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The medium-term impact of non-pharmaceutical interventions. The case of the 1918 Influenza in U.S. cities *

Guillaume Chapelle †

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Abstract

This paper uses a difference-in-differences (DID) framework to estimate the impact of Non-Pharmaceutical Interventions (NPIs) used to fight the 1918 influenza pandemic and control the resultant mortality in 43 U.S. cities. The results suggest that NPIs such as school closures and social distancing, as implemented in 1918, and when applied for a relatively long and sustained time, might have reduced individual and herd immunity and the population general health condition, thereby leading to a significantly higher number of deaths in subsequent years.

J.E.L. Codes: I18, H51, H84
Keywords: Non-pharmaceutical interventions, 1918 influenza, difference-in-differences, health policies

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1 Introduction

Since the emergence of the global Covid-19 pandemic, a growing stream of contributions has sought to help policymakers to improve their understanding of the crisis by analyzing past pandemics. In this context, the 1918 flu might offer an interesting opportunity to evaluate the potential impact of pandemics on economic activity (Barro, Ursúa, and Weng 2020) and the potential benefits of non-pharmaceutical interventions (NPIs) such as school closures and social distancing (Correia, Luck, and Verner 2020).

This paper is motivated by Figure 1 which displays the evolution of the average reported death rate in cities with the implementation of longer or shorter NPIs. I develop several measurements of mortality in large U.S. cities and estimate the impact of NPIs on the number of deaths by utilizing a difference-in-differences (DID) approach. I show that cities that responded more aggressively and rapidly to the 1918 pandemic with NPIs had similar trends before 1918 but ended with relatively higher mortality levels in the subsequent years- in particular, when the intervention last for a long period of time. I tackle the potential endogeneity of the implementation of NPIs, controlling for city's sociodemographic characteristics and exploiting variation of NPIs within regions. This allows to identify the impact of NPIs comparing cities with similar demographic characteristics or location but different duration or speed of NPIs. I also employ age group mortality to control for the demographic structure of cities. I find that the results remain qualitatively unchanged.

While this is not the first paper to document the impact of NPIs implemented in US cities in 1918, it contributes to the literature in several ways. First, this is the first paper to investigate the impact of NPIs during the pandemic on various mortality indicators. I show that the negative short term impact of NPIs as documented in Markel et al. (2007) and Correia, Luck, and Verner (2020) is not reflected in total mortality and is not robust to the inclusion of cities' fixed effect. Second, this is also the first paper to document the medium run impact of these policies and the rebound in mortality during the subsequent years.

This second result could be explained by the fact that NPIs might have reduced individual and herd immunity[1]. Gostic et al. (2016) indicate that the first flu that an individual contracts in one's life might have a long-lasting effect on the probability to die from other strains of influenza during one's entire lifetime. Consequently, reducing the spread of the disease might cause a city's population to become more vulnerable in the medium run, thereby increasing the overall mortality rate. Moreover, it might be believed that herd immunity could also allow a decrease in the spread of the next influenza as argued by Fox et al. (1971) and Fine

1. Herd immunity is defined as *"The resistance of a group to attack by a disease to which a large proportion of the members are immune, thus lessening the likelihood of a patient with a disease coming into contact with a susceptible individual"* (Agnew 1965)

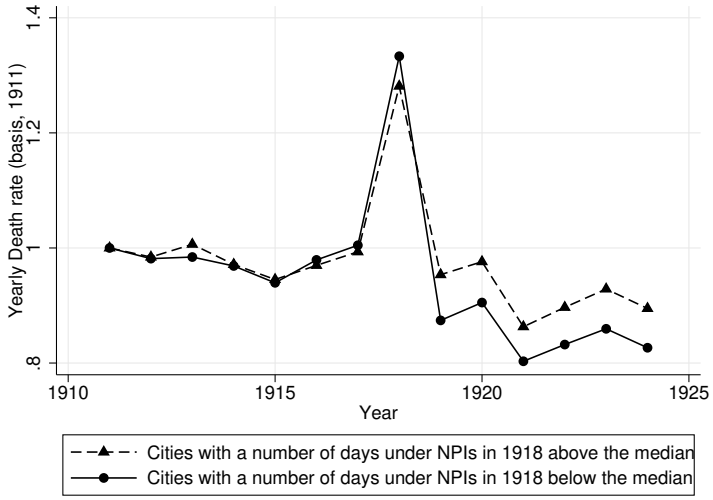
(1993). Finally, medical literature such as Douglas et al. (2020) and Markel, Stern, and Cetron (2008) fear that NPIs might have negative consequences on the general health status of the population by reducing physical activity and through economic and psycho-social impacts.

These findings suggest that the potential short-term benefits of NPIs documented in Markel et al. (2007), Correia, Luck, and Verner (2020), and Barro (2020) -particularly the fact that NPIs enabled a flattening of the epidemic curve- might be counterbalanced on the medium run by the lower immunity and health condition of the population. Moreover, these results might have implications on the current discussion on the tradeoff between health policies and economic growth. In particular, it sheds a new light on the potential medium-run economic impact of NPIs in U.S. cities during the 1918 influenza as discussed in Correia, Luck, and Verner (2020) and Lilley, Lilley, and Rinaldi (2020). Indeed, NPIs did not appear to preserve human capital thereby raising questions regarding the potential channels that are likely to explain their economic benefits.

It is important to remind that this study is specific to the 1918 influenza and has a limited external validity. It would be difficult to infer any implication on the potential impact of NPIs as implemented in 2020 for several reasons. First, as emphasized in Cohen-Kristiansen and Pinheiro (2020) , the Covid-19 and the 1918 influenza are two different viruses with different ways of transmission and health consequences. Moreover, the types of NPIs implemented in US cities in 1918 are different from the one used during the first wave of the Covid-19 as no lock down of the population was implemented. The most used NPIs were school closures, public gathering bans and quarantines. In addition, the 1918 NPIs were implemented at the city level while in 2020 NPIs appeared much more coordinated at the national or state scale. Finally, pharmaceutical technologies were less developed back then as compare to what they are today and the capacity to find a treatment or to produce a new vaccine is much higher.

The remainder of this paper is organized in the following manner. Section 2 presents the background and the current state of our knowledge on the 1918 pandemic including its potential effect on economic activity. Section 3 presents the data utilized in this paper. Section 4 develops a DID approach to estimate the impact of NPIs on mortality. Section 5 presents the results and the robustness checks. Section 6 presents the conclusion.

Figure 1: Evolution of the yearly death rate before and after the 1918 flu in 43 cities that implemented non-pharmaceutical Interventions in 1918 for different durations



Reading notes: Cities that implemented NPIs for a longer duration witnessed their death rates increase less than cities that had shorter NPIs in 1918. On the other hand, the death rate remained relatively higher during the following years for these cities

Computation of the author from the Bureau of Census Mortality Tables published in 1920 and 1924

Data on NPIs come from Markel et al. (2007)

Average death rate computed for a sample of 43 cities: Albany (NY), Baltimore, Birmingham, Boston, Buffalo, Cambridge, Chicago, Cincinnati, Cleveland, Columbus, Dayton, Denver, Fall River, Grand Rapid, Indianapolis, Kansas City, Los Angeles, Louisville, Lowell, Milkwaukee, Minneapolis, Nashville, New Haven, New Orleans, New York, Newark, Oakland, Omaha, Philadelphia, Pittsburgh, Portland, Providence, Richmond, Rochester, Saint Louis, Saint Paul, San Fransisco, Seattle, Spokane, Syracuse, Toledo, Washington, and Worcester.

2 Background and literature review

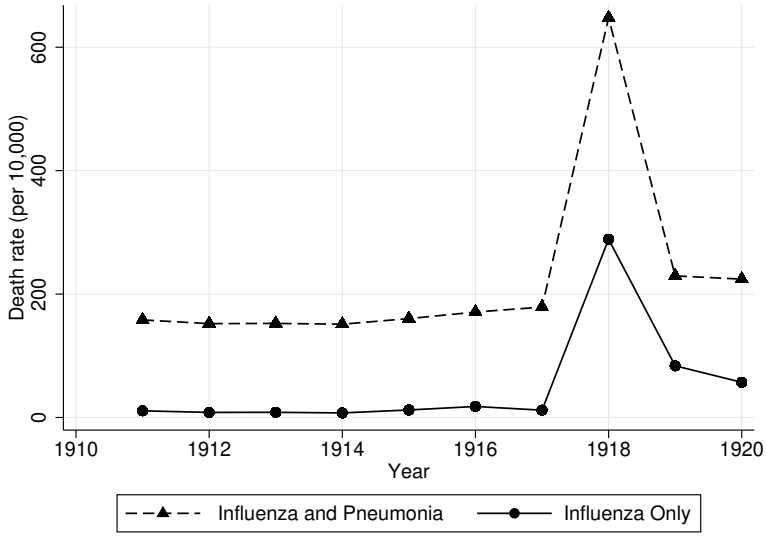
2.1 Policy responses to the 1918 influenza

The year 2020 has witnessed a severe global health crisis in the form of the Covid-19 pandemic, with over 50% of the world population under relatively strict NPIs. The crisis most similar to this one from which sufficient data is available is the 1918 flu that spread throughout the world at the end of the First World War and infected approximately one-fourth of the world's population at that time (Taubenberger and Morens 2006). It also had long run consequences on children born during this period (Almond 2006; Brown and Thomas 2018; Beach, Ferrie, and Saavedra 2018). The flu mostly affected active people with an unusual casualty rate concentrated in the age groups of between 15 and 45 years.

In the U.S., the flu was probably spread by troops who returned from Europe, thereby leading to a dramatic increase in the death rate in the autumn of 1918. There were three waves of illness from 1918 to 1919. The first one took place in

March 1918, in particular in military camps like Camp Funston in Kansas where 100 cases were reported. The second wave, which was the deadliest, came in Fall 1918 and was responsible for most of the deaths attributed to the pandemic. Finally, a third wave occurred in Winter 1918 and the flu subsided in Summer 1919. It is also noteworthy that the death rate due to influenza decreased in the subsequent years but remained at higher levels when compared with years prior to 1918 as illustrated in Figure 2. The virus mutated and continued to affect people in the following years. Indeed, Taubenberger and Morens (2006) emphasize that the virus at the origin of the 1918 pandemic gave birth to most of the subsequent influenza strains, with the exception of the avian flu. According to Fine (1993) *"prior to 1977, only a single major [influenza] virus (shift) sub-type was found circulating in the human population worldwide at any time"*. Moreover, Spinney (2017) explains that *"pandemic flu don't start and stop [...] they invade seasonal flu cycle [...], defining a pandemic's limit is an essentially arbitrary task."*

Figure 2: Evolution of the death rate caused by influenza and influenza and pneumonia



Author's computation from Bureau of the Census, Mortality Statistics 21st Annual Report published in 1920. Average death rate computed for a sample of 43 cities: Albany, Baltimore, Birmingham, Boston, Buffalo, Cambridge, Chicago, Cincinnati, Cleveland, Columbus, Dayton, Denver, Fall River, Grand Rapids, Indianapolis, Kansas City, Los Angeles, Louisville, Lowell, Milkwaukee, Minneapolis, Nashville, New Haven, New Orleans, New York, Newark, Oakland, Omaha, Philadelphia, Pittsburgh, Portland, Providence, Richmond, Rochester, Saint Louis, Saint Paul, San Fransisco, Seattle, Spokane, Syracuse, Toledo, Washington, and Worcester.

The federal government in the U.S. did not coordinate a national response (Correia, Luck, and Verner 2020) leaving cities to manage the pandemic by implementing local measures. The timing of the response appears to be correlated with the geographical longitude, thereby suggesting that cities located in the West had more time to prepare using the experience of cities in the East that had been more rapidly

affected and, thus, overwhelmed. Indeed Markel et al. (2007) show that the pandemic waves began in the East during the second week of September 1918, in the Midwest in the last week of September 1918 and in the West in the second week of October 1918. They show that all cities that they investigated implemented NPIs in some form-such as quarantines, social distancing and school closures- but that some were stricter and took action more promptly as compared to the others. Their data also documents a certain heterogeneity in the responses within each region. For example, New York responded rapidly to the pandemic and managed to flatten the epidemic curve by implementing strictly enforced isolation and quarantine procedures. According to Markel et al. (2007) this enabled the city to experience the lowest death rate on the East Coast. On the other hand, Pittsburgh only took action in the beginning of October 1918 and closed schools at the end of the month. This resulted in the highest excess mortality burden in the sample studied for the state.

2.2 The impact of NPIs during the 1918 influenza

This paper is intended as a contribution to the econometric and epidemiological literature. For example, Anderson, Charles, and Rees (2020) study the impact of public health efforts as water filtration on mortality in 25 US cities. I complement the econometric and statistical literature that documents the main drivers of mortality in U.S. cities during the 1918 influenza. Acuna-Soto, Viboud, and Chowell (2011) show that smaller cities experienced the worst outcome during the pandemic and that mortality during the pandemic was partially pre-determined by pre-pandemic pneumonia death rates. The authors suggest that this phenomenon might be explained by the physical and social structure of each city. This hypothesis was confirmed by three subsequent papers- Feigenbaum, Muller, and Wrigley-Field (2019) highlight the role of race during the 1918 pandemic, documenting that African Americans had a higher rate of death from infectious disease during this period. Moreover, Clay, Lewis, and Severnini (2018) also indicate that poor air quality contributed to higher mortality rates during the pandemic. Clay, Lewis, and Severnini (2019) document the role of several socioeconomic factors to explain the differences in mortality between U.S. cities before 1940. Overall, this literature emphasizes the fact that mortality from the influenza was strongly correlated with previous mortality levels, and thus, with observable and unobservable characteristics of cities such as their organisation or their demographic structure. This paper contributes to this literature in two ways. First, I explore the role of NPIs on the level of mortality in US cities during the pandemic in a DID setting with cities fixed effect controlling for characteristics that do not vary on the short term as social and physical structures. Second, I also investigate the medium term consequences of NPIs on mortality levels after the 1918 pandemic.

This is not the first paper to explore the impact of NPIs in U.S. cities during the 1918 influenza. Markel et al. (2007) find that early and strong NPIs enabled the flattening of the epidemic curve and reduced cumulated mortality. Bootsma and Ferguson (2007) used a parametric approach and found similar results. Hatchett,

Mecher, and Lipsitch (2007) rely on a smaller sample and found that these policies reduced mortality at the beginning of the pandemic but caused cities to be more sensitive to the next waves of influenza. Two recent econometric papers complemented these studies. Barro (2020) does not find any significant impact of NPIs on mortality from the 1918 flu, while Correia, Luck, and Verner (2020) found that NPIs enabled the flattening of the epidemic curve and reduced cumulated mortality. This paper improves on these contributions in several ways. First, I use several measures of mortality: the death count, the reported mortality rates, and the ratio between the number of deaths and the population in 1910. I also investigate the impact of NPIs not only on the number of deaths caused by influenza and pneumonia but also for all causes of deaths. This is important since NPIs can affect the transmission of all infectious diseases and the general health condition of the population. Second, none of these previous papers that focus on NPIs control for cities fixed effect. Most of them include a limited number of controls. The closest result to this study is a robustness check in Clay, Lewis, and Severnini (2018) that does not find any significant effect of NPIs in 1918. In this paper, I employ a panel framework with cities fixed effect and control for numerous potential confounding factors and regional shocks to account for the potential endogeneity of NPI implementation and intensity. Third, I also investigate the medium-term consequences of NPIs. Part of my results tend to support the fact that long and sustained NPIs might have enabled a flattening of the epidemic curve in the short run (i.e during the second wave of the 1918 pandemic). However, the estimated negative impact on mortality-as reported in Correia, Luck, and Verner (2020) and Markel et al. (2007)-is not robust when using death for all causes. These results are in line with those of Clay, Lewis, and Severnini (2018) and Beach, Clay, and Saavedra (2020) and Barro (2020) but also with Hatchett, Mecher, and Lipsitch (2007) that find no significant impact of NPIs on mortality in 1918 or a greater vulnerability to the third wave.

My findings also support the concerns raised in Markel, Stern, and Cetron (2008) that NPIs are associated with large costs that must also be accounted for during their implementation. Indeed, the fact that cities that implemented long NPIs incurred higher death rates in the following months and years, tends to support the literature on the importance of individual immunity on the spread and lethality of the subsequent waves of influenza as indicated in Gostic et al. (2016). This might also be considered as a support to the literature on herd immunity (Fine 1993; Fine, Eames, and Heymann 2011). It also tends to support concerns that NPIs might deteriorate the general health condition of the population due to reduced physical activity and their overall socioeconomic impact.

Finally, the 1918 experiment can contribute to the growing literature that is attempting to identify the impact of NPIs implemented during the Covid-19 crisis. Indeed, Lin and Meissner (2020a) documents the similarity in the evolution of the Covid-19 and the 1918 influenza in U.S. cities. Several contributions estimated the impact of NPIs in 2020 and tend to be in line with the estimated impact of the 1918 policies, while these policies appeared to be stronger in magnitude for Covid-19. Lin and Meissner (2020b) found that NPIs implemented locally in 2020 only

had a small effect on disease transmission while a similar conclusion is reached in Allcott et al. (2020).

2.3 The economic consequences of pandemics and the net benefits of NPIs

I also contribute to the literature documenting the economic impact of pandemics. For example, in 1999, Meltzer, Cox, and Fukuda (1999) estimated the potential economic impact of the next pandemic without including economic disruption and analyzed the benefits of developing vaccines to prevent a pandemic. Smith et al. (2009) developed a general equilibrium model to measure the potential impact of a pandemic on the UK economy under different scenarios. The Covid-19 pandemic has also given rise to a number of studies that propose a wide range of estimates of its potential economic impact as Atkeson (2020), Kong and Prinz (2020), Takahashi and Yamada (2020), Barrot, Grassi, and Sauvagnat (2020), Chen, Qian, and Wen (2020), Lin and Meissner (2020b), Baek et al. (2020), Allcott et al. (2020), and Dave et al. (2020).

This research is more precisely related to the literature that documented the impact of past pandemics, in particular, the 1918 pandemic. Karlsson, Nilsson, and Pichler (2014) documented the impact of the pandemic on earnings and capital returns in Sweden. Barro, Ursúa, and Weng (2020) used a panel of countries and estimate that the flu had negative impacts on several countries' gross domestic product (GDP) and consumption, which were estimated to be approximately 6% and 8% , respectively. Aassve et al. (2020) also found a significant impact of this pandemic on trust between people. Dahl, Hansen Worm, and Sandholt Jensen (2020) and Carrillo and Jappelli (2020) look at the impact of the 1918 pandemic on local economic growth in Denmark and Italy respectively. Velde (2020) studied the short-term dynamics of the U.S. economy during the pandemic. Bodenhorn (2020) studied the short-term consequences of NPIs on business disruption.

My results can contribute to the debate on the existence of a tradeoff between health and economic objectives during a pandemic as discussed in the recent work of Correia, Luck, and Verner (2020) who document the kind of economic impact one can expect from NPIs and the influenza pandemic on the manufacturing and banking sectors. My results are in line with Lilley, Lilley, and Rinaldi (2020) and argue for caution regarding any inferred causal links between economic activity and the implementation of NPIs in U.S. cities. I find that in the medium term, NPIs appear to have led to a decreased immunity of the population leaving individuals more sensitive to the subsequent waves of the pandemic and strains of influenza. NPIs also caused a deteriorated overall global health status that resulted in higher mortality levels in subsequent years. My findings could also contribute to the economic literature investigating the optimal policy responses to pandemics, -for example, Alvarez, Argente, and Lippi (2020), Jones, Philippon, and Venkateswaran (2020), and Toda (2020)- as they suggest that optimal policies must also include an exit

strategy as vaccination campaign when implementing NPIs.

3 Data

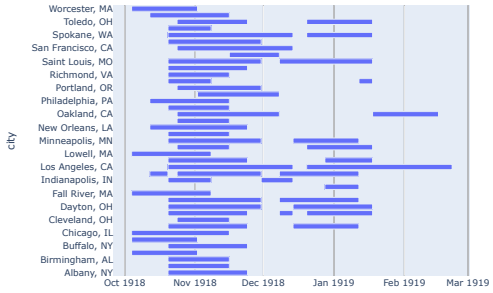
3.1 Measuring NPIs

I construct a panel of 43 cities with precise measures of NPIs. Similar to Correia, Luck, and Verner (2020), Barro (2020), and Velde (2020), I utilize the data on NPIs provided by Markel et al. (2007) that describes the number of days in which at least one NPI category as described in Figure 3 was implemented and the speed of the implementation of the NPIs after the mortality acceleration date in the city[2]. A negative speed number implies that the city took action after the acceleration date in the specific city while a positive number implies that action was taken earlier. On average, cities had at least one NPI implemented for three months and were found to have implemented the first NPI approximately a week after the acceleration date.

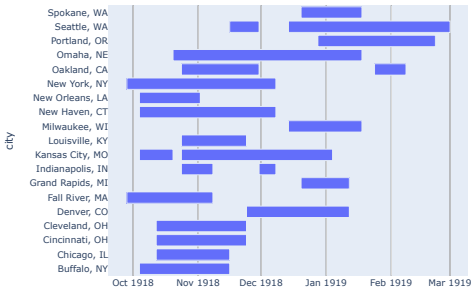
Figure 3 reports the timing of implementation of the four types of NPIs gathered by Markel et al. (2007). One can observe that the most widely used NPIs are school closures and public gathering bans that were implemented in 41 cities. On the other hand, isolation and quarantines were only implemented in 19 cities in the sample. Finally, 39 cities implemented other heterogeneous NPIs. For example, face masks could be mandatory or recommended as in Los Angeles or San Francisco. Public health messages were also published in newspapers as in Louisville or sent by mails as in Newark. New York, New Orleans and Los Angeles implemented staggered business hours while business hours were also restricted in Boston, Cincinnati or Columbus. Other NPIs also aimed to limit contamination in public transports; Louisville limited the capacity of streetcars while Milwaukee increased their frequency. Streetcars could also be cleaned and ventilated as in Albany. One can note that most of the NPIs were implemented during the second wave in October and November 1918 while fewer cities implemented these NPIs during the third wave. All these NPIs as facemasks, restrictions on business hours or public transport restrictions were classified as "other NPIs" by Markel et al. (2007).

2. The day the mortality rate exceeds twice its base

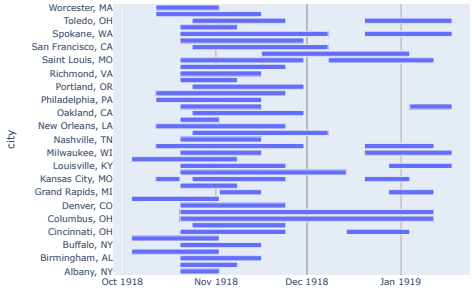
Figure 3: Timing of the four types of NPIs reported in Markel et al. (2007)



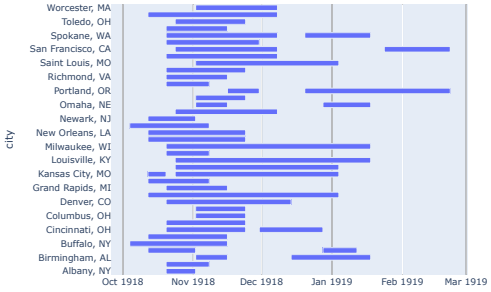
(a) School closures



(b) Isolation and quarantines



(c) Public gathering ban



(d) Other NPIs

Data were coded from the online appendix of Markel et al. (2007)
Other NPIS include mandatory or voluntary facemasks, street car ventilation , restricted business hours and diffusion of public health information.

3.2 Measurements of mortality

The most important issue in this paper is to measure mortality at the city level. I rely on the mortality tables for large cities published by the Census Bureau from 1905 to 1924. These reports are published yearly.[3]. One of the first sources are the report of 1920 and the report of 1924 that provide retrospective series of death rates for large cities by causes. The reports are consistent for overlapping years. I focus on death rates for all causes except stillbirths and deaths caused by influenza and pneumonia. I also utilize the 1911 reports to test the pre-trend for death rates for all causes back to 1908. There are five missing data points: Nashville, Tennessee in 1916 as no statistics for Tennessee are available for this year. Seattle and Los Angeles do not have death rates in 1923 and 1924 because the estimated population is not available. However, the total number of deaths is reported in the 1923 and 1924 reports. Thus, there is only one missing data point when using the two other

3. `https://www.cdc.gov/nchs/products/vsus/vsus_1890_1938.html`

mortality measurements described below.

The gross death rate is the ratio between the number of deaths and an estimated population number. As discussed in Correia, Luck, and Verner (2020) and Lilley, Lilley, and Rinaldi (2020) population census are conducted every 10 years (1890, 1900, 1910, 1920, 1930). Thus, the yearly estimates of the population rely on an extrapolation of the population based on the previous intercensus population growth and the redistricting of cities. In other words the population of 1911 is the result of the application of the yearly growth rate of the city from 1900 to 1910. A few additional adjustments might occur if the city annexed neighboring cities. The death rate might be subject to measurement error the further the years are from 1910 and from 1920. Thus, this problem is particularly important for the years 1918 and 1919.

I tackle this issue using three methods. First, when using reported death rates, I control systematically for the estimated population which allows to account for changes in the numerator due to the redistricting and extrapolations. Estimated population are usually found in the reports. They can also be obtained dividing the death rate from all causes with the number of deaths from all causes of the same year. Second, I utilize the number of deaths from all causes which is also reported in all yearly reports, (e.g Table 1 page 41 in the 1911 report) and ,thus, perform the analysis using the number of deaths divided by the population in 1910.[4] Third, in most specifications, I use the log of the number of deaths from all causes instead of a death rate. It must be noted that utilizing three different mortality indicators is an improvement with respect to the studies that rely on excess rates from Collins et al. (1930) which might be biased by the estimated population and focus on death from influenza and pneumonia. In robustness checks, I also gather data on the monthly number of deaths from all causes and the yearly number of deaths by age group from all causes from the same reports on mortality. Finally, I also use a number of deaths from influenza and pneumonia obtained multiplying the death rate from influenza and pneumonia with the estimated population of the corresponding year.

3.3 Other controls

I employ the exhaustive census for the years 1900, 1910, 1920 and 1930 which is downloaded on the IPUMS website and compiled by Ruggles et al. (2020) to control for the sociodemographic characteristics of the population as the share of black people, migration flows or the age structure. In addition, I also utilize the financial statistics reports to gather data on local authorities expenditures[5], in

4. An alternative could be to estimate the population between 1910 and 1920 with the growth rate between the corresponding census. However, Lilley, Lilley, and Rinaldi (2020) argues that the population in 1920 might be affected by NPIs.

5. `https://fraser.stlouisfed.org/title/financial-statistics-cities-164?browse=1900s`

Table 1: Descriptive Statistics for the 43 US Cities

	Mean	Std.Dev.	Obs	min	max
Demographics					
Population (1900)	328018.60	576706.40	43	36800	3437200
Population (1910)	441201.02	776807.64	43	100292	4770082
Population growth (1900-1910)	0.50	0.56	43	0	2
Sex Ratio (men/women) 1910	1.03	0.12	43	1	1
average age (1910)	28.39	1.32	43	25	31
First decile age (1910)	5.09	0.92	43	4	7
Median Age (1910)	26.42	1.56	43	23	30
Ninth decile age (1910)	53.51	1.88	43	49	58
Health					
NPI days (1918)	88.28	46.43	43	28	170
NPI Speed (1918)	-7.35	7.84	43	-35	11
Death Rate (1917)	179.10	61.53	43	59	380
Death Rate (1918)	647.14	187.53	43	283	1244
Health Expenditures per head (1900)	0.19	0.11	43	0	1
Health Expenditures per head (1917)	1.84	0.61	43	1	3

Author's computation from the Bureau of the Census, Mortality Statistics 21st Annual Report published in 1920 and 1924 and Financial Statistics of Cities Having a Population of Over 30,000 for the years 1900 and 1917. NPI variables are from Markel et al. (2007). Data on age and population are from from the US census gathered by Ruggles et al. (2020).

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

particular health expenditures in 1917 before the influenza. I also use the monthly temperature at the state level.[6]. The main variables used are summarized in Table 1. For the robustness checks, I also utilize the data gathered in Clay, Lewis, and Severnini (2018, 2019) to control for air quality, distance to military camps and other confounding factors highlighted in these studies. I also digitize the Statistical Abstract of the United States from the Census Bureau to extract information on the number of wage workers, aggregate wages, the total output and the added value for the 43 cities from 1899 to 1923.

6. `https://www.ncdc.noaa.gov/cag/statewide/time-series/`

4 Measuring the impact of NPIs on mortality in the medium term

4.1 Empirical specification

Epidemiological studies investigate how NPIs enable the flattening of an epidemic curve by examining high frequency (weekly) data (Markel et al. 2007; Bootsma and Ferguson 2007) and the mortality peak (Barro 2020; Correia, Luck, and Verner 2020). I follow a different approach in order to study the impact of NPIs in the medium term. I employ a DID approach as detailed in Bertrand, Duflo, and Mullainathan (2004) or in Dimick and Ryan (2014) for health policies. To check for the underlying hypothesis, in particular the pre-trend assumption, I first utilize an event study following a growing econometric literature (Duflo 2001; Autor 2003; Fetzer 2019; Correia, Luck, and Verner 2020); this is also used in ongoing studies documenting the impact of NPIs during the Covid-19 pandemic -such as Kong and Prinz (2020), Lin and Meissner (2020b), and Allcott et al. (2020). I estimate the following equation to explain mortality at the city level:

$$\begin{aligned} Mortality_{i,t} =& \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + Z_{i,t}\theta \\ &+ \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t} \end{aligned} \tag{1}$$

Mortality is measured for two main causes: all deaths but stillbirths and deaths from influenza and pneumonia (used in Bootsma and Ferguson (2007), Markel et al. (2007), and Correia, Luck, and Verner (2020) as people contracting the flu often die from pneumonia). X_i controls for the population in 1900 and health expenditures per capita in 1917. These controls capture the potential diverging behaviour of cities with different characteristics. I also control for time varying variables-the minimum and maximum monthly average temperature of the year as in Barro (2020) and the estimated population when mortality is measured with the original death rate reported in the reports. There are two continuous NPI terms reported in Markel et al. (2007). The first term, NPI Speed, measures the rapidity of the response with respect to the acceleration date in the city, and the second term, NPI Days, measures the duration that NPIs such as social distancing and school closures were implemented. Using the log of the number of days does not affect the results. β_t is used to understand whether cities that responded more aggressively to the pandemic had different trends from 1911 to 1920.

To compute the net effect, I also estimate a simpler DID specification:

$$\begin{aligned} Mortality_{i,t} =& \delta_i + \gamma_t + \beta \times Post \times NPI_{1918,i} \\ &+ \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t} \end{aligned} \tag{2}$$

where *Post* takes value of 1 when the year is beyond 1917.β is used to measure the net impact of NPIs implemented in 1918 from the year 1918 until the end of

the observations (up to 1924). Both equations are estimated by the ordinary least squares method and standard errors are clustered at the city level.

4.2 Potential endogeneity concerns

One might be concerned by the fact that the intensity of NPIs might be endogenous. In other words, cities that were more sensitive to the flu might implement stronger NPIs to mitigate their consequences (Barro 2020). The first means to test the potential endogeneity of NPIs and the comparability of cities with different levels of NPIs is to investigate pre-trends. Nevertheless, one might fear that the specific characteristic of cities as the age distribution, the share of black people or their geographic location make them more sensitive to influenza when compared with all previous causes of deaths and infectious diseases or lead them to behave differently after the pandemic. For example, cities that implemented stronger NPIs were concentrated on the West Coast that could go through a specific economic and social development after the first World War. Thus, I control for regional shocks in robustness checks by interacting year dummies with regional fixed effects thereby indicating whether the city is located in the Midwest, the North East, the West or the South. Moreover, Demographics and migration might also be a concern if cities that implemented stronger NPIs had a population that is more sensitive to the influenza. I take advantage of additional data sets, particularly the 1910 and 1920 census, to control for population's characteristics as the median age or the racial composition. Moreover, as the age structure might be key to understanding the evolution of mortality, I also collect additional data with the number of deaths in each city by age group in order to control for the age of those who died. Section 5.2 and 5.3 perform robustness checks to control for these potential endogeneity issues.

5 Results

5.1 Results of the event study

Figures 4 and 5 display the estimates of β_t. It appears that the common trend assumption is fulfilled before the 1918 pandemic and that cities with high and low NPIs had similar mortality trends for all three mortality measures.These policies do not appear to have any significant impact in 1918. This is particularly striking when one observes the log mortality. This might be explained by the fact that the influenza may also be reflected more in the total death rate if those who die from influenza have other co morbidity factors. Moreover, classifying death in the middle of a pandemic might lead to errors (Spinney 2017). On the one hand, it is possible that overwhelmed cities attribute an excessive number of deaths to the ongoing pandemic. On the other hand, more voluntary cities might have also attributed less death to the influenza. If the duration and speed of implementation of NPIs seems to be associated with a slightly lower but not significant reported death rate from influenza and pneumonia, this pattern disappears with the two alternate measures

of mortality. These results are consistent with those of Clay, Lewis, and Severnini (2018) and more recently with Barro (2020); the latter suggests that NPIs enabled a flattening of the epidemic curve, thereby reducing the peak mortality without significantly decreasing overall mortality because they were implemented for a short period of time. Ferguson et al. (2020) also emphasize that NPIs do not necessarily reduce the number of cases when implemented for a short period of time. The results are slightly different from those of Correia, Luck, and Verner (2020) and Markel et al. (2007), both of which are probably based on an excess death rate for influenza and pneumonia computed in Collins et al. (1930) that might be affected by measurement errors in the population estimates and in the number of deaths as discussed above. Under light of the current econometric studies on the impact of NPIs during the Covid-19 crisis, the insignificant impact of mild NPIs implemented in 1918 are not surprising as much stronger NPIs are only found to have a relatively weak effect on the spread of the virus (Lin and Meissner 2020b; Allcott et al. 2020; Dave et al. 2020).

More interestingly, one can observe a significant rebound of mortality in these cities from 1919 onward. This phenomenon might be explained by several phenomena brought to light by the epidemiological literature. First Hatchett, Mecher, and Lipsitch (2007) emphasize that cities that implemented stronger NPIs during the second wave appear to be hit harder by the third wave; this appears to be confirmed from the monthly series in Section 5.2. Second, it is worth noting that the 1918 pandemic gave birth to most of the subsequent virus strains (Taubenberger and Morens 2006). Consquently, the 1918 virus was probably the first influenza virus that afflicted a majority of U.S. citizen. This fact is important as Gostic et al. (2016) reveal that the first influenza virus that emerged is key to understanding one person's lifelong immunity response to influenza. The authors indicate that the spread of influenza in the past enables the prediction of the diffusion of influenza and mortality patterns in subsequent years. Indeed, Gostic et al. (2016) indicate that individuals that contracted certain particular strains of influenza are then less likely to die from influenza during their life. Therefore, by flattening the epidemic curve, NPIs might have reduced the number of infections even without significantly decreasing the number of deaths (Barro 2020) and thus increased the population's susceptibility to the subsequent strains of influenza. The fact that the results are clearer for NPI duration likely indicate this. Indeed, the longer people are isolated from each other, the lower their exposure to the initial strain of influenza, the lower the population's immunity, and, thus, the higher the death rate. It may be believed that school closures might have reduced the exposure of children, thereby making them more susceptible to the virus in subsequent years. The 1918 pandemic might have acted as a lifelong vaccine changing the immune responses of the contaminated population for their entire lives reducing the likelihood of them dying from influenza.

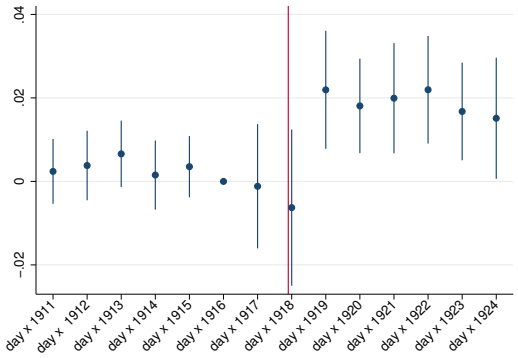
Moreover, this individual immunity might be complemented by the development of a herd immunity. Indeed, Fine, Eames, and Heymann (2011) reported that *"one proposal has been to reduce community spread of [influenza] by concentrating on vaccination of schoolchildren, as transmission within crowded classrooms leads to rapid*

dispersal throughout the community, and into the homes where susceptible adults reside". Consequently, it may believed that NPIs might have actually prevented the development of an herd immunity by reducing the level of individual immunity and decreasing the contact among people, thereby facilitating the circulation not only of the next strains of influenza but also of other infectious diseases that accounted for a large part of deaths at that time.

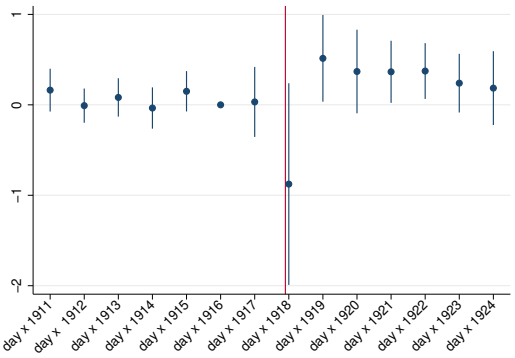
Finally, Douglas et al. (2020) emphasize that NPIs might be associated with adverse health outcomes. For example, a reduction in physical activity might increase the death rate for all causes (Gregg et al. 2003). Moreover, NPIs might also be associated with socioeconomic disruptions (Kong and Prinz 2020; Gregg et al. 2003), which might also have adverse health consequences (Markel, Stern, and Cetron 2008) and could eventually contribute to increased mortality.

An alternate explanation could be that cities that implemented NPIs in 1918 had a higher number of deaths and, thus, a jump in life expectancy, because a lot of people with bad health conditions were purged from the population while the people who were left behind were healthier (Spinney 2017). However, the positive impact of NPIs on mortality from October 1918 to February 1919 does not indicate anything in this direction. The number of deaths should have been lower in 1918 for the purged effect to be valid. Additional robustness checks were performed by interacting the level of mortality in 1918 with year dummies from 1919 to 1924. This did not change the results.

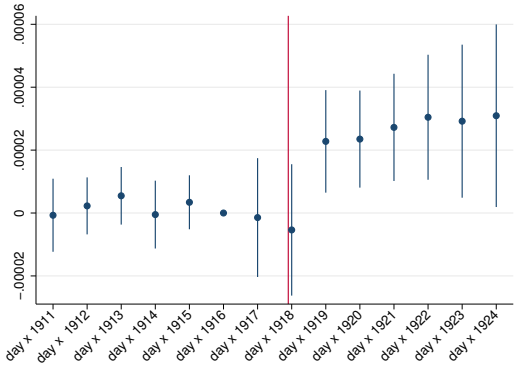
Figure 4: Event study: Estimates of the aggregate impact of NPI implementation duration on mortality



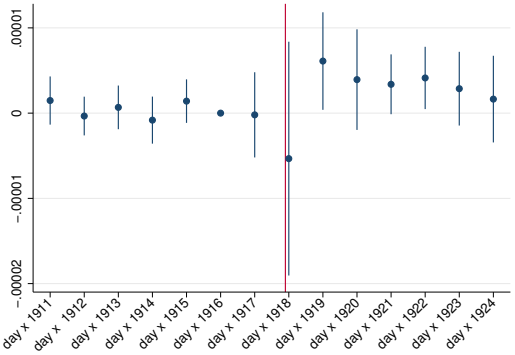
(a) Reported Death rate:
All causes of death



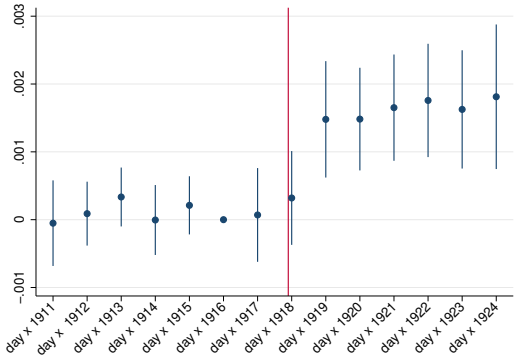
(b) Reported Death rate:
Influenza and Pneumonia



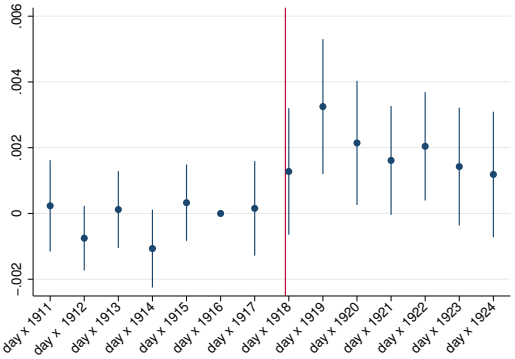
(c) Deaths/$Population_{1910}$:
All causes of death



(d) Deaths/$Population_{1910}$:
Influenza and pneumonia



(e) log(deaths):
All causes of death



(f) log(death):
Influenza and pneumonia

Reading notes: Cities that implemented NPIs for a longer duration saw their death rates increase less than cities that had shorter NPIs in 1918. On the other hand the death rate was relatively higher in 1919 and 1920 for these cities

Estimates of the difference in difference equation:

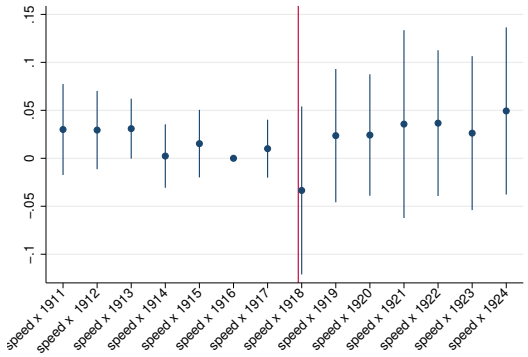
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$

Controls include health expenditures in 1917, population in 1910, years and cities' fixed effects

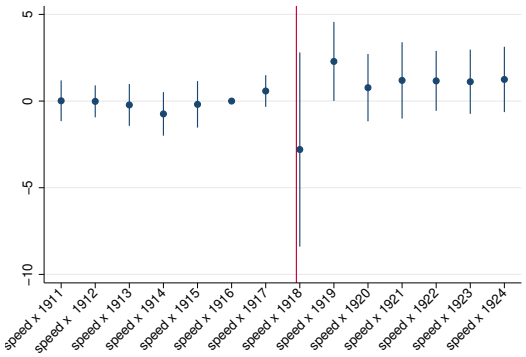
95% confidence interval clustered at the city level

NPI days is the number of days in which at least 1 NPI category was implemented

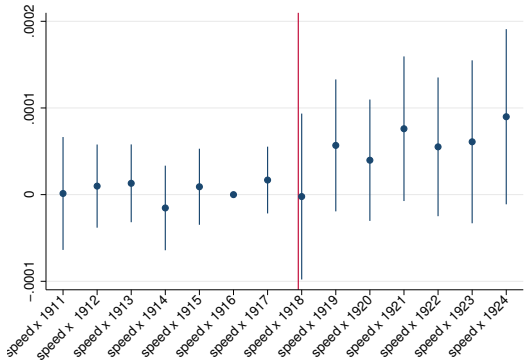
Figure 5: Event study: Estimates of the aggregate impact of NPI implementation speed on mortality



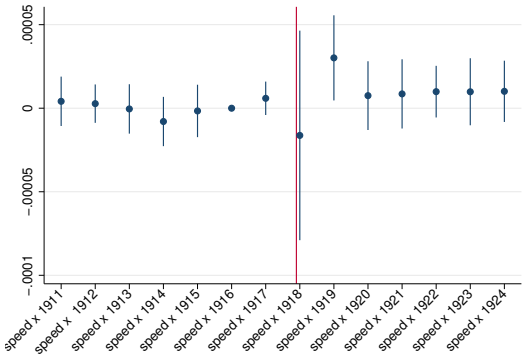
(a) Reported Death rate:
All causes of death



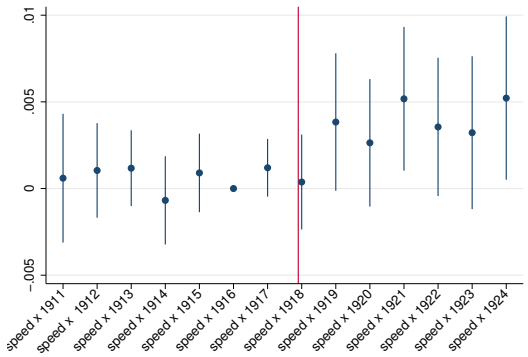
(b) Reported Death rate:
Influenza and Pneumonia



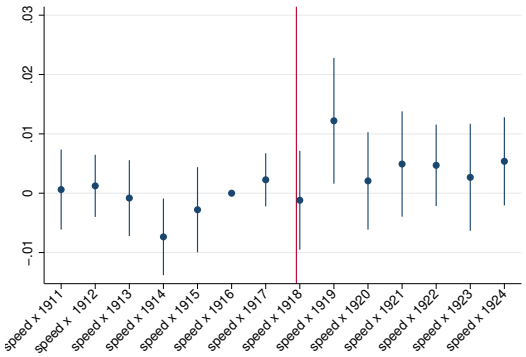
(c) Deaths/$Population_{1910}$:
All causes of death



(d) Deaths/$Population_{1910}$:
Influenza and pneumonia



(e) log(deaths):
All causes of death



(f) log(death):
Influenza and pneumonia

Reading notes: Cities having adopted more rapidly NPIs saw their death rates increase less than cities that were slower in 1918. On the other hand the death rate was relatively higher in 1919 and 1920 for these cities

Estimates of the difference in difference equation:

$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$

Controls include health expenditures in 1917, population in 1910, years and city fixed effects

95% confidence Interval clustered at the city level

NPI speed is the timing of implementation of the First NPI w.r.t the acceleration date of the pandemic in the city

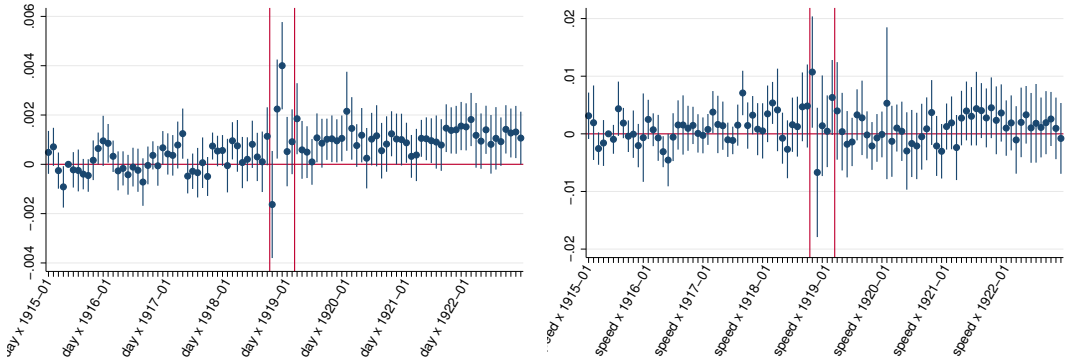
5.2 Robustness checks

I perform several robustness checks to verify the underlying hypothesis, to investigate the short run impact of NPIs, and to control for the influence of the demographic structure of cities and geography before and after the 1918 pandemic.

Additional tests of the common trend assumption. The data for the total number of deaths and the total death rate are available for all cities of the sample from 1908. I perform a robustness check adding 1908,1909 and 1910 and control for the estimated population with all mortality indicators in Figure B.1 in the Appendix. The pre-trends continue to be fulfilled and results remained unchanged. I also control for variation in the surface of the city between 1910 and 1920 to control for redistricting, this does not change the results.

Evidence from monthly data. I collect data on the monthly number of deaths at the city level from 1915 to 1922 (the year 1916 is still missing for Nashville). I can reproduce the event study with year x month fixed effects, control for the monthly temperature in the specific state and the timing of the pandemic (time between the first case in the city and the first case in the sample as well as time between the acceleration date in the city and the first acceleration date in the sample). The results are reported in Figure 6. The two red lines represent the date of implementation of the first NPI in the sample (September 27, 1918) and of withdrawal of the last NPI in the sample (February 28, 1918). The observed aggregate patterns remain valid. There is no trend observed in the preceding month. Moreover, it is noteworthy that during the first wave of the 1918 influenza, cities with low and high NPIs behaved in an extremely similar manner. Panel a) reports the coefficients on the number of days. It appears that cities with longer NPIs began performing slightly better in October 1918, with a relatively lower number of deaths, but the mortality then increased in November and December 1918. These patterns logically suggest that cities that implemented longer NPIs in the fall during the second wave were those hit harder in winter by the third wave of the pandemic. These patterns are consistent with the results in Hatchett, Mecher, and Lipsitch (2007), who find that cities that implemented early and continuous NPIs during the first wave were more sensitive to the next wave and explain why long NPIs did not have any significant effect in 1918 on the annual number of deaths. Moreover, from panel b), it is also noteworthy that the speed of implementation is never significantly associated with a lower or higher mortality level.

Figure 6: Event study: Estimates of the aggregate impact of NPIs on monthly mortality (All causes of death)



(a) log(deaths)
Number of Days

(b) log(deaths)
Speed of implementation

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 191502} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, monthly temperature in the state, yearsx-month fixed effects and cities' fixed effects.
95% confidence interval clustered at the city level
NPI days is the number of days in which at least 1 NPI category was implemented
NPI speed is the timing of implementation of the First NPI w.r.t the acceleration date of the pandemic in the city

Controlling for excess mortality in 1918 Spinney (2017) emphasizes the fact that the 1918 influenza was followed by a rise in life expectancy as people who died from the influenza were the most fragile. Provided the insignificant correlation between NPIs and mortality in 1918, this channel is unlikely to explain the results. However, to check that the rise of mortality in cities with high implementation of NPIs is not due to this effect, I also control for the excess mortality in 1918. The results remain unaffected as illustrated in Figure B.3 in the Appendix.

Controlling for air pollution Clay, Lewis, and Severnini (2018) stress the fact that air pollution explains a portion of the variation of mortality among cities in 1918. We utilize their indicator on air pollution (coal fire plant capacity within 30 miles) and their additional controls (share of white people, the distance from military camps and the timing of the pandemic in the city) using their data set that encompasses 32 cities of their sample that have data on NPIs. The patterns and results remain unchanged as illustrated in Figure B.4 in Appendix.

Differentiated trends between the East and the West. As discussed in Correia, Luck, and Verner (2020), the pandemic spread from the East to the West, thereby providing the West more time to adjust. One potential confounding factor could be that cities on the West Coast began behaving differently from the East Coast after the First World War due to certain regional shocks. I control for this

eventuality adding regional shocks, that is interacting years fixed effects with a fixed effect to indicate which of the four regions the city belongs to (West, South, North East, Midwest), Confidence intervals are wider and point estimates are slightly lower as there is less within region variation however the results remain statistically significant as illustrated in Figure B.2 in the appendix. Similar robustness checks are performed for the monthly series in Figure B.6.

Migrations. The flows of migrants after the first World War might have contributed to increase the mortality in some cities. To my knowledge, there is no statistics on the yearly flows of migrants at the city level. However, in the census, migrants declare their year of arrival in the US. I thus create a proxy for yearly flows of migrants using their arrival date and their city of residence in 1920 and 1930 to approximate the yearly number of migrants arrived from 1911 to 1919 and from 1920 to 1924 respectively. This measure is not perfect but might allow to proxy the main differences between cities. The inclusion of this estimated number of migrants does not affect the patterns as reported in Figure B.5 in the Appendix. Additional tests, Available upon request, including one or two lags were performed. This did not affect the results.

Demographic structure. An alternate explanation could be that cities with an aggressive policy may have a different demographic structure that could explain their divergence in terms of mortality after 1918. Tables B.1, B.2, B.3 and B.4 compare the demographic structure of these cities (population, population growth, sex ratio, average age, age distribution, share of each cohort and age groups) in 1910 and 1920. It is noteworthy that cities that implemented longer and earlier NPIs were younger, had higher population growth rates and had proportionally more males in 1910 and 1920. This reflects the fact that these cities tend to be located on the West Coast. I follow the epidemiological literature as Markel et al. (2007) and also control explicitly for the difference in population growth for each decade and the sex ratio, the median age, the share of white people in 1910, before the pandemic, or in 1920, immediately following the pandemic; in all such cases, the results are similar when controlling for regional shocks as illustrated in Figures B.7 and B.8. Similar robustness checks are performed for the monthly series in Figure B.6. In order to control for the implications of the demographic structure, I go further and also collect detailed mortality tables by age groups[7] from 1913 to 1922. Thus, I can estimate the same model but including age group fixed effects, age groups shocks and even age groups x city fixed effects. Figure B.9 presents the estimations of the

7. Age groups are groups of five years: less than 5, 5-9 etc...

following equations:

$$\begin{aligned} ln(death_{i,g,t}) =& \delta_i + \eta_g + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + Z_{i,t}\theta \\ &+ \sum_{t \neq 1916} \lambda_t \times 1_{t(i,g)=t} \times X_i \\ &+ \sum_{g \neq [0;4]} \sum_{t \neq 1916} \pi^{t,g} \times 1_{t(i,g)=t} \times 1_{g(i,t)=g} + \epsilon_{i,t} \end{aligned} \tag{3}$$

$$\begin{aligned} ln(death_{i,g,t}) =& \delta_{i,g} + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + Z_{i,t}\theta \\ &+ \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t} \end{aligned} \tag{4}$$

where $death_{i,g,t}$ is the number of deaths in city i; and age group g during the year t. η_g is an age group fixed effect, $\eta_{i,g}$ is a city x year fixed effect, and $\pi^{g,t}$ captures shocks specific to each age group in year t. The patterns reported in Figure B.9 remain unchanged. Exploiting variation within groups for age does not affect the results, thereby relieving the concern that the sudden change after 1918 could be driven by demographic shocks affecting certain cities with a particular demographic structure or a change in the demographic structure of cities.

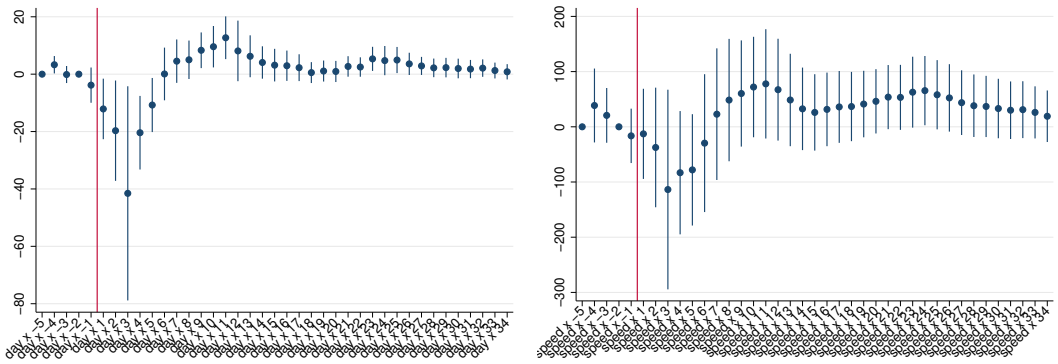
Using weekly data from Collins et al. (1930) Ongoing studies documenting the impact of NPIs during the Covid-19 pandemic such as Kong and Prinz (2020), Lin and Meissner (2020b), Allcott et al. (2020), and Dave et al. (2020) estimate event studies that compare counties or states that have implemented NPIs with counties or state that have not done so. Unfortunately, their framework is not suited for the data available for 1918. All cities in the sample are treated and applied some NPIs; thus there is no control group. I adapt the empirical specification to accommodate weekly data and to account for the timing of the pandemic in each city:

$$\begin{aligned} Excess_{i,t} =& \delta_i + \gamma_t + \sum_{ws=-5;ws\neq-4}^{38} \pi^{ws} \times 1_{t(i)=ws} + \sum_{ws=-5;ws\neq-4}^{38} \beta^{ws} \times 1_{t(i)=ws} \times NPI_{1918,i} \\ &+ \sum_{w=-3,w\neq-2}^{30} \omega^t \times 1_{t(i)=w} + \sum_t \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t} \end{aligned} \tag{5}$$

$Excess_{i,t}$ is the weekly excess death rate from influenza and pneumonia used in Bootsma and Ferguson (2007) and Markel et al. (2007) and computed in Collins et al. (1930); it is only available for flu epidemics and no data for death from all causes are available. This excess death rate has the same problems as the yearly death rates and relies on population estimates for the years 1918 and 1919 but also of the one used in the median computed from the years prior 1918 (see Collins

et al. (1930) and Bodenhorn (2020) for a discussion of the method). Moreover, several cities have missing points for certain weeks, with the most important being Birmingham where there is a missing week in the middle of the epidemic curve. In addition, Weekly data with no treatment on the number of deaths for influenza and pneumonia or total number of deaths are not available. There are only a few data points to test for pre-trends before the epidemic. γ_t is a time fixed effect. $\sum_{ws=-4}^{38} 1_{t(i)=ws}$ are dummies indicating the time elapsed from the acceleration date to account for the timing of the pandemic in the city. λ_t is a time fixed effect interacted with control variables X_i. One might fear that the acceleration date might be affected by the implementation date of NPIs. However, it is worth noting that only four cities implemented their first NPI before the acceleration date and the expected effect of NPIs is always after the acceleration date except for New York. In addition, I also control for the distance with respect to the implementation date of NPIs ($\sum_{w=-3,w\neq-2}^{30} \omega^t \times 1_{t(i)=w}$). The results are displayed in Figure 7. These patterns are consistent with the findings of Bootsma and Ferguson (2007), Markel et al. (2007), and Correia, Luck, and Verner (2020) that long and sustained NPIs managed to flatten the epidemic curve as the duration of the implementation of NPIs is associated with a decrease in the excess death rate from influenza during the five weeks after the acceleration date. It is also associated with higher excess mortality, thereby reflecting that these cities might be more sensitive to the second wave as illustrated with the monthly data. This also tends to support the findings in Hatchett, Mecher, and Lipsitch (2007) which indicate that faster cities were also hit harder by the third wave. These results are in line with those Ferguson et al. (2020) which explain that NPIs might flatten the epidemic curve without reducing the number of cases. These results also support the interpretation that a certain proportion of the rebound in mortality from 1919 might be explained by a lower herd and individual immunity. Following the recommendation from Allcott et al. (2020) I perform robustness checks eliminating fixed effects of cities and using a dummy to indicate when the observations are out of the event study windows. This does not change the results.

Figure 7: Event study: Estimates of the aggregate impact of NPIs on weekly excess mortality (Influenza and pneumonia)



(a) Excess death rate
Number of Days

(b) Excess death rate
Speed of implementation

Controls include health expenditures in 1917, population in 1910, week fixed effects and cities' fixed effects, fixed effect for the distance from the acceleration date, fixed effects for the distance since the implementation of the first NPI
The red line materializes the acceleration date of the epidemic
95% confidence interval clustered at the city level
NPI days is the number of days in which at least 1 NPI category was implemented
NPI speed is the timing of implementation of the First NPI w.r.t the acceleration date of the pandemic in the city

5.3 The medium-term impact of NPIs on total mortality

In order to obtain an idea of the net benefits of NPIs, I run a DID specification for estimating equation 2. Table 2 displays the main results for mortality from all causes. Columns (1) to (4) utilize the reported total death rate as dependent variable; columns (5) to (8) utilize the total number of deaths on the population of 1910 while columns (9) to (12) utilize the log of the total number of deaths. Panel a) reports the coefficients for the number of days for which NPIs were implemented. Columns (1), (5) and (9) have no further control than city and year fixed effects. The estimated impact of the duration of NPIs is similar for the three indicators, one extra day of NPIs is associated with an increase of 0.014 deaths per 1000 in the estimated population, 0.02 increase in the number of deaths per 1000 people living in the city in 1920 and an increase of the number of deaths of 0.16%. This implies that an increase of standard deviation (46 days) in the duration of NPIs is associated with an increase in the number of deaths by approximately 7 percent. Columns (2) , (6) and (10) control for additional variables -the population in 1900, the minimum and maximum monthly temperature of the year in the State, the Estimated Population for column (2) and the amount of municipal health expenditures per capita in 1917. Results are similar in magnitude. Finally, columns (3), (7), and (11) control for regional shocks interacting year fixed effects with regional dummies (Midwest, West, North East and South). As the implementation of NPIs is related

to the geographical location of cities the identifying variation is lower. The results remain significant but the point estimate is lower particularly for the log number of deaths. One extra day of NPIs is associated with an increase of 0.011 in the number deaths per 1000 people in the estimated population, an increase of 0.02 deaths per 1000 people in the 1920 population and an increase of 0.07% of the number of deaths. As expected, including demographics control as in columns (4) , (8) and (12) has a similar impact on the estimated coefficients as including regional shocks as demographic discrepancies between low and high NPIs cities are driven by the difference in East Vs West, both of which are correlated with the implementation of NPIs. Estimates for the two rates are significant at the 85% significance level while estimates for the log number of deaths is significant at the 90% significance level and is the same as when including regional shocks. The same specification is performed in Panel b) but using the speed of implementation as a measure of NPIs. The results are also positive but never statistically significant. Additional robustness checks are presented in Table 4 using the number of deaths by age group instead of the total number of deaths substituting $\sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i}$ with $Post \times NPI_{1918,i}$ in equations 3 and 4. It is worth noting that the results are of similar magnitude when accounting for differences in age groups between cities and regional shocks or other demographics. Table B.5 in Appendix reports the same estimates with mortality from influenza and pneumonia as a dependent variable. As for mortality from all causes point estimates are always positive but only significant when looking at the impact of NPI duration on the log number of deaths.

A similar exercise is performed in Table 3 but the log monthly number of deaths from all causes is utilized. Columns (1) to (4) use monthly observations from January 1915 to February 1919. Columns (5) to (8) extend the series to December 1920 while columns (9) to (12) run through December 1922. The results are similar in magnitude as those displayed in columns (8) to (12) in Table 2. In this specification, including demographic controls and yearly regional shocks affect the coefficient estimates, to a lesser extent. This could be interpreted as the fact that NPIs modified the subsequent seasonality of mortality. High NPIs cities could have higher mortality during the month where influenza strains were active. The impact of the duration of NPIs is always statistically significant. It is stronger in 1918 which implies that the observed rebound in Figure 6 at the beginning of the third wave in winter erased the benefits of the first wave, thereby explaining the non significant impact of NPIs on the total number of deaths in 1918. The estimated impact progressively decreases as the time length is extended suggesting that the impact of NPIs on mortality is progressively fading. Panel b) reports the coefficients of NPI speed. The results are closer to Hatchett, Mecher, and Lipsitch (2007), as the point estimate is negative but never statistically significant.

Table 2: Medium Run Impact of NPIs on Mortality (All causes of death) (1911-1924)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Reported Rate				Deaths/$Population_{1910}$				ln(Deaths)			
Panel a) Impact of NPI Days on Mortality												
Days NPI x Post	0.0138***	0.0125***	0.0110*	0.00670	0.0223***	0.0209**	0.0117	0.0131*	0.0016***	0.0013***	0.0007*	0.0007*
	(0.0040)	(0.0044)	(0.0063)	(0.0046)	(0.0064)	(0.0083)	(0.0077)	(0.0076)	(0.0004)	(0.0004)	(0.0004)	(0.0004)
R^2	0.878	0.888	0.929	0.910	0.866	0.858	0.892	0.882	0.993	0.994	0.996	0.995
N	597	597	597	597	597	601	601	601	601	601	601	601
Panel b) Impact of NPI Speed on Mortality												
Speed NPI x Post	0.00560	0.00630	-0.00290	-0.00890	0.0395	0.0488	0.0150	0.0280	0.00180	0.00290	0.000800	0.00140
	(0.0308)	(0.0271)	(0.0272)	(0.0236)	(0.0307)	(0.0317)	(0.0272)	(0.0319)	(0.0021)	(0.0019)	(0.0016)	(0.0019)
R^2	0.867	0.879	0.926	0.908	0.846	0.845	0.889	0.878	0.992	0.993	0.995	0.995
N	597	597	597	597	597	601	601	601	601	601	601	601
Controls												
Time FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Pop1900	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
temperature	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
Est. pop	Y	Y	Y	Y	N	N	N	N	N	N	N	N
Health exp.	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
Region shocks	N	N	Y	N	N	N	Y	N	N	N	Y	N
Demographics	N	N	N	Y	N	N	N	Y	N	N	N	Y

Post is a dummy indicating observations after 1917 while speed NPI indicates the speed at which the city implemented their NPI. Days NPI describes the length the NPI measures were in place.

Estimates of the difference in difference equations:

$Deathrate_{i,t} = \delta_i + \gamma_t + \beta \times Post \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$

Controls include health expenditures per capita in 1917, population in 1900. Est. Population corresponds to the estimated population of the city for each yea. Non varying variables are interacted with year fixed effects. I also includes years and city fixed effects. Temperature include the monthly temperature in the state. Temperature include minimum and maximum monthly temperature of the year in the State. Demographics control for population growth in the decade and interact share of whites, median age and sex ratio in 1910 with years fixed effects. Regional shocks interact regional dummies (Midwest, West, North East, South) with years fixed effects.

standard errors clustered at the city level.

NPI days is the number of days in which at least 1 NPI category was implemented

NPI speed is the timing of implementation of the First NPI w.r.t the acceleration date of the pandemic in the city

Table 3: The Impact of NPIs on Mortality (All causes of death) using monthly data

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	ln(deaths)											
	(From 01/1915 to 02/1919)				(From 01/1915 to 12/1920)				(From 01/1915 to 12/1922)			
Panel a) Impact of NPI Days on Mortality												
Days NPI x Post	0.0020***	0.0020***	0.0020**	0.0015**	0.0014***	0.0013***	0.0012**	0.0010**	0.0014***	0.0013***	0.0010**	0.0010**
	(0.0007)	(0.0006)	(0.0008)	(0.0006)	(0.0004)	(0.0004)	(0.0005)	(0.0004)	(0.0004)	(0.0004)	(0.0004)	(0.0004)
R^2	0.972	0.972	0.974	0.973	0.972	0.973	0.974	0.974	0.974	0.976	0.977	0.976
N	2138	2138	2138	2138	2568	2568	2568	2568	4116	4116	4116	4116
Panel b) Impact of NPI Speed on Mortality												
Speed NPI x Post	-0.00120	-0.00150	-0.00210	-0.00170	-0.000500	-0.000600	-0.00110	-0.000700	-0.000400	0.0000	-0.000500	-0.000400
	(0.0026)	(0.0026)	(0.0030)	(0.0025)	(0.0015)	(0.0017)	(0.0018)	(0.0018)	(0.0013)	(0.0016)	(0.0017)	(0.0018)
R^2	0.971	0.972	0.973	0.973	0.972	0.973	0.974	0.973	0.973	0.975	0.976	0.976
Obs	2138	2138	2138	2138	2568	2568	2568	2568	4116	4116	4116	4116
Controls												
Time FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Pop1900	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
Timing	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
temperature	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
Health exp.	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
Region shocks	N	N	Y	N	N	N	Y	N	N	N	Y	N
Demographics	N	N	N	Y	N	N	N	Y	N	N	N	Y

Post is a dummy indicating observations after September 1917 while speed NPI indicates the speed at which the city implemented their NPI. Days NPI describes the length the NPI measures were in place.

Estimates of the difference in difference equation:

$Mortality_{i,t} = \delta_i + \gamma_t + \beta \times Post \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{y(i)=t} \times X_i + \epsilon_{i,t}$

Controls include health expenditures per capita in 1917, population in 1900, and the timing of the pandemic (time between the first case (resp. the acceleration date) in the city and the first case (resp. the acceleration date) in the sample. Non varying variables are interacted with year fixed effects. I also includes years, city fixed effects. Temperature include the monthly temperature in the state. Demographics control for population growth in the decade and interact share of whites, median age and sex ratio in 1910 with years fixed effects. Regional shocks interact regional dummies (Midwest, West, North East, South) with years fixed effects.

standard errors clustered at the city level.

NPI days is the number of days in which at least 1 NPI category was implemented

NPI speed is the timing of implementation of the First NPI w.r.t the acceleration date of the pandemic in the city

Table 4: The Impact of NPIs on Mortality (All causes of death, 1913-1922) using mortality by age group

	(1)	(2)	(3)	(4)	(5)	(6)
			log(deaths)			
Panel a) Impact of NPI Days on Mortality						
Days NPI x Post	0.0011***	0.0007**	0.0008**	0.0011***	0.0007**	0.0008**
	(0.0003)	(0.0003)	(0.0003)	(0.0003)	(0.0003)	(0.0003)
R^2	0.964	0.965	0.964	0.978	0.979	0.978
Obs	8151	8151	8151	8151	8151	8151
Panel b) Impact of NPI Speed on Mortality						
Speed NPI x Post	0.0033**	0.00180	0.0023*	0.0033**	0.00180	0.0023*
	(0.0015)	(0.0013)	(0.0013)	(0.0016)	(0.0014)	(0.0013)
R^2	0.964	0.965	0.964	0.978	0.979	0.978
Obs	8151	8151	8151	8151	8151	8151
Time FE	Y	Y	Y	Y	Y	Y
City FE	Y	Y	Y	N	N	N
Age FE	Y	Y	Y	N	N	N
age x city FE	N	N	N	Y	Y	Y
pop1900	Y	Y	Y	Y	Y	Y
Temperature	Y	Y	Y	Y	Y	Y
Health	Y	Y	Y	Y	Y	Y
Age shocks	Y	Y	Y	N	N	N
Region shocks	N	Y	N	N	Y	N
Demographics	N	N	Y	N	N	Y

Post is a dummy indicating observations after September 1917 while speed NPI indicates the speed at which the city implemented their NPI. Days NPI describes the length the NPI measures were in place.

Estimates of the difference in difference equations 3 and 4

Controls include health expenditures per capita in 1917, population in 1900. Non varying variables are interacted with year fixed effects. I also includes years, city and age groups fixed effects or city x age group fixed effects. Temperature include the monthly temperature in the state. Regional and age group shocks interact regional (Midwest, West, North East, South) or age group dummies with years fixed effects.

standard errors clustered at the city level.

NPI days is the number of days in which at least 1 NPI category was implemented

NPI speed is timing of implementation of the First NPI w.r.t the acceleration date of the pandemic in the city

6 Conclusion

In this paper, I investigate the 1918 pandemic in the U.S. to assess the potential health benefits of NPIs at the city level. My findings can be summarized in the following manner: first, in the short run, evidence from weekly data on excess mortality from influenza tends to confirm that NPIs managed to flatten the epidemic curve. However, I do not find any significant negative impact of early and long implementation of NPIs on total Mortality from all causes. Indeed, the use of total

mortality instead of influenza specific mortality combined with cities fixed effects suggest that long NPIs are associated with a rise in mortality during the pandemic, while the rapid implementation of NPIs has no significant impact. Moreover, I find that cities that implemented NPIs for a longer duration underwent a relatively higher number of deaths in all subsequent years. These results are robust to the inclusion of numerous controls, such as regional shocks or the demographic structure and estimated migration flows. It sheds a new light on the impact of NPIs as they were implemented in 1918: NPIs are associated with higher mortality levels. These findings do not deny the short term benefits of these policies that might lower the peak of the pandemic and prevent overcrowding of the health system (Markel et al. 2007; Hatchett, Mecher, and Lipsitch 2007). However, they lead to the raising of some caution on their potential impact on health and mortality when they are repealed (Hatchett, Mecher, and Lipsitch 2007; Markel, Stern, and Cetron 2008) in the medium run. Thus, policymakers should prepare exit strategies to prevent NPIs from leading to higher deaths in their aftermath. Overall, my results regarding the impact of NPIs are in line with the literature extensively reviewed in Balinska and Rizzo (2009) and Markel, Stern, and Cetron (2008) that raise cautions on the net benefits of NPIs.

Furthermore, my results can also shed a new light on the current debate on the economic impact of NPIs during the 1918 pandemic (Correia, Luck, and Verner 2020; Lilley, Lilley, and Rinaldi 2020). While Correia, Luck, and Verner (2020) argue that NPIs might be associated with better economic outcome in the medium-run, their impact on mortality raises questions on the potential channels underlying these effects. As cities with long NPIs underwent higher levels of mortality, it is difficult to attribute their economic rebound to the potential lower mortality achieved by these policies. However, their benefits might be driven by the flattening of the epidemic curve (Eichenbaum, Rebelo, and Trabandt 2020) that could reduce the medium term business disruption.

The last word is a word of caution. As any study based on an historical natural experiment, this paper has limited external validity and thus applicability to current public health policies. The 1918 pandemic was an unprecedented event in the history of health and led to the emergence of most strains of seasonal influenza until 1977 which continue to kill up to 650,000 people yearly worldwide (World Health Organization 2007; Paget et al. 2019). It would be difficult to draw any inference regarding the predicted impact of NPIs as implemented during the Covid-19 crisis, not least because the magnitude and scale of the two pandemics are different and that the influenza and the Covid-19 are two entirely different viruses (Cohen-Kristiansen and Pinheiro 2020). In 2020, NPIs are mainly being implemented on a national (or state) scale, rather than at the city level. Moreover, pharmaceutical technologies were less developed back then as compare to what they are today, and the capacity to produce a new vaccine within a reasonable time was much lower back in 1918 (Ni et al. 2020; Callaway 2020).

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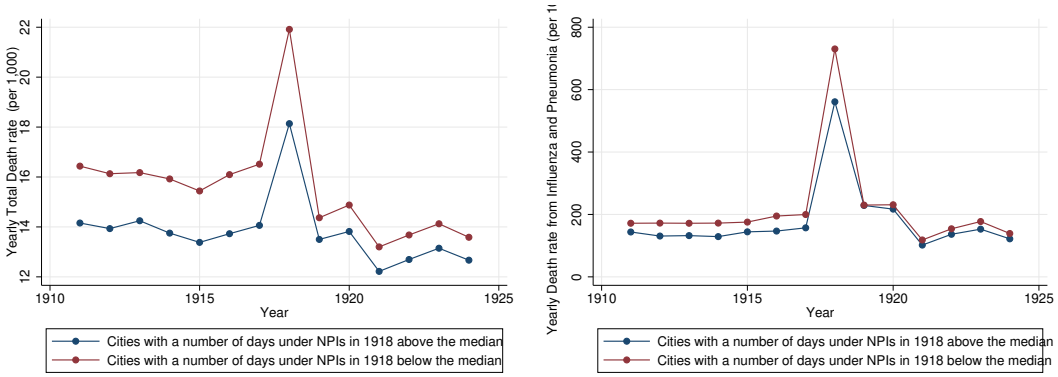
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World Health Organization. 2007. "Up to 650 000 people die of respiratory diseases linked to seasonal flu each year." Accessed April 11, 2020. `https://www.who.int/en/news-room/detail/14-12-2017-up-to-650-000-people-die-of-respiratory-diseases-linked-to-seasonal-flu-each-year`.

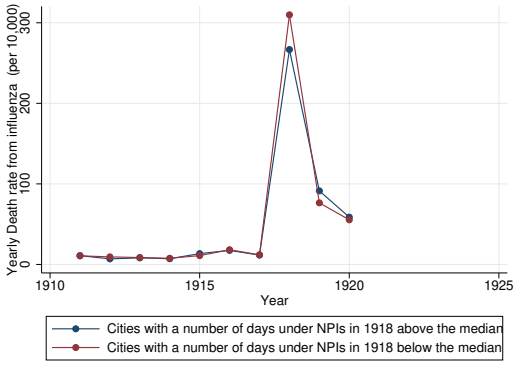
A Additional Series

Figure A.1: Evolution of the death rates by level of NPI in 1918



(a) All causes of death

(b) Influenza and Pneumonia



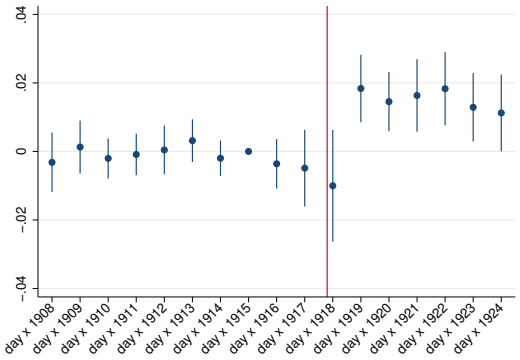
(c) Influenza Only

Reading notes: Cities that implemented NPIs for a longer time saw their death rates increase less than cities that had shorter NPIs in 1918. On the other hand the death rate was relatively higher in the next years for these cities

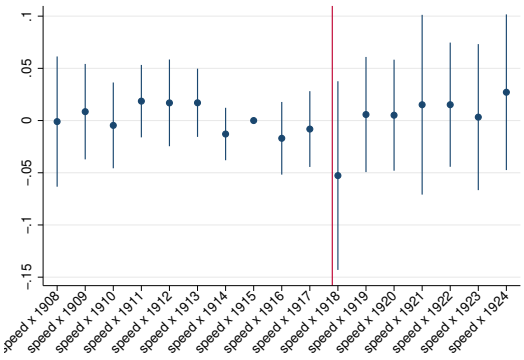
B Robustness Checks

B.1 Extending the series, controlling for regional shocks or variables in Clay, Lewis, and Severnini (2019)

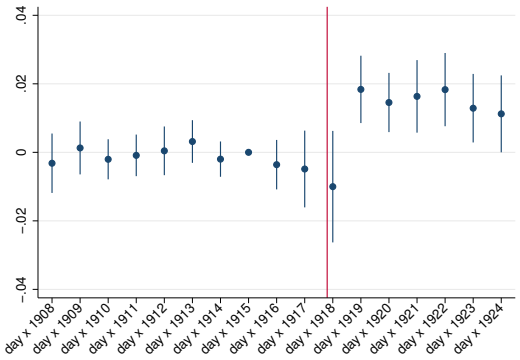
Figure B.1: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **from 1908**



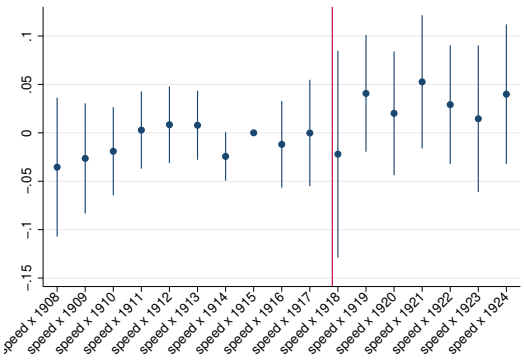
(a) Reported Death rate: Number of Days



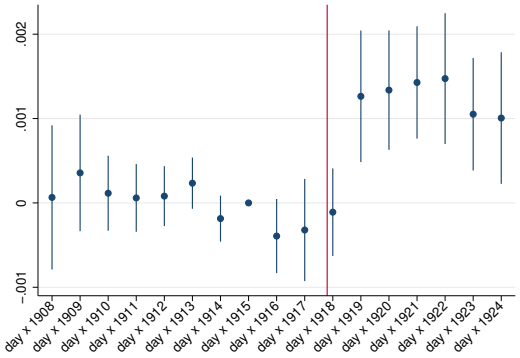
(b) Reported Death rate: Speed of implementation



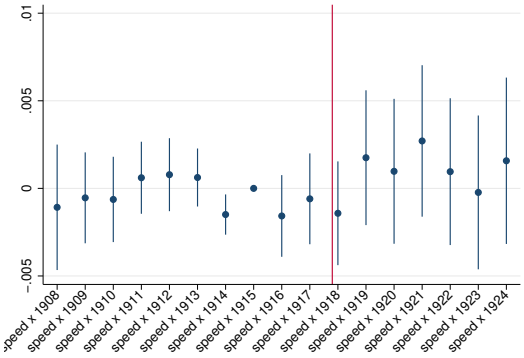
(c) Deaths/$Population_{1910}$: Number of Days



(d) Deaths/$Population_{1910}$: Speed of implementation



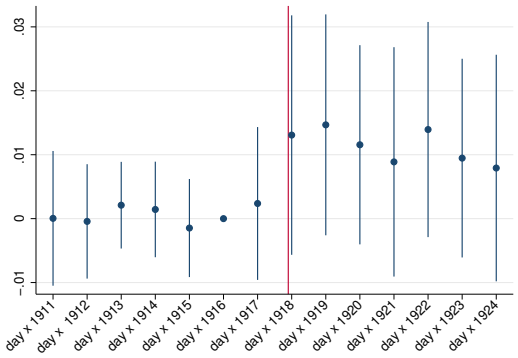
(e) log(deaths): Number of Days



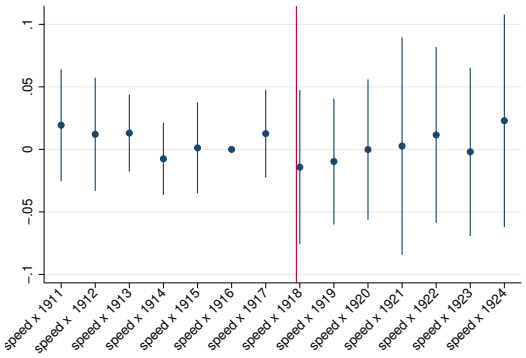
(f) log(death): Speed of implementation

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, years and cities' fixed effects
95% confidence interval clustered at the city level

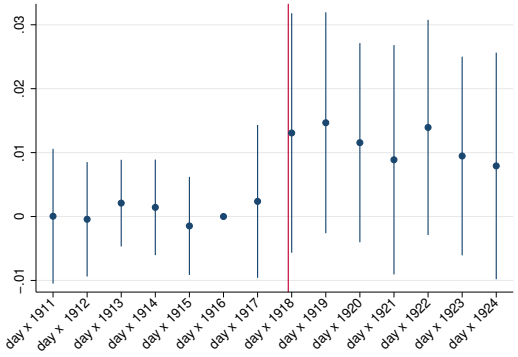
Figure B.2: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for regional shocks**



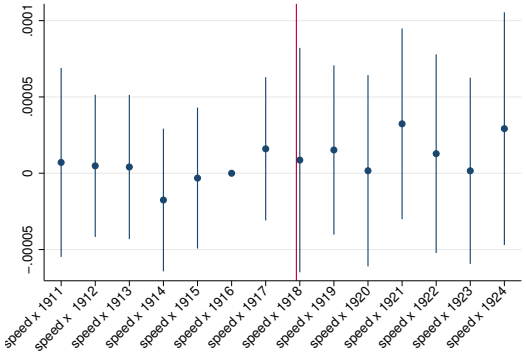
(a) Reported Death rate: Number of Days



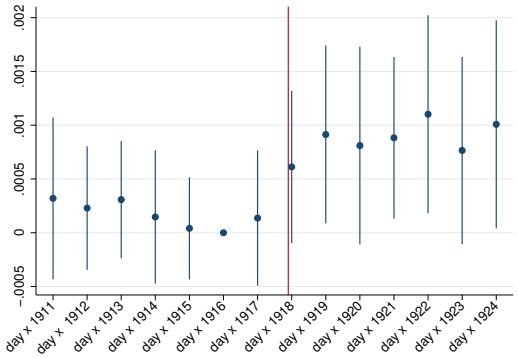
(b) Reported Death rate: Speed of implementation



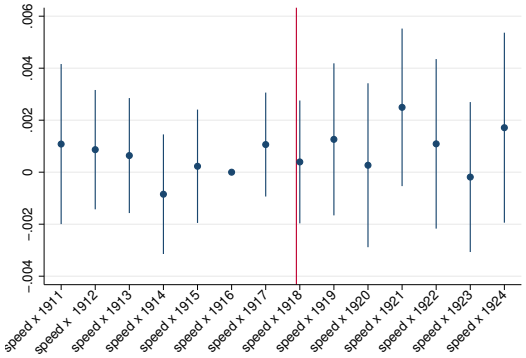
(c) Deaths/$Population_{1910}$: Number of Days



(d) Deaths/$Population_{1910}$: Speed of implementation



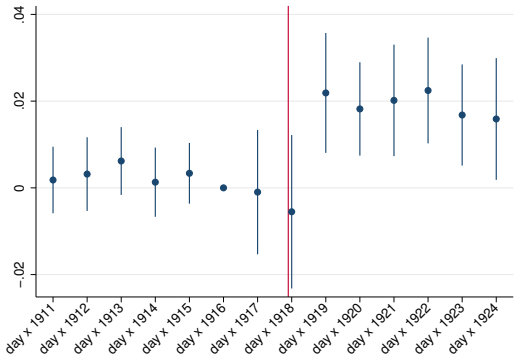
(e) log(deaths): Number of Days



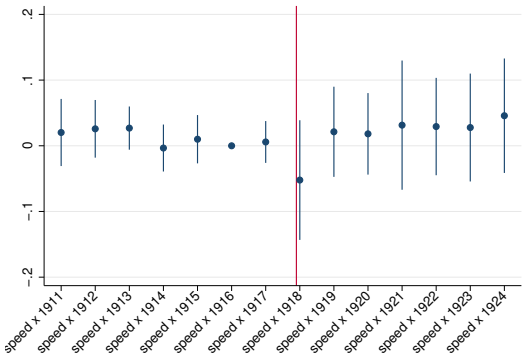
(f) log(death): Speed of implementation

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, yearly estimated Population, years and cities' fixed effects and interaction terms between the cities' region (West, Midwest, South, North East) and years fixed effects.
95% confidence interval clustered at the city level

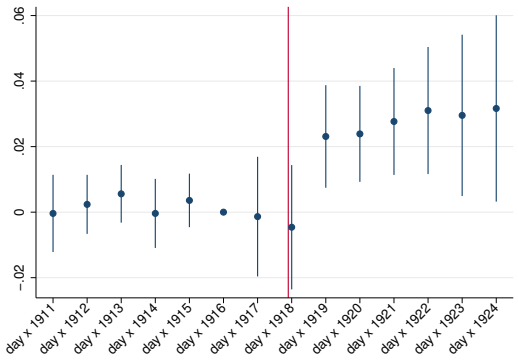
Figure B.3: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for excess mortality in 1918**



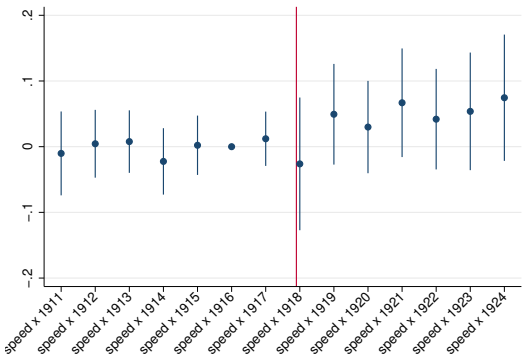
(a) Reported Death rate: Number of Days



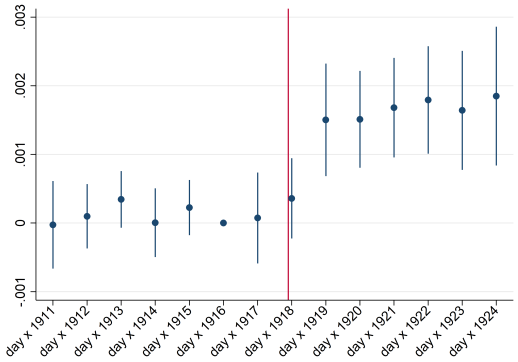
(b) Reported Death rate: Speed of implementation



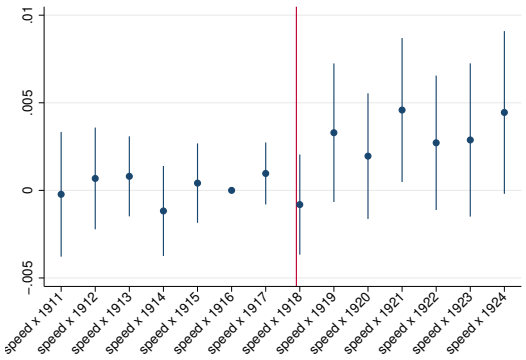
(c) Deaths/$Population_{1910}$: Number of Days



(d) Deaths/$Population_{1910}$: Speed of implementation



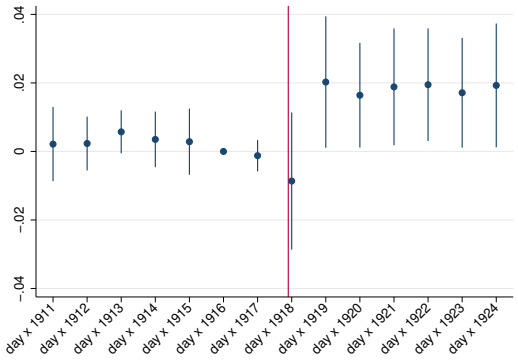
(e) log(deaths): Number of Days



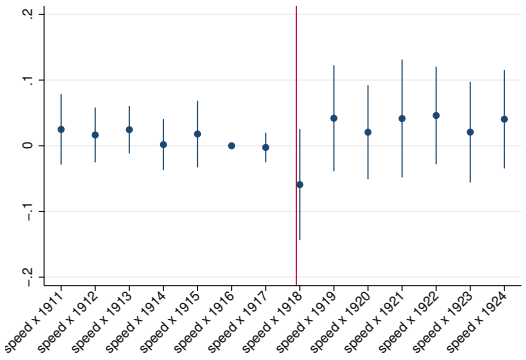
(f) log(death): Speed of implementation

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, years and cities' fixed effects and excess mortality in 1918 (Growth rate of mortality between 1917 and 1918, several alternate indicators were tried as excess mortality in 1918 as measured in Markel et al. (2007) or simply death rate in 1918, results remain unchanged)
95% confidence interval clustered at the city level

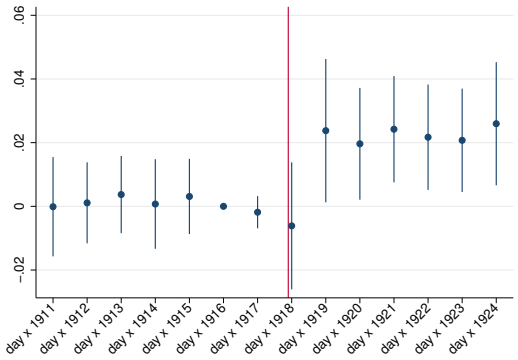
Figure B.4: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **using Clay et al (2019) control variables**



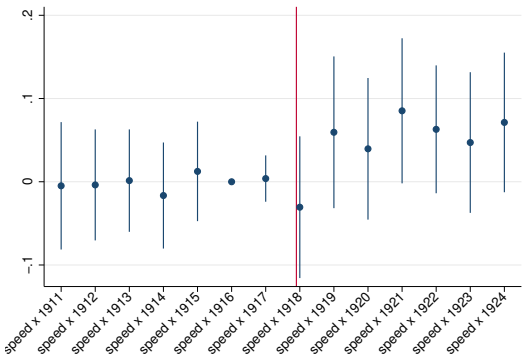
(a) Reported Death rate: Number of Days



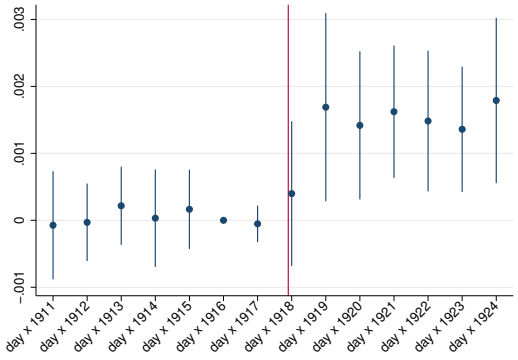
(b) Reported Death rate: Speed of implementation



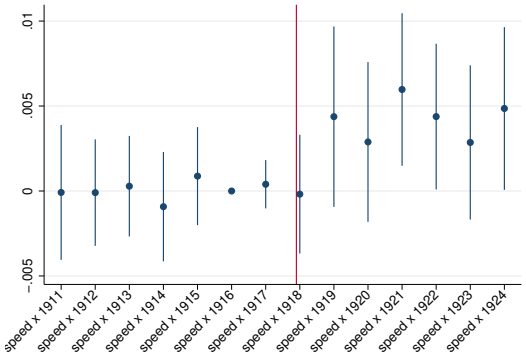
(c) Deaths/$Population_{1910}$: Number of Days



(d) Deaths/$Population_{1910}$: Speed of implementation



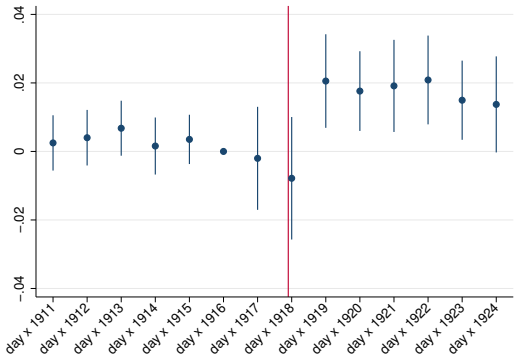
(e) log(deaths): Number of Days



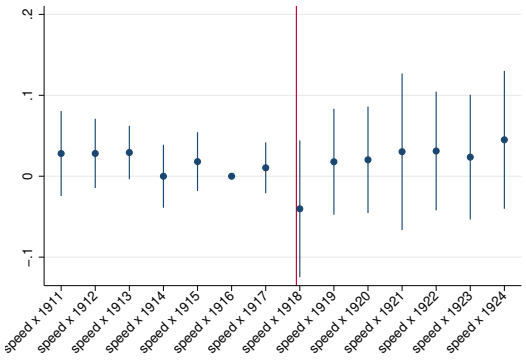
(f) log(death): Speed of implementation

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, years and cities' fixed effects,coal fired plant capacity within 30 miles, share of white, distance to the closest WWI military camp, late arrival of the pandemic
95% confidence interval clustered at the city level
Sample: 32 Cities in Clay, Lewis, and Severnini (2019) with Markel et al. (2007)

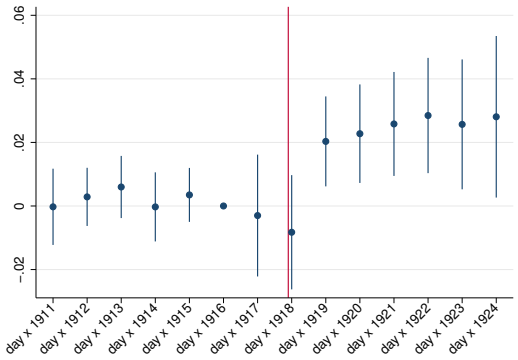
Figure B.5: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for estimated migration flows**



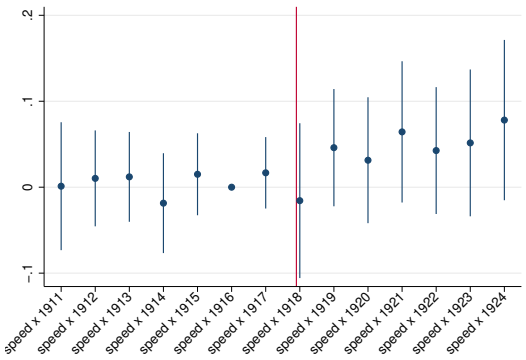
(a) Reported Death rate: Number of Days



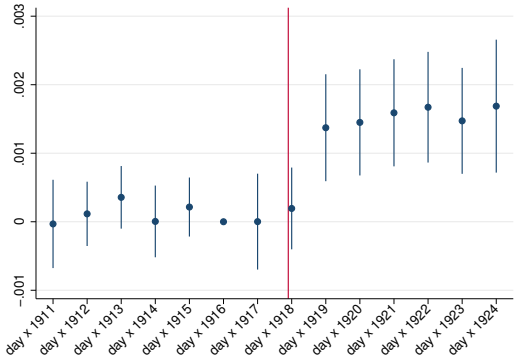
(b) Reported Death rate: Speed of implementation



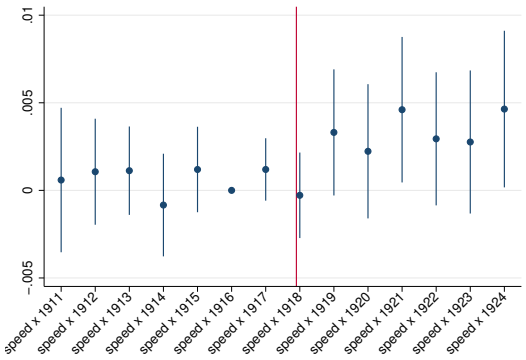
(c) Deaths/$Population_{1910}$: Number of Days



(d) Deaths/$Population_{1910}$: Speed of implementation



(e) log(deaths): Number of Days

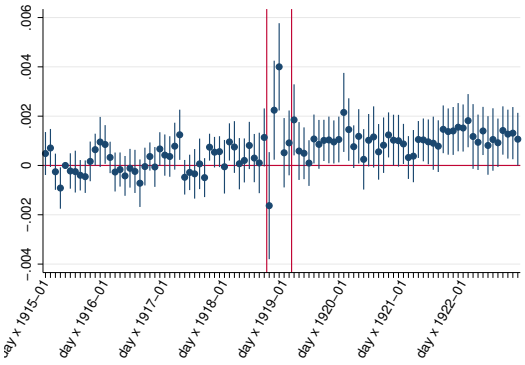


(f) log(death): Speed of implementation

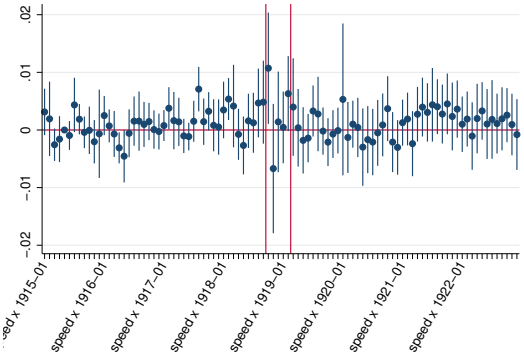
Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, years and cities' fixed effects and the log of the estimated migration flows
95% confidence interval clustered at the city level

B.2 Evidence from Monthly deaths

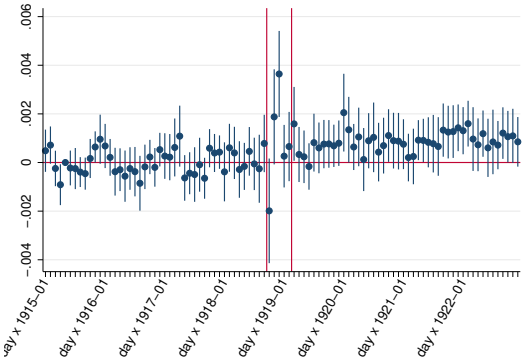
Figure B.6: Event study: Estimates of the aggregate impact of NPIs on monthly mortality (All causes of death) **Robustness checks**



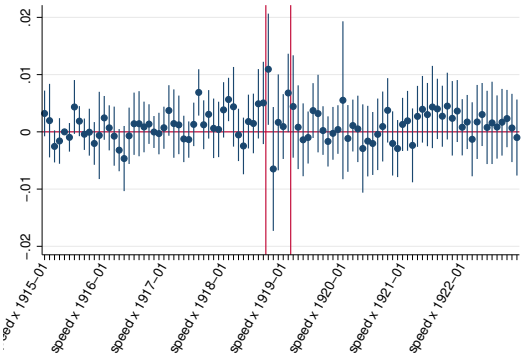
(a) log(deaths)
Number of Days
controlling for regional shocks



(b) log(deaths)
Speed of implementation
controlling for regional shocks



(c) log(deaths)
Number of Days
controlling for demographics



(d) log(deaths)
Speed of implementation
controlling for demographics

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, yearly estimated Population, years and cities' fixed effects and interaction terms between the cities' region (West, Midwest, South, North East) and years fixed effects.
95% confidence interval clustered at the city level

B.3 Controlling for differences in the demographic structures

B.4 Balance tests

Table B.1: Balance test, demographics in 1910 by length of NPIs

		Below the Median			Above the Median			Difference		
variable	year	Average	Standard Deviation	Obs	Average	Standard Deviation	Obs	Difference	Tstat	pvalue
POP	1910	310610	326523	22	578011	1.057e+06	21	-267402	-1.132	0.264
POPgrowth	1910	0.346	0.436	22	0.655	0.630	21	-0.309	-1.878	0.0675
ratio	1910	0.988	0.0923	22	1.073	0.138	21	-0.0844	-2.364	0.0229
average_age	1910	28.15	1.321	22	28.65	1.300	21	-0.505	-1.262	0.214
age_q1	1910	4.818	0.795	22	5.381	0.973	21	-0.563	-2.081	0.0437
age_q5	1910	26.05	1.463	22	26.81	1.601	21	-0.764	-1.635	0.110
age_q9	1910	53.82	1.967	22	53.19	1.778	21	0.628	1.096	0.280
share_a0001	1910	0.0208	0.00309	22	0.0184	0.00294	21	0.00240	2.607	0.0127
share_a0104	1910	0.0750	0.00850	22	0.0684	0.00924	21	0.00659	2.435	0.0193
share_a0514	1910	0.169	0.0170	22	0.154	0.0201	21	0.0146	2.575	0.0137
share_a1524	1910	0.200	0.0129	22	0.202	0.0108	21	-0.00230	-0.632	0.531
share_a2534	1910	0.192	0.0158	22	0.207	0.0198	21	-0.0154	-2.827	0.00722
share_a3544	1910	0.148	0.00971	22	0.154	0.0113	21	-0.00640	-1.999	0.0522
share_a4554	1910	0.101	0.00769	22	0.105	0.00807	21	-0.00389	-1.619	0.113
share_a5564	1910	0.0553	0.00708	22	0.0541	0.00717	21	0.00125	0.575	0.568
share_a6574	1910	0.0286	0.00536	22	0.0265	0.00490	21	0.00213	1.358	0.182
share_a7584	1910	0.00947	0.00206	22	0.00863	0.00177	21	0.000838	1.431	0.160
share_a8500	1910	0.00157	0.000372	22	0.00138	0.000312	21	0.000198	1.888	0.0661
share_c0001	1910	.	.	.	.	.	.	.	.	.
share_c0104	1910	.	.	.	.	.	.	.	.	.
share_c0514	1910	0.131	0.0153	22	0.119	0.0162	21	0.0124	2.585	0.0134
share_c1524	1910	0.168	0.0156	22	0.156	0.0197	21	0.0127	2.344	0.0240
share_c2534	1910	0.209	0.0143	22	0.217	0.0127	21	-0.00721	-1.743	0.0889
share_c3544	1910	0.183	0.0145	22	0.196	0.0181	21	-0.0129	-2.588	0.0133
share_c4554	1910	0.137	0.00942	22	0.143	0.0104	21	-0.00627	-2.078	0.0440
share_c5564	1910	0.0905	0.00773	22	0.0935	0.00792	21	-0.00298	-1.249	0.219
share_c6574	1910	0.0495	0.00700	22	0.0477	0.00707	21	0.00178	0.830	0.411
share_c7584	1910	0.0236	0.00460	22	0.0218	0.00422	21	0.00177	1.312	0.197
share_c8500	1910	0.00763	0.00173	22	0.00689	0.00143	21	0.000743	1.531	0.134
share_c99999	1910	.	.	.	.	.	.	.	.	.

Table B.2: Balance test, demographics in 1920 by length of NPIs

		Below the Median			Above the Median			Difference		
variable	year	Average	Standard Deviation	Obs	Average	Standard Deviation	Obs	Difference	Tstat	pvalue
POP	1920	369174	385078	22	711416	1.249e+06	21	-342242	-1.226	0.227
POPgrowth	1920	0.187	0.110	22	0.282	0.191	21	-0.0949	-2.003	0.0518
ratio	1920	0.968	0.0607	22	1.015	0.0519	21	-0.0465	-2.693	0.0102
average_age	1920	29.01	1.330	22	29.98	1.520	21	-0.964	-2.216	0.0323
age_q1	1920	4.955	0.899	22	5.476	0.928	21	-0.522	-1.872	0.0683
age_q5	1920	27.18	1.593	22	28.71	1.793	21	-1.532	-2.966	0.00501
age_q9	1920	55.41	1.894	22	55.81	2.089	21	-0.400	-0.659	0.513
share_a0001	1920	0.0198	0.00268	22	0.0167	0.00220	21	0.00307	4.086	0.000199
share_a0104	1920	0.0759	0.0102	22	0.0680	0.00961	21	0.00789	2.607	0.0127
share_a0514	1920	0.173	0.0183	22	0.160	0.0149	21	0.0134	2.624	0.0122
share_a1524	1920	0.176	0.0142	22	0.171	0.0115	21	0.00519	1.315	0.196
share_a2534	1920	0.185	0.0127	22	0.196	0.0105	21	-0.0112	-3.141	0.00312
share_a3544	1920	0.151	0.0114	22	0.161	0.0112	21	-0.0106	-3.083	0.00365
share_a4554	1920	0.112	0.00899	22	0.115	0.0115	21	-0.00349	-1.113	0.272
share_a5564	1920	0.0647	0.00843	22	0.0684	0.00930	21	-0.00374	-1.381	0.175
share_a6574	1920	0.0307	0.00476	22	0.0311	0.00548	21	-0.000414	-0.265	0.793
share_a7584	1920	0.0105	0.00202	22	0.0106	0.00214	21	-8.32e-05	-0.131	0.897
share_a8500	1920	0.00218	0.000378	22	0.00217	0.000447	21	9.49e-06	0.0753	0.940
share_c0001	1920	0.0193	0.00279	22	0.0171	0.00256	21	0.00221	2.706	0.00989
share_c0104	1920	0.0751	0.00934	22	0.0682	0.00865	21	0.00686	2.496	0.0167
share_c0514	1920	0.167	0.0171	22	0.154	0.0137	21	0.0127	2.676	0.0107
share_c1524	1920	0.186	0.0158	22	0.184	0.0113	21	0.00236	0.562	0.578
share_c2534	1920	0.180	0.0126	22	0.194	0.0105	21	-0.0137	-3.875	0.000377
share_c3544	1920	0.142	0.0105	22	0.150	0.0109	21	-0.00758	-2.324	0.0252
share_c4554	1920	0.100	0.00890	22	0.105	0.0116	21	-0.00417	-1.327	0.192
share_c5564	1920	0.0574	0.00757	22	0.0606	0.00864	21	-0.00321	-1.297	0.202
share_c6574	1920	0.0256	0.00424	22	0.0259	0.00491	21	-0.000293	-0.210	0.835
share_c7584	1920	0.00740	0.00145	22	0.00756	0.00157	21	-0.000153	-0.333	0.741
share_c8500	1920	0.00145	0.000337	22	0.00145	0.000339	21	7.99e-07	0.00774	0.994
share_c99999	1920	0.0386	0.00510	22	0.0336	0.00450	21	0.00503	3.421	0.00143

Table B.3: Balance test, demographics in 1910 by speed of NPIs

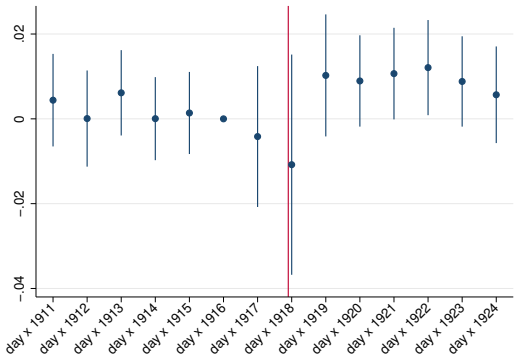
		Below the Median			Above the Median			Difference		
variable	year	Average	Standard Deviation	Obs	Average	Standard Deviation	Obs	Difference	Tstat	pvalue
POP	1910	326922	320429	22	560922	1.063e+06	21	-234001	-0.987	0.329
POPgrowth	1910	0.365	0.436	22	0.636	0.639	21	-0.271	-1.635	0.110
ratio	1910	0.994	0.0903	22	1.067	0.143	21	-0.0736	-2.028	0.0491
average_age	1910	28.10	1.259	22	28.70	1.342	21	-0.603	-1.519	0.136
age_q1	1910	4.864	0.834	22	5.333	0.966	21	-0.470	-1.709	0.0949
age_q5	1910	26.05	1.430	22	26.81	1.632	21	-0.764	-1.635	0.110
age_q9	1910	53.64	1.733	22	53.38	2.061	21	0.255	0.441	0.662
share_a0001	1910	0.0208	0.00311	22	0.0184	0.00294	21	0.00239	2.586	0.0134
share_a0104	1910	0.0750	0.00853	22	0.0684	0.00924	21	0.00653	2.408	0.0206
share_a0514	1910	0.170	0.0173	22	0.154	0.0194	21	0.0157	2.805	0.00767
share_a1524	1910	0.199	0.0122	22	0.203	0.0114	21	-0.00399	-1.108	0.274
share_a2534	1910	0.192	0.0160	22	0.206	0.0203	21	-0.0135	-2.433	0.0194
share_a3544	1910	0.149	0.0101	22	0.153	0.0114	21	-0.00454	-1.384	0.174
share_a4554	1910	0.101	0.00795	22	0.105	0.00774	21	-0.00411	-1.716	0.0937
share_a5564	1910	0.0548	0.00646	22	0.0547	0.00781	21	9.90e-05	0.0454	0.964
share_a6574	1910	0.0281	0.00485	22	0.0270	0.00559	21	0.00108	0.675	0.503
share_a7584	1910	0.00922	0.00188	22	0.00889	0.00204	21	0.000331	0.553	0.583
share_a8500	1910	0.00151	0.000348	22	0.00144	0.000365	21	7.47e-05	0.687	0.496
share_c0001	1910	.	.	.	.	.	.	.	.	.
share_c0104	1910	.	.	.	.	.	.	.	.	.
share_c0514	1910	0.131	0.0154	22	0.119	0.0161	21	0.0126	2.628	0.0120
share_c1524	1910	0.169	0.0160	22	0.155	0.0191	21	0.0133	2.489	0.0170
share_c2534	1910	0.209	0.0138	22	0.217	0.0128	21	-0.00876	-2.156	0.0370
share_c3544	1910	0.184	0.0147	22	0.195	0.0186	21	-0.0111	-2.167	0.0361
share_c4554	1910	0.138	0.00971	22	0.142	0.0106	21	-0.00424	-1.368	0.179
share_c5564	1910	0.0902	0.00790	22	0.0938	0.00760	21	-0.00358	-1.512	0.138
share_c6574	1910	0.0489	0.00629	22	0.0483	0.00784	21	0.000595	0.275	0.785
share_c7584	1910	0.0231	0.00417	22	0.0223	0.00480	21	0.000816	0.596	0.555
share_c8500	1910	0.00740	0.00158	22	0.00713	0.00169	21	0.000279	0.561	0.578

Table B.4: Balance test, demographics in 1920 by speed of NPIs

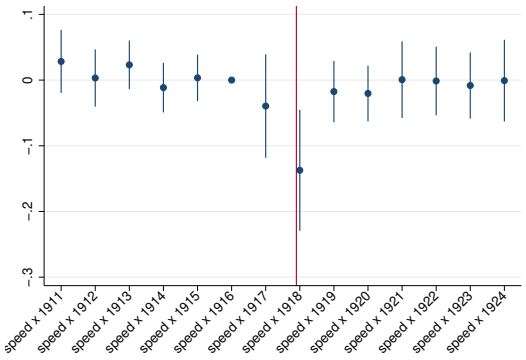
		Below the Median			Above the Median			Difference		
variable	year	Average	Standard Deviation	Obs	Average	Standard Deviation	Obs	Difference	Tstat	pvalue
POP	1920	388825	377175	22	690830	1.257e+06	21	-302005	-1.078	0.287
POPgrowth	1920	0.193	0.106	22	0.275	0.197	21	-0.0828	-1.729	0.0913
ratio	1920	0.978	0.0609	22	1.005	0.0588	21	-0.0262	-1.433	0.159
average_age	1920	29.04	1.402	22	29.95	1.469	21	-0.911	-2.080	0.0438
age_q1	1920	5	0.976	22	5.429	0.870	21	-0.429	-1.517	0.137
age_q5	1920	27.27	1.723	22	28.62	1.746	21	-1.346	-2.545	0.0148
age_q9	1920	55.27	1.882	22	55.95	2.061	21	-0.680	-1.130	0.265
share_a0001	1920	0.0196	0.00290	22	0.0169	0.00221	21	0.00264	3.340	0.00180
share_a0104	1920	0.0751	0.0107	22	0.0689	0.00974	21	0.00622	1.994	0.0528
share_a0514	1920	0.172	0.0192	22	0.160	0.0145	21	0.0119	2.296	0.0269
share_a1524	1920	0.177	0.0137	22	0.170	0.0116	21	0.00682	1.753	0.0871
share_a2534	1920	0.186	0.0127	22	0.195	0.0115	21	-0.00942	-2.549	0.0146
share_a3544	1920	0.151	0.0115	22	0.161	0.0115	21	-0.00988	-2.821	0.00734
share_a4554	1920	0.112	0.0106	22	0.115	0.0102	21	-0.00243	-0.769	0.446
share_a5564	1920	0.0644	0.00889	22	0.0687	0.00871	21	-0.00426	-1.587	0.120
share_a6574	1920	0.0303	0.00448	22	0.0316	0.00566	21	-0.00126	-0.814	0.421
share_a7584	1920	0.0104	0.00187	22	0.0107	0.00227	21	-0.000378	-0.597	0.554
share_a8500	1920	0.00218	0.000384	22	0.00216	0.000442	21	2.19e-05	0.174	0.863
share_c0001	1920	0.0191	0.00287	22	0.0173	0.00263	21	0.00182	2.161	0.0366
share_c0104	1920	0.0743	0.00987	22	0.0691	0.00866	21	0.00520	1.833	0.0741
share_c0514	1920	0.166	0.0177	22	0.155	0.0133	21	0.0119	2.491	0.0169
share_c1524	1920	0.187	0.0154	22	0.183	0.0115	21	0.00419	1.004	0.321
share_c2534	1920	0.181	0.0125	22	0.193	0.0115	21	-0.0122	-3.316	0.00192
share_c3544	1920	0.142	0.0104	22	0.150	0.0111	21	-0.00723	-2.202	0.0334
share_c4554	1920	0.101	0.0111	22	0.104	0.00964	21	-0.00282	-0.885	0.381
share_c5564	1920	0.0571	0.00772	22	0.0609	0.00834	21	-0.00388	-1.585	0.121
share_c6574	1920	0.0252	0.00390	22	0.0263	0.00514	21	-0.00106	-0.763	0.450
share_c7584	1920	0.00735	0.00139	22	0.00761	0.00162	21	-0.000264	-0.575	0.569
share_c8500	1920	0.00146	0.000351	22	0.00143	0.000324	21	2.34e-05	0.227	0.821
share_c99999	1920	0.0383	0.00546	22	0.0340	0.00445	21	0.00430	2.820	0.00736

B.5 Event Studies controlling for the demographic structure

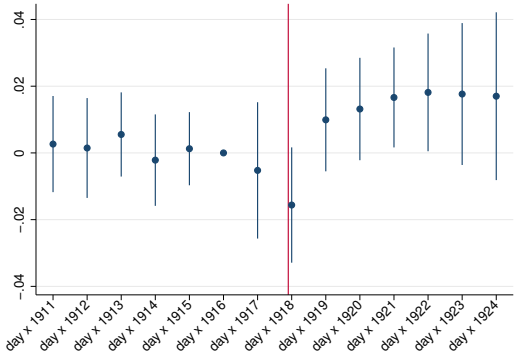
Figure B.7: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for demographic characteristics in 1910**



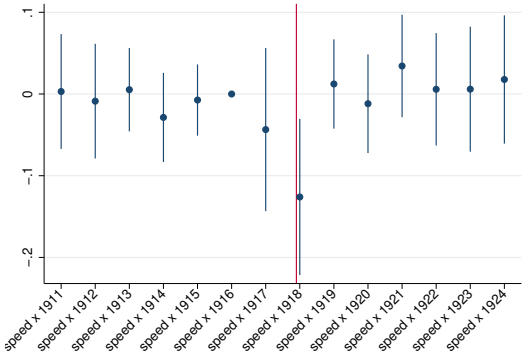
(a) Reported Death rate: Number of Days



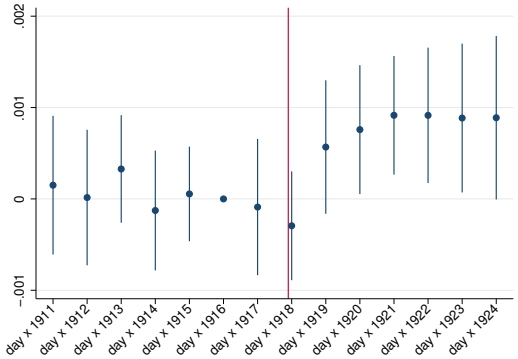
(b) Reported Death rate: Speed of implementation



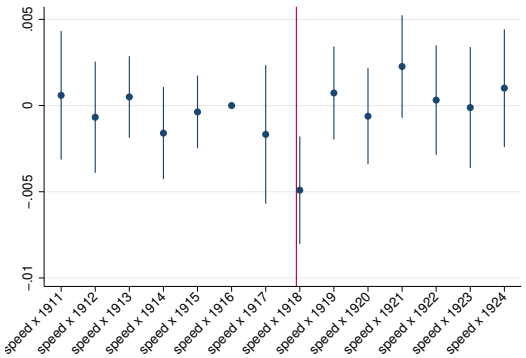
(c) Deaths/$Population_{1910}$: Number of Days



(d) Deaths/$Population_{1910}$: Speed of implementation



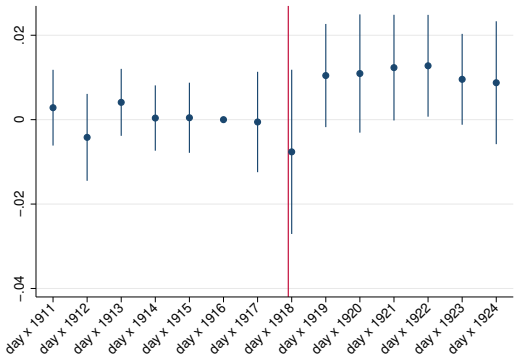
(e) log(deaths): Number of Days



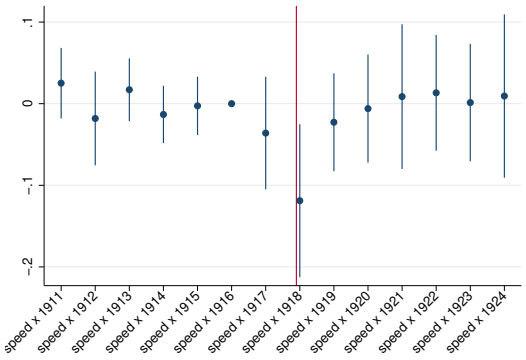
(f) log(death): Speed of implementation

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, yearly estimated Population, years and cities' fixed effects and years fixed effects.
Demographic controls include median age, the first and ninth age decile and the sex ratio.
95% confidence interval clustered at the city level

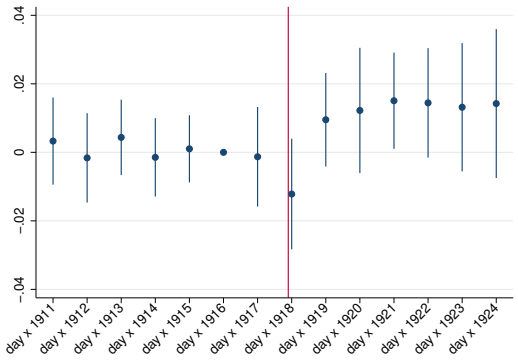
Figure B.8: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for demographic characteristics in 1920**



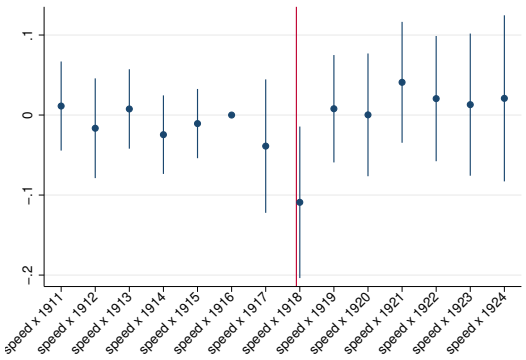
(a) Reported Death rate: Number of Days



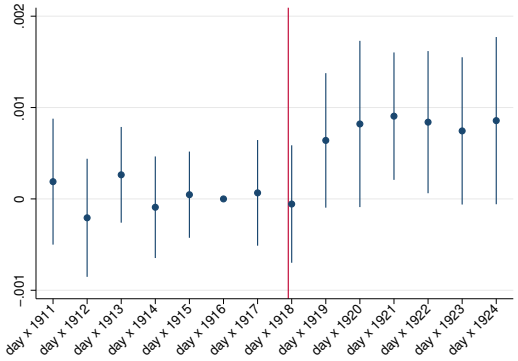
(b) Reported Death rate: Speed of implementation



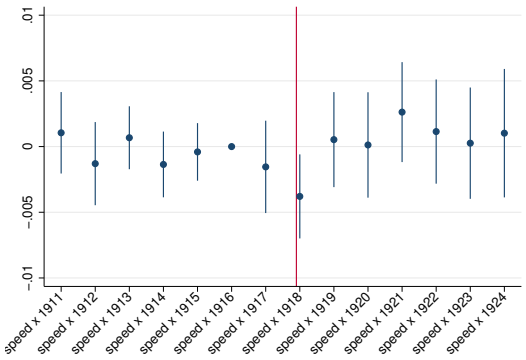
(c) Deaths/$Population_{1910}$: Number of Days



(d) Deaths/$Population_{1910}$: Speed of implementation



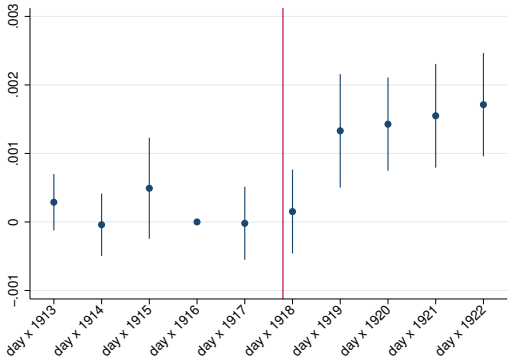
(e) log(deaths): Number of Days



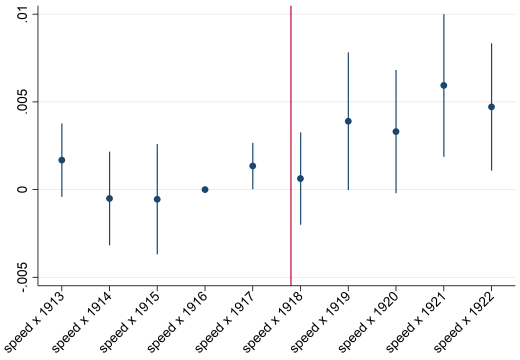
(f) log(death): Speed of implementation

Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, yearly estimated Population, years and cities' fixed effects and years fixed effects.
Demographic controls include median age, the first and ninth age decile and the sex ratio in 1920
95% confidence interval clustered at the city level

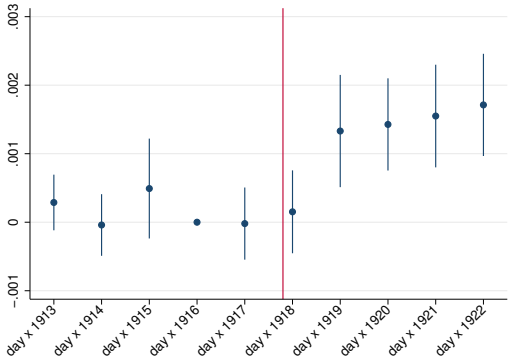
Figure B.9: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **by age groups**



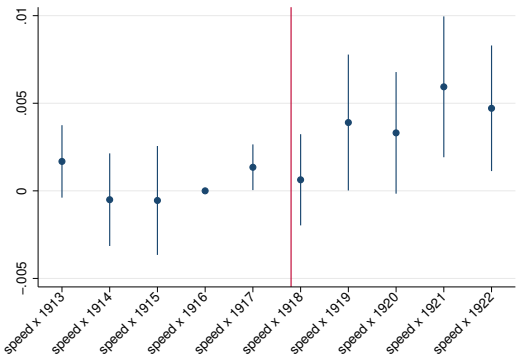
(a) log(deaths):
Number of Days
Controlling for age groups fixed effects and shocks



(b) log(death):
Speed of implementation
Controlling for age groups shocks



(c) log(deaths):
Number of Days
Controlling for city x age groups fixed effects



(d) log(death):
Speed of implementation
Controlling for city x age groups fixed effects

Estimates of the difference in difference equation:
$Deathrate_{i,g,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \sum_{g \neq <5} \eta^t \times 1_{group(i)=g} + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910, yearly estimated Population, years fixed effects
Panels a) and c) include age groups x time fixed effects and cities fixed effects
Panels b) and d) include age groups x cities fixed effects
Age groups are bins of five years from 0 to 94 years old.
95% confidence interval clustered at the city level

B.6 Results with mortality from influenza and pneumonia

Table B.5: Medium Run Impact of NPIs on Mortality (Deaths from influenza and pneumonia) (1911-1924)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Reported Rate				Deaths/$Population_{1910}$				ln(Deaths)			
Panel a) Impact of NPI Days on Mortality												
Days NPI x Post	.150	0.106	0.209	0.0590	0.259	0.158	0.162	0.105	0.0028***	0.0022***	0.00100	0.0012**
	(0.1214)	(0.1286)	(0.1303)	(0.1234)	(0.1606)	(0.1731)	(0.2143)	(0.1459)	(0.0006)	(0.0006)	(0.0006)	(0.0005)
R^2	0.898	0.904	0.944	0.923	0.907	0.911	0.937	0.931	0.976	0.978	0.984	0.983
N	597	597	597	597	597	597	597	597	597	597	597	597
Panel b) Impact of NPI Speed on Mortality												
Speed NPI x Post	0.693	0.769	0.844	0.178	1.3833**	0.959	0.671	0.333	0.00410	0.0062*	0.00220	0.00230
	(0.5425)	(0.5786)	(0.5724)	(0.4847)	(0.6814)	(0.7637)	(0.7473)	(0.6310)	(0.0040)	(0.0032)	(0.0020)	(0.0028)
R^2	0.898	0.904	0.944	0.923	0.907	0.911	0.937	0.930	0.972	0.977	0.984	0.983
N	597	597	597	597	597	597	597	597	597	597	597	597
Controls												
Time FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
¨Pop1900	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
temperature	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
Est. pop	Y	Y	Y	Y	N	N	N	N	N	N	N	N
Health exp.	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y
Region shocks	N	N	Y	N	N	N	Y	N	N	N	Y	N
Demographics	N	N	N	Y	N	N	N	Y	N	N	N	Y

Post is a dummy indicating observations after 1917 while speed NPI indicates the speed at which the city implemented their NPI. Days NPI describes the length the NPI measures were in place.

Estimates of the difference in difference equations:

$Deathrate_{i,t} = \delta_i + \gamma_t + \beta \times Post \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$

Controls include health expenditures per capita in 1917, population in 1900. Est. Population corresponds to the estimated population of the city for each yea. Non varying variables are interacted with year fixed effects. I also includes years and city fixed effects. Temperature include the monthly temperature in the state. Temperature include minimum and maximum monthly temperature of the year in the State. Demographics control for population growth in the decade and interact share of whites, median age and sex ratio in 1910 with years fixed effects. Regional shocks interact regional dummies (Midwest, West, North East, South) with years fixed effects.

standard errors clustered at the city level.

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