910-1916, 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. 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, 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)

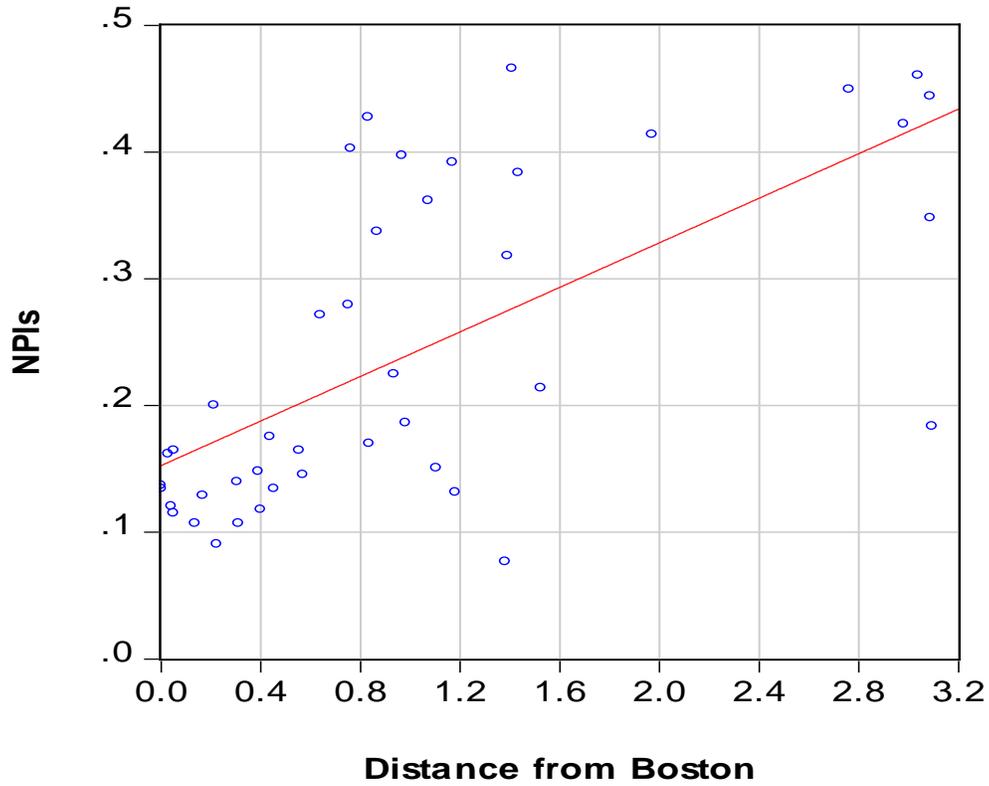
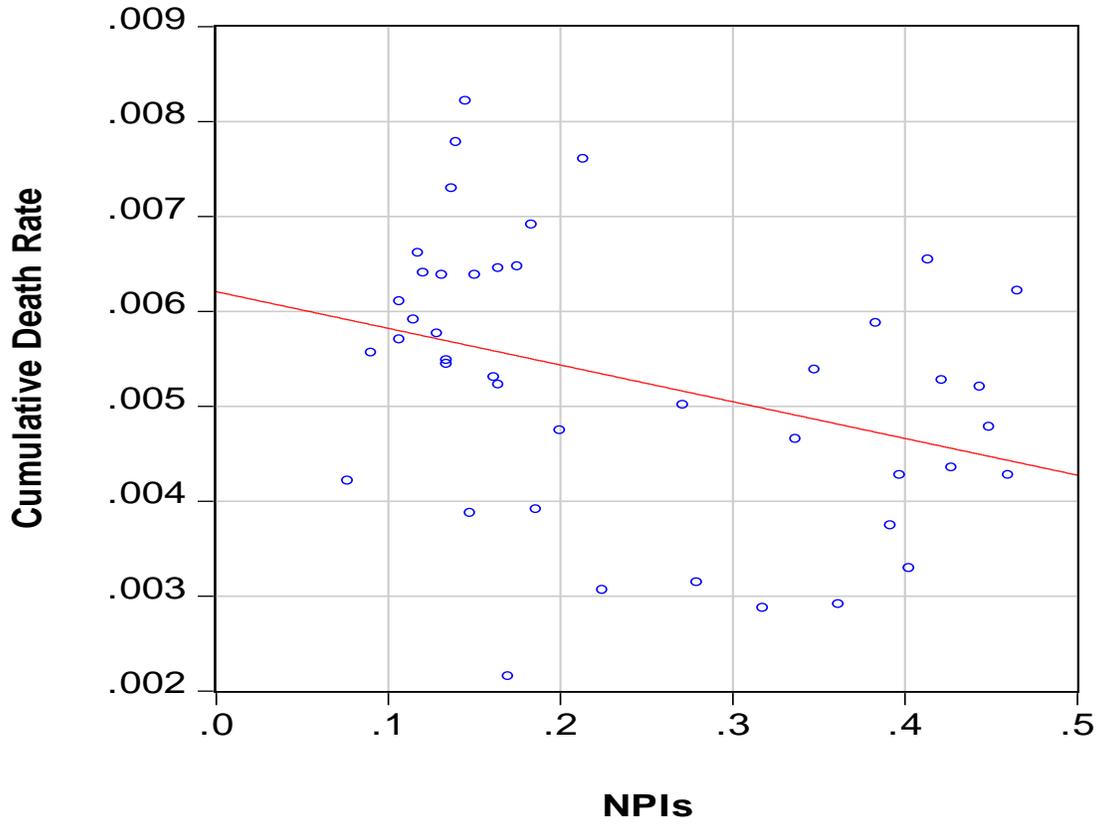


Figure 2

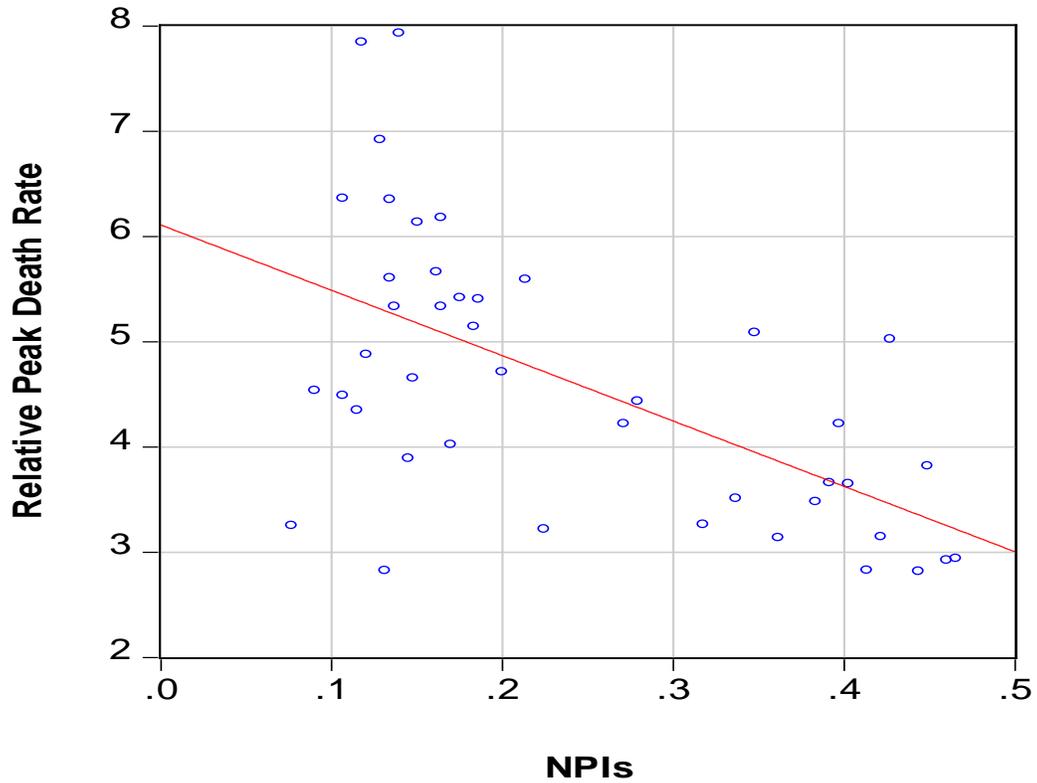
Relationship between NPIs and Cumulative Death Rate



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

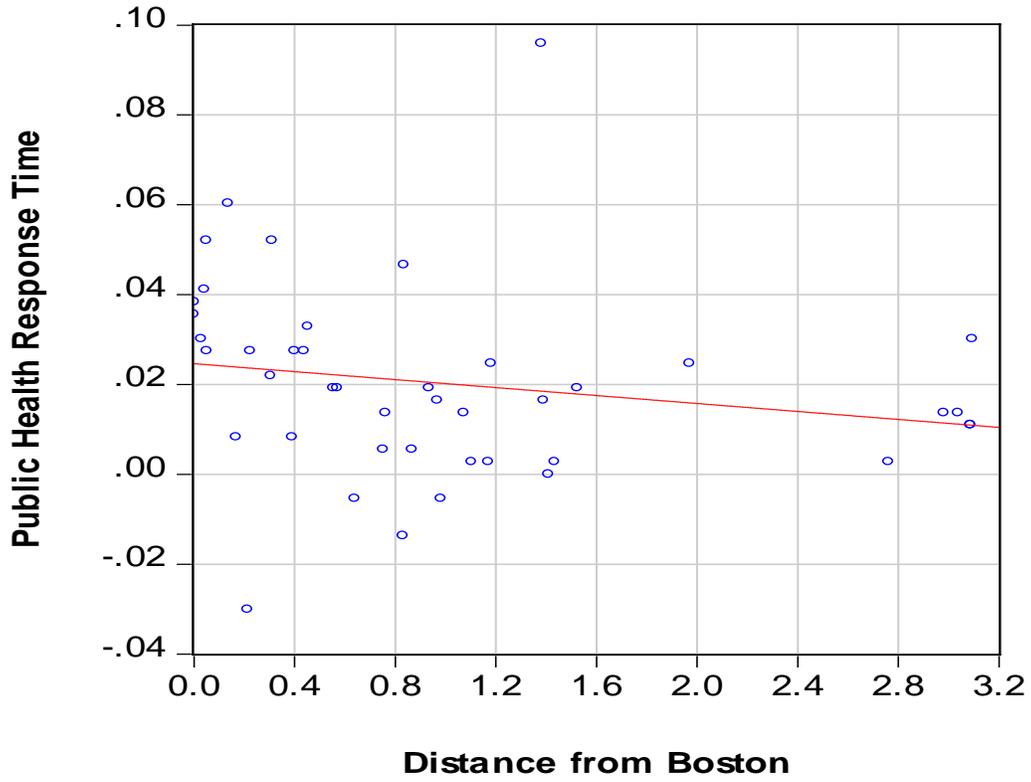
Relationship between NPIs and Relative Peak Death Rate



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).