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DP17666

DO PANDEMICS CHANGE HEALTHCARE? EVIDENCE FROM THE GREAT INFLUENZA

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Melissa Thomasson

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Discussion Paper DP17666
Published 14 November 2022
Submitted 05 November 2022

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Abstract

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JEL Classification: I11, I14, J10, N32

Keywords: N/A

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Acknowledgements

We thank Haley Drake, Jeffrey Liu, and Nikolaos Souslous for their excellent research assistance. Mitchener thanks the Center for Advanced Studies of Behavioral Sciences for their support in 2021-22. We thank Robby Aronowitz, Marco del Angel, and seminar and conference participants at the 2022 NBER Summer Institute, the University of Arizona, Universita' Bocconi, CUNEF University, and the University of Southern Denmark for comments and helpful feedback.

Do Pandemics Change Healthcare?

Evidence from the Great Influenza

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November 5, 2022

Abstract

Using newly digitized U.S. city-level data on hospitals, we explore how pandemics alter preferences for healthcare. We find that cities with higher levels of mortality during the Great Influenza of 1918-1919 subsequently expanded hospital capacity by more than cities experiencing less influenza mortality: cities in the top half of the mortality distribution increased their count of hospitals by 8-10 percent in the years after the pandemic. This effect persisted to 1960 and was driven by increases in non-governmental hospitals. Growth responded most in richer cities, exacerbating existing inequalities in access to healthcare. We do not find evidence that government-run hospitals or other types of city-level spending related to healthcare responded to pandemic intensity, suggesting that large health shocks do not necessarily lead to increased public provision of health services.

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Keywords: hospitals, healthcare, influenza, pandemics, local public goods

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I. Introduction

The global pandemic of 2020-22 has dramatically upended the delivery of healthcare. One of the most visible effects of the disruption caused by COVID-19 has been the dramatic increase in the use of electronic information and telecommunications technologies to reduce in-person visits (Koma, Cubanski, and Neuman 2021). In the U.S., telehealth utilization especially increased in areas where the pandemic was more severe, accounting for upwards of 80 percent of all visits in these locations (Karimi et al. 2022). Prior to the pandemic, uptake of telehealth by patients and health facilities had been slow, with a mere 0.15 percent of all health visits in 2019 conducted through telehealth, even though 24 percent of U.S. healthcare organizations already had programs for telehealth delivery.¹ Part of this adaptation was supply-driven, as providers ramped up telehealth efforts to accommodate patient needs as the pandemic led them to cancel in-person care and payors expanded the kinds of visits they would reimburse.

But COVID-19 may have also changed individual preferences for healthcare provision, with 76 percent of patients recently surveyed stating that they were likely to continue to use telehealth going forward, compared to just 11 percent in 2019 (Bestsenny et al. 2021). Given the ongoing nature of the global pandemic, it is currently difficult to project whether these trends will persist.

The magnitude of the strain on healthcare delivery mechanisms seen during the COVID-19 pandemic is similar to that witnessed during the Great Influenza (GI) of 1918-19. That pandemic struck the U.S. during World War I, when many nurses and doctors were overseas, creating staffing shortages at home. In places where the influenza pandemic was most severe, existing facilities were overrun with patients, leading to temporary, makeshift “influenza hospitals” constructed in gymnasiums, performance halls, and even in the open air. Responses to these two global pandemics thus raises an interesting question that, to date, has not been addressed in the literature. Do public health shocks generate lasting changes in the provision of healthcare?

In this paper, we use the Great Influenza to investigate the effects of large health shocks on the private and public provision of healthcare services in the years after the 1918-19 pandemic. The GI was a massive public health crisis that struck near the birth of what we now consider the

¹Figures from FairHealth (2020) and Becker Hospital Review (2020).

modern hospital and healthcare system in the United States. Precisely how local governments and private providers would respond to it and whether it would alter household preferences for the delivery of healthcare was not clear at the time. In the early 20th century, hospitals predominantly served indigent persons and members of the working class. Middle- and upper-class persons would be admitted for surgical procedures, but still preferred to have private physicians and nurses visit their homes for acute illnesses. On the other hand, the GI was proving to be a massive killer, far worse than prior years of seasonal influenza. As such, the GI provides a unique setting to test how healthcare delivery systems respond to crises in both the short run and long run.

To estimate the effects of the GI on healthcare provision, we focus on the provision of healthcare through hospitals for several reasons. First, during the Progressive Era, hospitals were evolving into their modern form of fee-for-service providers with on-site clinicians. Advances in medical training and technology, which leveraged clinical work in hospitals, were creating a professionalized staff of doctors and nurses that could care for patients in a centralized location. Scientific discoveries and advances in clinical techniques were also beginning to be reflected in improved patient outcomes, shifting hospitals from providing nursing care for the working poor to admitting middle- and upper-class patients for surgery. When the pandemic struck, higher income people no longer had the option to stay at home. Entire families were struck with illness, with no one to care for them. The GI provided a substantial shock to a fledgling industry. As a result, we can examine whether the pandemic was merely a transient disruption, or whether it led to long-run changes in the demand for hospital care.

Second, given that hospitals were operated by private (often not-for-profit) entities and municipal governments, the hospital response to the pandemic allows us to examine both public and private responses to public health shocks. Ex ante, the nature of the supply-side response to increased demand caused by the pandemic is not obvious. It might be that, regardless of ownership, all hospitals expanded capacity to serve their patient populations. On the other hand, if the pandemic shocked endowments unequally and made it more difficult to agree over the provision of local public goods, rising demand by higher-income households would have to be met through the market, and we might see expansion only in hospitals operated by private entities. We leverage a

rich, new dataset on the location and attributes of hospitals to shed light on how the healthcare system responded.

We construct a novel dataset of hospitals spanning the period 1909 to 1975, combining newly digitized information from the *American Medical Directory* and the *Journal of the American Medical Association* with Finkelstein (2007)'s American Hospital Association (AHA) directory data that begins in 1948. We standardize across these three data sources to create the first consistent long-run measure of hospital access in American cities. Our dataset spans the period when the modern hospital industry emerged in the first quarter of the 20th century to the 1970s. Importantly, our standardized data includes both the number of hospitals in each city and the number of beds, allowing us to measure both new hospital entries and existing hospital expansion. For each hospital, we also observe whether it is operated by local government or by a private entity (either a for-profit or non-profit organization). We combine these new data with information on pandemic (and pre-pandemic) city-level mortality and estimate the effects of pandemic severity on the local provision of healthcare. We estimate dynamic event studies and difference-in-differences models that allow us to trace out the effects of pandemic mortality over time. Results show that pandemic intensity was not selected on hospital growth *before* 1918, but that cities experiencing higher pandemic mortality rates subsequently built more hospitals and increased hospital capacity (beds) — an effect that persists through the 1960s.

We also explore potential mechanisms and sources of heterogeneity that affect our main results. Increased hospital capacity was driven by growth in private facilities as opposed to hospitals operated by municipal or county governments.² Similarly, we find no evidence that pandemic intensity had any effect on municipal healthcare spending during the pandemic or in the subsequent time period. Results also suggest that the pandemic's effects on hospital capacity were not evenly distributed. Given the lack of health insurance coverage prior to World War II, it is perhaps not surprising that our analysis shows that cities with higher occupational income responded more than cities with below-median income. We also find greater effects for smaller cities than larger

²State-operated healthcare facilities mostly catered to mental health patients or specific groups, such as in prison hospitals. Federal facilities were largely confined to the military and veterans. This sector grew in importance after 1930 when president Hoover's consolidated it into the Veterans Administration.

cities. Smaller cities had fewer beds per capita in 1918 than larger cities, and it appears that one effect of the pandemic was to encourage these cities to catch up to their larger peers in hospital access.

Our paper makes several contributions to the existing literature. First, we examine how external shocks may alter preferences for the provision of healthcare in the short- and longer-run, a topic that has thus far received little attention. The empirical literature on local public goods has suggested that large external shocks, such as pandemics, can alter the provision of local public goods through three mechanisms: changed preferences (Foster and Rosenzweig 1995; Gustafsson, Biel, and Garling 2000; Banerjee and Somanathan 2007; Cárdenas et al. 2017; Duchoslav 2017; Cecchi and Duchoslav 2018), collective action (Alesina, Baqir, and Easterly 1999; de Janvry, Dequiedt, and Sadoulet 2014), and budget constraints (Feler and Senses 2017; Jerch, Kahn, and Lin 2020). This literature suggests that the expected effects of a large public health shock are *ex ante* unclear. Standard political economy arguments suggest that healthcare provision by local governments could increase in response to public health shocks, at least in the short run, in order to reduce mortality, and possibly in the medium and long run to mitigate the impact of future health shocks (Meltzer and Richard 1981; Page and Shapiro 1992; Iversen and Soskice 2001). On the other hand, if global health shocks and other types of widespread disasters lead to greater variance in outcomes for voters (in terms of health and incomes), cities may find it harder to agree on increased public-service provision (Anderson, Mellor, and Milyo 2008; Cárdenas 2003; de Oliveira, Croson, and Eckel 2015). At the same time, non-homothetic preferences potentially make demand for medical treatment quite income elastic. In this case, even in the face of differing preferences for public goods across income groups, there could still be a large differential increase in demand for non-public healthcare provision in cities where the pandemic struck with greater intensity. In this paper, we explore whether communities responded to public health shocks by providing public services, or if the response was driven by an increase in demand for private healthcare provision—potentially exacerbating inequality in access to healthcare.

Second, to our knowledge this is one of the first papers in economics that sheds light on factors influencing the development of the modern hospital industry, which today accounts for one-third

of all healthcare spending, or about six percent of gross domestic product.³ In related work, Finkelstein (2007) finds that the introduction of Medicare in 1965 led to a dramatic expansion in the hospital system. Chung, Gaynor, and Richards-Shubik (2017) find that subsidies introduced as part of the Hill-Burton program allowed hospitals to expand through the 1950s and 1960s. We contribute to this literature by focusing on an earlier, formative period in the hospital industry and by studying the effects of a health shock.

Finally, our paper also relates to a new literature that revises our understanding of the effects of the Great Influenza.⁴ Recent papers study how non-pharmaceutical interventions influenced economic activity (Correia, Luck, and Verner 2020; Lilley, Lilley, and Rinaldi 2020), how school closures affected educational attainment (Ager et al. forthcoming), how the pandemic affected the business cycle (Bodenhorn 2020; Barro, Ursúa, and Weng 2020; Dahl, Hansen, and Jensen 2021) and trust levels among survivors (Aassve et al. 2021), how political preferences changed following the crisis (Blickle 2020), and whether *in utero* exposure to the flu had lasting consequences (Beach, Brown, et al. 2022). We contribute to this literature by analyzing whether the GI altered preferences for healthcare provision, and whether the severity of the GI also increased local public health spending.

II. The 1918 Global Influenza and Medical Care in the U.S.

When the influenza pandemic struck the U.S. in 1918, hospitals were just assuming their modern forms as places people would go to relieve acute illness or to have surgery. Prior to this transition, which began toward the end of the 19th century, the public had a negative perception of hospitals. Early hospitals emerged from almshouses and institutions that had wards to care for sick patients. The first hospitals formed in urban areas to care for sick patients who were viewed as the “worthy poor,” such as hard workers lodging in boarding houses (**Rosenberg**). Following English tradition, these early hospitals were funded by donations or sponsored by charitable organizations. The first hospital in the United States, the Pennsylvania Hospital, was founded in 1751 by Benjamin Franklin and physician Thomas Bond. According to Franklin, a hospital

³Our research thus complements pioneering historical studies of the rise of US hospitals, in particular Rosenberg (1987) and Stevens (1989).

⁴See Beach, Clay, and Saavedra (2022) for a survey.

provided a means of caring for visitors seeking advice from Philadelphia's physicians as well as for caring for "...the poor Inhabitants of this City, tho' they had Homes, yet were therein but badly accommodated in Sickness, and could not be so well and so easily taken Care of in their separate Habitations, as they might be in one convenient House" (Franklin 1754, p. 3). In truth, medical science could do little, but patients could benefit from rest, warmth, and food. With a limited number of beds, early hospitals only admitted the "morally worthy" (Rosenberg 1987, p. 23). Prostitutes, alcoholics, and people suffering from contagious illnesses and cancer who could not be cared for at home were treated in almshouses.

Even though medical care was still ineffective (drugs to deal with bacterial infections or viruses did not exist and surgery often had poor outcomes due to infection), industrialization and urbanization after the Civil War led to an increase in the number of hospitals because workers living in cities were often boarders, with no one to care for them if they became sick or injured. In addition, working-class families often needed the income of more than one family member to survive, so nursing care may not have been available.⁵ Like Franklin and Bond over 100 years before, community-minded philanthropists and religious orders began to build hospitals to provide care for "...respectable and otherwise deserving" poor Americans who found themselves sick or injured and who could not care for themselves at home, and municipal hospitals for indigent patients evolved from the medical wards of almshouses. (Rosenberg 1987, p. 103).⁶

Early American hospitals primarily cared for indigent and working-class patients while middle- and upper-class people continued to receive care in their homes until after the turn of the century. Their willingness to go to hospitals for medical treatment evolved more slowly. A number of supply-side pressures and an increasing awareness of the benefits of the scientific revolution in medicine appear to have been important in altering middle and upper-class preferences. On the supply-side, it was not until the late-19th century that many physicians trained or worked in hospitals. Prior to this, most physicians trained in the United States received a very poor medical education. Medical schools did not require undergraduate degrees, and medical students

⁵Common treatments for infection included enemas, topical rubs, and phlebotomy (Smith, Watkins, and Hewlett 2012).

⁶Rosenberg (1987, pp. 101–09) provides greater discussion on the rise of these hospitals.

did not typically engage in laboratory or clinical work (Ludmerer 1985). By contrast, physicians and scientists at European universities were pushing the frontiers of medical science, including major discoveries in areas such as germ theory and pathology. As a result, American physicians who trained in Europe returned home and became leaders in the reform of medical education — developing curricula that emphasized science, laboratory work, and, most importantly, clinical work (Field 1968). Johns Hopkins set a new standard in 1893 when it became the first medical school to require an undergraduate diploma for admission and elevated the importance of clinical education by building a teaching hospital (Ludmerer 1985). The American Medical Association (AMA) disseminated these innovations as best-practice standards in medical education, and in the first decades of the 20th century, states began to require these for medical licensing.⁷

In addition to receiving training from hospitals, physicians could only take advantage of some of the newer technological breakthroughs in hospitals. For example, the first X-ray machines (1897) were large and not portable (Howell 2016). Improved surgical practices were also more easily implemented in the controlled environment of modern surgical suites than in homes. These included using aseptic techniques in surgery and the utilization of newer, more effective disinfectants than carbolic acid (Blevins and Bronze 2010).⁸ Hospitals provided physicians with on-call nursing assistance and pathology laboratories. Physicians came to prefer hospitals to perform surgery and treat serious illness, and the size of general hospitals increased over time as hospital administrators sought to take advantage of economies of scale.

Furthermore, major discoveries, such as Pasteur’s development of a vaccine for rabies (1885), Behring and Kitisato’s antitoxin for diphtheria treatment (1891), and salvarsan for the treatment of syphilis (1910) provided the public with growing evidence of the value of medicine. The therapeutic effectiveness of medicine was still relatively limited: doctors could now more accurately diagnose

⁷See Moehling et al. (2019) for further discussion and Fernández (2021) for similar developments in European hospital care.

⁸Surgery began to take advantage of Koch’s work in 1876 (building on Lister’s earlier discoveries about asepsis), who demonstrated the role of bacteria in infection and convinced many surgeons to use aseptic techniques in surgery. With aseptic techniques, surgery became safer, but was still high-risk; one study in Britain, for example, noted an 8.1 percent mortality rate for cesarean section between 1906 and 1910. In the United States, mortality ranged between three percent in selected obstetric units and 13 percent in community hospitals between 1920 and 1930 (Low 2009). All the while, more surgeries were performed in patients’ homes than in hospitals until around 1920 (Rosenberg 1987).

illness, remove inflamed appendices and infected tonsils, set broken bones, and discuss hygiene, but they could not cure infectious diseases, treat patients using intravenous therapy, nor use cardiopulmonary resuscitation. Despite this, the increasingly scientific nature of medicine started to shift public opinion in favor of hospitals, at least for surgery. World War I led to further discoveries, such as blood transfusions (Barr et al. 2019), and the popular press reinforced the notion that hospitals could save lives. For example, a 1916 *New York Times Magazine* headline read “Miracles of Surgery on Men Mutilated in War.”⁹

The 1918 influenza pandemic may have further contributed to this shift in public opinion by revealing the inadequacy of existing healthcare facilities, particularly in communities that were hard-hit by the virus. The pandemic was caused by a highly contagious H1N1 influenza virus that induced severe respiratory distress.¹⁰ The state of medical technology in 1918 meant that care for infected individuals was limited to nursing care, but with an estimated 25 percent of Americans contracting the virus, the sheer number of victims quickly overwhelmed hospitals. Henry A. Christian, the physician-in-chief at Peter Bent Brigham Hospital in Boston noted that the hospital could not handle the load of influenza patients, stating:

The hospital coöperated with the Board of Health and took, in the main, cases selected by them – patients who could not be cared for at home, or those in almost dying condition that it was necessary to get out of their homes to ease the problem of home management of less seriously sick ones. Many died in a few hours after being brought to the hospital. The wards were filled with patients extremely ill with the pneumonia that accompanied influenza (Christian 1915-1918).

Contemporaries reported that staffing shortages of nurses and doctors (in part due to World War I), and limited hospital capacity further hampered the ability to respond to the pandemic (Clay, Lewis, and Severnini 2018; Jester et al. 2019). Consequently, the fast-spreading infectious disease overwhelmed medical facilities (Guimbeau, Menon, and Musacchio 2020; Ojo 2020; Crosby 2003; Byerly 2010). Emergency temporary hospitals were created in schools, large halls, and even outdoors in “open air” hospitals (Crosby 2003). In about 30 days, cumulative death rates increased

⁹January 16, 1916, p. 6.

¹⁰Little consensus has emerged on its underlying causes. For a discussion, see Crosby (2003), Kolata (2001), Brainerd and Siegler (2003), and Huntington (1923)

from 1.3 to 100 deaths per 100,000 (Lin and Meissner 2020).¹¹

In many cities across the country, private entrepreneurs, community-based initiatives, public authorities and physicians started promoting the expansion of hospital capacity. Several of these initiatives explicitly cited the pandemic as the impetus. For example, in 1921, a group of forty-five physicians incorporated as an association in Sacramento, CA, to promote the construction of a new modern hospital in the city. To raise funds, the Sutter Hospital Association reminded the community that:

For a long time the people of Sacramento have keenly felt the need of better hospital facilities in this vicinity...During the great influenza epidemic of 1918 Sacramento was in a desperate condition for want of a place to take care of the large number of sick and dying victims. During normal times our present hospitals are crowded to capacity—in abnormal times often wretched makeshifts are even inadequate. (*The Sacramento Bee*, October 11, 1923, S.3.)

The association between the pandemic and the need for more hospitals was probably felt keenly in this Californian city, where the mortality rate from the pandemic (9.7 per thousand) was significantly above the national average of 7 per thousand.¹² In Nashville, TN, another city heavily exposed to the pandemic (9 deaths per thousand), the Baptist community organized to open a new hospital. The *Nashville Tennessean* reported in 1920 that hospital advocates similarly justified their efforts as a response to the influenza pandemic:

For many years the people of Nashville have suffered for lack of adequate hospital beds and facilities ...further intensified by the presence of influenza: applicants for hospital treatment and service were being turned away from the few public and private institutions of the city possessing such facilities, literally by the hundreds. (*Nashville Tennessean*, June 6, 1920, p. 5.)¹³

Even in cities less affected by the pandemic there was a sense of civic pride in the construction of new hospitals. In Houston, TX (5.4 deaths per thousand), the local press hearkened back to the pandemic while hailing the simultaneous development of four new hospitals in 1923:

¹¹Over the span of two years, the Great Influenza (GI) killed an estimated 39 million people (675,000 in the U.S.), with a mean global death rate of 2.1 percent (Barro, Ursúa, and Weng 2020). Low-end estimates put the figure at around 20 million, which is still 2.5 times more than combat-related deaths from World War I (Royde-Smith and Showalter 2020).

¹²When the Sutter Hospital opened in 1923, it increased Sacramento's hospital bed capacity by one quarter. From these promising beginnings, the Sutter association continued to grow and is now one of largest healthcare providers in Northern California.

¹³The Saint Thomas Midtown Hospital, as it is now known, is currently the largest nonprofit hospital in the city.

Great disasters, widespreading epidemics—they come infrequently, but they come. In late years they have found Houston utterly unable to cope with them ...Not only were the few Houston hospitals helpless before unusual calls on their facilities, but with the swiftly growing city and its developing tributary county they were overtaxed ...The building now under way will put Houston’s agencies of mercy on a level with the city. (*The Houston Post*, July 1st, p. 34)

This need for permanent hospitals was highlighted even while the pandemic was ongoing. In October 1918, an editorial writer in Greenville, North Carolina bemoaned that a prior effort to fund a hospital failed, and dedicated himself to advocating for a future hospital project (which would eventually succeed in 1924):

I am simply calling to the attention of the voters of Pitt County, the mistake we made when we had the privilege of voting to establish a Community Hospital in Pitt County. ...If we had voted for the building of this hospital, in my opinion it would have meant the saving of many lives. We would have had sufficient time to have built and equipped the Hospital, and at this time would have had it completely filled with Influenza patients, where they could have had the proper sanitary and medical attention ...Even though the crisis has passed, we have no assurance that this same condition will not exist again, so let us made haste and establish a Community Hospital. (*Greenville Daily News*, October 25, 1918, p. 1)

Similar narrative accounts are prevalent throughout the U.S. and suggest that preferences for healthcare may have changed in response to the influenza pandemic. We now turn to testing this hypothesis more formally by constructing a data set that covers 466 U.S. cities.

III. Data and Summary Statistics

We combine city-level data from several sources to examine the relationship between pandemic intensity, healthcare provision, and expenditures on public services and infrastructure over the period 1909 to 1975.

First, we gather data on hospitals to measure healthcare provision. No single source provides consistent information on hospitals over our sample period, so we combine data from three different sources. From 1909 to 1923, we digitize the *American Medical Directory* (AMD), which reports the name and location of the hospital, the ownership type (e.g., private or public), and the number of beds.¹⁴ Starting in 1925, we digitize data collected by the American Medical Association’s

¹⁴These files are available for 1909, 1911, 1912, 1916, 1918, 1921, and 1923.

Council on Medical Education and published in the *Journal of the American Medical Association*; it includes similar data fields as the AMD.¹⁵ We also draw on American Hospital Association (AHA) directory data for 1948 to 1975 compiled by Finkelstein (2007), which similarly provides the name and location of hospitals, ownership status, and number of beds. We geocode all three sources using the Google Maps API to address misspelling and OCR errors. This process creates a consistent measure of place that we use to aggregate our hospital level data into a city-year panel.

We use both hospital counts and bed numbers by city-year as our outcomes. Our baseline definition of a hospital (and its beds) excludes institutions that were either not built in response to local demand or that served distinct sub-populations that lie outside the scope of our study. For example, we exclude sanatoria, mental health facilities, asylums, and “recovery homes” from the baseline analysis. We also drop military hospitals, Federal Veteran’s Association facilities, and smaller facilities such as “infirmaries” and “clinics.”¹⁶ Figure 1 shows the growth of hospitals and hospital beds over this time period using our baseline definition of hospitals. Despite our data originating from three different sources, the resulting time-series are relatively smooth, and show an increase in both hospitals and beds over time. Figure 1 shows that the relative importance of governmental hospitals declined over our time period — government beds were nearly 30% of all beds in 1912 but fell to 22% by 1975.

Second, to measure pandemic severity, we use city-level death counts by year and cause from 1918 reported in the Department of Commerce – Bureau of the Census *Mortality Statistics*. This publication reports data on deaths for cities with populations greater than or equal to 10,000 and recorded in vital statistics registration areas. For our baseline measure of influenza mortality, we use the reported deaths from flu and pneumonia in 1918 per 1,000 residents.¹⁷ Figure 2 plots a histogram of pandemic death rates by city and illustrates that the pandemic’s effect was unequally distributed. Figure 3 displays the cities with mortality data, with darker dots corresponding to higher levels of mortality. It shows that there is no simple relationship between geography and

¹⁵These files are available for 1920, 1925, and yearly from 1927 to 1947.

¹⁶We show that our results are robust to more expansive definitions of hospitals.

¹⁷In order to avoid a changing denominator due to deaths in 1918, we use the population estimate from the 1917 *Mortality Statistics* to construct this number. We show results using an excess mortality calculation and additional years of mortality in the robustness section.

pandemic effects: even within states, there is often a large variation in death rates.¹⁸

Third, to analyze city-level government spending, we digitize annual data from the Department of Commerce, Bureau of the Census, *Financial Statistics of Cities Having a Population over 30,000*. This government serial contains detailed information on municipal revenues and expenditures, which we use to examine the local response to the pandemic of government spending through current expenditures (noted “payments” in the sources) and capital expenses (“outlays”). In particular, we use city statistics data from 1910-12, 1915-19 and 1921-29.¹⁹

Finally, we collect city-level covariates from the publicly available 1910 and 1930 U.S. censuses using IPUMS (Ruggles et al. 2021). From the 1910 census, these covariates include population, the share of the population that is Black, the labor force participation rate, the mean occupational score of workers, and the share of the employed population in each of the first-digit 1950 occupational groupings.²⁰ From the 1930 census, we gather information on the number of veterans in each city who served in World War I as well as whether these veterans worked as doctors or nurses. To supplement these time-invariant controls, we use a time-series of city population constructed by Schmidt (2018).²¹ Table 1 provides summary statistics for key variables used in our city-level analysis.

IV. Estimating the Effects of Pandemic Intensity on Hospitals

To measure the effect of influenza mortality on subsequent healthcare provision, we estimate a simple event-study model of the form:

$$Outcome_{it} = \gamma_i + \sum_{j \neq 1916} \beta_j Mortality1918_i \mathbb{1}_{j=t} + \epsilon_{it} \quad (1)$$

¹⁸In some versions of our empirical approach, we explicitly use this variation by including state fixed effects.

¹⁹No volumes were published in 1913, 1914, and 1920.

²⁰The occupation score is the IPUMS variable OCCSCORE, which is calculated using 1950 wages for each indicated occupation.

²¹Schmidt (2018) collects these data from multiple sources, but his primary source is scraped tables from city pages on Wikipedia. We interpolate population linearly between decennial census years.

where $Outcome_{it}$ is hospitals or hospital beds for city i in year t .²² $Mortality1918_i$ is a measure of city i 's influenza mortality rate in 1918 as defined earlier, and γ_i are city fixed effects. We allow the influenza impact to vary over time through an interaction with year fixed effects. Our key coefficients are in vector β_j and track the impact of mortality on outcomes over time. Throughout our analysis, we cluster our standard errors at the city level. We estimate models using several different measures of $Mortality1918_i$, with our baseline measure being an indicator for whether a city is in the top half of the mortality distribution. Alternative specifications consider additional cutoffs and continuous measures of mortality as our treatment variable.²³

Our identifying assumption is that there are no omitted variables correlated with both the severity of the 1918 pandemic and subsequent outcomes. While we cannot test this assumption directly, examination of the pre-1918 β_j coefficients is informative: a lack of pre-period effects suggests that there is no pre-existing trend relationship between outcome variables and pandemic mortality. However, our findings could still be affected by any omitted variable that is correlated with the severity of the pandemic and (like the pandemic) had an effect on outcomes after 1918. To explore this possibility, we also estimate the following model, which includes a vector X_i of city-specific controls measured in 1910 (the census preceding the GI):

$$Outcome_{it} = \gamma_i + \sum_{j \neq 1916} [\beta_j Mortality1918_i \mathbb{1}_{j=t} + \delta_j X_i \mathbb{1}_{j=t}] + \epsilon_{it} \quad (2)$$

where X_i includes the average age of the population, labor force participation, the share of the population that is Black, the average 1950 occupational score, and the share of the workforce in 1-digit 1950 occupational categories.²⁴ To the extent that these variables are correlated with both the pandemic severity and future outcomes, we would expect the β_j vector coefficients to change across specifications (1) and (2).

We also estimate versions of equation (2) that account for population by controlling for time-

²²To deal with zero values, we transform the outcome variables using the inverse hyperbolic sine (IHS) function.

²³Recent work has examined the properties of the standard continuous difference-in-differences and found that the comparisons that this approach makes are difficult to interpret (Callaway, Goodman-Bacon, and Sant'Anna 2021).

²⁴These categories are the share of workers in professional/technical fields, farmers, clerical and kindred, sales workers, craftsmen, operatives, service workers, farm laborers, and laborers.

varying log city population. These models test whether observed effects are driven by population growth in cities that were differentially hit by the pandemic. This would be the case, for example, if the pandemic were more severe in larger cities, larger cities in turn grew even larger after the pandemic, and there were a positive correlation between city growth and outcomes.²⁵

Figure 4 shows results from an event-study model based on equation (1), with the inverse hyperbolic sine of the number of hospitals as the outcome variable.²⁶ The treatment variable in these regressions is an indicator for whether a city was in the top half of the 1918 pandemic mortality distribution. The outcome variable in Panel A of Figure 4 is all hospitals in our data. The two lower panels of this figure display results for relevant subsets of the hospital universe: Panel B includes only non-governmental hospitals while Panel C is limited to government hospitals. All three figures display the coefficients from the vector β_j in equation (1). Estimated coefficients reflect the change in hospitals in cities in the top half of the mortality distribution relative to those in the bottom half of the distribution over time with 1916 as the omitted year.

All three panels of Figure 4 indicate that mortality was not related to the number of hospitals in a city prior to 1918. However, there is a positive relationship between mortality and local hospitals after 1918. In particular, starting in the early 1920s, being in the top half of the mortality distribution is associated with an approximately 10-percent increase in the number of hospitals in a city. Panels B and C of Figure 4 show that our results are driven by non-governmental hospitals, i.e., private hospitals that were either for-profit or non-profit hospitals. Panel B shows an increase of non-governmental hospitals of over 10 percent — a difference that persists through the 1960s before starting to erode. By contrast, the bottom panel shows that pandemic intensity was not related to government-run hospital growth.

These results suggest that preferences for public provision of hospital services did not increase in response to the pandemic. Cities increased their provision of healthcare facilities, but primarily did so through private hospitals, both of which catered to paying customers.²⁷ While it is difficult

²⁵We do not control directly for population in our baseline specification since it is plausible that population growth itself was affected by the pandemic. Our results are also similar if we use per-capita measures of hospitals instead of controlling continuously for log population.

²⁶Throughout, all results are similar if we instead use the log of hospitals plus 1 as the treatment variable.

²⁷Given the features of our data, we cannot distinguish consistently between private for-profit and nonprofit hospitals.

to estimate the general equilibrium effect of the pandemic on the entire hospital industry given our empirical strategy, the pattern of these results is consistent with the declining role of direct public provision of healthcare in the United State over this time period. In particular, the nonprofit sector, which followed a model of charging patients fees, came to quickly dominate the market for “in hospital” healthcare. By 1935, a large majority of hospital beds were already accounted for by nonprofit institutions (White 1982).²⁸

Figure 4 shows that, after persisting through the 1950s, the effect of the pandemic on hospital provision wanes toward the end of our sample period. This persistence is unsurprising given the nature of the hospital industry. Per-capita hospital beds in a given city are serially correlated, suggesting that hospital investment is “sticky” once established.²⁹ Furthermore, since hospitals often serve areas outside of their immediate city boundaries, a hospital that establishes itself in an early period can crowd out future investors and maintain its position over time.

The timing of the decay in our treatment effects suggests an association with a number of federally-funded programs affecting local healthcare markets that were enacted and authorized for funding in the 1960s. Finkelstein (2007) showed that the enactment of Medicare in 1965 increased hospital beds nationwide by about 30 percent between 1965 and 1970. In a setting of imperfect health insurance markets and liquidity-constrained households, government-subsidized healthcare (Medicare and Medicaid) could have the effect of leveling-up the intensity of local demand for healthcare.³⁰ Alternatively, as the “memory” of the 1918-19 pandemic faded and hospitals became more organized, networked, and commercialized, it is plausible that cities began to converge to similar levels of provision due to competitive pressures and opportunities.

To summarize these event studies, Table 2 presents the results from estimating a difference-in-differences model comparing the average effect in the post-1918 period relative to the pre-period.

²⁸Initially, payment for hospital services was mostly out of pocket because private health insurance was very limited as a workplace fringe benefit until World War II (Thomasson 2002).

²⁹The correlation coefficient between per-capita beds in 1920 and 1960 in our sample is approximately 0.5; the correlation coefficient for per-capita hospital counts over the same time period is higher, at approximately 0.6

³⁰Another federal program also acted on the supply side of hospital care. The Hill-Burton Act of 1946 provided federal funds to equalize the availability of hospital treatment across the nation (Chung, Gaynor, and Richards-Shubik 2017). Although enacted in 1946, the funds allocated initially favored the construction of new hospitals in rural and underserved areas. For this reason, it is unlikely that the Act had an effect on our sample of U.S. cities. However, a 1964 amendment released funds for hospital modernization with a preference given to urban hospitals (Clark et al. 1980).

In particular, we estimate:

$$Outcome_{it} = \beta_1 Post_t + \beta_2 Post_t \times Mortality1918_i + \gamma_i + \delta_t + \epsilon_{it} \quad (3)$$

where $Post_t$ is an indicator for observations after 1919, γ_i is a city fixed effect, and δ_t is a year fixed effect. Table 2 indicates that, on average, hospitals increased by between 7-7.5 percent in cities in the top half of the mortality distribution relative to those in the bottom half of the mortality distribution, with that effect being driven by a 11-12 percent increase in non-governmental hospitals. There is no detectable impact on non-governmental hospitals.

V. Alternative measures of hospital access, healthcare provision, and pandemic severity

In addition to measuring how pandemic mortality affected the count of hospitals, we explore whether the number of hospital beds in a city increased. The number of hospital beds can increase both through the construction of new hospitals and the expansion of existing facilities. Moreover, using hospital beds as an outcome can capture size differences in hospitals that are built.

Panel B of Figure 5 shows results from estimating equation (1) using the inverse hyperbolic sine number of hospital beds as the outcome variable. Panel B of Figure 5 exhibits a similar pattern as our hospital results: the number of hospital beds rises in places where pandemic severity was greater — a result that remains statistically significant decades after the pandemic ended. In Figure 6 we show the same results for non-government hospitals. These results are more precisely estimated than the overall findings. Being in the top half of the mortality distribution is associated with an increase in hospital capacity of about 10 percent and an increase in bed capacity of about 20 percent.

The overall evolution of the healthcare industry is consistent with the pattern observed in the results for total hospital beds. During our sample period, the average size of U.S. hospitals increased from 100 beds in 1920 to 161 in 1975. This increase was driven by changes in the cost structure of healthcare provision that encouraged scale economies as well as changes on the demand side. New surgical and diagnostic techniques that were developed in the early-20th century required

large-scale capital investment as well as the recruitment of specialized staff. One study in 1930 suggested that the average cost of a hospital bed built in New York City was \$18,000, described as a “far cry from the estimate of \$167 per bed” made for a Boston hospital in the 1860s. At the same time, the growing acceptance of the hospital as the location for care provided a demand-side channel that reinforced the tendency for larger scale (White 1990). As a consequence, healthcare in the U.S. moved away from the ‘cottage industry’ model of solo practitioners that had prevailed until the late-19th century. To the extent that the pandemic induced demand for new hospitals, it is likely that these hospitals would have been larger and more modern, creating a proportionately larger effect on beds.³¹

Panels C of Figures 5 and 6, respectively, show results focusing exclusively on the extensive margin — whether a city-year has any hospitals. These results are less precisely estimated than our hospital and bed results, but do suggest that there was an increase in the probability of observing a hospital in a city after the pandemic in locations where there was greater mortality from the pandemic.

We also consider alternative methods of specifying our treatment variable. Figure 7 shows a version of equation (1) estimated with a continuous measure of mortality as the treatment variable. In particular, these figures show the effects of a one-standard-deviation increase in city-level 1918 mortality on each outcome. We observe generally similar patterns of effects, though effect sizes are smaller when using this continuous specification since a one-standard-deviation change is smaller than the difference between the top and bottom half of the mortality distribution. We summarize these event studies for non-government hospitals with alternative right-hand-side variables in Table 3.

VI. Heterogeneity and mechanisms

The fact that non-governmental hospitals grew in response to pandemic intensity but government-run hospitals did not raises questions about how these changes may have affected access to health-

³¹Next to these internal economies of scale, there were also some external economies favoring horizontal and vertical integration. These included savings in management costs and in access to capital, monopoly power, and regulatory changes; however, the merger and integration wave in U.S. healthcare happened in the 1980s and 1990s, after the period we analyze (White 1990; Gaynor and Haas-Wilson 1999; Cutler and Scott Morton 2013; Gaynor, Ho, and Town 2015).

care. In this section, we explore a variety of city-specific characteristics to understand who benefited from the growth in hospital supply and why we observe little change in governmental hospital activity in response to pandemic severity.

First, we confirm that our government null results are not specific to our sample of hospitals by looking at an alternative data source for city-level outcomes. In particular, we estimate versions of equation (1) with city-level measures of government spending as our outcome variables. As noted above, we digitized new information on city-level spending using the Department of Commerce, Bureau of the Census, *Financial Statistics of Cities Having a Population over 30,000* for years between 1910-1929. Given that the Commerce Department only collected these data for cities of 30,000 people or greater, these estimates are based on a smaller sample of 185 cities. We estimate our baseline model, comparing local government spending on healthcare before and after the pandemic as a function of pandemic intensity. We focus on presenting results for two types of government spending that are most closely related to our analysis: on local health departments and related illness-prevention activities (Figure 8) and investment spending on government-supported city hospitals (Figure 9).³² We see little evidence that pandemic severity is related to either health or hospital government spending in the years after the pandemic, consistent with the null results that we find in the hospital data. These results hold when using either a discrete or a continuous treatment.

To explore heterogeneity, we estimate our baseline model from equation (1) using the median value of city characteristics to split the sample of cities. We start by comparing cities above and below the median population in 1917. Figure 10 shows the results for the baseline model for hospital beds. The two panels show that our results are mainly driven by smaller cities. It may have been easier for smaller cities to coalesce around the need for additional medical facilities after a crisis. In addition, smaller cities had less developed hospital systems before the pandemic, creating a larger opportunity for improvement. Cities in the bottom half of the population distribution had a median of 2.7 hospital beds per 1,000 residents before 1918; larger cities had 3.2 beds per 1,000, a 19 percent difference.

³²We also examined other forms of government spending and saw no evidence of a pandemic response.

Next, we turn to income heterogeneity. There is no consensus about the income elasticity of demand for hospitals or healthcare (OECD 2006). In the case of the U.S., recent studies have found elasticities of both below one (Acemoglu, Finkelstein, and Notowidigdo 2013) and above one (Hall and Jones 2007; Fogel 2009). Irrespective of healthcare being a necessity or a superior good, it is reasonable to expect that the impact of the shift in preferences studied in this paper could vary by income. To test for this, Figure 11 splits the sample of cities by median occupational score, where this variable is used to proxy for city income. Occupational score assigns each working resident from the 1910 Census the 1950 wage associated with their occupation, and is commonly used as a proxy for both individual and regional status (Sobek 1995). The results show larger effects for cities with higher occupational scores. This finding is consistent with the fact that our results are driven by a growth in non-governmental facilities. Since these facilities required out-of-pocket payments, it is unsurprising that we observe the largest responses in cities with higher incomes.³³

Finally, we check whether there is heterogeneity when we examine cities based on the share of the population that is foreign-born. Figure 12 shows results after splitting the sample above and below the median share of foreign-born. While the patterns of heterogeneity are not precisely estimated, it appears that effects are more prevalent in cities that have fewer foreign-born residents. It may have been easier in these cities to reach consensus on the value of new hospitals given their relative homogeneity of backgrounds and preferences, as has been seen in some studies on the prevalence of government-provided services (e.g., Alesina, Baqir, and Easterly 1999.) Table 4 provides a summary of these heterogeneity results generated from estimating equation (3), reporting the coefficients on the interaction between the post-period dummy and influenza mortality.

VII. Robustness Checks

To understand whether our results are sensitive to specification, sample, and measurement decisions, this section provides a number of additional tests. They are summarized in Table 5, which reports the average post-period effect coefficients generated from estimating equation (3) after making the indicated changes.

³³We obtain similar results when dividing the sample by average manufacturing wages in 1914 using available data for a smaller subsample of cities. These data come from Pawel Janas’s digitization of the 1914 US Bureau of the Census *Census of Manufactures*.

We first consider whether our findings are sensitive to the types of healthcare facilities included in the baseline analysis. To do so, we expand our definition beyond self-identified hospitals and include more specialized facilities — infirmaries, clinics, and sanatoriums. These were excluded from the baseline specification on the grounds that they may not capture a pandemic-induced change in preferences related to general hospital treatment (e.g., early in our sample period, sanatoriums were facilities used for the treatment of tuberculosis). Figure 13 indicates that our results are robust to including these additional types of medical facilities: the estimated coefficients are roughly the same size and remain statistically significant at conventional levels.

Second, we analyze whether our results are sensitive to our measurement of pandemic mortality. Figure 14 shows results for three alternative measures of mortality. Panel A shows results using 1918-19 pandemic deaths per person (instead of only 1918 deaths). Panel B shows resulting using an excess mortality calculation. To calculate excess deaths, we estimate city-specific trendlines for influenza and pneumonia deaths using pre-1918 data. We then predict a counterfactual number of deaths in 1918 that would have occurred but for the pandemic. We subtract this prediction from the actual number of deaths and divide by 1917 population to create a measure of pandemic severity. As shown in Panels A and B of Figure 14, the results are generally similar using either additional measure of pandemic severity. Panel C splits the 1918 mortality rates for cities into thirds rather than halves. The grey line indicates the treatment effect for cities in the top third of the mortality distribution relative to cities in the bottom third, while the darker black line represents a comparison between the first and second thirds of the distribution. This figure shows that the difference in outcomes are similar as we move from the first to the second and the second to the third terciles of the mortality distribution, with the exception of the post-1950 period, where the effects of being in the second tercile of deaths converge to baseline levels quicker than in the third tercile.

Next, we turn to specification robustness for our baseline results. Figure 15 shows a number of specification checks for the non-governmental hospital count. Panel A adds a vector of 1910 city-level covariates interacted with year fixed effects to the baseline model. Panel B adds the vector of 1910 city-level covariates plus time-varying log city population. In both cases, results are similar

to our main results. Panel C includes state-year fixed effects. When we include state-year fixed effects, our results are less precisely estimated but similar in magnitude. Figure 16 repeats these robustness exercises with non-governmental hospital beds as the outcome variable. The results for beds are larger and more precisely estimated than the corresponding hospital count results.

A different concern is the potential influence of World War I on healthcare preferences, an event which coincided with the influenza pandemic and may act as a confounder. For instance, when military personnel returned from service, they might have hastened the spread of the virus. If their presence is correlated with subsequent hospital outcomes and pandemic severity, our results could be biased. Returning military personnel could also have direct effects on hospital construction and expansion through an increased demand for medical care or because they served as doctors and nurses in the military (and thus increased the supply of medical practitioners in communities). To test for these potential impacts, we use the 1930 population census to compute the share of World War I veterans as well as the share of veterans employed as doctors or nurses for each city in our sample.³⁴ We also include the logged distance from each city to the nearest county with a military training camp using data on camp locations collected by Ferrara and Fishback (2020).³⁵ We include these variables as additional controls by interacting them with year fixed effects in our baseline regression model. Panel D of Figures 15 and 16 indicate that the inclusion of WWI controls does not affect the size or statistical significance of our baseline count or bed results.

As discussed in Section 2, a main finding of our analysis is that non-governmental hospitals were the segment of the healthcare sector that responded to pandemic mortality. An alternative explanation for this finding is that hospitals may have inadvertently changed their classification in our sample because we rely on multiple data sources to classify hospitals as public/government or non-governmental. To ensure that our results are not driven by such reclassification concerns, Figure 17 shows results where we exclude any city that had a government hospital before 1923 from the sample since 1923 is the transition year in our sample from AMD to JAMA data. Even

³⁴We use 1930 since it was the first census after the pandemic to ask about veteran status.

³⁵We explored using distance to camps as an instrumental variable for flu mortality. We did not observe a consistently strong first stage. In addition, in our context, it is unlikely that distance to camps satisfies the instrumental variable exclusion restriction assumption: returning military personnel played an important role in the development of the medical system in the years after WWI.

when these cities are excluded, we still observe a large increase in non-government hospitals as a function of pandemic severity, suggesting that classification changes resulting from changing data sources within our sample period are not affecting the results.

Finally, to address concerns that our results are unrelated to the pandemic — and perhaps instead related to a general difference in healthcare around 1918 that was correlated with future expansion — we run a placebo test. We construct city-level death rates in 1918 due to cancer using the same census publications on causes of death and methodology that we used to construct influenza death rates. We then use cancer mortality instead of influenza as our main treatment variable. Figure 18 indicates that there is no relationship between cancer death rates in 1918 and either the number of hospitals (Panel A) or of hospital beds (Panel B), either before or after the pandemic. These null results for cancer suggest that our findings are not driven by preexisting differences in mortality for illnesses that were unlikely to change around 1918.

VIII. Conclusion

The Great Influenza pandemic of 1918-1919 represented the first major public health shock of the 20th century. Much like the COVID-19 pandemic, the influenza pandemic stressed the healthcare system, and public health authorities struggled to treat victims in makeshift accommodations. Beyond these immediate effects, the COVID-19 pandemic may result in lasting changes in the provision of healthcare, including an increased reliance on telehealth and remote patient monitoring.

In this paper, we use data from U.S. cities to explore both the short- and long-run effects of the influenza pandemic on the fledgling American hospital industry. We find that cities that experienced greater mortality during the Great Influenza added more hospital capacity in later years. At a time when technological advances and changes in training of physicians favored the centralization of healthcare in hospitals, the influenza pandemic reinforced this trend by changing the public's preferences, particularly in cities where the pandemic exposed more clearly the deficiencies of local healthcare. At the start of our period, hospitals were seen as dangerous places for interning the poor or contagious. Gradually, they became perceived as the normal location for treatment, especially among middle-class patients. Ironically, it seems that two global pandemics

changed healthcare in the US in opposite directions — the first toward hospital care and the second away from it.

The differential effect of the pandemic persisted until the 1960s when the implementation of several federally-funded healthcare programs acted as another shock. We also find that these effects were unequal: less affluent cities added less capacity. The mixed nature of U.S. healthcare markets, combining public and private provision, allows us to distinguish the legacies of a large health shock on the preferences for public services and private treatment. Interestingly, we find that cities that added hospitals did so only in the fee-for-service, nonprofit and for-profit sector; pandemic intensity did not affect the provision of governmental hospitals. We verify our results by looking at whether city-level public health expenditures were affected by pandemic intensity. Consistent with our hospital results, we find that governmental expenditures on public health did not increase in cities that were more severely affected by the pandemic, suggesting that the public health shock of the influenza pandemic raised the demand for fee-based healthcare services rather than greater public healthcare provision.

Time will tell whether the COVID-19 health shock will also impart a comparable long-lasting legacy, but looking into the past is useful to identify what factors are likely to shape the form of that legacy.

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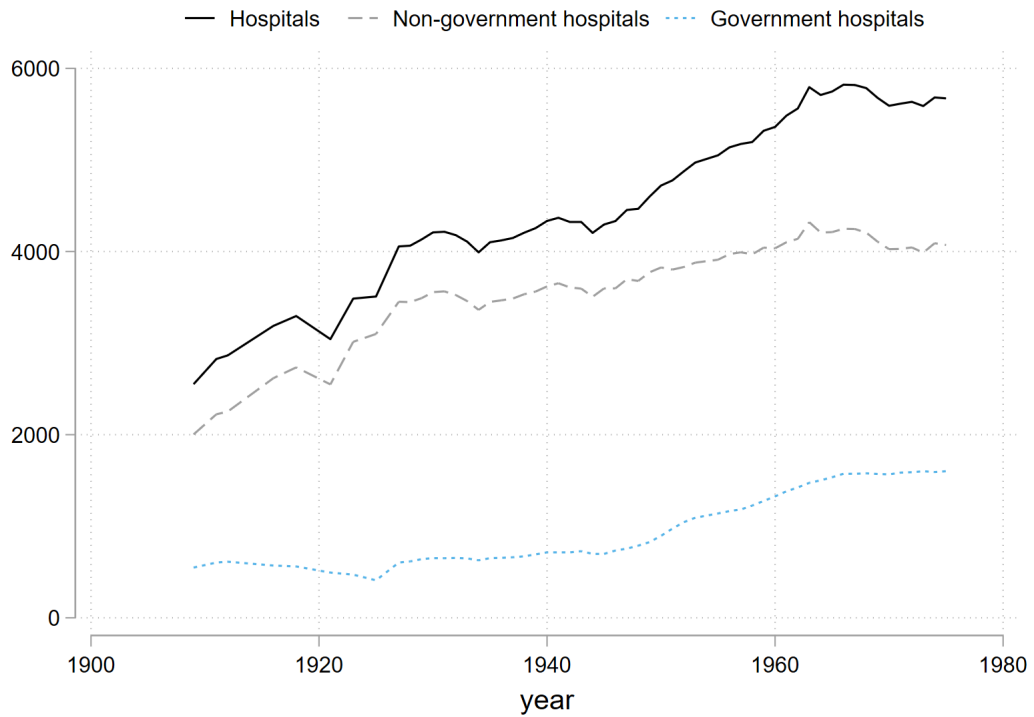
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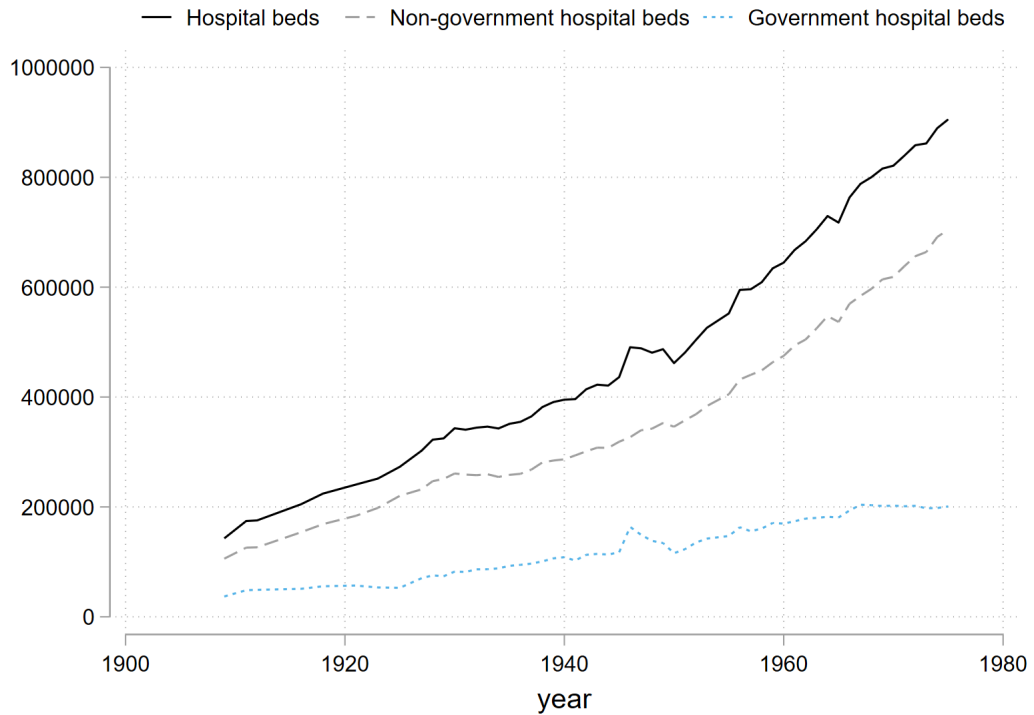
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Figure 1: Growth of hospital provision in the United States, 1909-1975

(a) Hospitals

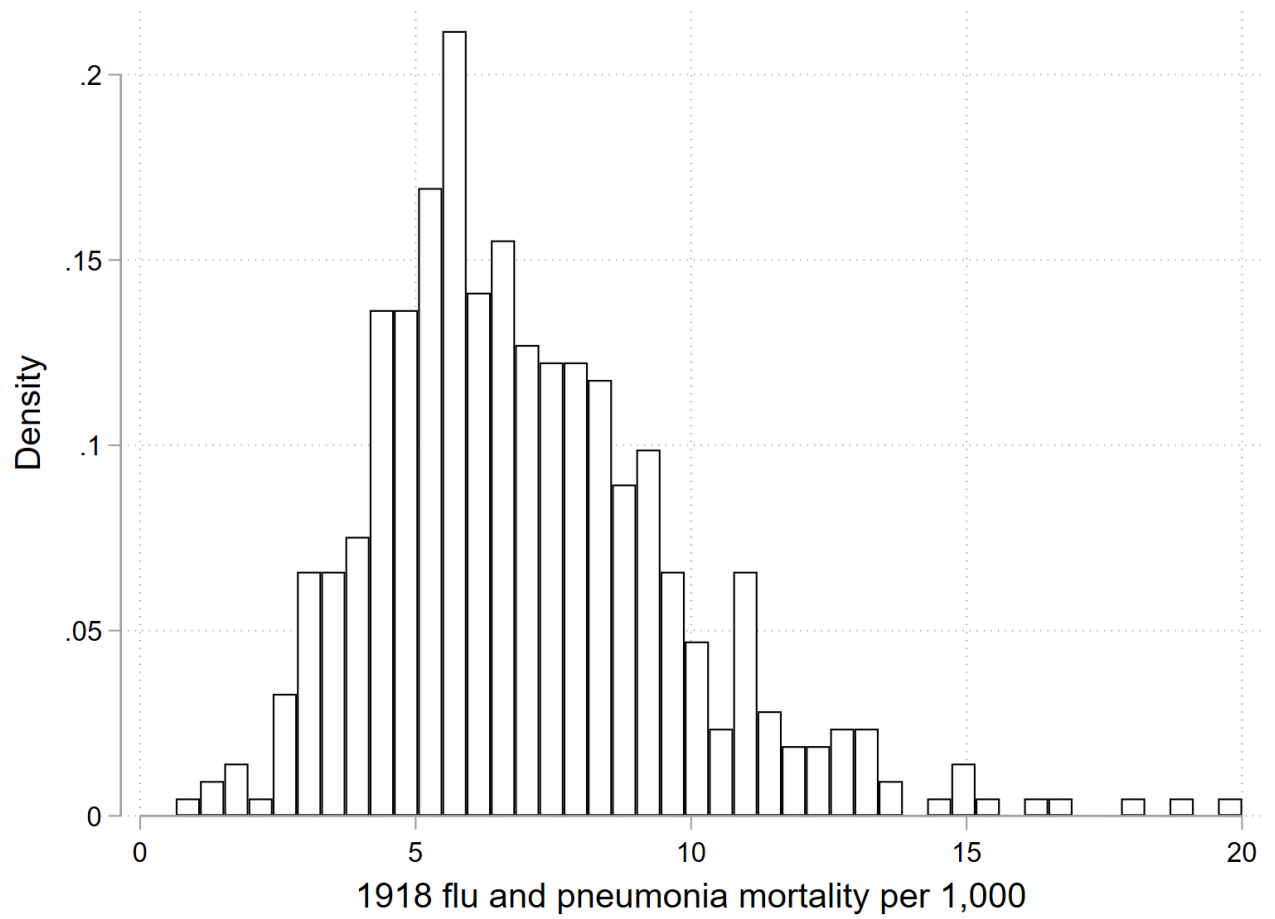


(b) Hospital beds



These figures show the growth of the number of hospitals (Panel a) and hospital beds (Panel b) in the United States from 1909-1975. The time series is limited to hospitals that provide general hospital services, and excludes specialized facilities such as mental-health hospitals.

Figure 2: Distribution of pandemic mortality across cities in 1918



Notes: This figure shows the distribution of mortality for our sample of 466 U.S. cities, with information on hospitals and 1918 flu mortality.

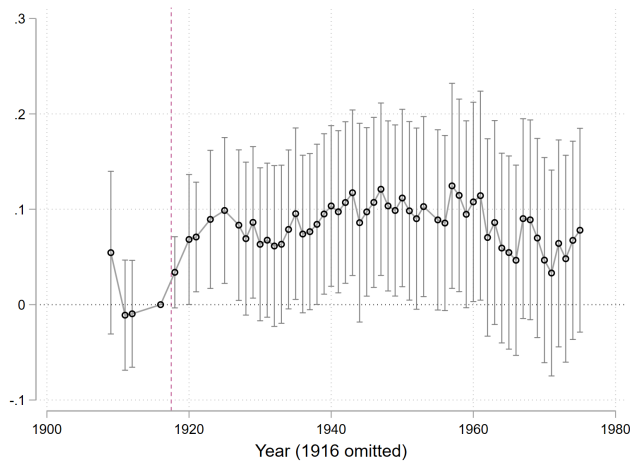
Figure 3: Geographic distribution of pandemic mortality across cities in 1918



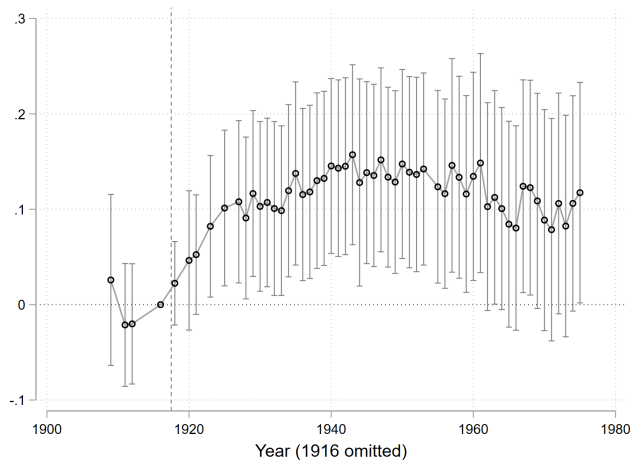
Notes: This figure shows the geographic distribution of mortality for our sample of 466 U.S. cities, with information on hospitals and 1918 flu mortality.

Figure 4: Effect of the 1918 flu pandemic on inverse hyperbolic sine number of hospitals

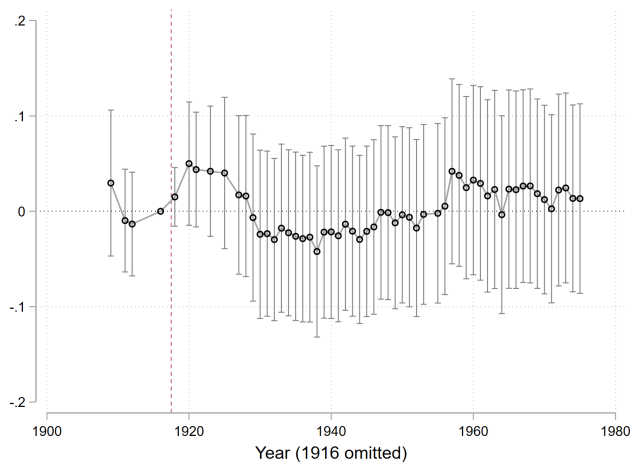
(a) All hospitals



(b) Non-government hospitals



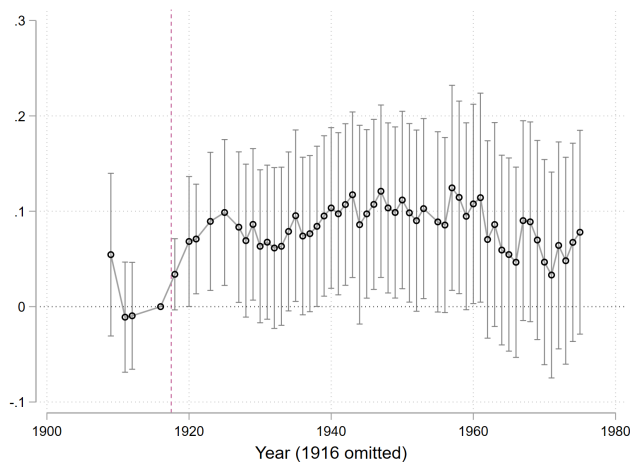
(c) Government hospitals



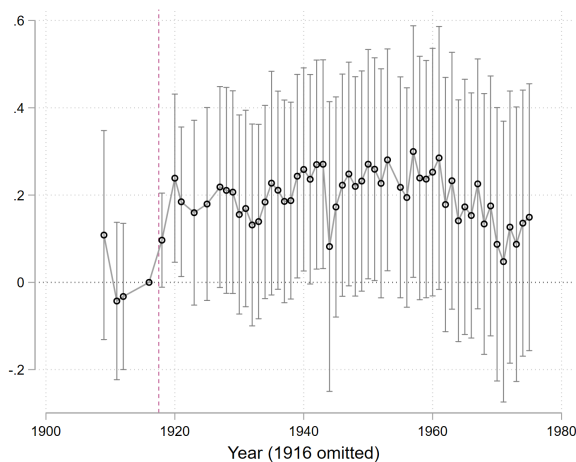
These figures show the effects of pandemic severity on the inverse hyperbolic sine of hospitals in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A uses all hospitals as the outcome variable, while Panels B and C respectively depict results using non-government and government hospitals as outcomes. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 5: Effect of the 1918 flu pandemic on alternative hospital measures

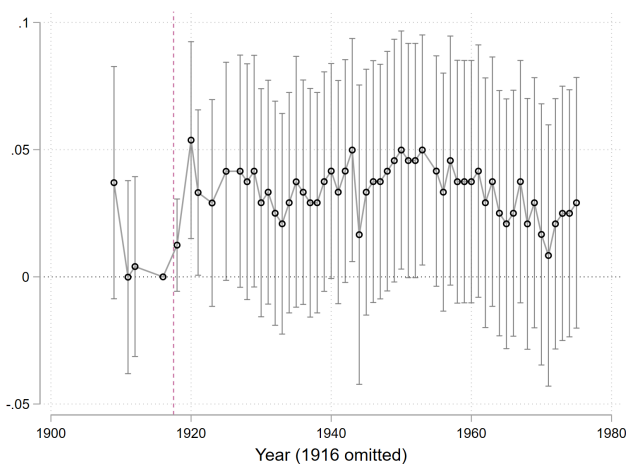
(a) Hospitals



(b) Hospital beds



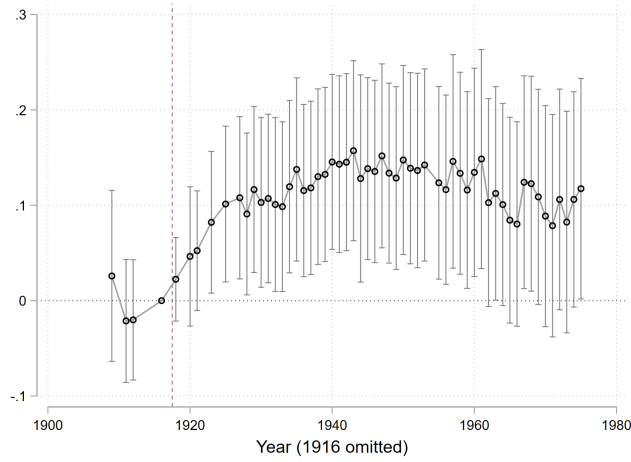
(c) Probability of having a hospital



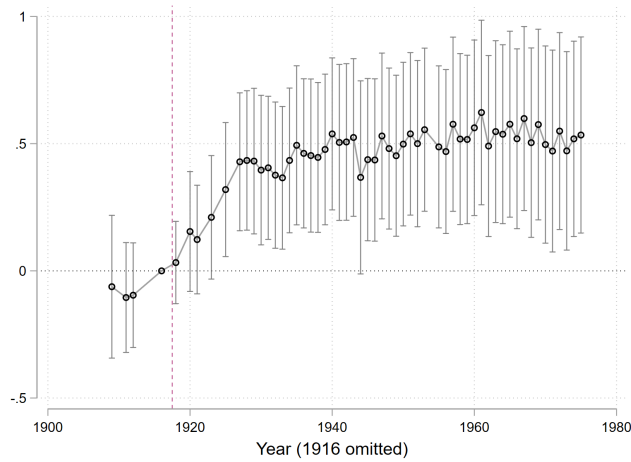
These figures show the effects of pandemic severity on alternative measures of hospitals in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A includes all hospitals as the outcome variable, while Panels B and C respectively depict results using hospital beds and the probability of observing a hospital as outcome variables. Hospital beds and hospitals are transformed using the inverse hyperbolic sine. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 6: Effect of the 1918 flu pandemic on the alternative non-government hospital measures

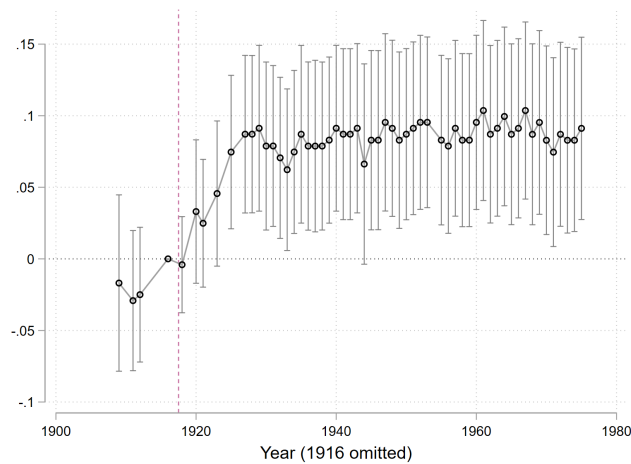
(a) Non-government hospitals



(b) Non-government hospital beds

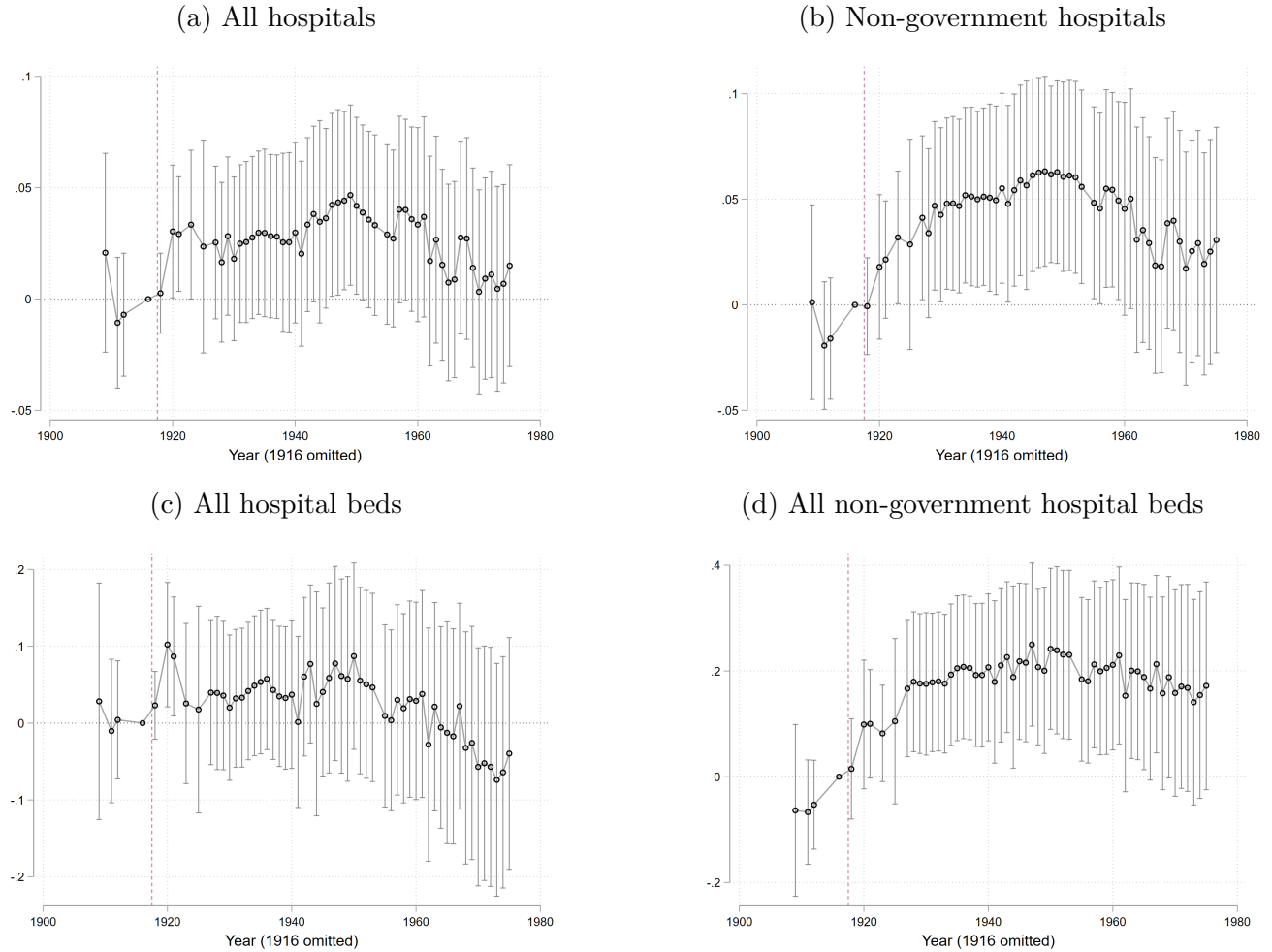


(c) Probability of having a non-government hospital



These figures show the effects of pandemic severity on alternative measures of non-government hospitals in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A includes all hospitals as the outcome variable, while Panels B and C respectively depict results using hospital beds and the probability of observing a hospital as outcome variables. Hospital beds and hospitals are transformed using the inverse hyperbolic sine. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 7: Effect of the 1918 flu pandemic on inverse hyperbolic sine hospitals and hospital beds (continuous treatment)



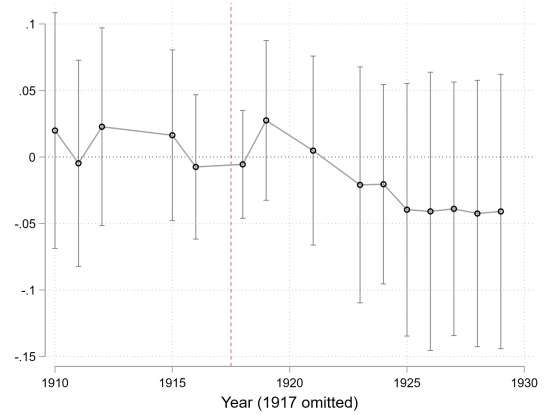
These figures show the effects of pandemic severity on hospitals and hospital beds in a given city, as estimated by the model in Equation (1). The treatment variable is a continuous measure of pandemic mortality normalized to show a 1-standard deviation increase in death rates. The outcome variables are labelled in each panel and presented using the inverse hyperbolic sine transformation. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 8: Effect of the 1918 flu pandemic on local government health spending

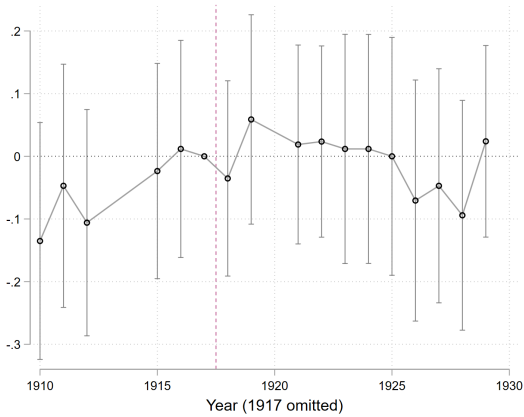
(a) Health spending, discrete treatment



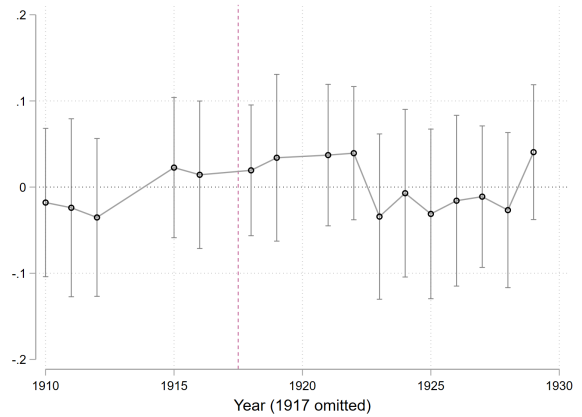
(b) Health spending, continuous treatment



(c) Had health outlay, discrete treatment



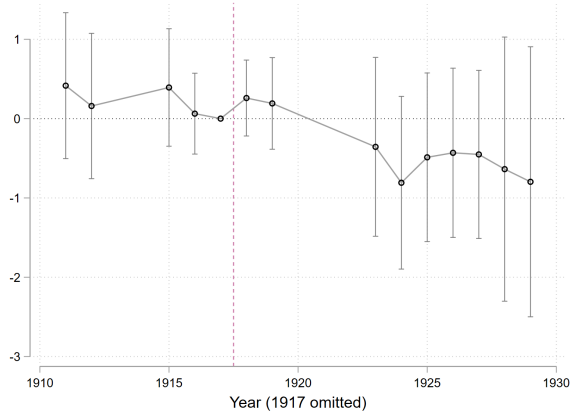
(d) Had health outlay, continuous treatment



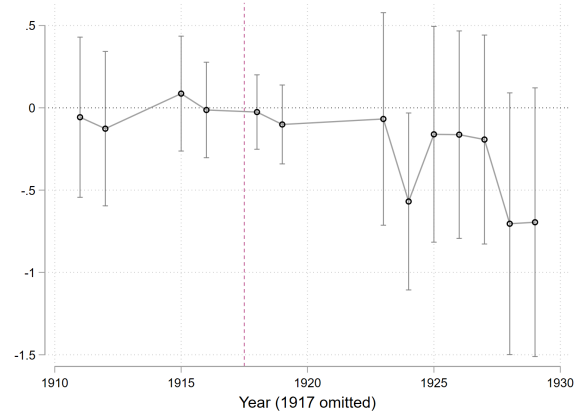
These figures show the effects of pandemic severity on alternative measures of government spending in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within this sample of cities or a continuous measure of mortality (measured in standard deviation units), as labelled in each panel. The outcome variables are labelled in each panel. Spending variables are transformed using the inverse hyperbolic sine. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 9: Effect of the 1918 flu pandemic on local government hospital spending

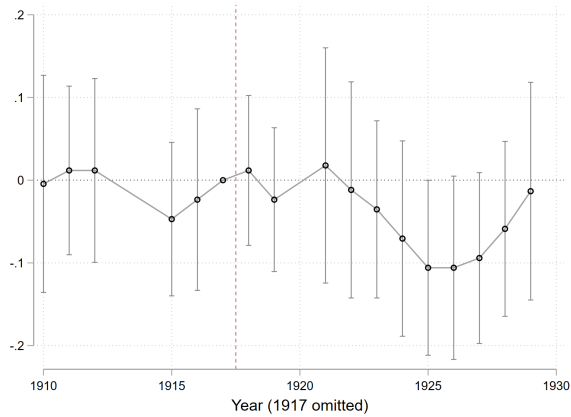
(a) Hospital outlays, discrete treatment



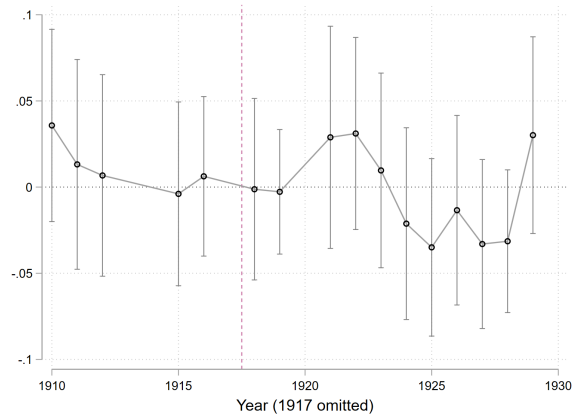
(b) Hospital outlays, continuous treatment



(c) Had hospital outlay, discrete treatment



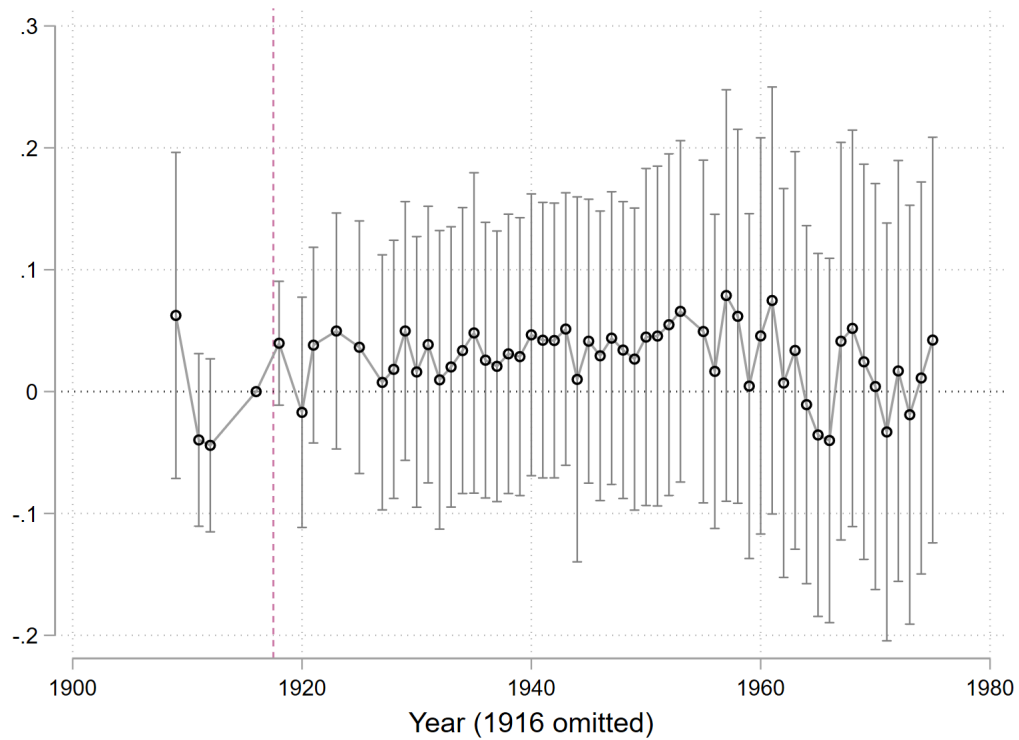
(d) Had hospital outlay, continuous treatment



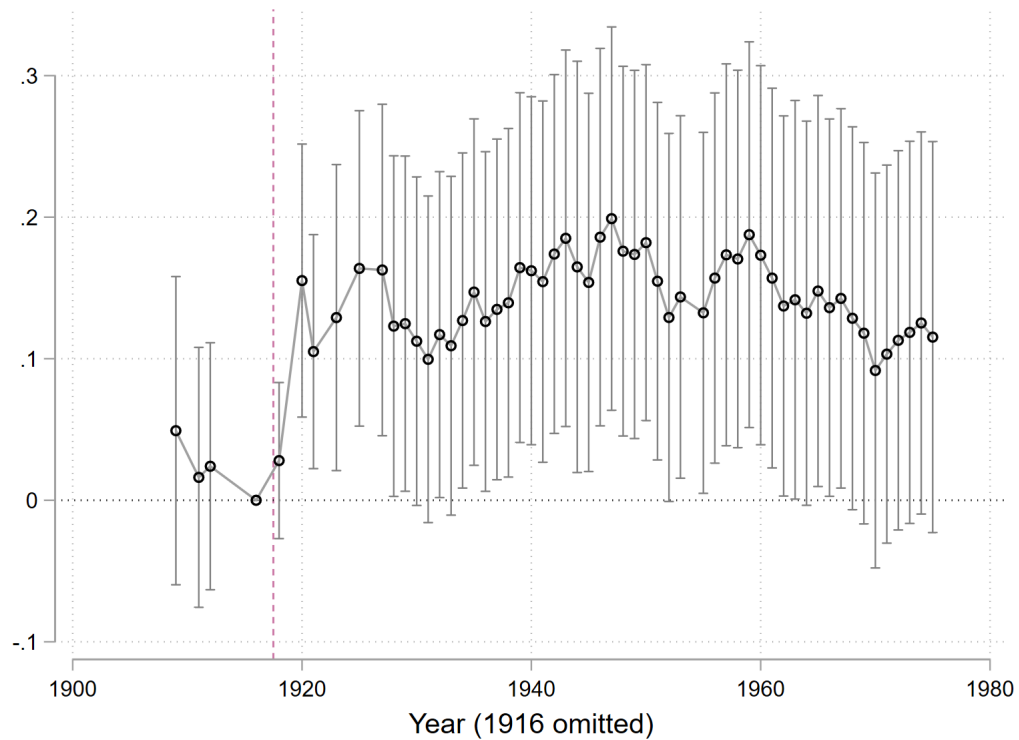
These figures show the effects of pandemic severity on measures of government spending in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within this sample of cities or a continuous measure of mortality (measured in standard deviation units), as labelled in each panel. The outcome variables are labelled in each panel. Spending variables are transformed using the inverse hyperbolic sine. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 10: Effect of the 1918 flu pandemic on hospital bed growth (heterogeneity by city size)

(a) Above median size



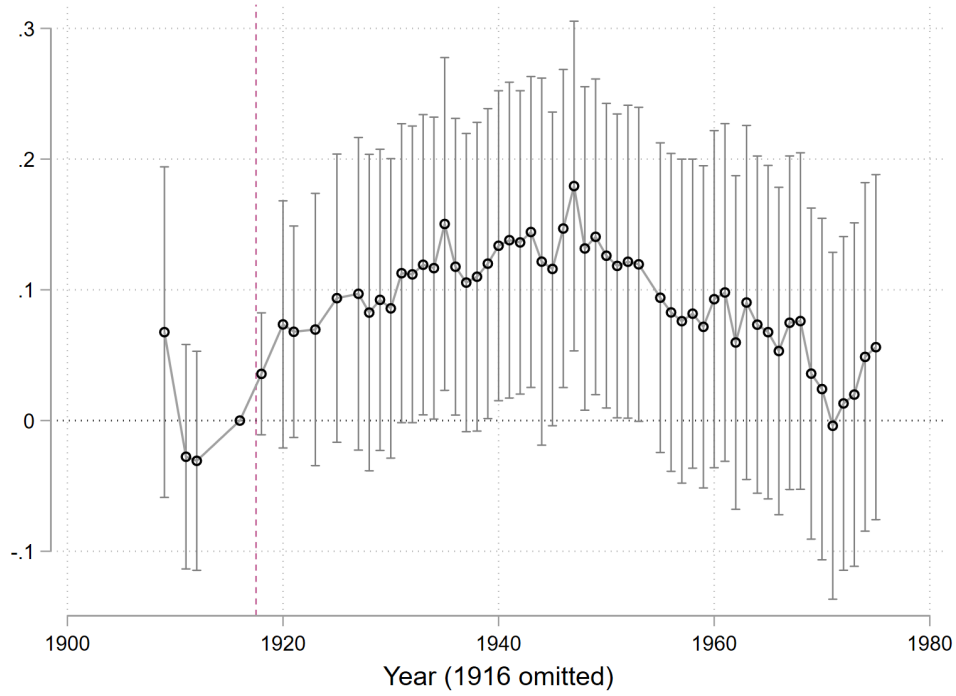
(b) Below median size



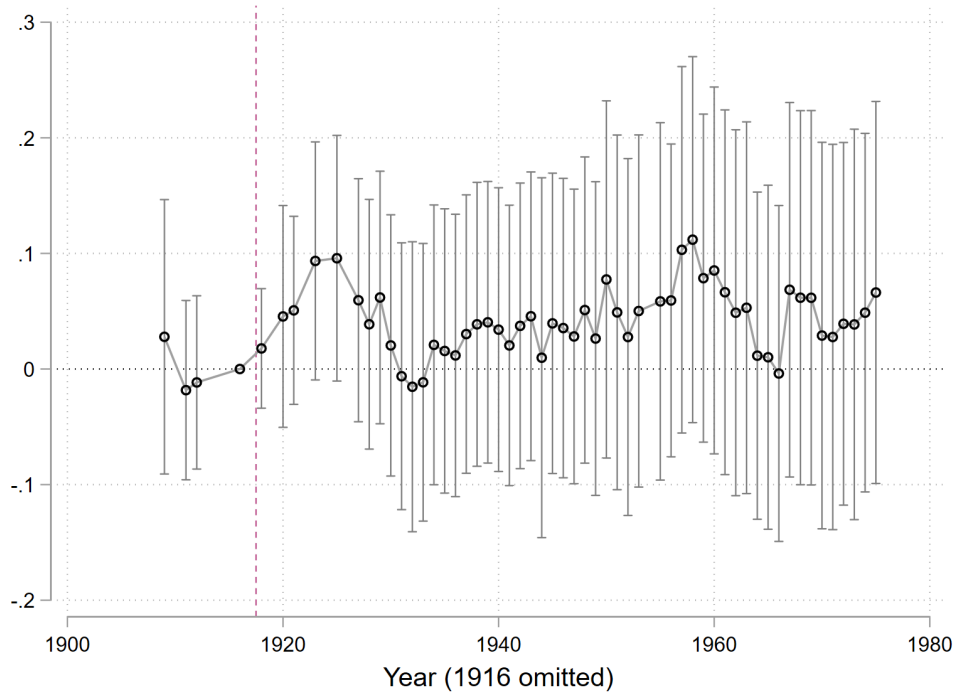
These figures show the effects of pandemic severity on the inverse hyperbolic sine number of hospital beds in a given city, as estimated by the model in equation (1). 95-percent confidence intervals are shown and standard errors are clustered by city. Panel A shows results for larger cities, panel B for smaller cities.

Figure 11: Effect of the 1918 flu pandemic on hospitals (heterogeneity by city occupation scores)

(a) Above median occupation score



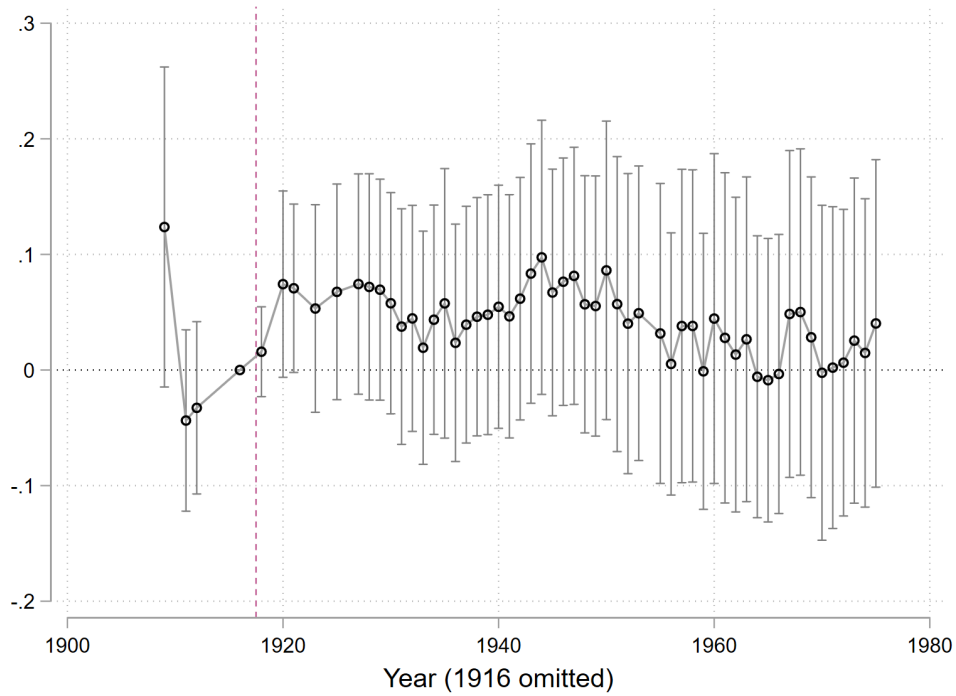
(b) Below median occupation score



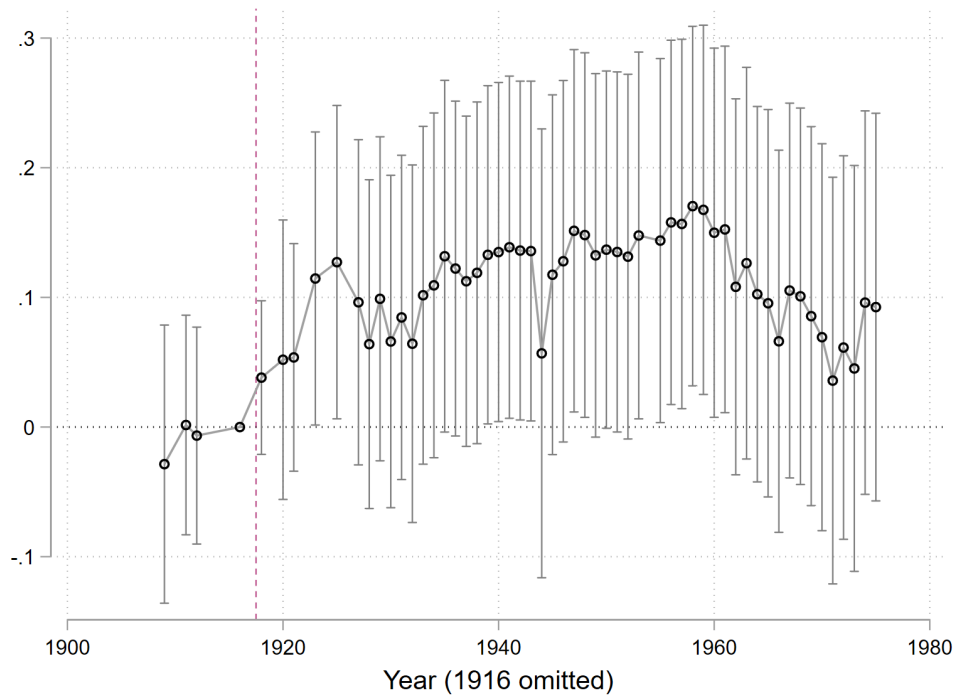
These figures show the effects of pandemic severity on the inverse hyperbolic sine number of hospital beds in a given city, as estimated by the model in equation (1). Panel A shows results for cities with above median occupation scores, panel B for cities with below median occupation scores. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within the overall sample of cities. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 12: Effect of the 1918 flu pandemic on hospitals (heterogeneity by city share foreign-born)

(a) Above median share foreign-born



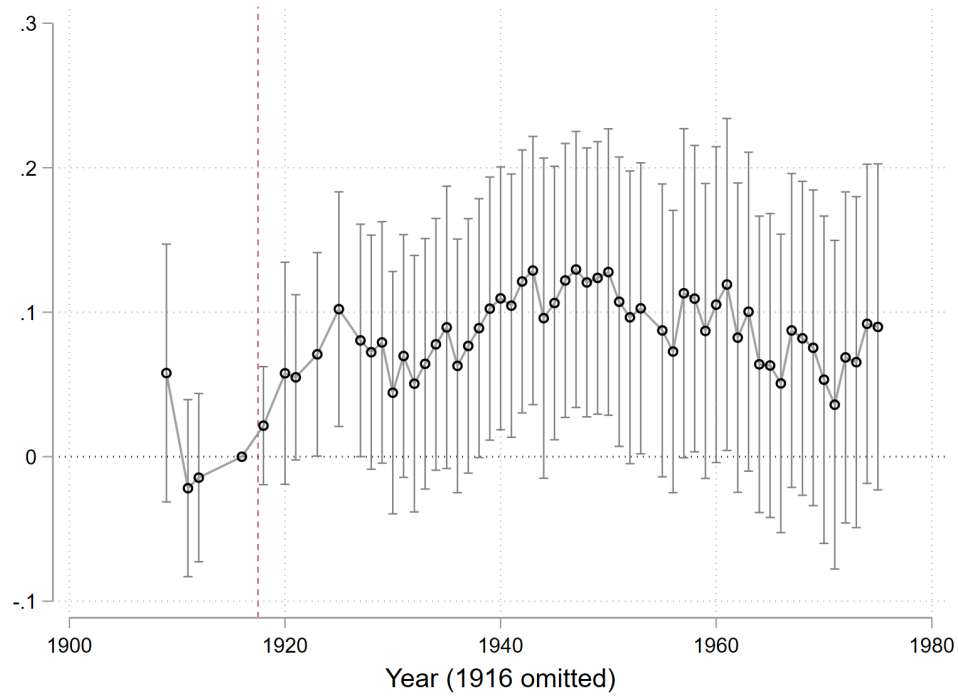
(b) Below median share foreign-born



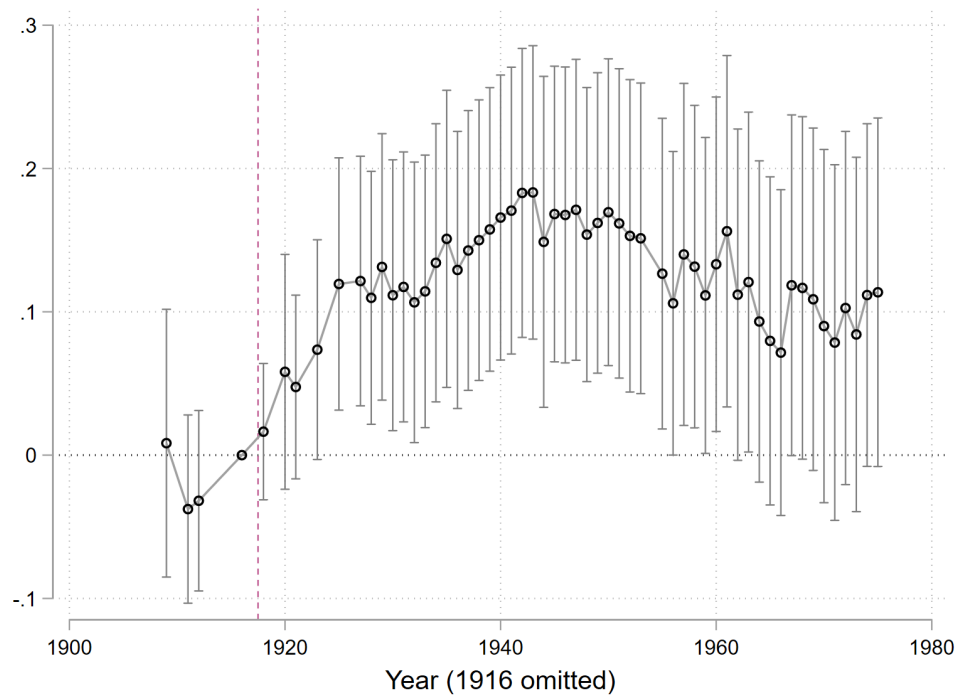
These figures show the effects of pandemic severity on the inverse hyperbolic sine number of hospital beds in a given city, as estimated by the model in equation (1). Panel A shows results for cities with above median share foreign-born residents, panel B for cities with below median share foreign-born residents. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within the overall sample of cities. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 13: Effect of the 1918 flu pandemic on hospitals (less strict hospital definition)

(a) All hospitals



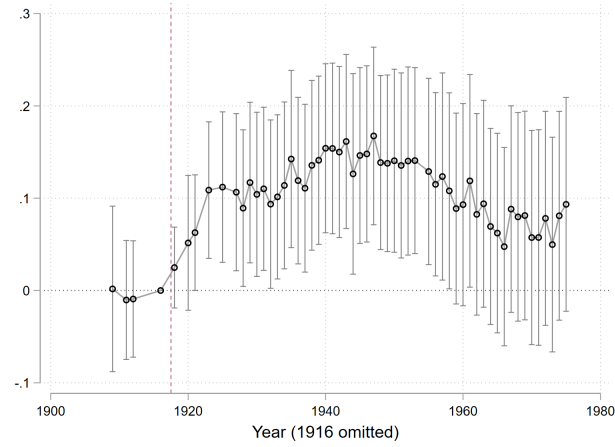
(b) Non-government hospitals



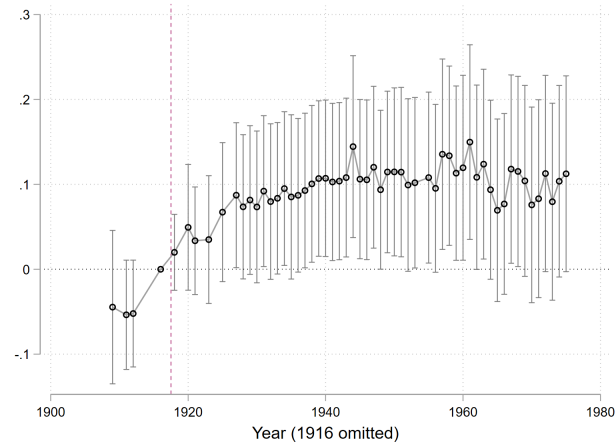
These figures show the effects of pandemic severity on the inverse hyperbolic sine number of hospitals and other medical facility beds like clinics in a given city, as estimated by the model in equation (1). 95-percent confidence intervals are shown and standard errors are clustered by city. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within the overall sample of cities.

Figure 14: Effect of the 1918 flu pandemic, alternative mortality measures

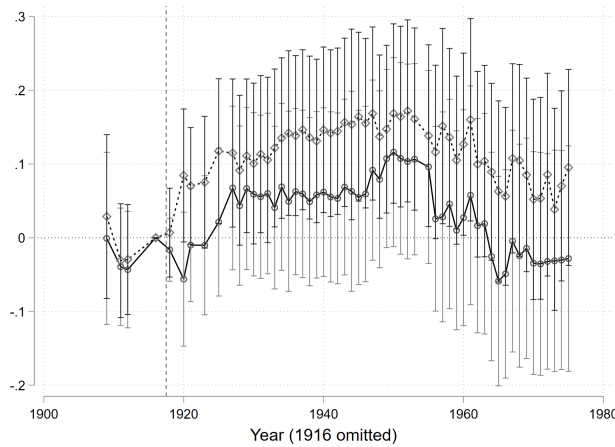
(a) Non-government hospitals, 1918-19 mortality



(b) Non-government hospitals, excess mortality



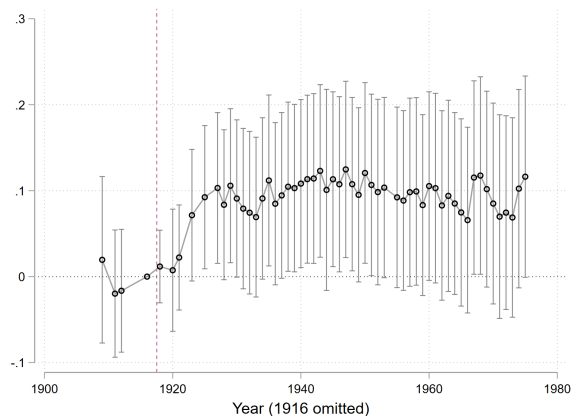
(c) Non-government hospitals, 1918 mortality thirds



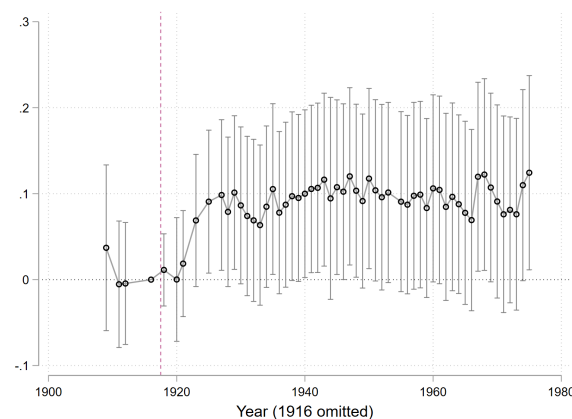
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A uses 1918-19 mortality to make this calculation, Panels B depict results using excess mortality calculated relative to the mortality trend before 1918 for each city, and Panel C shows results using 1918 mortality thirds instead of halves. The top third is shown in grey, the middle third in black. Effects are relative to the omitted third and 1916, the omitted year. Outcome variables are transformed using the inverse hyperbolic sine. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 15: Effect of the 1918 flu pandemic on non-government hospitals, specification robustness

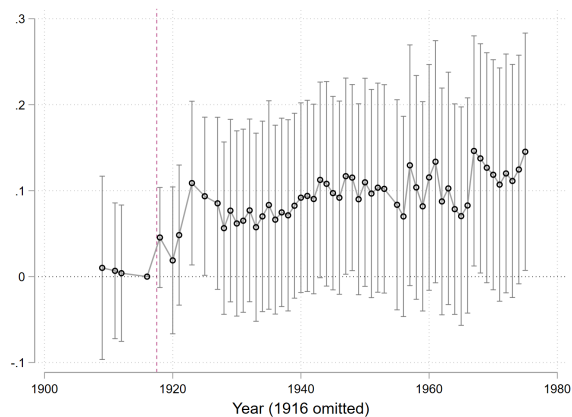
(a) Include interacted 1910 controls



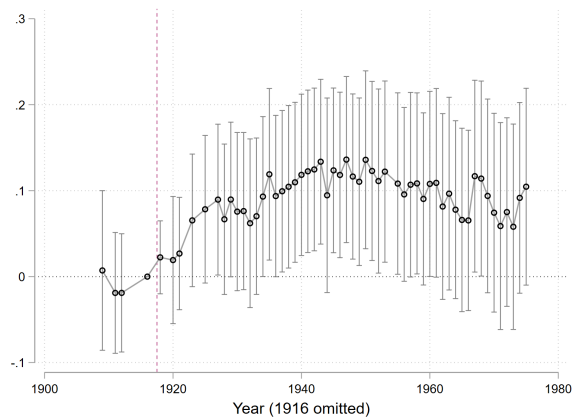
(b) Include interacted 1910 controls and time-varying population



(c) State-year fixed effects



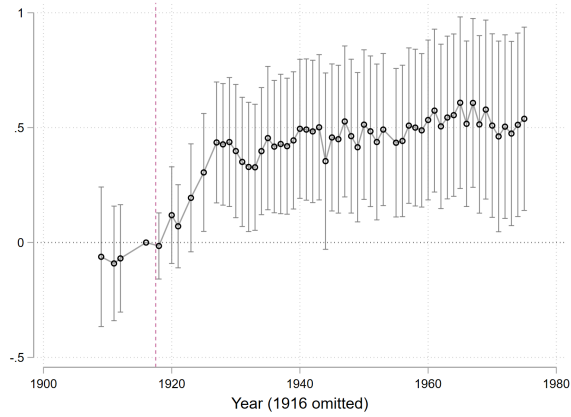
(d) Include 1930 WWI controls



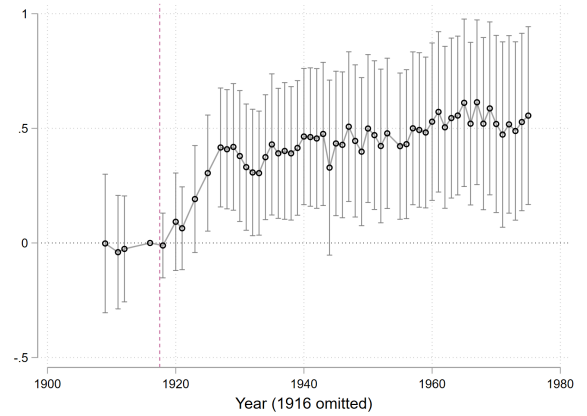
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each panel makes the indicated change to the baseline regression specification. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 16: Effect of the 1918 flu pandemic on non-government hospital beds, specification robustness

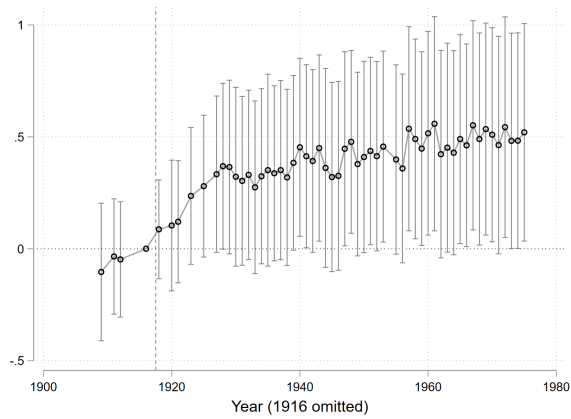
(a) Include interacted 1910 controls



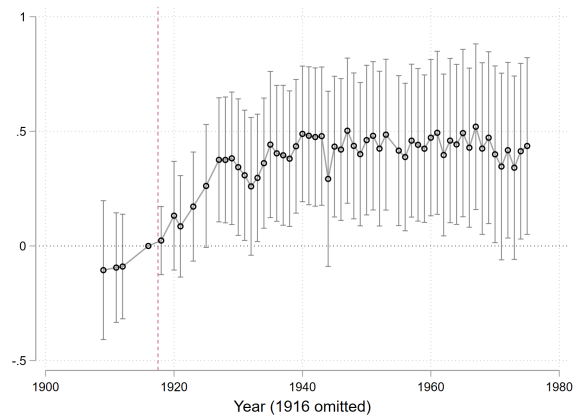
(b) Include interacted 1910 controls and time-varying population



(c) State-year fixed effects



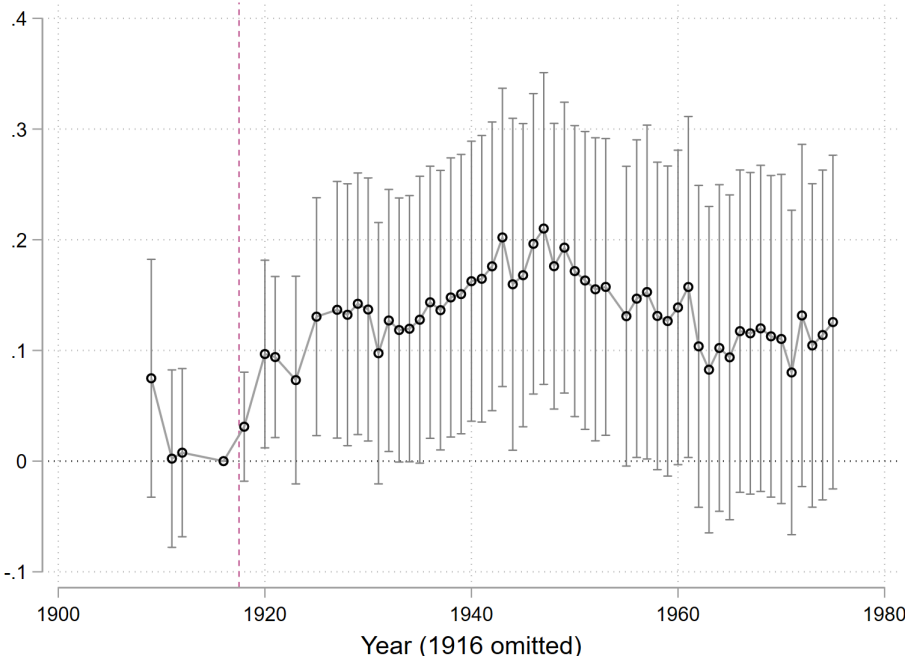
(d) Include 1930 WWI controls



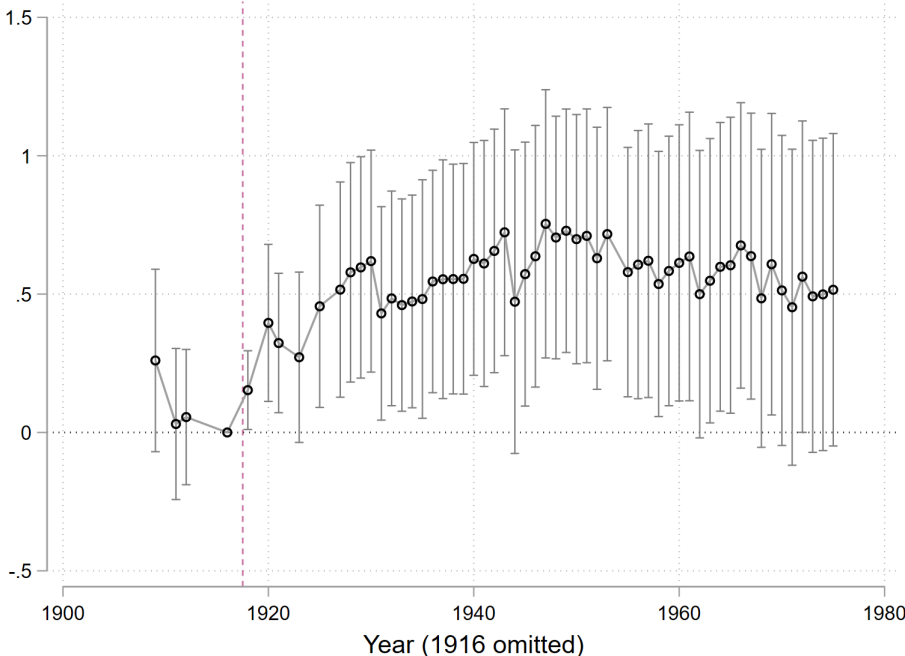
These figures show the effects of pandemic severity on the inverse hyperbolic sine number of hospital beds in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each panel makes the indicated change to the baseline regression specification. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 17: Effect of the 1918 flu pandemic on inverse hyperbolic sine hospitals, exclude places with a pre-1923 government hospital

(a) Non-government hospitals



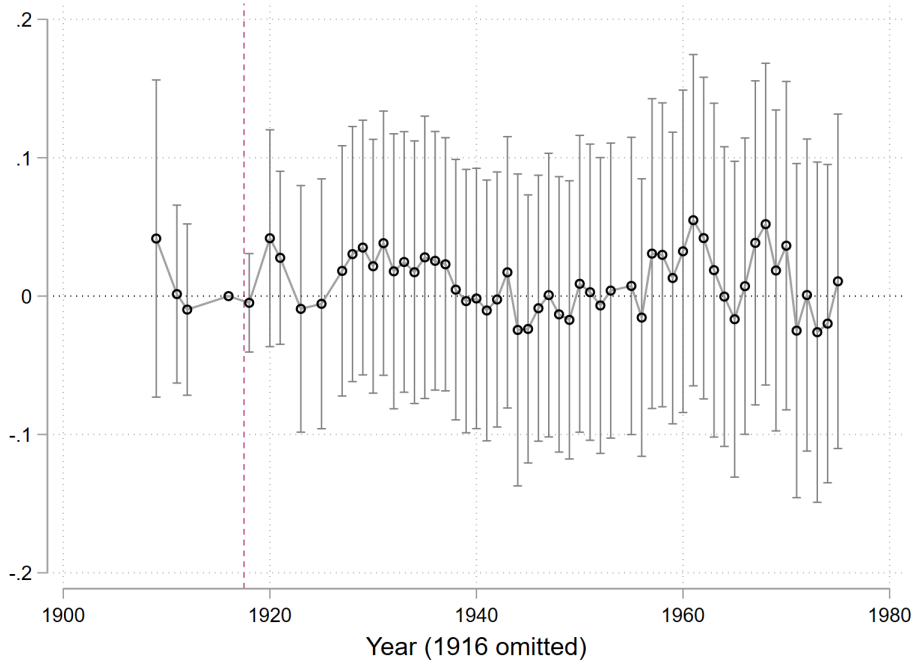
(b) Non-government hospital beds



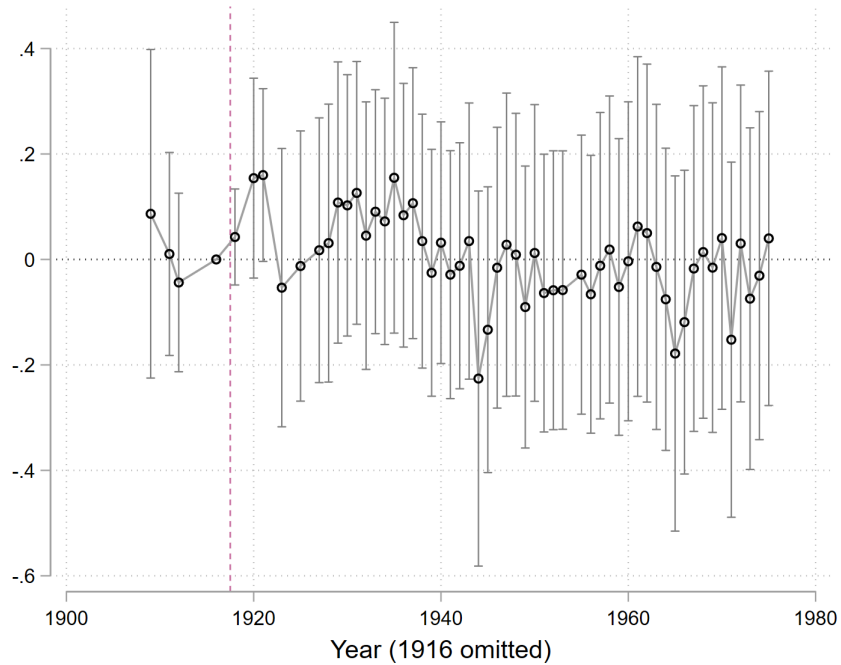
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. For this analysis, we drop all places that had a government hospital before 1923 from the estimation sample. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 18: Placebo test using cancer as city-level treatment

(a) Hospitals



(b) Hospital beds



These figures show the effects of pandemic severity on the inverse hyperbolic sine of hospitals and hospital beds in a given city, as estimated by the model in Equation (1). The treatment variable is an indicator for being in the top 50 percent of the cancer mortality distribution in 1918. Outcome variables are transformed using the inverse hyperbolic sine. 95-percent confidence intervals are shown and standard errors are clustered by city.

Table 1: Summary statistics

	Mean	Std. dev.
City-year variables (N = 27,531)		
IHS hospitals	1.433	0.812
IHS gov. hospital	0.310	0.526
IHS non-gov. hospital	1.300	0.829
IHS hospital beds	5.864	1.916
IHS gov. hospital beds	1.734	2.807
IHS non-gov. hospital beds	5.408	2.304
Had a hospital	0.937	0.242
Time-invariant variables (N = 466)		
1918 flu death rate per 1k	7.049	2.819
1918 cancer death rate per 1k	0.972	0.383
1917 city population (1k)	81.310	31.408
1910 time-invariant variables (N = 466)		
Share Black	0.067	0.125
Average OCCSCORE	24.346	1.633
Average age	28.245	2.111
Share in labor force	0.630	0.043
Share professionals	0.051	0.016
Share farmers	0.008	0.011
Share managers	0.066	0.016
Share clerical	0.062	0.020
Share sales	0.063	0.018
Share craftsmen	0.173	0.049
Share operatives	0.181	0.107
Share service	0.124	0.062
Share farm laborers	0.014	0.011
Share WW1 vet (1930)	0.034	0.007

Notes: This table shows summary statistics for the main variables and sample used in this paper. Outcome variables are transformed using the inverse hyperbolic sine (IHS) function. OCCSCORE refers to the IPUMS-constructed 1950 OCCSCORE variable, which assigns each observation the median earnings of workers with the same occupation in 1950.

Table 2: Effect of the pandemic on hospitals

Dependent variable and sample	Flu mortality \times post	Std. error	Mean of outcome
IHS hospitals			
Full sample	0.070	(0.038)	1.433
Pre-1960 sample	0.076	(0.036)	1.430
Pre-1950 sample	0.072	(0.035)	1.428
IHS non-government hospitals			
Full sample	0.113	(0.041)	1.300
Pre-1960 sample	0.118	(0.039)	1.297
Pre-1950 sample	0.113	(0.038)	1.295
IHS government hospitals			
Full sample	0.007	(0.042)	0.310
Pre-1960 sample	-0.001	(0.040)	0.317
Pre-1950 sample	-0.006	(0.039)	0.317

Notes: This table shows results from our baseline model estimating the effect of the pandemic on the inverse hyperbolic sine number of hospitals in a given city. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution in 1918. Each model includes city and year fixed effects. Standard errors are clustered by city.

Table 3: Effect of the pandemic on non-government hospitals, alternative hospital measures

Dependent variable and sample	Flu mortality \times post	Std. error	Mean of outcome
IHS hospitals			
Full sample	0.113	(0.041)	1.300
Pre-1960 sample	0.118	(0.039)	1.296
Pre-1950 sample	0.113	(0.038)	1.295
IHS beds			
Full sample	0.514	(0.141)	5.408
Pre-1960 sample	0.485	(0.134)	5.242
Pre-1950 sample	0.458	(0.131)	5.133
Have a non-government hospital			
Full sample	0.098	(0.026)	0.878
Pre-1960 sample	0.095	(0.026)	0.879
Pre-1950 sample	0.092	(0.026)	0.876

Notes: This table shows results from our baseline model estimating the effect of the pandemic on the indicated measure of non-government hospitals. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each model includes city and year fixed effects. Standard errors are clustered by city.

Table 4: City-level heterogeneity and the effects of the pandemic

Het. variable and sample	Flu mortality \times post	Std. error
Full sample		
Size of city		
Top-half	0.021	(0.055)
Bottom-half	0.120	(0.053)
Occupational income ranking		
Top-half	0.083	(0.051)
Bottom-half	0.039	(0.059)
Manufacturing wages		
Top-half	0.072	(0.055)
Bottom-half	0.037	(0.053)
Pre-1950		
Size of city		
Top-half	0.022	(0.048)
Bottom-half	0.124	(0.052)
Occupational income ranking		
Top-half	0.107	(0.050)
Bottom-half	0.028	(0.052)
Manufacturing wages		
Top-half	0.079	(0.050)
Bottom-half	0.050	(0.051)

Notes: This table shows results from our baseline model estimating the effect of the pandemic on the inverse hyperbolic sine number of hospitals in a given city. Each row contains estimates from the indicated subgroup of the population of cities. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each model includes city and year fixed effects. Standard errors are clustered by city.

Table 5: Robustness checks for the pandemic’s effect on healthcare provision

Specification	Flu mortality \times post	Std. error
Full sample IHS non-government hospitals		
Baseline results	0.113	(0.041)
Looser hospital definition	0.135	(0.044)
1910 controls	0.090	(0.043)
1910 controls and time-varying pop.	0.080	(0.041)
1930 controls	0.095	(0.041)
State-year fixed effects	0.079	(0.047)
1918-1919 flu mortality treatment	0.107	(0.041)
Excess deaths mortality treatment	0.122	(0.040)
Exclude places with pre-1923 gov hosp.	0.113	(0.053)
Continuous mort. treatment (1 sd)	0.052	(0.019)
IHS non-government beds		
Baseline results	0.514	(0.141)
Looser hospital definition	0.489	(0.138)
1910 controls	0.483	(0.146)
1910 controls and time-varying pop.	0.445	(0.140)
1930 controls	0.450	(0.141)
State-year fixed effects	0.419	(0.178)
1918-1919 flu mortality treatment	0.424	(0.142)
Excess deaths mortality treatment	0.454	(0.140)
Exclude places with pre-23 gov hosp.	0.466	(0.195)
Continuous mort. treatment (1 sd)	0.229	(0.069)

Notes: This table shows results from variations on our baseline model estimating the effect of the pandemic on the inverse hyperbolic sine (IHS) number of hospitals or beds. The baseline treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each model includes city and year fixed effects. Each row makes the indicated change to the baseline specification. Standard errors are clustered by city.