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

Guillaume Chapelle (CYU Cergy Paris Université)

CEPR CESifo SciencesPo.

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 relatively intensively, might have reduced individual and herd immunity reducing the life expectancy of people with co morbidity, thereby leading to a significantly higher number of deaths in subsequent years. It would be difficult to draw any inference regarding the predicted impact of NPIs as implemented during the Covid-19 crisis as influenza and Covid-19 are two entirely different viruses and nowadays' pharmaceutical technologies can limit these medium term impacts.

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

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†Assistant Professor of Economics, CY Cergy Paris Université, THEMA, CNRS, F-95000 Cergy, France and Affiliate researcher to Sciences Po, LIEPP, Paris, email: gc.chapelle@gmail.com; guillaume.chapelle@cyu.fr; guillaume.chapelle@sciencespo.fr. There are no conflicts of interest to declare

1 Introduction

Since the outbreak of the global Covid-19 pandemic, a growing stream of contributions has sought to help policymakers improve their understanding of the crisis by analyzing past pandemics. In this context, the 1918 influenza 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 more or less intense 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 mortality trends before 1918 but ended with relatively higher mortality levels in the subsequent years- in particular, when the intervention was intense. 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 intensities or speeds 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) was quickly followed by a rebound in mortality during the subsequent months and years.

This second result could be explained by the fact that NPIs might have reduced individual and herd immunity[1] in a period where any people were afflicted with co morbidity factors from influenza. 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 later in their lifetime. Consequently, reducing the spread of the disease might cause a city's population to become more vulnerable in the medium-run in particular when they were afflicted by other diseases. This ultimately contributed to an increase in the overall mortality rate. Moreover, it is possible that herd immunity could also allow a decrease in the spread of the next influenza as argued by Fox et al. (1971) and Fine (1993). Finally, medical literature such as Douglas et al. (2020) and Markel, Stern, and

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)

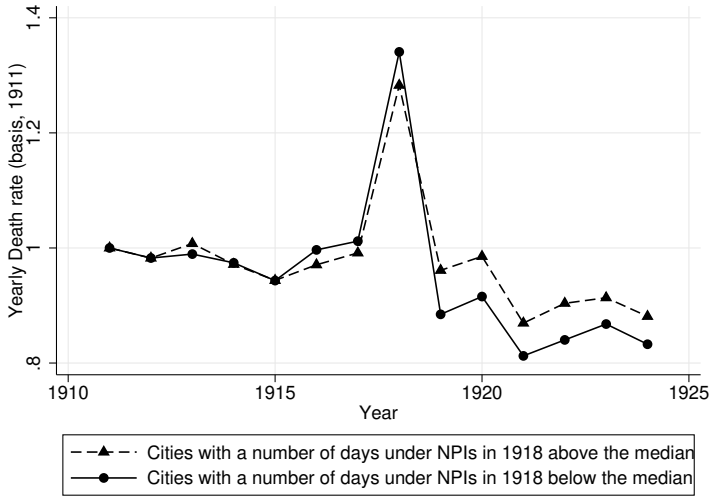
Cetron (2008) fear that NPIs might have negative consequences on the general health status of the population while a larger diffusion of influenza in the following years might be associated with adverse health outcomes for children suffering from in utero exposition (Almond 2006; Lin and Liu 2014).

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 in 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 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 note that this study is specific to the 1918 influenza and has 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), 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 those used during the first wave of the Covid-19 as no lock down of the population was implemented in 1918. 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 in 1918 than 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 main results and the robustness checks. Section 6 discusses these results while section 7 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 for different intensities



Reading notes: Cities that implemented more intense NPIs witnessed their death rates increase less than cities that had less intense 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.

Before 1918 the average death index in cities above the median is 0.97 while it is 0.98 for cities below. The difference is not statistically significant

After 1918, the average index in cities above the median .91 while it is 0.85 for cities below the median. The difference is -.056 (95% CI -.067 -0.466)

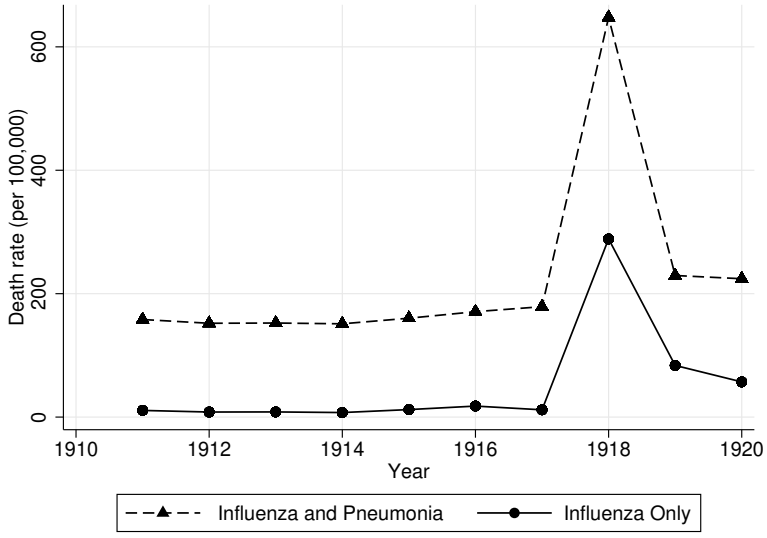
2 Background and literature review

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 and in the Midwest in the end of September 1918 and in the West in the beginning 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. 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 applied econometric and epidemiological literature. I complement the econometric and statistical literature that documents the main drivers of mortality in U.S. cities in the early XXth century and during the 1918 influenza. For example, Anderson, Charles, and Rees (2020) study the impact of public health efforts as water filtration on mortality in 25 US cities. Acuna-Soto, Viboud, and Chowell (2011) show that smaller cities experienced the worst outcome during the 1918 pandemic and that mortality during this 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, part of the lives saved in the first wave as put forward in Correia, Luck, and Verner (2020) and Markel et al. (2007)- were lost in the following months. 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. NPIs appeared to turn cities more vulnerable in the medium run.

My findings also support the concerns raised in Markel, Stern, and Cetron (2008) or Adda (2016) 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 phenomenon is also put forward to explain age profile of mortality in 1918 (Mamelund 2011) : people aged above 60-65 had lower than expected mortality which could be a consequence of the protection emanating from exposure to infections contracted before 1889. This might also be considered as a support to the literature on herd immunity (Fine 1993; Fine, Eames, and Heymann 2011).

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 which were stronger in magnitude and intensity and tend to be in line with the estimated impact of the 1918 policies in the short run. Lin and Meissner (2020b) and Allcott et al. (2020) found that NPIs implemented locally had a significant and sizeable effect on disease transmission in the short run. More specifically Dave et al. (2020) found that Stay In Place Order (SIPO) reduced the number of deaths and infections from Covid-19 by up to 50% .

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), Dave et al. (2020), and Eyméoud et al. (2021).

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 and find that longer and more intense NPIs are associated, if anything, with better economic outcomes in the medium run. In the same vein, Berkes et al. (2020) find that longer NPIs in 1918 were associated with more patents in the following years. 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. It also contribute to the literature assessing the impact of NPIs and their net benefits as Adda (2016) who investigate the net health and economic benefits of school and transport closures outside the context of a global pandemic.

3 Data

3.1 Measuring NPIs

To conduct this study, 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). The authors provide two measures of NPIs. The first one describes the cumulated number of days where the three main NPI categories (school closures, isolation and quarantines and public gathering bans) were implemented. This measure reflects both the intensity and the duration of NPIs as a calendar day where 2 different NPIs were applied will count twice. The second measure is 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 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.

Markel et al. (2007) online appendix describes briefly what kind of NPIs were implemented in the different cities. The most widely used NPIs were 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 mail 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. Mean-

2. The day the mortality rate exceeds twice its base

while, in Albany, streetcars were routinely cleaned and ventilated. Most of the NPIs were implemented during the second wave in October 1918 while fewer cities implemented these NPIs during the third wave.

3.2 Measurements of mortality

The most important issue in this paper is to measure mortality at the city level. To do so, I rely on the mortality tables for large cities published by the Census Bureau from 1905 to 1924. These reports are published yearly[3] and provide the total number of deaths, estimated death rates and the classification of the deaths for the 43 cities under scrutiny. These cities are part of the registration area for which the administration meets the required standard to enter in the mortality statistics. The most important variable gathered from these reports is the yearly death counts for deaths from all causes except stillbirths[4] (e.g Table 1 page 41 in the 1911 report). I also gather the monthly death count and the yearly death count by age group. There is one missing data point for the yearly death count: Nashville, Tennessee in 1916, as no statistics for Tennessee are available for this year.

There are two main advantages to using the total death count as a main measure of mortality when compared with previous papers investigating the impact of NPIs in 1918.

First, it is important to assess the impact of NPIs through the lens of mortality from all causes as the classification of deaths is not always reliable. For example a death certificate may contain several causes and deaths from influenza are often associated with comorbidity factors (Center for Disease Control and Prevention 2021; Martınez et al. 2019). Moreover, their accuracy might vary across regions as illustrated in yearly reports (e.g page 68 in the 1920 report). More specifically, several epidemiological papers report that the classification of deaths during the pandemic was not accurate and that excess mortality should be preferably measured from mortality from all causes (Chandra et al. 2018). All previous papers (Markel et al. 2007; Barro 2020; Correia, Luck, and Verner 2020) investigating the impact of NPIs on mortality focused on mortality from influenza and pneumonia.

Second, most previous papers investigated the impact of NPIs on gross death rate. The gross death rate is the ratio between the number of deaths in city i at time t ($Deaths_{i,t}$) and an estimated population number ($\widehat{Population}_{i,t}$).

$$GrossRate_{i,t} = \frac{Deaths_{i,t}}{\widehat{Population}_{i,t}}$$

As discussed in Correia, Luck, and Verner (2020) and Lilley, Lilley, and Rinaldi (2020), population censuses are conducted every 10 years (1890, 1900, 1910, 1920,

3. https://www.cdc.gov/nchs/products/vsus/vsus_1890_1938.html
4. still births are not reported in these reports

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 prevalent in the years 1918 and 1919. Using the original death counts instead of the reported death rates thus allows to avoid relying on this extrapolation. In most specifications, I use the ratio between the number of deaths from all causes and the population in 1910 or the log of the number of deaths from all causes. I then control for the estimated population or for the decennial city growth to account for redistricting and city growth[5].

Additionally, I gather data on gross rates from all causes and from specific causes of deaths (cancers, heat diseases, Nephritis, Tuberculosis, suicides, homicides and accidents) to perform robustness checks. I use the reports of 1920, 1922 and 1924 that provide retrospective series of death rates for large cities by causes. The reports are consistent for overlapping years. 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 when looking at the gross death rate from all causes: 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. Moreover, in some cities, the death rates from specific causes are sometimes also missing for a few years, and death rates from accidents are only provided from 1916."

Finally, when describing the short term dynamics of mortality with weekly data, I rely on the excess rate from influenza and pneumonia from Collins et al. (1930). These excess rates were computed by substracting the weekly death rate with an estimated median death rate for the corresponding week computed from the past years. Unfortunately, these rates were thus affected by the problems explained above as they rely on a classification of deaths and an estimated population not only for the years 1918 and 1917 but also for all previous years when computing the median. I was unable to access a weekly count of deaths neither for influenza or pneumonia nor for all causes. Markel et al. (2007) compute their own excess death rate; they use the weekly death rate from influenza and pneumonia reported in the weekly health index and substract the median death rate reported in Collins et al. (1930) for the corresponding month in order to account for time invariant city level unobserved heterogeneity in mortality.

Table 1: Descriptive Statistics for the 43 US Cities

	Mean	Std.Dev.	Obs	min	max
Demographics					
Population 1900	329555.33	580082.35	43	36863.00	3460139.00
Population in 1910	441201.02	776807.64	43	100292.00	4770082.00
Population growth (1910 to 1920)	0.50	0.56	43	0.06	2.20
Average age in 1910	28.39	1.32	43	25.44	31.19
Men/Women (1910)	1.03	0.12	43	0.90	1.44
NPIs					
NPI days (1918)	88.28	46.43	43	28.00	170.00
NPI speed (1918)	-7.35	7.84	43	-35.00	11.00
Mortality					
From pneumonia and influenza (per 100,000) in 1917	179.10	61.53	43	58.90	380.40
From all causes (per 1,000) in 1917	15.32	2.74	43	8.60	22.90
From pneumonia and influenza (per 100,000) in 1918	647.14	187.53	43	282.70	1243.60
From all causes (per 1,000) in 1918	20.07	3.82	43	13.70	27.00

Author's computation from the Bureau of the Census, Mortality Statistics. 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.

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. I also utilize the financial statistics reports to gather data on local authorities expenditures[6] particularly health expenditures as well as the monthly temperature at the state level[7]. 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.

5. 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.

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

7. 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). To study the impact of NPIs in the medium-term, I follow a different approach. 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, particularly the pre-trend assumption, I first utilize an event study following a growing econometric literature (Duflo 2001; Autor 2003; Adda 2016; 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), Allcott et al. (2020), and Dave 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 yearly 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 reported gross death rates. 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 and intensity of NPIs as it measures the cumulated number of days public gathering bans, school closures and quarantines were implemented. With the second variable, a calendar year where two distinct NPIs were implemented would count twice. β_t is used to understand whether cities that responded more aggressively to the pandemic had different trends in mortality from 1911 to 1924.

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

$$\begin{aligned} Mortality_{i,t} =& \delta_i + \gamma_t + \beta \times Post \times NPI_{1918,i} \\ &+ Z_{i,t}\theta + \lambda \times Post \times X_i + \epsilon_{i,t} \end{aligned} \tag{2}$$

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 that, contrary to Adda (2016), the measure of NPIs might be endogenous. In particular, the estimation of the impact of NPIs intensity on mortality can be subject to two main biases: the reverse causality bias and the unobserved variable bias. In this section, I discuss the sources of endogeneity and the strategies developed to tackle these issues thanks to a series of robustness checks performed throughout the study.

The reverse causality bias One might fear that places that responded more aggressively to the 1918 pandemic were experimenting with more lethal flu pandemics. In other words, cities that were more sensitive to the flu might implement stronger NPIs (Barro 2020). This bias might be extremely important in the short run as the main independent variable used in the literature is the intensity of NPIs. The resultant positive correlation between NPIs and mortality could lead one to underestimate the capacity of NPIs to mitigate mortality during the pandemic, i.e in the short run. The concerns about reverse causality are less stringent in the medium run once the pandemic and NPIs are over. By using an event study to carefully investigate the timing of the treatment and the pre-trend, we can attempt to relieve our concerns about this bias checking that cities that implemented more aggressive NPIs did not appear more affected by previous seasonal flus and had similar mortality dynamics before 1918.

The unobserved variable bias Mortality and NPIs might be correlated with other characteristics of cities. This can affect the medium term impact of NPIs if these characteristics have a direct impact on the yearly evolution of mortality. As such, it might be important to control for the influence of the potential confounding factors. Overall there are three different categories of potential unobserved factors that might affect the results that can be dealt with by exploiting different strategies and robustness checks.

Firstly, some relatively fixed characteristics of cities might also influence the evolution of mortality during and after the pandemic. Indeed, the literature pointed at several characteristics likely to influence the mortality dynamics in 1918, such as health expenditures, density, air quality, proximity to military camps, or racial composition. This might create a bias if these characteristics influence both policymakers' responses to a pandemic and the sensitivity of a city to a particular disease. For example, the demographic structure might be important as the 1918 influenza was particularly deadly for young people. One might think that cities with a larger share of young people might adopt stronger policy response but also suffer more from the pandemic. This bias remains related to the reverse causality bias and might be controlled thanks to the inclusion of the city's fixed effect and the investigation of pre-trends. This should reassure on the comparability of cities with

varying NPI intensity. Nevertheless, this might not always be the case if the influence of these factors suddenly changed from 1918 for other reasons. 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. I check for this eventuality by explicitly controlling for the influence of these variables on the yearly mortality dynamics interacting these variables with years fixed effects. In terms of geography, I thus also investigate the impact of NPIs within regions controlling for regional shocks. I also perform these kinds of robustness checks for the city's demographic characteristics as the share of black people, the median age and other variables mentioned in the literature (distance to military camps, density, air quality and health expenditures).

Secondly, some cities' characteristics that influenced their mortality dynamics might also change along with NPIs or for external reasons from 1918. For example, as the end of the pandemic coincided with the end of World War I, one might be concerned that cities with high levels of NPIs might be affected by different migration flows or demographic changes from 1918. There might also exist a substitution between NPIs and health expenditures as some cities might have chosen to improve their health systems instead of relying too heavily on NPIs, thus having long-term consequences on mortality once the pandemic ended. To control for these dimensions, I rely on three methods following the data availability. First, when yearly data are available, as for estimated migrations flows or temperature, I explicitly control for these variables. Second, when considering variables from the census which are only available every 10 years or on health expenditure, I also perform placebo tests in order to check whether NPIs are not significantly correlated with a change in these variables after 1918. These placebo tests can also be informative on the potential mechanisms likely to drive the results. Finally, I also follow Adda (2016) and control for the influence of the city's demographic structure relying on more disaggregated data and investigating the change in mortality within age group.

Thirdly, one might also be concerned by measurement errors in the dependent variables that could be correlated with the NPI variable. For example if some cities with a particular NPI intensity changed their method of counting deaths between two years without signaling this change in the original source. I check for this eventuality using two methods. First, I investigate whether there exists any trend break between 1918 and 1919 with monthly data from these reports for high and low NPIs cities. Second, I also perform placebo tests on outcomes less likely to be affected by NPIs as accidents, homicides, or suicides.

5 The medium term impact of NPIs

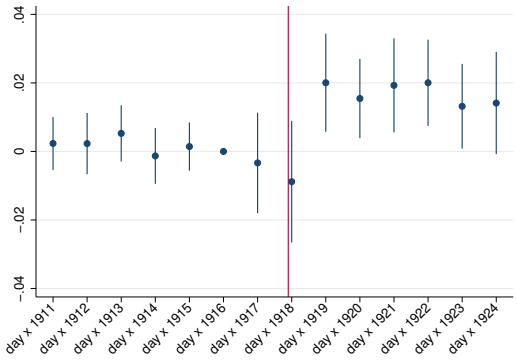
5.1 Results of the event study

Figures 3 and 4 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

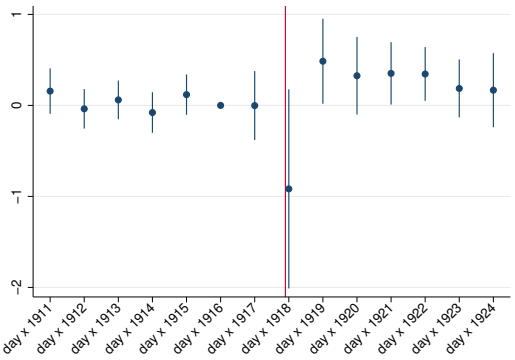
NPIs had similar mortality trends for all three mortality measures.These policies appear to be associated with a lower level of mortality in 1918. Moreover, provided that the pandemic started in Autumn, an attenuation bias could also explain these results. Indeed one can observe from the monthly series in Figure 5 that cities with more aggressive NPIs performed better during the first wave of the pandemic. These results are consistent with those of Clay, Lewis, and Severnini (2018) and Markel et al. (2007) and more recently with Barro (2020) and Correia, Luck, and Verner (2020); the latter suggests that NPIs enabled a flattening of the epidemic curve, thereby reducing the peak mortality even if results are not always statistically significant. This dimension will be discussed further when investigating the short-run impact of NPIs in the next section.

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. One key mechanism was is described in Gostic et al. (2016) who reveal that the first influenza virus that emerged in a region is key to understanding an individual'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 from the initial strain of influenza and thus increased the population's susceptibility to the subsequent strains. The fact that the results are clearer for NPI duration and intensity likely indicates 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. This phenomenon might be complemented by other mechanisms as discussed in Section 6.

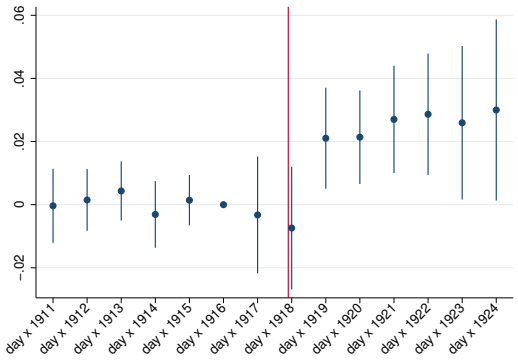
Figure 3: Event study: Estimates of the aggregate impact of NPI implementation duration and intensity on mortality



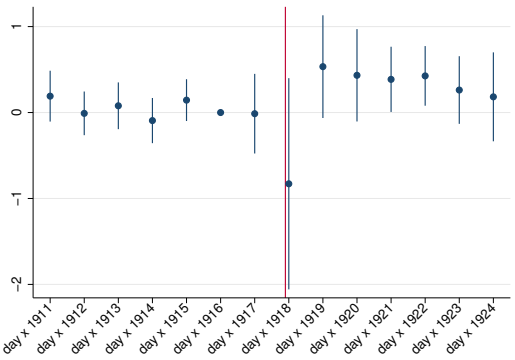
(a) Reported Death rate:
All causes of death



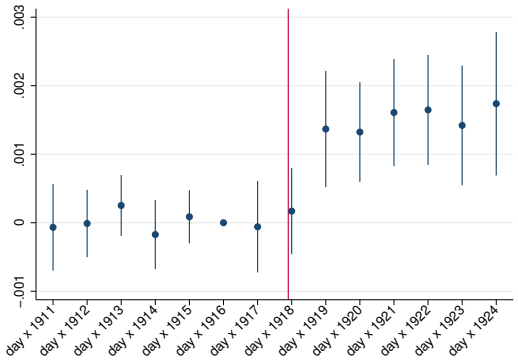
(b) Reported Death rate:
Influenza and Pneumonia



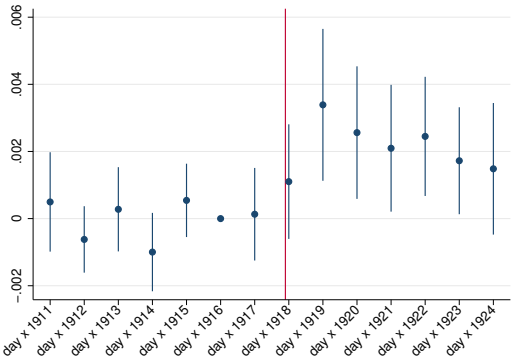
(c) Deaths/$Population_{1910}$:
All causes of death



(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 and with a higher intensity 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:

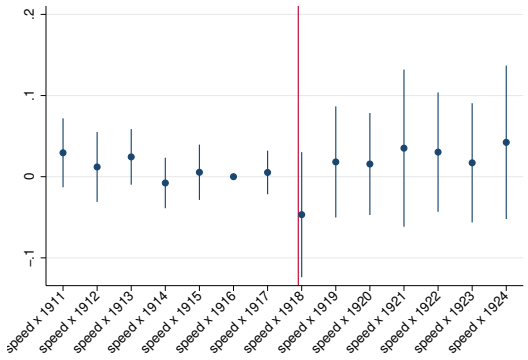
$Mortality_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$

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

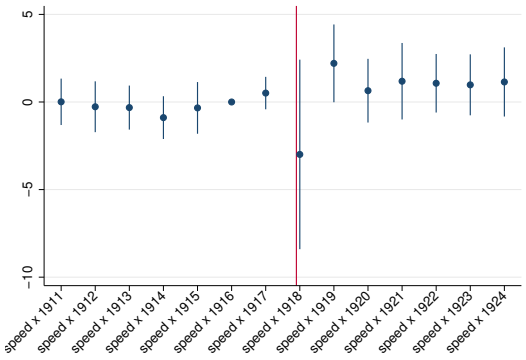
95% confidence interval clustered at the city level

NPI days is the cumulated number of days under NPIs

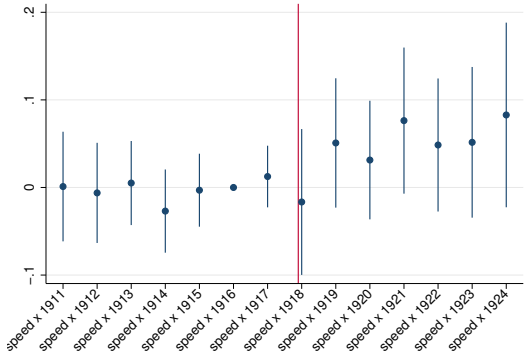
Figure 4: Event study: Estimates of the aggregate impact of NPI implementation speed on mortality



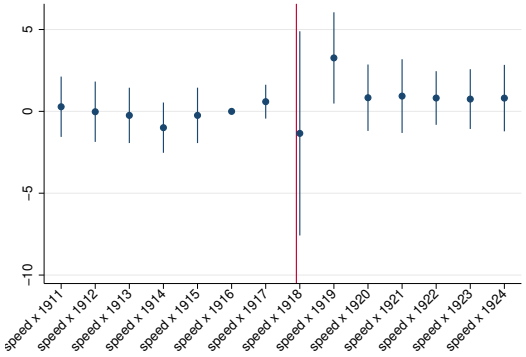
(a) Reported Death rate:
All causes of death



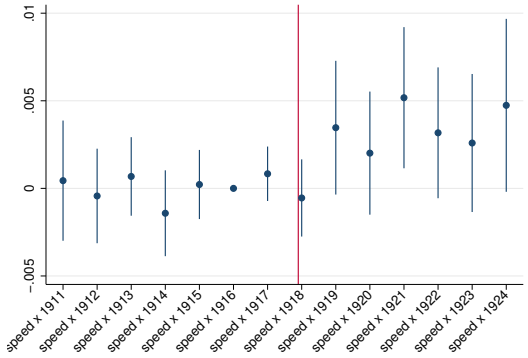
(b) Reported Death rate:
Influenza and Pneumonia



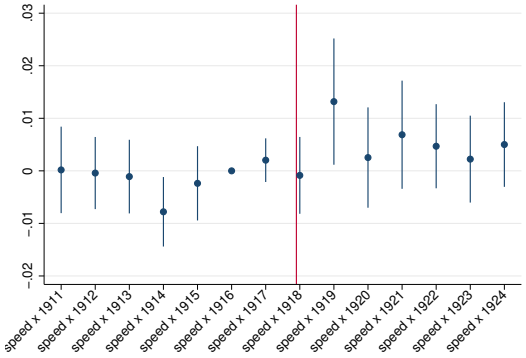
(c) Deaths/$Population_{1910}$:
All causes of death



(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:

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

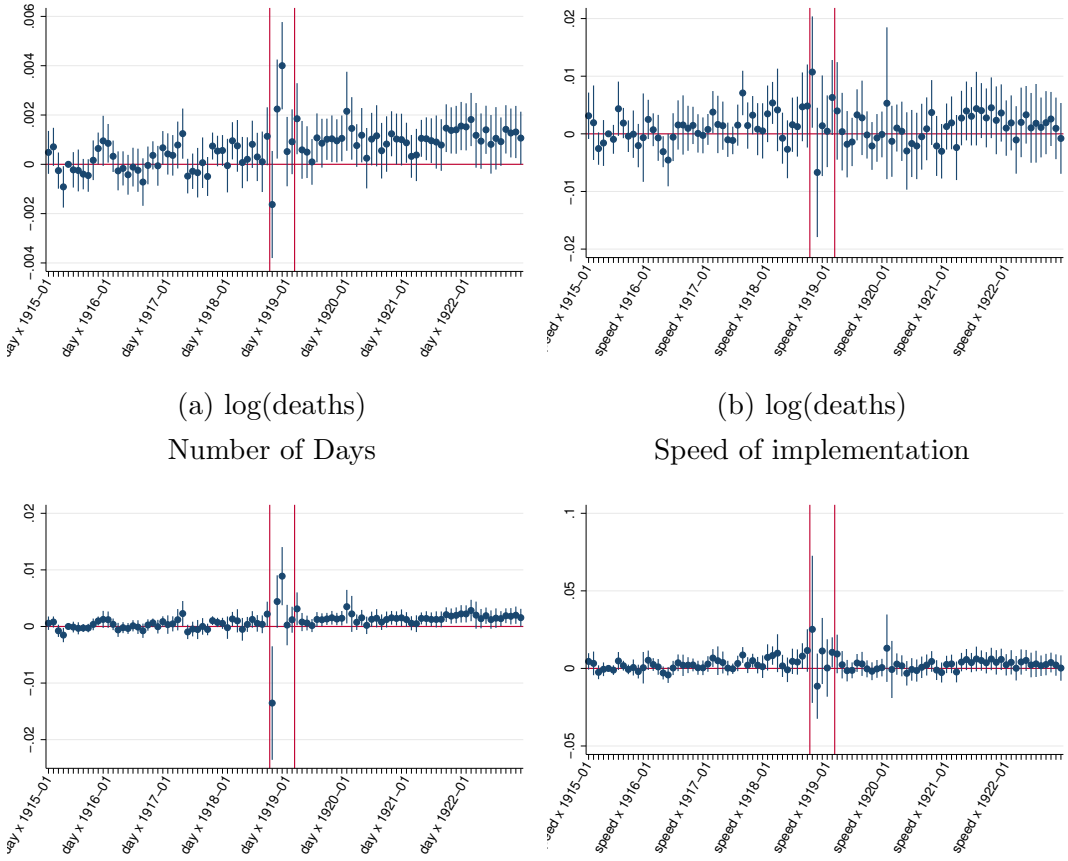
Controls include health expenditures in 1917, population in 1910, years and city fixed effects

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

Evidence from monthly data. I collect data on the monthly number of deaths at the city level from 1915 to 1922. 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 5. 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, 1919). 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 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 the winter by the third wave. These patterns are consistent with the results in Hatchett, Mecher, and Lipsitch (2007), who find that the 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 5: 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

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

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

Estimates of the difference in difference equation:
$Mortality_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 191502} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
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 cumulated number of days under NPIs
NPI speed is the timing of implementation of the First NPI w.r.t the acceleration date of the pandemic in the city

These patterns are robust to a wide range of robustness checks reported in Appendix:

- **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 A.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.

- **Controlling for excess mortality in 1918** Spinney (2017) emphasizes the fact that the 1918 influenza was followed by a rise in life expectancy as those who died from the influenza were the most fragile. 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 A.3 in the Appendix.

- **Controlling for air pollution and distance from camps** 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 of 32 cities that have data on NPIs. The patterns and results remain unchanged as illustrated in Figure A.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 allowing the West more time to prepare and 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 by 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 A.2 in the Appendix. Similar robustness checks are performed for the monthly series in Figure A.7.

- **Suicides.** Markel, Stern, and Cetron (2008), Giuntella et al. (2020), and Altindag, Erten, and Keskin (2020) suggest that NPIs might be associated with large economic and psychological costs. A lower economic performance might be associated with social and psychological problems leading to higher number of suicides. Robustness checks in Figure A.6 do not find any statistical association between the implementation of NPIS and suicide. The results on total mortality net of suicide remain unchanged.

- **Migration.** The flows of migrants after World War I might have contributed to the increase in mortality in some cities. To my knowledge, there are 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 that arrived

from 1911 to 1919 and from 1920 to 1924 respectively. This measure might allow to proxy the main differences in migration flows between cities. The inclusion of this estimated number of migrants does not affect the patterns as reported in Figure A.5 in the Appendix.

- **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 A.1 and A.2 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 had a lower average age, 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 A.8 and A.9. Similar robustness checks are performed for the monthly series in Figure A.7. In order to control for the implications of the demographic structure, I also collected detailed mortality tables by age groups[8] 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 A.10 presents the estimations of the 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 A.10 remain unchanged. Exploiting variation within groups for age does not affect the results, thereby relieving the concern that the sudden

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

change after 1918 could be driven by demographic shocks affecting certain cities with a particular demographic structure or a change in the demographic structure.

- **Change in the data quality** One might fear that there could be some changes in the data collection method between 1918 and 1919 that could drive these results. While the original sources do not mention such a change, I also check for this eventuality by inspecting whether monthly series are smooth between December 1918 and January 1919 in Figure A.11 in the Appendix. No discontinuity appears neither for cities with low nor high NPIs. The yearly series on suicides, homicides and accident do not indicate any change in the collected number of deaths when the cause of death is unrelated to NPIs.

5.2 The net medium-term impact of NPIs on mortality

In order to obtain an idea of the net benefits of NPIs, I run a DID specification for estimating equation 2. Table 3 displays the main results for mortality from all causes. Columns (1) to (4) utilize the reported total death rate as the 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 cumulated number of days of NPIs is similar for the three indicators, one extra day of NPI 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 1910 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 state's yearly minimum and maximum monthly temperature, the estimated number of migrants that came to the city in during the year, 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 of deaths per 1000 people in the estimated population, an increase of 0.02 deaths per 1000 people in the 1910 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 are significant at the 90% significance

level and are 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. Table A.4 in the 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 the cumulated number of days under NPIs on the log number of deaths.

A similar exercise demonstrated in Table 2 but using monthly data from 1915 to 1922. The results are similar in magnitude to those displayed in Table 3. When relying on monthly data, the coefficient estimates remain more stable when including controls. 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 cumulated number of days under NPIs is always statistically significant. Panel b) reports the coefficients of NPI speed. The results are never statistically significant. Finally, Table 4 uses 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.

I perform further robustness checks in Table 5 in estimating placebo tests to check whether intense NPIs are associated with a significant observable change from the census between 1910 and 1920 (panel A) , a change in health expenditures between 1918 and 1919 (panel B) or change in deaths that shouldn't be influenced by NPIs and immunity mechanisms as homicides, car accidents or suicides after 1917 (panel C). One does not observe any significant association between the share of black people, the sex ratio, the share of foreign born, or the share of literate people with NPI intensity. Moreover, there was no significant change in the level of health expenditures after the pandemic in cities with an intense level of NPIs. Finally, as for suicides, neither car accidents nor homicides are significantly associated with NPIs.

Ultimately, as the sample size remains relatively small, one might fear that the results could be influenced by some extreme events or few observations. To relieve these concerns on inference, I perform two additional robustness checks. First, I estimate the baseline specification by successively removing each city from the sample. Point estimates and standard errors are reported in Figure A.12 in the Appendix. The point estimates appear extremely stable and do not appear to depend on one single observation. Second, I follow Heß (2017) to perform permutation tests within statistical regions to compute p-values for the baseline estimates in Table A.3. The p-values tend to increase slightly while results remain statistically significant.

Table 2: Medium Run Impact of NPIs on Mortality (All causes of death, monthly data) (1915-1922)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Deaths/$Population_{1910}$				ln(Deaths)			
Panel a) Impact of NPI Days on Mortality								
Days NPI x Post	0.0018***	0.0017***	0.0010*	0.0011**	0.0013***	0.0013***	0.0009**	0.0008**
	(0.0006)	(0.0006)	(0.0006)	(0.0005)	(0.0004)	(0.0003)	(0.0003)	(0.0003)
R^2	0.696	0.810	0.818	0.813	0.975	0.983	0.984	0.983
N	4116	4116	4116	4116	4116	4116	4116	4116
Panel b) Impact of NPI Speed on Mortality								
Speed NPI x Post	0.000200	0.000900	0.000300	0.000100	0.000010	0.000700	0.000200	0.000300
	(0.0017)	(0.0022)	(0.0021)	(0.0022)	(0.0012)	(0.0015)	(0.0016)	(0.0017)
R^2	0.692	0.806	0.817	0.812	0.974	0.982	0.983	0.983
N	4116	4116	4116	4116	4116	4116	4116	4116
Time FE	Y	Y	Y	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y	Y	Y	Y
Pop1900	N	Y	Y	Y	N	Y	Y	Y
Timing	Y	Y	Y	Y	Y	Y	Y	Y
temperature	N	Y	Y	Y	N	Y	Y	Y
Health exp.	N	Y	Y	Y	N	Y	Y	Y
Region shocks	N	N	Y	N	N	N	Y	N
Demographics	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:

$Mortality_{i,t} = \delta_i + \gamma_t + \beta \times Post \times NPI_{1918,i} + \lambda \times Post \times X_i + \epsilon_{i,t}$

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 include 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 3: 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***	0.0121***	0.0110*	0.00640	0.0223***	0.0195***	0.0110	0.0126*	0.0016***	0.0013***	0.0007*	0.0007*
	(0.0040)	(0.0043)	(0.0062)	(0.0046)	(0.0064)	(0.0070)	(0.0070)	(0.0073)	(0.0004)	(0.0004)	(0.0004)	(0.0004)
R^2	0.878	0.882	0.888	0.890	0.866	0.856	0.863	0.867	0.993	0.994	0.994	0.994
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	0.00110	-0.00760	-0.0100	0.0395	0.0401	0.0108	0.0206	0.00180	0.00250	0.000600	0.00100
	(0.0308)	(0.0260)	(0.0249)	(0.0219)	(0.0307)	(0.0284)	(0.0255)	(0.0331)	(0.0021)	(0.0018)	(0.0015)	(0.0019)
R^2	0.867	0.874	0.884	0.888	0.846	0.845	0.861	0.863	0.992	0.993	0.994	0.994
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
Migrations	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:

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

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 include 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 cumulated number of days under NPIs

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 1917 while speed NPI indicates the speed at which the city implemented their NPI. Days NPI the cumulated number of days NPIs 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 include 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 cumulated number of days when NPIs are implemented

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

Table 5: Placebo tests

	(1)	(2)	(3)	(4)	(5)	(6)
Panel a) Characteristics of the population						
	share other origin	share black	share foreign born	share literate	ln(pop)	sex ratio
NPI Days X Post	0.0110	0.00670	0.000600	-0.00250	0.000700	-0.000800
	(0.0118)	(0.0054)	(0.0082)	(0.0032)	(0.0007)	(0.0005)
R^2	0.995	0.995	0.993	0.974	0.995	0.837
N	86	86	86	86	86	86
Panel b) Mortality rates						
	suicide	homicide	Total (1+2)	Accident	Total (3+4)	
NPI Days X Post	-0.0180	0.0129	-0.00280	-0.0324	-0.0509	
	(0.0124)	(0.0092)	(0.0136)	(0.0397)	(0.0492)	
R^2	0.801	0.889	0.846	0.470	0.772	
N	429	422	422	258	254	
Panel c) Health Expenditures per capita						
	health conservation	charities	Total (1+2)			
NPI Days X Post	-0.000400	-0.00200	-0.00240			
	(0.0010)	(0.0025)	(0.0030)			
R^2	0.857	0.922	0.928			
N	291	290	290			
Year FE	Y	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y	Y

Estimate of placebo tests :
$Y_{i,t} = \delta_i + \gamma_t + NPI\ Days_i \times Post_t + \epsilon_{i,t}$
NPI days is the cumulated number of days NPIs were in place in 1918.
Post is a dummy taking value 1 after 1918
Panel a) variables are from the US census performed in 1910 and 1920 collected in Ruggles et al. (2020)
Panel b) data are from the 1922 reports on mortality Statistics
Panel c) statistics are from the financial statistics of city for the years 1911, 1914, 1915, 1916 1917, 1918, 1920
NPI variable are from Markel et al. (2007)

6 Discussion of the results

The previous section demonstrated that cities that responded more aggressively to the second wave of influenza in autumn 1918 suffered from a rise in mortality in the following months and years. This phenomenon might be explained by the findings described in Gostic et al. (2016) who reveal that the first influenza virus that emerged in a region is key to understanding people'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. They also indicate that individuals that contracted certain particular strains of influenza are then less likely to die from influenza later in their life. Therefore, by flattening the epidemic curve, NPIs might have reduced the number of infections in October 1918, when the strain of influenza was the most contagious and thus increased the population's susceptibility to the subsequent strains.

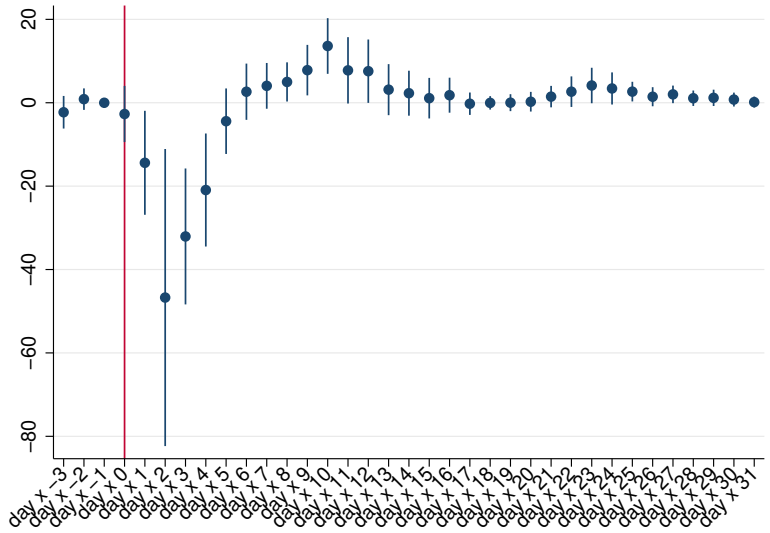
The hypothesis that NPIs reduced the number of contagions at the peak of

the pandemic is confirmed when investigating the short term impact of NPIs in October 1918, during the main and deadliest wave of the influenza epidemic. To illustrate this, I use the data from Collins et al. (1930) and follow 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) that estimate event studies that compare counties or states that have or have not implemented NPIs. I adapt their specification to my sample where all cities in the sample are treated and applied NPIs and to accommodate weekly data while accounting for the timing of the pandemic in each city:

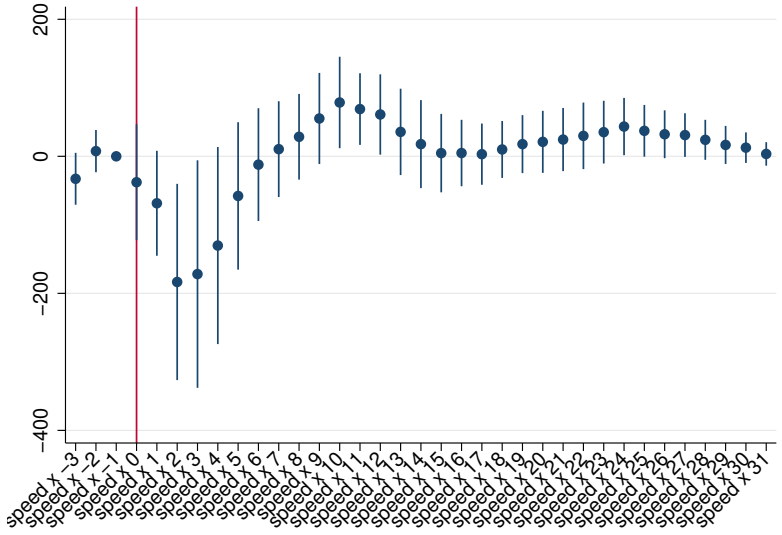
$$\begin{aligned} Excess_{i,t} =& \delta_i + \gamma_t + \sum_{ws=-5;ws\neq-4}^{38} \pi^{ws} \times 1_{t(i)=ws} + \sum_{ws=-5;ws\neq-4}^{38} \beta^{ws} \times 1_{t(i)=ws} \times NPI_{1918,i} \\ &+ \sum_{w=-3,w\neq-2}^{30} \omega^t \times 1_{t(i)=w} + \sum_t \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}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

$Excess_{i,t}$ 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. There are only a few data points to test for pre-trends before the epidemic as the series start at the beginning of the pandemic. γ_t is a time fixed effect. $\sum_{ws=-4}^{38} 1_{t(i)=ws}$ are dummies indicating the time elapsed from the implementation of the first NPI in the city. λ_t is a time fixed effect that interacted with control variables X_i. In addition, I also control for the distance with respect to the acceleration date of the epidemic ($\sum_{w=-3,w\neq-2}^{30} \omega^t \times 1_{t(i)=w}$). The results are displayed in Figure 6. These patterns are consistent with the findings of Bootsma and Ferguson (2007), Markel et al. (2007), and Correia, Luck, and Verner (2020) that intense NPIs managed to flatten the epidemic curve as the cumulated number of days under NPIs is associated with a decrease in the excess death rate from influenza during the five weeks after the acceleration date. 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. The impact of NPIs on mortality in October, i.e. during the peak of the pandemic, is also confirmed when relying on monthly data as illustrated in Table 6, and in the event study reported in the previous section. Estimates in Table 6 report that one cumulated day of NPI is associated with a drop of 0.022% of the mortality in October which translates into 0.016 lives saved per 1,000 inhabitants. This means that a city of 100,000 inhabitants with an average number of NPIs (88 days) avoided about 170 deaths in October 1918.

Figure 6: Event study: Estimates of the aggregate impact of NPIs on weekly excess mortality from influenza and pneumonia



(a) Number of Days



(b) Speed of implementation

Estimate of the event study

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

Controls include health expenditures in 1917, population in 1910, week fixed effects and cities' fixed effects, fixed effect for the distance from the implementation of the first NPI, fixed effects for the distance since the acceleration date

The red line materializes the week of implementation of the first NPI

95% confidence interval clustered at the city level

NPI days is the cumulated number of days under NPIs

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

Table 6: Short-Run Impact of NPIs on Mortality (All causes of death, monthly data) (1915/01-1918/10)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Deaths/$Population_{1910}$				ln(Deaths)			
Panel a) Impact of NPI Days on Mortality								
Days NPI x Post	-0.0160***	-0.0161***	-0.0156***	-0.0160***	-0.0021*	-0.0022*	-0.0022*	-0.0023**
	(0.0054)	(0.0054)	(0.0054)	(0.0054)	(0.0011)	(0.0011)	(0.0011)	(0.0011)
R^2	0.839	0.823	0.846	0.840	0.984	0.981	0.985	0.985
N	1966	1966	1966	1966	1966	1966	1966	1966
Panel b) Impact of NPI Speed on Mortality								
Speed NPI x Post	0.0198	0.0208	0.0211	0.0208	0.00850	0.00920	0.0093*	0.00940
	(0.0304)	(0.0296)	(0.0287)	(0.0301)	(0.0060)	(0.0057)	(0.0055)	(0.0058)
R^2	0.835	0.821	0.844	0.837	0.985	0.982	0.986	0.985
N	1966	1966	1966	1966	1966	1966	1966	1966
Time FE	Y	Y	Y	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y	Y	Y	Y
Pop1900	N	Y	Y	Y	N	Y	Y	Y
Timing	Y	Y	Y	Y	Y	Y	Y	Y
temperature	N	Y	Y	Y	N	Y	Y	Y
Health exp.	N	Y	Y	Y	N	Y	Y	Y
Region shocks	N	N	Y	N	N	N	Y	N
Demographics	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 acceleration date in the city and the the acceleration date in the sample. Non varying variables are interacted with year fixed effects. I also include 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

The short-term impact on the number of contagions might have been translated in the medium term asa lower number of deaths in cities that implemented less aggressive NPIs through several amplifying factors.

First, it is important to keep in mind that the 1918 pandemic gave birth to most of the subsequent virus strains (Taubenberger and Morens 2006); the 1918 virus was probably the original strain that afflicted a majority of U.S. citizen. As a consequence cities that implemented less aggressive NPIs in Autumn 1918 suffered from higher infection rates but might have developed a better immune response. It is worth noting that the virus strains of early autumn 1918 were probably the most contagious given that they were associated with a much higher number of deaths

in all cities. Unfortunately, data on contagions in this period are not available. It is also likely that intense NPIs mights have contributed to reduce more infections than deaths if one considers that some of them, such as school closures, reduced the exposition of people less likely to die. Intense NPIs fighting against the strains of autumn 1918 might have reduced the exposition to a particular strain associated with a better long-term immune response of the cities' population during their whole life.

Second, this improvement in the immune response of the population was of particular importance in a period where most of the deaths were caused by infectious diseases and other causes that were important co morbidity factors associated with influenza (Center for Disease Control and Prevention 2021; Lenzi et al. 2012). Indeed, in 1920, the most important causes of deaths were, in order Pneumonia and Influenza (14%), organic diseases of the heart (11%), Tuberculosis (7.8%), Acute Nephritis (6.8%) or Cancer (6.4%). It is noteworthy that all these identified causes of deaths are also co-morbidity factors associated with influenza. Consequently, by making people more sensitive to the subsequent strains of influenza, NPIs might have reduced the life expectancy of people afflicted by other co morbidity factors. Table 7 likely indicates this as NPIs appear to be associated with an increase in mortality from these comorbidity factors and this bridges the gap between the impact of NPIs on the sole deaths from influenza and pneumonia and their impact on the total number of deaths. If this can reflect misclassification in the cause of death, one might think that by reducing the immune response to influenza, NPIs also reduced the life expectancy of people afflicted with other chronic diseases in these cities.

Table 7: Impact on deaths with comorbidity

	(1)	(2)	(3)	(4)	(5)
	Cancer	Heart	Nephritis	Tuberculosis	Total
NPI days X Post	0.0475	0.1986***	0.0705	0.1932***	0.5098***
	(0.0310)	(0.0591)	(0.0539)	(0.0643)	(0.1575)
R^2	0.790	0.875	0.454	0.927	0.836
N	429	429	429	429	429
Time FE	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y

Post is a dummy indicating observations after September 1917 while NPI Days NPI describes the cumulated number of days under NPIs.
Estimates of the difference in difference equation:
$Mortality_{i,t} = \delta_i + \gamma_t + \beta \times Post \times NPI_{1918,i} + \epsilon_{i,t}$
Mortality rates by cause are from the 1922 report.
NPI variable is from Markel et al. (2007)

Third, the development of individual immunity during autumn 1918 might be

complemented by the development of 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"*. As such, it is possible 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. Consequently, this facilitates 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.

Fourth, by reducing the diffusion of influenza over the next periods, less aggressive NPIs in autumn might have reduced the likelihood of pregnant women contracting influenza. Thus, this potentially limited in utero exposition and its negative consequences on the health of children and future young adults even in the long-run as documented in Almond (2006) and Lin and Liu (2014) who find that in utero exposition decrease the future earnings of young adults but also the likelihood to have serious health problems including kidney disease, circulatory and respiratory problems, and diabetes.

The combination of these factors might explain the long-term and medium-term impact of NPIs. The lower exposure to the initial strain of influenza might have increased the sensitivity of the population to the circulation of the next strains reducing their life expectancy, especially in these afflicted by other co morbidity factors such as tuberculosis or heart diseases. However, one should keep in mind that if these increases offset the short-term benefits of the lives saved during the first wave of influenza, they remain relatively modest. For instance, a city with 100,000 inhabitants that implemented the average cumulated number of NPI days (88) would suffer from a yearly loss of about 100 lives. The particular context of the early XXth century is probably very different from nowadays as the share of infectious disease in the number of deaths is much smaller in contemporaneous developed economies. Moreover, the development of vaccines and improvements in medical science tends to alleviate our concerns regarding the medium-term impact of NPIs as implemented in 2020 while stressing the importance of exit strategies and vaccination campaigns once these NPIs are repealed.

7 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 and from monthly data on total mortality confirm that NPIs flattened the epidemic curve and reduced the number of deaths in October 1918 during the deadliest wave. However, I find that cities that implemented more intense and longer NPIs underwent a relatively higher number of deaths in subsequent

months and years. These results are robust to the inclusion of numerous controls, such as regional shocks, the demographic structure and estimated migration flows. This sheds 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 warn of 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. This study also illustrates that the benefits of NPIs should be evaluated in terms of years of life saved rather than by an absolute number of lives.

Furthermore, my results can also shed new light on the current debate surrounding 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 no or better economic outcomes 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 rebounds 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 a 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 influenza and 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 significantly less developed back then as compared to 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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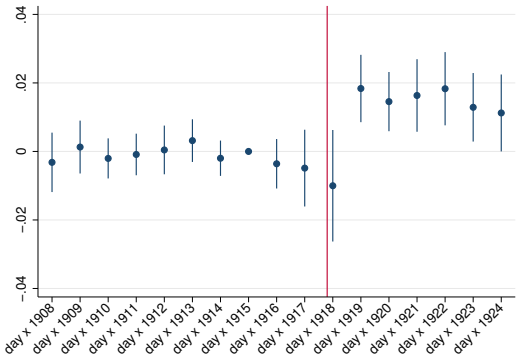
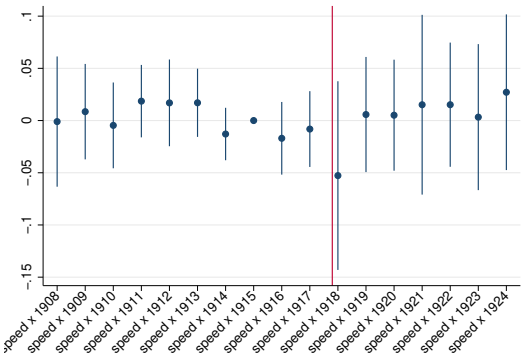
A Robustness Checks

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

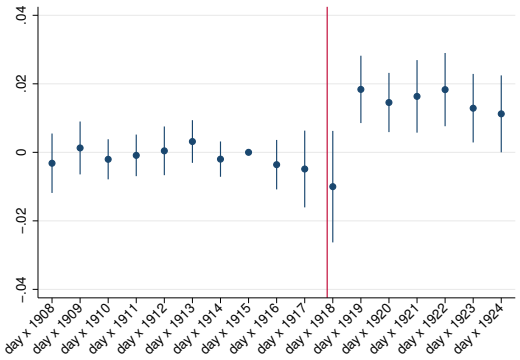
Figure A.1: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **from 1908**



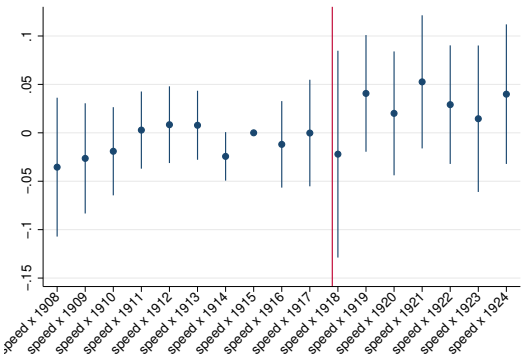
(a) Reported Death rate: Number of Days



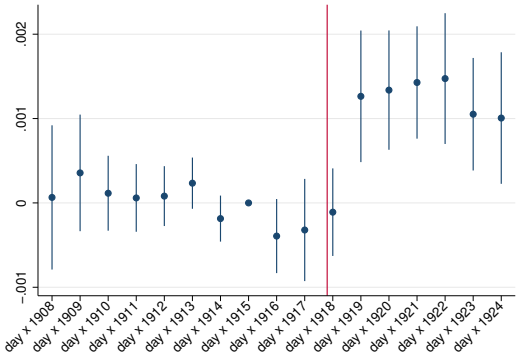
(b) Reported Death rate: Speed of implementation



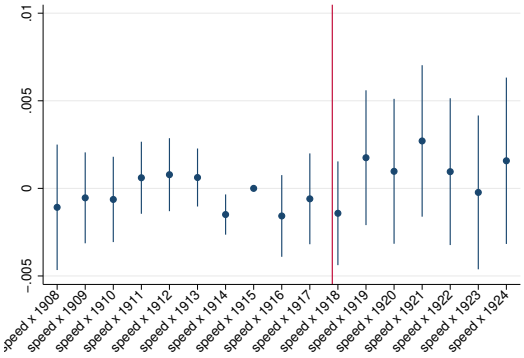
(c) Deaths/$Population_{1910}$: Number of Days



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



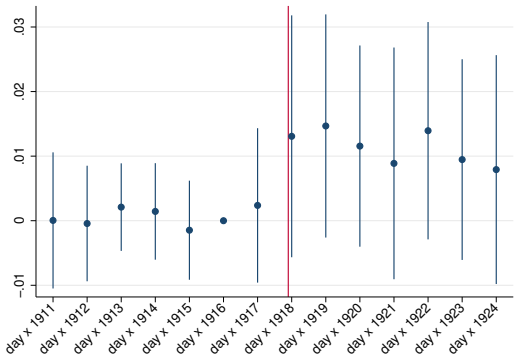
(e) log(deaths): Number of Days



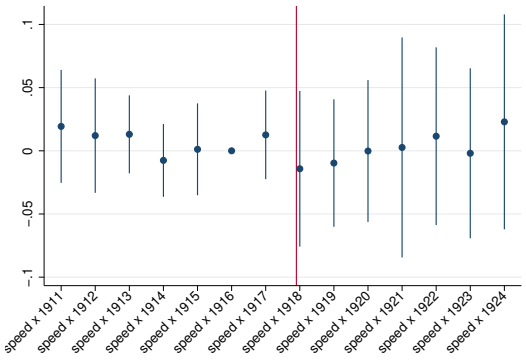
(f) log(death): Speed of implementation

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

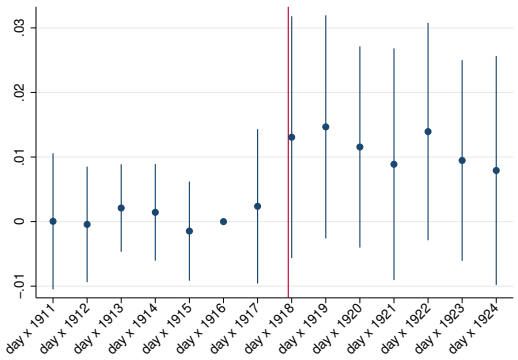
Figure A.2: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for regional shocks**



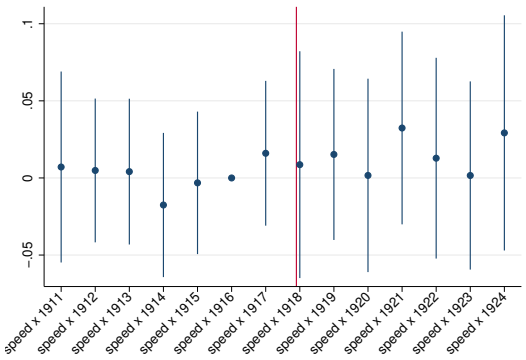
(a) Reported Death rate: Number of Days



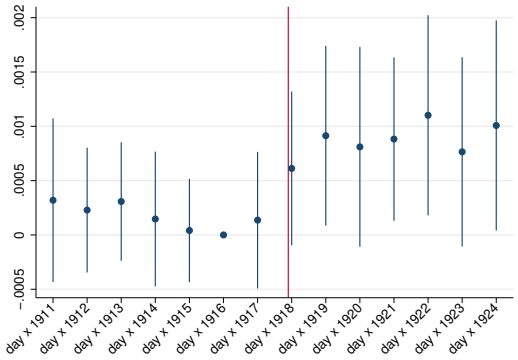
(b) Reported Death rate: Speed of implementation



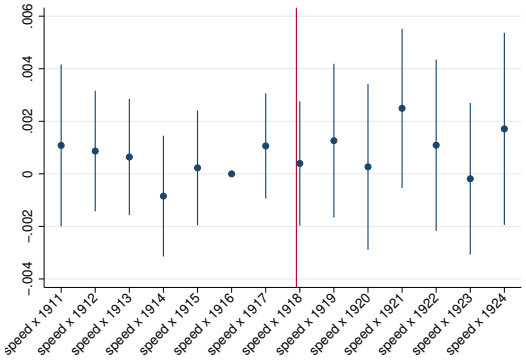
(c) Deaths/$Population_{1910}$: Number of Days



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



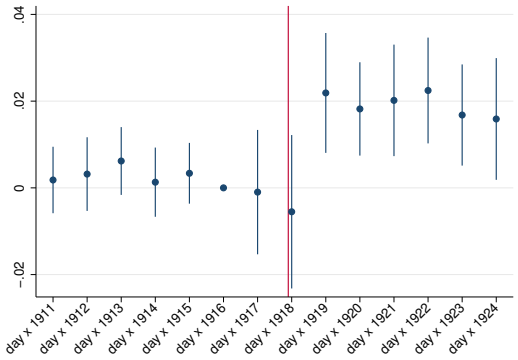
(e) log(deaths): Number of Days



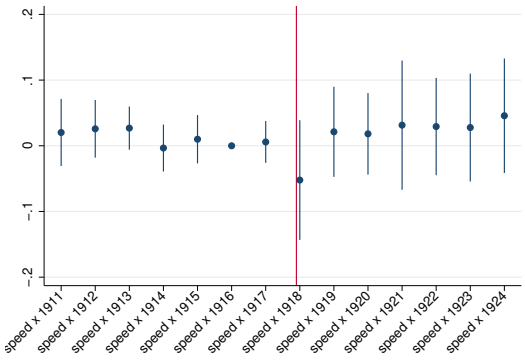
(f) log(death): Speed of implementation

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

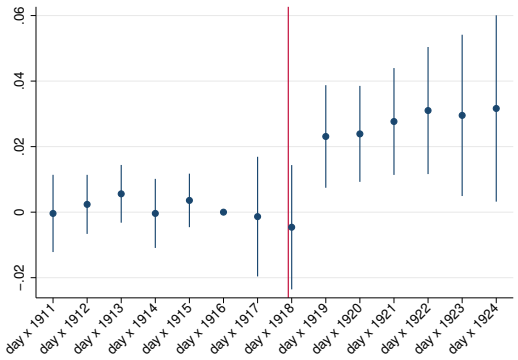
Figure A.3: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for excess mortality in 1918**



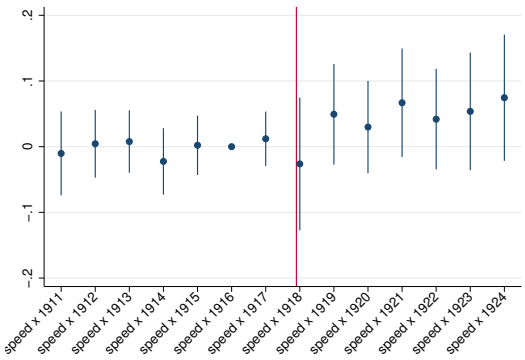
(a) Reported Death rate: Number of Days



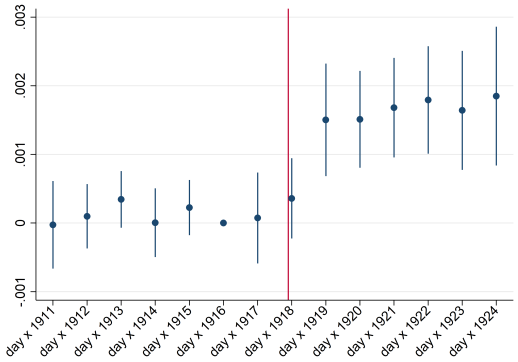
(b) Reported Death rate: Speed of implementation



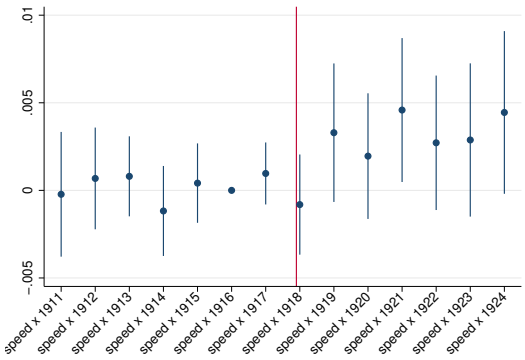
(c) Deaths/$Population_{1910}$: Number of Days



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



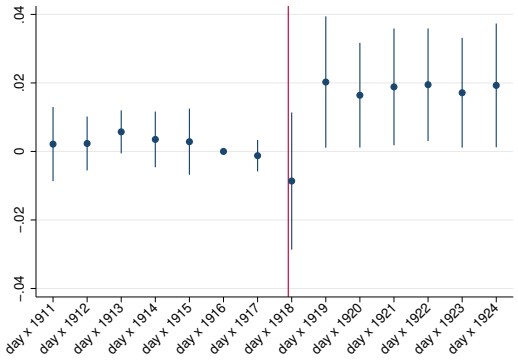
(e) log(deaths): Number of Days



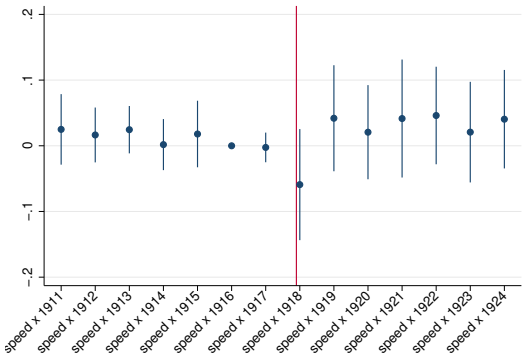
(f) log(death): Speed of implementation

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

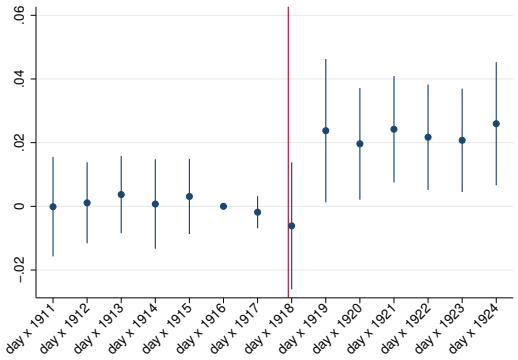
Figure A.4: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **using Clay et al (2019) control variables**



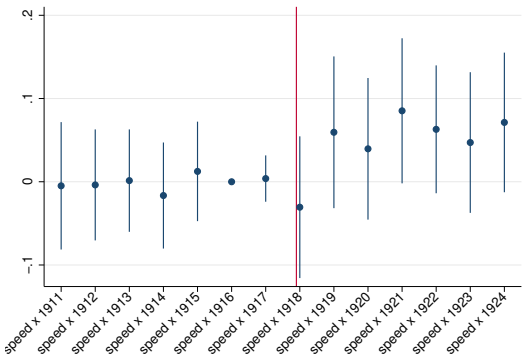
(a) Reported Death rate: Number of Days



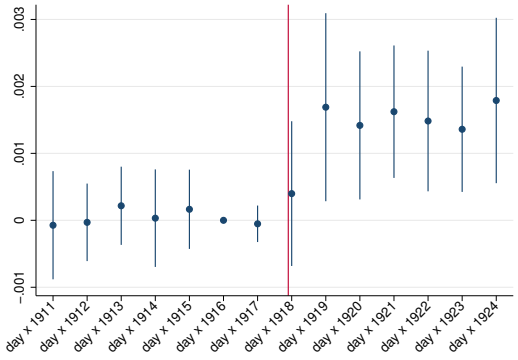
(b) Reported Death rate: Speed of implementation



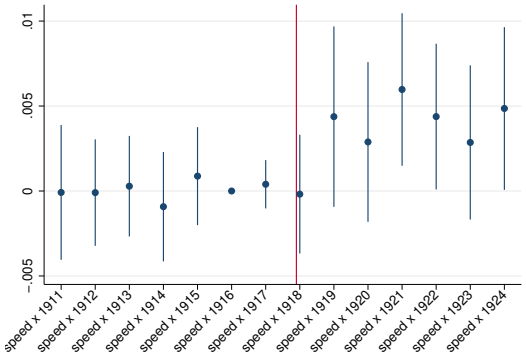
(c) Deaths/$Population_{1910}$: Number of Days



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



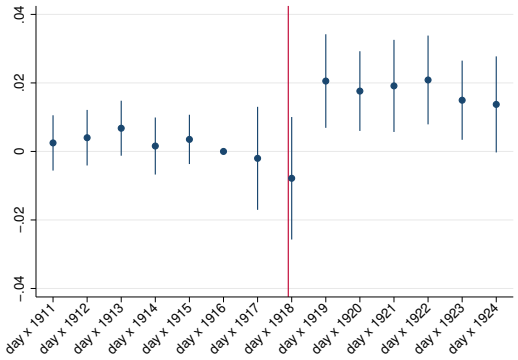
(e) log(deaths): Number of Days



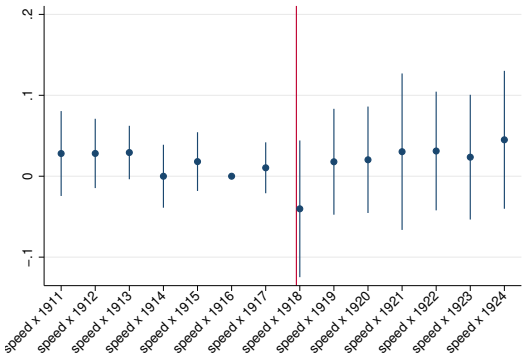
(f) log(death): Speed of implementation

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)

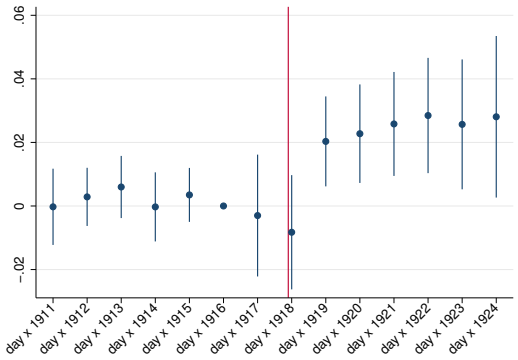
Figure A.5: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for estimated migration flows**



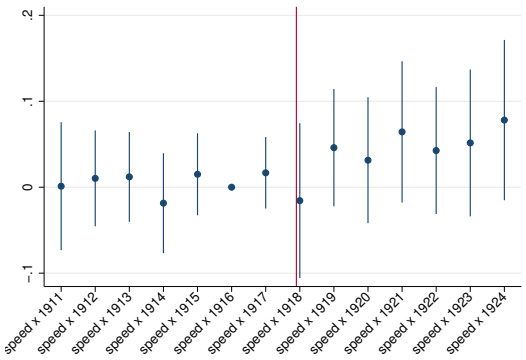
(a) Reported Death rate: Number of Days



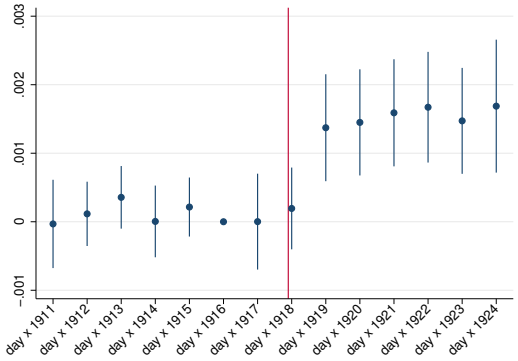
(b) Reported Death rate: Speed of implementation



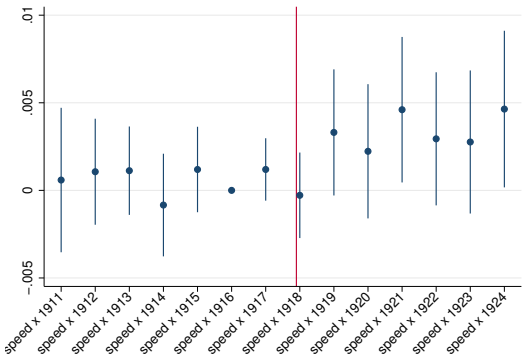
(c) Deaths/$Population_{1910}$: Number of Days



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



(e) log(deaths): Number of Days

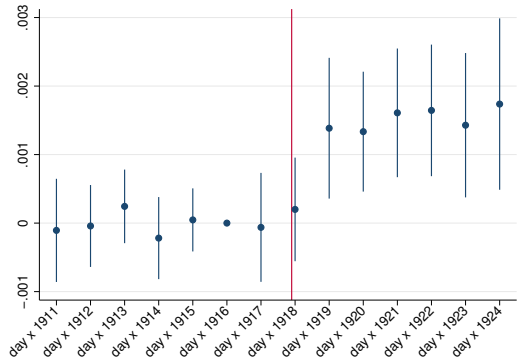


(f) log(death): Speed of implementation

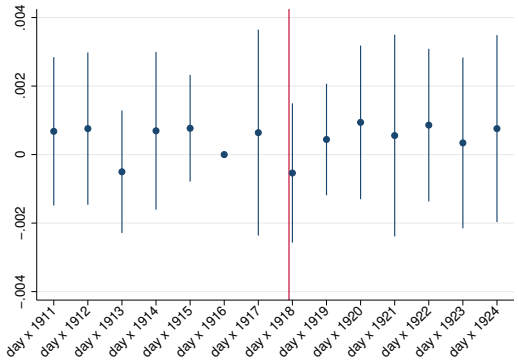
Estimates of the difference in difference equation:
$Deathrate_{i,t} = \delta_i + \gamma_t + \sum_{t \neq 1916} \beta_t \times 1_{t(i)=t} \times NPI_{1918,i} + \sum_{t \neq 1916} \lambda_t \times 1_{t(i)=t} \times X_i + \epsilon_{i,t}$
Controls include health expenditures in 1917, population in 1910,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

A.2 Investigating the role of suicide

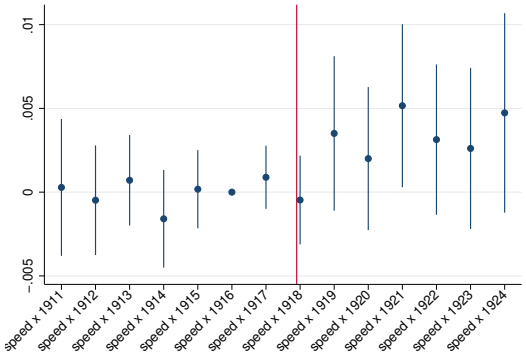
Figure A.6: Event study: Estimates of the aggregate impact of NPIs on mortality (net of suicides) and on suicides



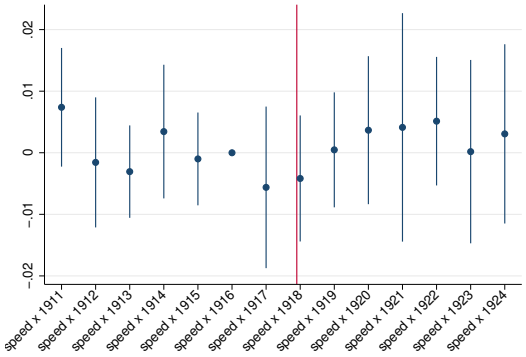
(a) log(deaths-suicides)



(b) log(suicides)



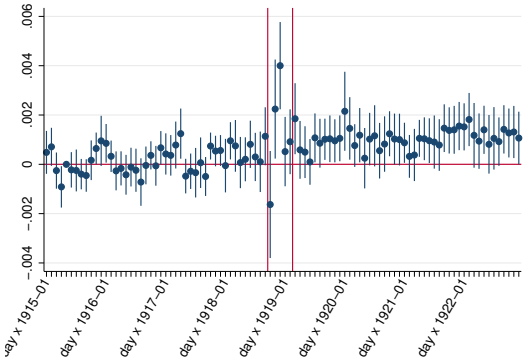
(c) log(deaths-suicides)



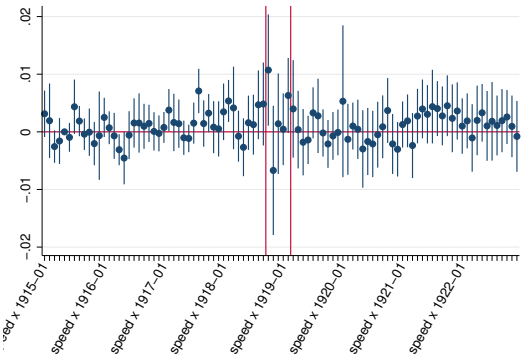
(d) log(suicides)

A.3 Evidence from Monthly deaths

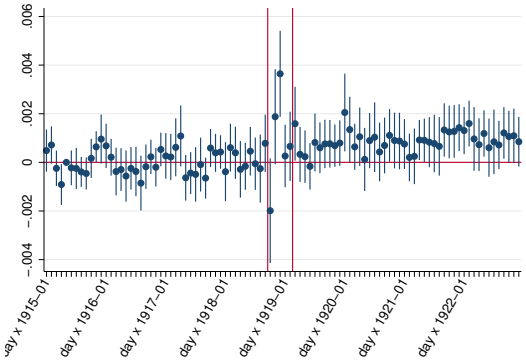
Figure A.7: Event study: Estimates of the aggregate impact of NPIs on monthly mortality (All causes of death) **Robustness checks**



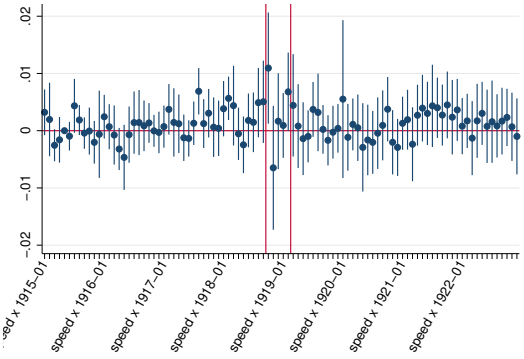
(a) log(deaths)
Number of Days
controlling for regional shocks



(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

A.4 Controlling for differences in the demographic structures

A.4.1 Balance tests

Table A.1: Balance test, demographics by length of NPIs

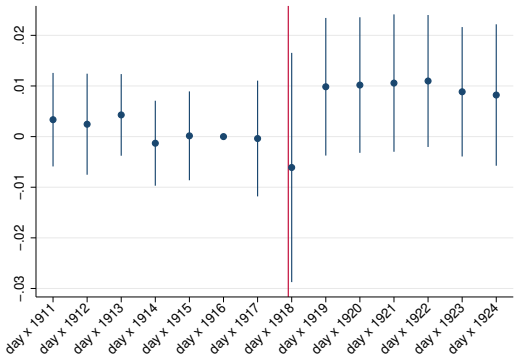
		Below the Median			Above the Median			Difference		
variable	year	Average	Standard Deviation	Obs	Average	Standard Deviation	Obs	Difference	Tstat	pvalue
Population	1910	310610	326523	22	578011	1.057e+06	21	-267402	-1.132	0.264
Growth	1910	0.346	0.436	22	0.655	0.630	21	-0.309	-1.878	0.0675
Average age	1910	28.15	1.321	22	28.65	1.300	21	-0.505	-1.262	0.214
Median age	1910	26.05	1.463	22	26.81	1.601	21	-0.764	-1.635	0.110
share white	1910	91.42	13.00	22	94.27	6.209	21	-2.849	-0.910	0.368
share foreign born	1910	24.78	11.90	22	20.46	9.597	21	4.320	1.307	0.199
Sex ratio	1910	0.988	0.0923	22	1.073	0.138	21	-0.0844	-2.364	0.0229
Population	1920	369174	385078	22	711416	1.249e+06	21	-342242	-1.226	0.227
Growth	1920	0.187	0.110	22	0.282	0.191	21	-0.0949	-2.003	0.0518
Average age	1920	29.01	1.330	22	29.98	1.520	21	-0.964	-2.216	0.0323
Median age	1920	27.18	1.593	22	28.71	1.793	21	-1.532	-2.966	0.00501
share white	1920	91.72	11.89	22	93.42	6.013	21	-1.706	-0.589	0.559
share foreign born	1920	21.59	10.37	22	16.63	8.748	21	4.958	1.690	0.0986
Sex ratio	1920	0.968	0.0607	22	1.015	0.0519	21	-0.0465	-2.693	0.0102

Table A.2: Balance test, demographics by speed of NPIs

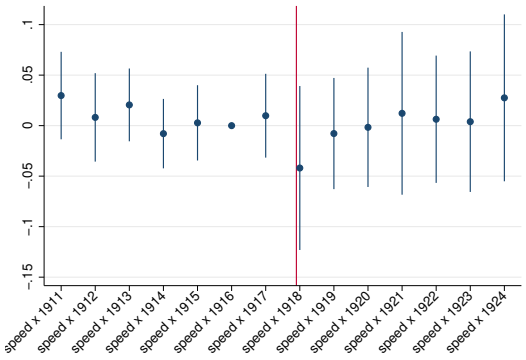
		Below the Median			Above the Median			Difference		
variable	year	Average	Standard Deviation	Obs	Average	Standard Deviation	Obs	Difference	Tstat	pvalue
Population	1910	326922	320429	22	560922	1.063e+06	21	-234001	-0.987	0.329
Population growth	1910	0.365	0.436	22	0.636	0.639	21	-0.271	-1.635	0.110
Average age	1910	28.10	1.259	22	28.70	1.342	21	-0.603	-1.519	0.136
Median age	1910	26.05	1.430	22	26.81	1.632	21	-0.764	-1.635	0.110
share white	1910	91.23	12.29	22	94.47	7.486	21	-3.240	-1.038	0.306
share foreign born	1910	24.20	11.97	22	21.08	9.745	21	3.119	0.934	0.356
Sex ratio	1910	0.994	0.0903	22	1.067	0.143	21	-0.0736	-2.028	0.0491
Population	1920	388825	377175	22	690830	1.257e+06	21	-302005	-1.078	0.287
Population growth	1920	0.193	0.106	22	0.275	0.197	21	-0.0828	-1.729	0.0913
Average age	1920	29.04	1.402	22	29.95	1.469	21	-0.911	-2.080	0.0438
Median age	1920	27.27	1.723	22	28.62	1.746	21	-1.346	-2.545	0.0148
Share white	1920	91.35	11.43	22	93.81	6.773	21	-2.460	-0.853	0.398
share foreign born	1920	20.91	10.59	22	17.34	8.838	21	3.571	1.197	0.238
Sex ratio	1920	0.978	0.0609	22	1.005	0.0588	21	-0.0262	-1.433	0.159

A.4.2 Event Studies controlling for the demographic structure

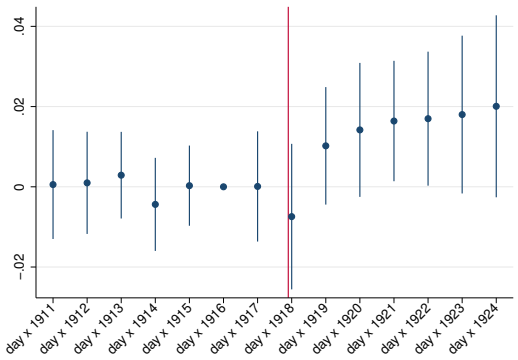
Figure A.8: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for demographic characteristics in 1910**



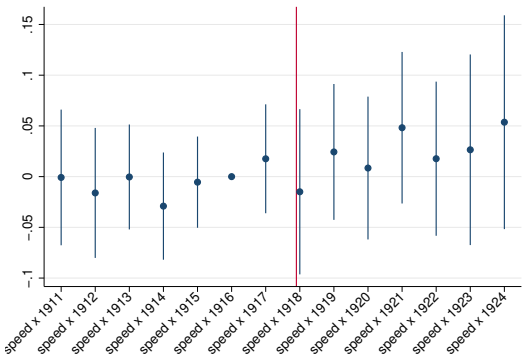
(a) Reported Death rate: Number of Days



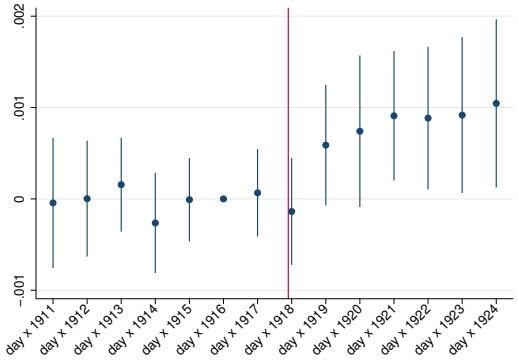
(b) Reported Death rate: Speed of implementation



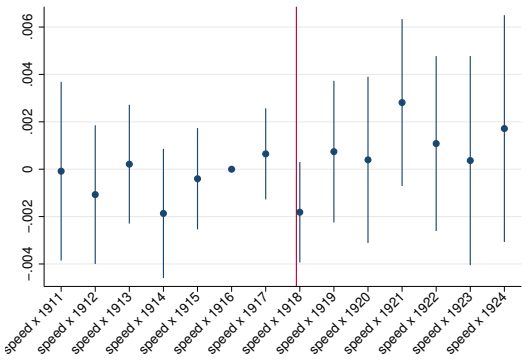
(c) Deaths/$Population_{1910}$: Number of Days



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



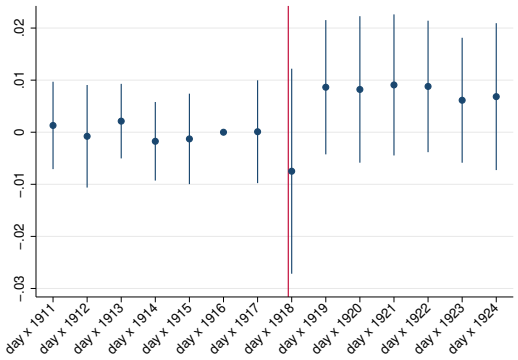
(e) log(deaths): Number of Days



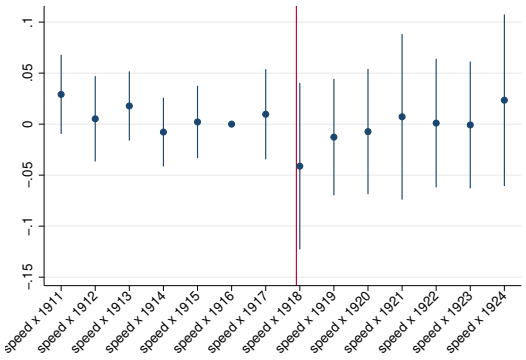
(f) log(death): Speed of implementation

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

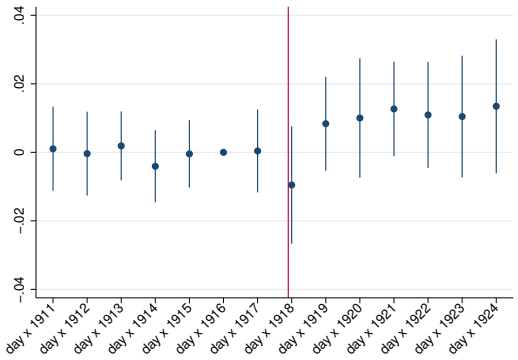
Figure A.9: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **controlling for demographic characteristics in 1920**



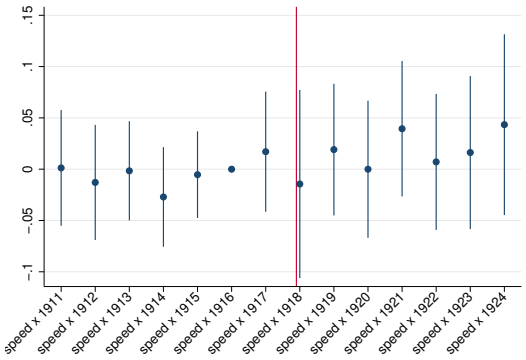
(a) Reported Death rate: Number of Days



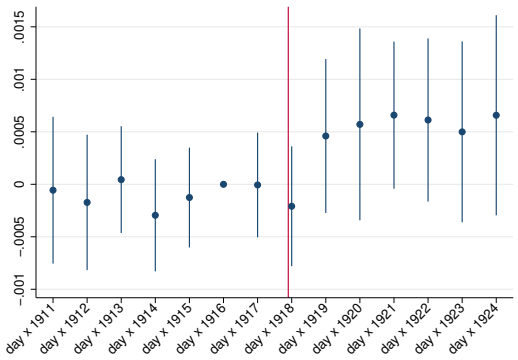
(b) Reported Death rate: Speed of implementation



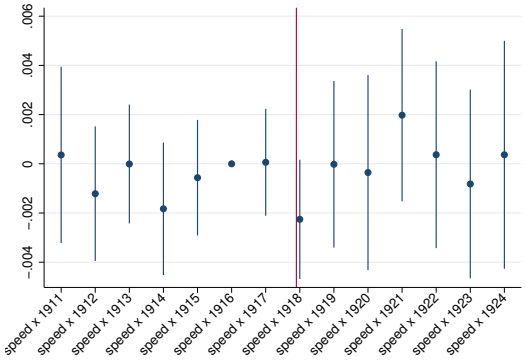
(c) Deaths/$Population_{1910}$: Number of Days



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



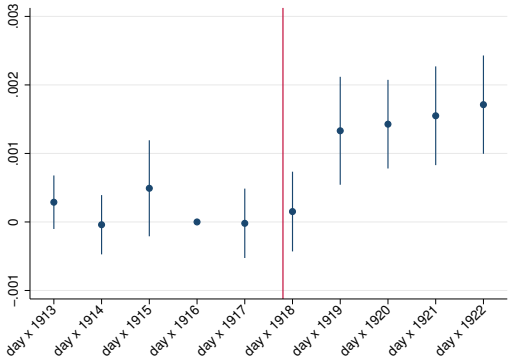
(e) log(deaths): Number of Days



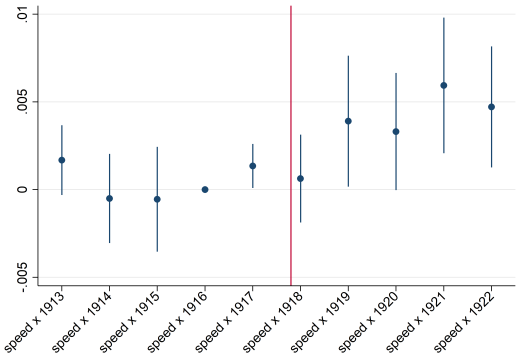
(f) log(death): Speed of implementation

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

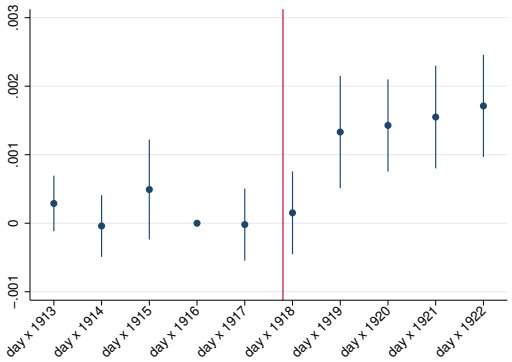
Figure A.10: Event study: Estimates of the aggregate impact of NPIs on mortality (All causes of death) **by age groups**



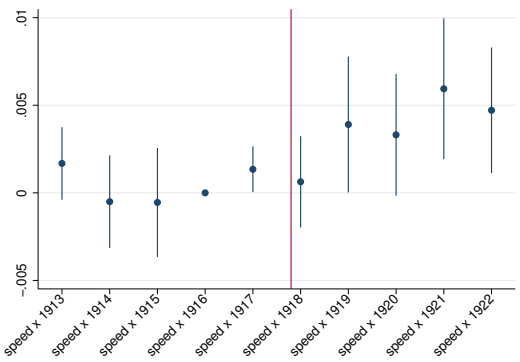
(a) log(deaths):
Number of Days
Controlling for age groups fixed effects and shocks



(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

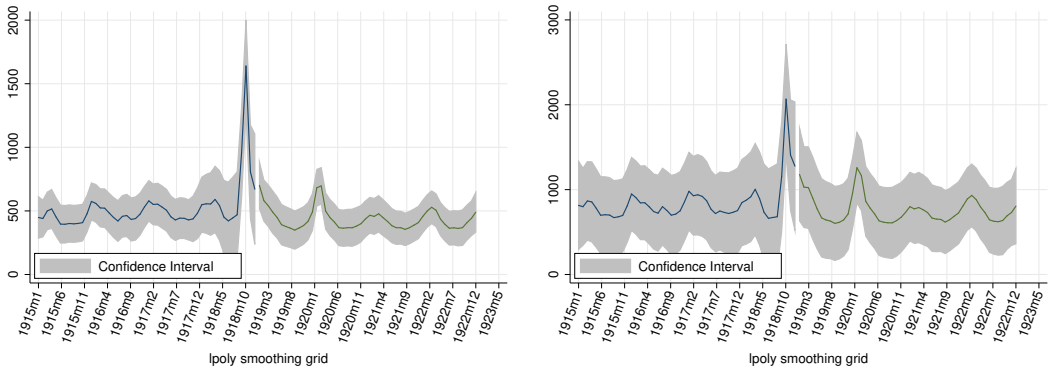


(d) log(death):
Speed of implementation
Controlling for city x age groups fixed effects

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

A.5 Descriptive statistics on mortality series following the level of NPIs

Figure A.11: Smoothness of the original monthly mortality series between 1918 and 1919 **by level of NPIs**



(a) Cities with a number of NPI days below the median

(b) Cities with a number of NPI days above the median

Separate non parametric regression from 01/1915 to 12/1918 and from 01/1919 to 12/1922
Epaniechnikov kernel estimator

A.6 Statistical Inference

Table A.3: Permutation Tests

	Yearly data			Monthly data	
	Gross Rate	$Deaths/Pop_{1910}$	ln(Deaths)	$Deaths/Pop_{1910}$	ln(Deaths)
NPI Days $\times Post$	0.0138	0.02	0.00156	0.0018	0.001329
p-value	0.001	0.001	0.001	0.002	0.001
p-value (RI)	0.02	0.096	0.059	0.026	0.016
Year FE	Y	Y	Y	Y	Y
City FE	Y	Y	Y	Y	Y

Permutation tests for Random Inference. 1000 permutation were performed within Statistical regions and clustered at the city level following Heß (2017).
Standard errors are clustered at the city level

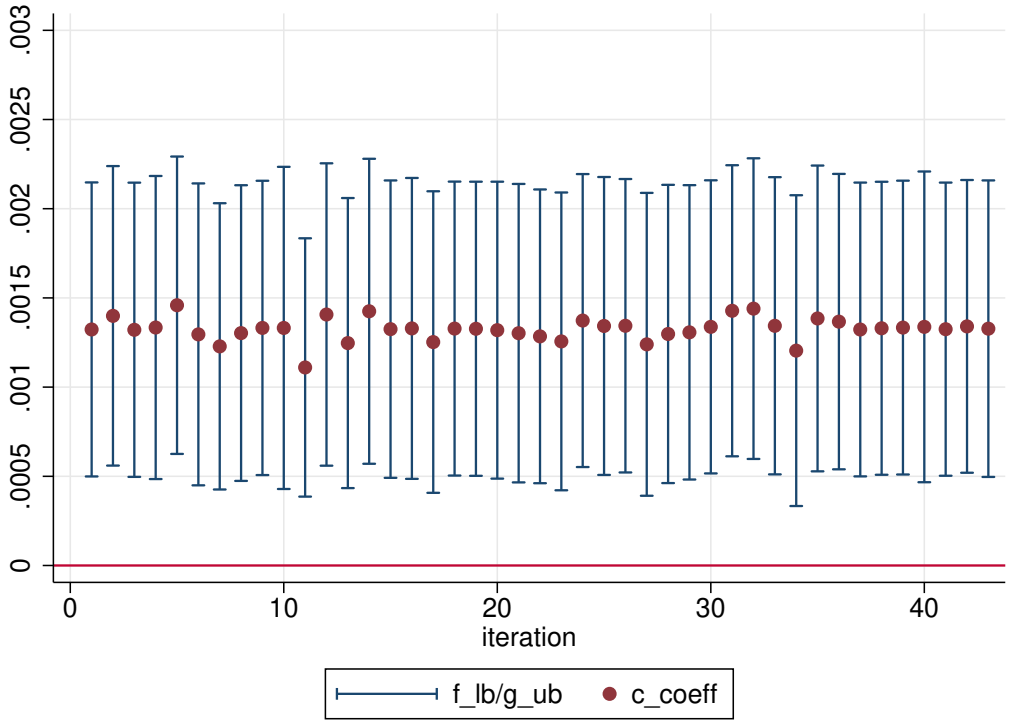


Figure A.12: Robustness checks, eliminating each of the 43 cities in the baseline regression

A.7 Results with mortality from influenza and pneumonia

Table A.4: 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	0.150	0.0901	0.2202*	0.0596	0.259	0.139	0.172	0.108	0.0028***	0.0022***	0.0011*	0.0012**
d_SE	(0.1214)	(0.1247)	(0.1268)	(0.1176)	(0.1606)	(0.1670)	(0.2011)	(0.1360)	(0.0006)	(0.0006)	(0.0006)	(0.0005)
R^2	0.898	0.899	0.903	0.902	0.907	0.908	0.911	0.913	0.976	0.977	0.979	0.980
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	0.568	0.667	0.140	1.3833**	0.860	0.688	0.294	0.00410	0.0059*	0.00240	0.00240
	(0.5425)	(0.5763)	(0.5742)	(0.4684)	(0.6814)	(0.7356)	(0.7069)	(0.5902)	(0.0040)	(0.0030)	(0.0019)	(0.0027)
R^2	0.898	0.899	0.902	0.902	0.907	0.908	0.911	0.913	0.972	0.976	0.979	0.980
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
Migrations	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 cumulated number of days under NPIs

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 include 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.