Do Pandemics Change Healthcare?

Evidence from the Great Influenza

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Abstract

Using newly digitized U.S. city-level data on hospitals, we explore how pandemics alter preferences for healthcare. We find that cities in the top half of the mortality distribution during the Great Influenza of 1918-1919 subsequently increased hospital capacity by 12-14 percent more than cities with lower levels of mortality. This growth was driven by the construction and expansion of non-governmental hospitals, persisted until the 1970s, and was driven by smaller cities with less well-developed hospital systems before the pandemic. Other types of public expenditure in healthcare did not respond to pandemic intensity, suggesting that large health shocks may not lead to increased public provision of health services.

JEL: I11, I14, J10, N32

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I. Introduction

Large-scale health shocks, such as pandemics, can severely strain healthcare systems. Surges in demand for treatment lead to shortages of beds and staff, exposing the healthcare system to capacity constraints that can be difficult to change in the short run as they often require high-cost investments in technology and infrastructure. An important question arising from large-scale health shocks is whether they have lasting effects on the delivery of healthcare.

An illustration of this issue recently arose during the COVID-19 pandemic of 2020-22. In order to reduce infection rates and alleviate capacity constraints by reducing in-person visits, hospitals increasingly made use of electronic information and communication technologies (Koma, Cubanski, and Neuman 2021). Telehealth visits in the U.S. increased especially in areas where the pandemic was more severe, accounting for upwards of 80 percent of all visits in these locations (Karimi et al. 2022). On the delivery side, physicians in office-based settings that utilized telehealth appointments for patient care in their practices rose from 16.0% in 2019 to 80.5% by 2021 (Peters et al. 2024). Uptake of telehealth by patients and health facilities was slow prior to the pandemic despite roughly a quarter of all U.S. healthcare organizations already having made initial investments in programs for telehealth delivery: a mere 0.15 percent of all health visits in 2019 were conducted through telehealth, but the pandemic may have altered patient preferences. Recent survey data on Medicare patients suggests that telehealth visits peaked at around 47% in Q2:2022, have subsequently declined to 12.7% (latest data for Q3:2023), but remain roughly twice that relative to rates just prior to the pandemic (Cottrill, Cubanski, and Neuman 2024). It remains to be seen how widespread its use will be ten years after the pandemic.

Whether COVID-19 will permanently shift more patients to telehealth is a question that time will eventually answer, but it is not the first time that a pandemic has forced this question. The magnitude of the strain on healthcare during the Great Influenza of 1918-19 was similar to that witnessed during COVID-19, and it occurred at a moment of even greater fragility. Not only was the hospital system still in its infancy, but the Great Influenza struck the U.S. during World War I, when many nurses and doctors were overseas, creating staffing shortages at home. In places where the 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. In this paper, we use this historical episode to ask: can large-scale public health shocks generate lasting changes in the provision of healthcare?

The answer, we argue, is yes, but with important qualifications. The response to the Great Influenza ran through private markets rather than public provision, and was concentrated in smaller communities where baseline infrastructure gaps were largest. These findings suggest that the durability of pandemic-induced healthcare changes depends on where unmet need is most acute and whether communities are positioned to act on it — conditions that vary considerably across America today. Rural areas are experiencing a wave of hospital closures that is recreating precisely the kind of infrastructure deficit that, after 1918, made smaller cities the most responsive to a health shock. Whether COVID-19 will generate a comparable private-market response in these communities is an open question our findings cannot answer, but one they help to frame.

In this paper, we investigate the effects of the Great Influenza (GI) on the provision of healthcare services in the US over the five decades following the 1918-19 pandemic. The GI was a massive public health crisis that struck near the birth of what we now consider the modern hospital and healthcare system in the United States. In the early 20^{th} century, hospitals were evolving from institutions that primarily served the indigent and working class into places where middle- and upper-class households increasingly sought care, although many still preferred to have private physicians and nurses visit their homes. The GI, however, proved to be a massive killer, far worse than prior years of seasonal influenza, and potentially changed preferences. As such, the GI provides a unique setting to test how healthcare delivery systems respond to crises. Hospital investments, once made, are durable and observable over decades, giving us an ideal window into lasting preference change, and allowing us to trace the pandemic's legacy over the long run in a way that is not yet possible for COVID-19.

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, created 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 improve patient outcomes. When the pandemic struck in 1918, even higher-income households no longer had the option to stay at home because entire families were affected. Nurses, already in shortage due to WWI, were not available to visit homes. The GI provided a substantial shock to a fledgling industry. Since our new data set tracks the evolution of healthcare over the long run, we can examine whether the pandemic was merely a transient disruption or whether it led to lasting changes in the demand for and supply of hospitals.

Specifically, we examine several hypotheses. First, we analyze whether pandemic severity is causally related to an increase in hospital capacity as measured by the number of hospitals or the number of beds in a city. Second, we predict that effects were concentrated in smaller and medium-sized cities. Two mechanisms point in this direction. Smaller cities entered the pandemic with substantially less developed hospital infrastructure than larger ones, leaving a larger gap between latent demand and existing supply that the pandemic could reveal and activate. At the same time, collective action theory predicts that smaller, more homogeneous communities find it easier to organize around shared needs — the free-rider problems that complicate large-scale public good provision are more tractable when the affected community is smaller and the shared experience of the shock more visible (Alesina, Baqir, and Easterly 1999). Together, these mechanisms predict that smaller cities both needed more hospitals and were better positioned to build them, consistent with a catch-up story in which the pandemic hastened convergence toward the higher per-capita hospital capacity already present in larger cities.

Third, we predict that the supply response came through private, fee-for-service hospitals rather than public ones. This prediction follows from both the historical structure of American hospital provision and the nature of the demand shock. Non-indigent patients seeking hospital care had always done so through private institutions. The American hospital system had long operated as a hybrid in which charitable and later fee-for-service private institutions served patients with some ability to pay (even if they did price discriminate) while public hospitals remained a distinct and separate track for the morally less respectable (alcoholics, unwed mothers, etc). The pandemic delivered a sharp demand shock precisely among these potential fee-for-service

patients: non-indigent families, who had previously preferred home care, suddenly found that option unavailable as entire households fell ill simultaneously and nurses were in short supply due to WWI. These households could express their revealed preference for hospital care through the market, without needing to build political coalitions or overcome disagreement about public spending priorities. Crucially, the collective action that we argue was easier in smaller cities was private rather than political in nature — physicians, philanthropists, and community members organizing to finance and build fee-for-service hospitals. Compelling increased public provision is a fundamentally different process, requiring majority agreement through municipal government and facing the classic free-rider problems of public goods provision. We therefore expect private expansion, not public, as the primary channel of response.

To examine these questions, we build a dataset of U.S. hospitals spanning the period 1898 to 1975, combining newly digitized information from R.L. Polk & Co's *Medical and Surgical Register*, 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 20$^{\text{th}}$ century to the 1970s. Importantly, our 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).

By combining these new data with information on pandemic and pre-pandemic city-level mortality, we are able to estimate dynamic event studies and difference-in-differences models that allow us to trace out the effects of pandemic severity on the local provision of healthcare over time. Results indicate 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 1970s.

Our results indicate that post-GI increases in hospital capacity were driven by growth in small- and medium-sized cities – those with populations roughly between 10,000-50,000 – and that had

fewer beds per capita in 1918 than larger cities. In these cities, moving from the bottom to the top half of the mortality distribution is associated with a 12-14 percent increase in the number of hospitals. These findings suggest that the pandemic encouraged catch-up growth in hospital capacity in these cities relative to larger ones. Absent the Great Influenza, small- and medium-sized cities might have eventually converged toward the per capita capacity of larger cities, but the pandemic appears to have hastened this convergence in hospital access. The results were more pronounced in cities with fewer Black or foreign-born residents, suggesting that interests may have been more easily aligned to increase hospital capacity in cities where demographic differences were less pronounced.

We also find that the increase in capacity in small- and medium-sized cities was concentrated in privately operated hospitals rather than in hospitals operated by municipal or county governments, continuing a long-standing American pattern in which private organizations provided most general hospital care while public hospitals primarily served those unable to afford access to privately run institutions. Federal provision was largely confined to the military and, after 1930, to veterans through the consolidated Veterans Administration — populations with pre-committed entitlements that were distinct from the general civilian market for hospital care.[1] Consistent with this pattern, we find no evidence that pandemic intensity affected municipal healthcare outlays on capital expenditure either during the pandemic or in the following decade. Our analysis of city-expenditure does not reveal any short-term increases in public health expenditure in response to the GI.

Our paper makes several contributions to the existing literature. First, we examine how large external shocks may alter preferences for healthcare provision in the short- and longer-run, a topic that has thus far received little attention. Prior work on local public goods suggests 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

[1]For a detailed history of "voluntary" hospitals and the hybrid public-private system in the U.S., see Rosenberg (1987) and Stevens (1989). State-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 administration consolidated it into the Veterans Administration.

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 2023).

Theoretically, the expected effects of a large public health shock are *ex ante* unclear. Standard political economy arguments imply 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). In contrast, widespread shocks that lead to greater dispersion in health and income outcomes for voters may make 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 if preferences for public goods diverge across income groups, cities experiencing more severe effects from the pandemic could see large increases in demand for privately provided healthcare.

Adding complexity to this debate is that the hospital industry in the United States has long operated as a hybrid system between non-public provision of care for "worthy" patients and the deserving poor, and governmental care for those deemed less deserving.

Second, to the best of our knowledge, our research is one of the first in economics to shed 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 GDP.[2] 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 federal subsidies introduced as part of the Hill-Burton program allowed hospitals to expand through the 1950s and 1960s. Hollingsworth et al. (2024) show how a large philanthropic endowment, set up by industrialist James Buchanan Duke, had a significant impact on North Carolina's healthcare sector, namely, by funding the creation of new hospitals or the modernization of existing ones. We contribute to this literature by analyzing the previously underexplored formative period in the hospital industry and by studying the lasting effects of a health shock (rather than policy changes) on hospital capacity.

[2]Our research thus complements pioneering historical studies of the rise of US hospitals, in particular Rosenberg (1987), Stevens (1989), and Starr (2017).

Finally, our paper relates to the new literature that revises our understanding of the effects of the Great Influenza.[3] Recent papers study how non-pharmaceutical interventions influenced economic activity (Correia, Luck, and Verner 2022; Lilley, Lilley, and Rinaldi 2020), how school closures affected educational attainment (Ager et al. 2024), 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.

II..A. The Infancy of Modern U.S. Hospitals

When the influenza pandemic struck the U.S. in 1918, hospitals were just assuming their modern form as places people would go to relieve acute illness or to have surgery. Prior to this transition, which began toward the end of the 19$^{\text{th}}$ 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 workers lodging in boarding houses (Rosenberg 1987, p. 103). 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 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). At that time, medical science offered little in terms of diagnostic or clinical help, but patients might have nevertheless benefited from rest, warmth, and food. With a limited number of beds, early

[3]See Beach, Clay, and Saavedra (2022) for a survey.

hospitals only admitted the "morally worthy" (Rosenberg 1987, p. 23). Prostitutes, alcoholics, and people suffering from contagious illnesses 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.[4] In addition, working-class families often needed the income of more than one family member to survive, so nursing care by family members may not have been available. 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).[5]

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-19$^{\text{th}}$ 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 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. 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

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

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

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 same standards for medical licensing.[6]

In addition to receiving training in hospitals, some newer technological advances could only be used in hospitals. For example, the first X-ray machines (1897) were large and non-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).[7] Hospitals provided physicians with on-call nursing assistance and pathology laboratories, and physicians came to prefer hospitals to perform surgery and treat serious illnesses. The size of general hospitals increased over time as hospital administrators sought to take advantage of economies of scale and scope.

Just as physicians came to view hospitals as essential workshops, major discoveries, such as Pasteur's development of a vaccine for rabies (1885), Behring and Kitasato'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 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

[6]See Moehling et al. (2020) for further discussion and Fernandéz (2021) for similar developments in European hospital care.

[7]Surgery 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).

(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."[8]

II..B. The Global Influenza Pandemic and Hospitals

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.[9] The state of medical technology in 1918 meant that care for infected individuals was limited to nursing, 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 from 1.3 to 100 deaths per 100,000 (Lin and Meissner 2020).[10]

[8] January 16, 1916, p. 6.

[9] Little consensus has emerged on its underlying causes. For a discussion, see Crosby (2003), Kolata (2001), and Brainerd and Siegler (2003)

[10] Over 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).

In many cities across the country, private entrepreneurs, community-based initiatives, public authorities, and physicians started promoting the expansion of hospital capacity. Many 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.[11] 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.)[12]

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:

> 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

[11]When 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 the largest healthcare providers in Northern California.

[12]The Saint Thomas Midtown Hospital, as it is now known, is currently the largest non-profit hospital in the city.

> 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 make haste and establish a Community Hospital. (*Greenville Daily News*, October 25, 1918, p. 1)

In 1923, three physicians in Greenville, one of whom had been involved with the earlier, failed attempt to build a public hospital, mortgaged their property and borrowed money to build a 42 room private hospital that opened in 1924 (Kammerer 1986).

Similar narrative accounts appear throughout the U.S. and suggest that preferences for health-care may have shifted in response to the influenza pandemic. Figure 6 provides more direct evidence using digitized newspaper archives. We search Newspapers.com for mentions of "need more doctors", "need more nurses", and "need more nurses" combined with "flu" for the years 1918-1925. For each city, we count the number of matches and normalize by the total number of newspaper pages digitized on Newspapers.com. We then plot the response by city-size bins.

Results show that cities of all sizes reported on medical system shortages. However, the largest cities have the smallest per-page mention frequency. For the term "need more nurses", we see that results are largest for cities in the 100k-250k range, but largest for small cities once we condition on pages that also mention the flu.

III. Data and Summary Statistics

To move from narrative evidence to causal identification, we combine city-level data from several sources to examine the relationship between pandemic intensity, healthcare provision, and expenditures on public services and infrastructure from 1898 to 1975.

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 deaths for cities with populations of at least 10,000 that were included in vital statistics registration areas. Our baseline measure of influenza intensity is reported deaths from flu and pneumonia in 1918 per 1,000 residents in 1917.[13] Figure 1 plots a histogram of pandemic death rates by city and illustrates that the pandemic's effect on mortality was unequally distributed. Figure 2 displays the cities reporting mortality, with darker dots corresponding to higher levels of mortality. It shows that there is no simple relationship between geography and pandemic mortality: even within states, there is often a large variation in death rates.[14]

Next, 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 newly digitized sources with existing data that starts in 1948. For 1898, 1900, 1904, and 1906 we digitize data from R.L Polk & Co's *Medical and Surgical Register*, which reports the name and location of the hospital, the ownership type (e.g., private or public), and the number of beds. From 1909 to 1923, we digitize the *American Medical Directory* (AMD), which reports similar information.[15] Starting in 1925, we digitize similar data collected by the American Medical Association's Council on Medical Education and published in the *Journal of the American Medical Association*.[16] 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 four sources using the Google Maps API to address

[13] We use the population estimate from the 1917 *Mortality Statistics* to avoid a changing denominator due to deaths in 1918. We show results using an excess mortality calculation and additional years of mortality in the robustness section.

[14] In some versions of our empirical approach, we explicitly use this variation by including state-by-year fixed effects.

[15] These files are available for 1909, 1911, 1912, 1916, 1918, 1921, and 1923.

[16] These files are available for 1920, 1925, and yearly from 1927 to 1947.

misspellings 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 hospital counts, bed numbers, and the probability of observing certain types of hospitals 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 mental health facilities, asylums, and "recovery homes" from the baseline analysis. We also drop military hospitals and Veterans Association facilities.[17]

Figure 3 shows the growth of hospitals and hospital beds over this time period using our baseline definition of hospitals in the sample of cities that we can link to mortality data. Although the data are drawn from four different sources, the resulting time-series are relatively smooth.

In our sample, the number of hospitals increases sharply at the beginning of the 20th century, stabilizes in the interwar period, and then follows a gentle but persistent downward trend after World War II. This aggregate trend, however, masks changes in the *types* of hospitals that became more common. Over time, smaller hospitals closed and were replaced or merged into larger facilities. Panel (b) of Figure 3 illustrates that the number of hospital beds grew during our sample period, even as the number of facilities declined. The figure also indicates that the relative importance of governmental hospitals diminished over time, with the majority of growth coming from non-government hospitals.

To analyze the response of local authorities to the pandemic, we gather data on 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, and we focus on two classes of healthcare expenditure: 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.[18]

Next, we collect city-level covariates from the publicly available 1910 and 1930 U.S. censuses

[17] We show that our results are robust to alternative definitions of hospitals in Section VI..

[18] No volumes were published in 1913, 1914, and 1920.

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.[19] 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. In addition to these fixed characteristics of cities, we also construct a time-varying count of workers in medical occupations and the healthcare industry in each city.

To supplement the census, we use a time-series of city population constructed by Schmidt (2018).[20] Table A1 provides summary statistics for the key variables used in our analysis. As our estimates of the pandemic effect are concentrated in small- and medium-sized cities, we show summary statistics for both the full sample of cities and cities in the bottom 75 percent of the 1917 city size distribution. Apart from measures of size (population or size of healthcare system), all other pre-pandemic controls are indistinguishable by city size.

IV. Estimating the Effects of Pandemic Intensity on Hospitals

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

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

where $Outcome_{it}$ is a hospital-related outcome for city i in year t. $Mortality1918_i$ is a measure of city i's influenza mortality rate in 1918, and γ_i are city 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.[21]

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

[20] Schmidt (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.

[21] Alternative treatment specifications consider other distribution cutoffs, continuous measures of mortality, and mortality calculations that include 1919 data. Recent work has examined the difficulty of interpreting standard

For count outcomes, we use a Poisson fixed-effects estimator to estimate models analogous to Equation 1.[22] For dichotomous outcomes, like the probability of observing a certain type of hospital, we use linear probability models.

Our identifying assumption is that there are no omitted variables correlated with both the severity of the 1918 pandemic and subsequent outcomes. For example, perhaps cities that were hit harder by the pandemic had less established health systems that would have changed even if no pandemic occurred. 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 had an effect on outcomes after 1918. To explore this possibility, Equation 1 includes a vector X_i of city-specific controls measured in 1910 (the census preceding the GI) and 1917 (the last year before the pandemic with available population estimates) interacted with year fixed effects. 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.[23] We also include logged 1917 population in X_i, estimated in the 1917 mortality reports.[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 when we include them; we show in robustness exercises that our coefficients are similar if we remove them.

Baseline results

Figure 4 shows results from an event-study model based on Equation 1, with the count of hospitals as our outcome variable. Panel A includes all cities. Panel B restricts the sample to cities in the bottom 75th percentile of the city-size distribution. Estimated coefficients reflect the

continuous difference-in-difference models (Callaway, Goodman-Bacon, and Sant'Anna 2024).

[22] Counts of hospitals and hospital beds are both count variables. We show robustness to alternative functional forms in Section VI.

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

[24] We do not control directly for time-varying population in our baseline specification since it is plausible that population growth itself was affected by the pandemic. That said, Figure A14 shows that our results are only slightly attenuated when we include logged time-varying population as a control.

change in hospitals in cities in the top half of the mortality distribution relative to those in the bottom half, with 1916 as the omitted year. Both panels of Figure 4 indicate that mortality was not related to the number of hospitals in a city prior to 1918. In the full sample of cities, we see a null effect. However, Panel B shows that there is a positive relationship between mortality and local hospitals after 1918 for cities in the bottom 75th percentile of the size distribution. In particular, starting in the early 1920s, being in the top half of the mortality distribution is associated with an approximately 15 percent increase in the number of hospitals in a city.

The concentration of effects in the smaller- and medium-sized cities partly reflects the properties of our estimator. The Poisson estimator, while well-suited for count data like hospital numbers, is sensitive to dispersion in the outcome variable, a feature that large cities with more variable hospital counts exhibit more strongly. The largest cities appear not to have reacted to the pandemic, and this lack of a reaction disproportionately affects the overall sample estimate.

More substantively, the differential response of smaller cities reflects the two mechanisms we previously identified. Smaller cities entered the pandemic with less developed hospital infrastructure. Prior to 1918, cities in the bottom half of the population distribution had a median of 2.7 hospital beds per 1,000 residents, compared with 3.2 beds per 1,000 in larger cities, a 19 percent gap. Thus, these smaller cities had a larger unmet need that could be revealed by the pandemic[25] At the same time, it easier for groups in smaller cities to coalesce around a perceived need for and financing of new capacity after a health crisis, consistent with the collective action logic that we outlined above. The Sutter (California) Hospital Association's founding appeal captured both forces: the physicians who organized it noted that the community was "...freshly recovered from the influenza epidemic of 1918, which brought out as never before the need for a large hospital" formed the Sutter Hospital Association to raise money for a hospital project (The Sacramento Bee, October 11, 1923, p. 1). Accordingly, throughout the remainder of the paper, we focus our analysis on these smaller cities.

Figure 4 shows that the effect of the pandemic on hospital provision persists through the 1950s before slightly waning toward the end of our sample period. This persistence is unsurprising given

[25]This was confirmed by a landmark study, undertaken between 1927 and 1935 by the Committee on the Costs of Medical Care (**CCMC**)

the nature of hospital investment. Per capita hospital beds are serially correlated, suggesting that hospital capacity, once established, is sticky. Furthermore, since hospitals typically 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.

We next examine whether this response is also reflected in hospital capacity, as measured by bed counts. Unlike the hospital count, beds capture both new construction and the expansion of existing facilities, and thus pick up intensive-margin responses as well as differences in the scale of investment across hospitals.

Panel B of Figure 5 reports estimates from Equation 1 using bed counts as the outcome variable. The pattern is similar to that for hospitals: cities experiencing higher pandemic mortality witnessed larger increases in beds, with effects persisting decades after the pandemic ended. Smaller cities in the top half of the mortality distribution saw approximately 15 percent more hospitals and roughly 20 percent more beds relative to less-affected cities.

Panel C of Figure 5 focuses exclusively on the extensive margin — whether a city-year has any hospitals. We find no effect on this margin, which is unsurprising given that all cities in our sample had at least 10,000 people in 1917 and rarely lacked hospital facilities entirely before the pandemic.

The overall evolution of the healthcare industry is consistent with the pattern observed for total hospital beds. The average size of U.S. hospitals in our sample increased from 100 beds in 1920 to 161 in 1975.[26] This growth reflected changes in the cost structure of healthcare provision, which encouraged economies of scope and scale, and shifts in demand. New surgical and diagnostic techniques that were developed in the early 20$^{\text{th}}$ century required substantial capital investment as well as 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 (Rorem 1930). A 1935 'Business Census' of US hospitals estimated that equipment averaged 15% of all costs in general hospitals, but the share ranged from 25% in hospitals with up to 25 beds to only 14% for larger institutions above 150 beds (Pennell,

[26] White (1990) reports that the average size of U.S. hospitals increased from 78 beds in 1929 to 173 in 1985.

Mountin, and Pearson 1939).

Despite this capital deepening, contemporary studies found little evidence of economies of scale in hospital operations(Pennell, Mountin, and Pearson 1939; Hayes 1955). Unit costs were either flat or slightly increasing with size. Since hospital size was closely tied to population, the same reports similarly found no relationship between city size and unit costs of hospital provision. One report attributed this to the fact that larger hospitals provided a wider range of diagnostic and therapeutic services, requiring specialized staff and equipment that offset potential scale economies (Hayes 1955, pp. 108–09).[27] Hospitals, in other words, were not assembly lines for standardized care but increasingly complex units whose costs grew with their scope. This apparent absence of scale economies is consistent with the moderate growth in average hospital size observed over our period.

Health historians have also noted that internal economies of scale were limited during the spread of US hospital care (White 1990; Starr 2017).[28] Technology was not the only constraint; organizational economics mattered as well. Starr (2017) describes the tension between pressures for greater scale–to economize on fixed costs–and physicians' resistance to larger organizations that might erode their control over medical practice. The AMA campaigned for the professionalization of hospital management under its own affiliates, while doctors resisted incentives to merge or expand existing facilities. This was not contradictory: the AMA sought to institutionalize the hospital as the prime site of treatment while ensuring that physicians retained authority over what happened inside it. As Starr (2017) observes, private physicians regarded hospitals as "doctors' workshops" auxiliary to their office practices, while administrators saw them as "medical centers" serving the community as the main coordinators of health services (Starr 2017, p. 178).

Ethnic and religious diversity reinforced this fragmentation. Local communities saw sponsorship of their own healthcare facilities as a guardrail against discrimination. However, by fundraising for hospitals primarily serving their populations, these groups contributed to the multiplication of small hospitals and blocked their merger into larger healthcare organizations until the 1980s.[29]

[27] The same report found that the number of therapeutic or diagnostic services had a stronger positive correlation with the unit costs per bed than size or occupancy rates.

[28] White (1990) states that even by the 1970s the optimal hospital scale was around 300 beds.

[29] In addition to internal economies of scale, there were also some external economies favoring horizontal and

Starr (2017) refers to this as a case of "blocked institutional development."

A seeming corroboration comes from trends in medical labor markets. Expanding hospital capacity necessarily requires a parallel expansion in the healthcare workforce. Figure A1 shows that, using decennial census data for our baseline sample of small- and medium-sized cities, pandemic intensity predicts an increase in the log number of nurses and doctors–gains of roughly 10 percent,consistent in magnitude with our main hospital results, although estimated with less precision.

At the same time, the growing acceptance of the hospital as the primary site of care created a demand-side channel that reinforced these supply-side pressures, pulling U.S. for hospital care away from the 'cottage industry' model of solo practitioners that had prevailed until the late-19th century. (White 1990). To the extent that the pandemic induced demand for new hospitals, that demand was likely met first through an expansion in the number of facilities. Over time, as those hospitals modernized and grew in scale, bed capacity expanded faster than the number of institutions, which is why our estimates for beds consistently exceed those for hospital counts.

Figures 7 and 8 estimate our baseline models separately for non-government and government hospitals. Figure 7 shows that growth in non-governmental hospitals– private for-profit and non-profit institutions– drives our findings, with pandemic-exposed cities seeing increases of over 20 percent (shown in Panel A) that persist through the 1960s before modestly eroding. By contrast, Figure 8 shows that pandemic intensity was not related to government-run hospital growth.

The public provision of hospital services, in other words, did not expand where the pandemic hit hardest. Cities built more hospital capacity, but primarily did so through fee-for-service private and non-profit hospitals catering to paying customers.[30] While it is difficult 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 broader trajectory of American healthcare over this

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).

[30]Given the features of our data, we cannot consistently distinguish between private for-profit and non-profit hospitals, though the majority of non-government hospitals both today and in the past were affiliated with non-profits.

period, in which the non-profit sector operating under a fee-for-service model, came to dominate the market for inpatient care. By 1935, non-profit institutions already accounted for a large majority of hospital beds (White 1982).[31]

To summarize these event studies, Table A2 presents the results from estimating a difference-in-differences model comparing the average effect in the post-1918 period relative to the pre-period. In particular, we estimate:

$$Outcome_{it} = \beta_1 Post_t + \beta_2 Post_t \times Mortality1918_i + \sum_{j \neq 1916} [\delta_j X_i \ \mathbb{1}_{j=t}] + \gamma_i + \delta_t + \epsilon_{it} \qquad (2)$$

where $Post_t$ is an indicator for observations after 1918, γ_i is a city fixed effect, and δ_t is a year fixed effect. Table A2 indicates that, on average, hospitals increased by between 12–14 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 an 18 percent increase in non-governmental hospitals, with no detectable impact on governmental hospitals. Table A3 reports analogous results for non-governmental beds and the probability of observing a non-governmental hospital.

V. Heterogeneity

The fact that non-governmental hospitals grew in response to pandemic intensity while government-run hospitals did not raises questions about how these changes may have affected access to health-care. In this section, we explore a variety of city-specific characteristics to understand who benefited from the growth in hospital supply and why governmental hospital activity was largely unresponsive to pandemic severity.

We first confirm that the government null result is not an artifact of our hospital sample by drawing on an alternative data source for city-level outcomes. In particular, we estimate versions of Equation 1 using city-level government spending as our outcome variables, combining newly digitized data on city-level spending from 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 these data were collected only for cities of 30,000 or more, these estimates are based on a

[31] Initially, 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).

smaller sample of 185 cities. We focus on two categories of government spending that are most closely related to our analysis: expenditures on on local health departments and related illness-prevention activities, and capital investment in government-supported city hospitals.[32] We see little evidence that pandemic severity is related to government healthcare 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 the full sample (Figure 9) or the subsample of cities in the bottom 75 percentile of the population distribution in our hospital sample (Figure A2).

To explore heterogeneity, we estimate our baseline model from Equation 1 using the median value of city characteristics in our baseline sample of smaller cities. We start by comparing cities above and below the median population of smaller cities in 1917. Figure A3 shows the results for the baseline model for non-governmental hospitals. The two panels show that our results are mainly driven by smaller cities, even after we limit our sample to exclude the biggest cities. As discussed in the previous section, it was likely easier for smaller cities to coalesce around the need for additional medical facilities after a crisis and they had more scope to improve, given that larger cities often had more established medical facilities prior to the pandemic.

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 possible impact of the pandemic on preferences could vary by income. To test for this, Figure A4 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 similar effects across occupational income groups. This result may appear in tension with the demand-side mechanism we emphasize, which centers on non-indigent households as the primary source of the post-pandemic preference shift. However, the relevant distinction in our framework is institutional

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

rather than strictly income-based: the key threshold separates households with access to fee-for-service care from those dependent on public provision, a divide that does not map cleanly onto occupational income rankings within the sample of small and medium cities we analyze. Since our sample already excludes the largest cities — where public hospitals were most developed and where the indigent share of the patient population was highest — the within-sample variation in occupational score may capture relatively little variation in the underlying institutional access margin that our theory emphasizes.

We also examine the measured effects based on city-level differences in the shares of the population that are foreign-born or Black. Figure A5 shows results after splitting the sample above and below the median share of foreign-born. While these patterns of heterogeneity are not precisely estimated, it appears that effects are more prevalent in cities that have fewer foreign-born residents. Similarly, Figure A6 shows that our results are strongest in areas with a lower share of the Black population. 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 A4 provides a summary of these heterogeneity results generated from estimating Equation 2, reporting the coefficients on the interaction between the post-period dummy and influenza mortality for each indicated subgroup of cities.

VI. Robustness Checks

To understand whether our results are sensitive to specification, sample, and measurement decisions, this section describes and reports a number of additional tests. They are summarized in Table A5, which reports the average post-period effect coefficients generated from estimating equation (2) after making the indicated changes. All results, unless otherwise indicated, are shown for our sample of cities in the bottom 75th percentile of the population distribution.

First, we consider whether our findings are sensitive to the types of healthcare facilities included in the baseline analysis. To do so, we exclude infirmaries, clinics, and sanatoriums. These were included in the baseline sample because they appear to respond to healthcare demand and had been

used to treat other large-scale contagious diseases, such as tuberculosis. Moreover, in a number of cases, we observe these facilities changing their name and converting to "hospitals," suggesting that the distinction between these facilities was less well-defined at this point in history. Figure A7 indicates that our results are similar, though attenuated, when we exclude these additional types of medical facilities. Next, we test whether our results differ by hospital size. Figure A8 shows that the magnitude of the pandemic effect is similar when we restrict the sample to hospitals with more than 25 beds.

Second, we analyze whether our results are sensitive to our measurement of pandemic mortality. Figure A9 shows a version of Equation 1 estimated with a continuous measure of mortality as the treatment variable. 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 halves of the mortality distribution.

Next, Figure A10 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 results using an excess mortality calculation. To calculate excess deaths, we use average influenza and pneumonia death rates in each city from 1913-1917 and use this as our counterfactual in 1918, and subtract this prediction from the actual death rate.[33] The results are generally similar using the two alternative measures of pandemic severity, though the excess mortality results are somewhat attenuated relative to using actual mortality. Panel C splits the 1918 mortality rates for cities into thirds rather than halves. The blue line indicates the treatment effect for cities in the top third of the mortality distribution relative to cities in the bottom third, while the black line represents a comparison between the first and second thirds of the distribution. This figure shows that the difference in outcomes is similar as we move from the first to the second (black line) and from the second to the third (blue line) terciles of the mortality distribution.

The core findings are stable across a range of specification choices. Figure A11 documents this across a number of different specifications using the number of non-governmental hospitals

[33]We only do this exercise for cities that appear in at least four of the possible five pre-1918 mortality reports.

as a dependent variable. Dropping the 1910 city-level covariates (Panel A), the 1917 population interactions in addition (Panel B), and including state-year fixed effects (Panel C) leaves the result essentially unchanged, although the specification reported in Panel C is less precisely estimated. The same holds for non-governmental hospital beds (Panels A-C of Figure A12). Controlling for time-varying population yields similar estimates (Table A5), suggesting that population growth does not explain our findings.

World War I presents a related concern, since it coincided with the pandemic and could have plausibly shaped hospital outcomes through several channels. Returning military personnel may have hastened the spread of the virus, and if their presence is correlated with subsequent hospital investment and pandemic severity, our estimates could be biased. Veterans may have directly stimulated hospital construction, either by generating demand for medical care, or by expanding the local supply of physicians and nurses. We address this by augmenting the baseline model with three controls interacted with year fixed effects: the share of World War I veterans in each city, the share of veterans employed as doctors or nurses, both drawn from the 1930 census, and a city's distance to its nearest military training camp using data on camp locations collected by Ferrara and Fishback (2024). footnoteWe use 1930 since it was the first census after the pandemic to ask about veteran status. We also explored using distance to camps as an instrumental variable for flu mortality. We did not observe a consistently strong first stage in our setting. 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. Panel D of Figures A11 and A12 show that the inclusion of these controls does not affect the size or statistical significance of our baseline results.

The Hill-Burton Act of 1946, which provided federal funds to equalize hospital access across the nation, presents a more specific concern for our long-run estimates (Chung, Gaynor, and Richards-Shubik 2017). It initially favored the construction of new hospitals in rural and underserved areas, making it unlikely to have directly impacted our sample of U.S. cities. A 1964 amendment, however, released funds for hospital modernization with a preference given to urban hospitals (Clark et al. 1980). While the timing of our effects predates Hill-Burton. If the medically underserved

communities targeted later by the program also had higher pandemic intensity in 1918, some of our long-run results could be driven by the Hill-Burton subsidies. Conditioning on logged Hill-Burton funding for each city interacted with year fixed effects leaves the results unchanged, as shown in Figure A13. These results confirm that the effects we document reflect the legacy of the pandemic rather than the Hill-Burton program.

Population growth is another potential confounder: cities hit harder by the pandemic may have been growing faster, generating hospital demand independent of the pandemic itself. However, the pandemic itself could have affected population growth and migration decisions, so including time-varying population might be a "bad control" that soaks up part of the effect that we attempt to measure.[34] Nevertheless, controlling for time-varying log population yields similar estimates to our main specification, as shown in Figure A14, suggesting that population growth is not driving our results.

A different concern arises from our use of multiple data sources to classify hospitals as public or non-governmental: apparent differences in the private-sector response could reflect coding changes across sources rather than real variation. To ensure that our results are not driven by such compositional concerns, we exclude any city that had a government hospital before 1923 – the transition year in our sample from AMD to JAMA data, and still observe a large increase in the number of non-governmental hospitals as a function of pandemic severity, as shown in Figure A15. These results confirm that classification changes are not driving the results.

As a final check, we replace influenza mortality with city-level cancer death rates in 1918 as a placebo, constructed from the same census publications and methodology that we used to construct influenza death rates. Figure A16 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), 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.

[34] Our baseline model does control for 1917 population interacted with year fixed effects.

VII. Conclusion

The Great Influenza of 1918-1919 left a lasting imprint on the American hospital industry. Cities that experienced greater pandemic mortality subsequently built more hospitals and expanded bed capacity, with effects persisting for decades. The response ran entirely through private markets: fee-for-service private and non-profit hospitals expanded in harder-hit cities, while government hospital provision and public health expenditures did not respond to pandemic intensity.

The effects were concentrated where they might be expected: smaller cities that entered the pandemic with less hospital infrastructure and more room to catch up. In these communities, the pandemic revealed unmet need and spurred collective action – not through municipal government, but through physicians, philanthropists, and community organizations building fee-for-service institutions. Cities with greater ethnic and racial diversity added less capacity, consistent with the logic of collective action: shared need is more easily translated into coordinated investment where preferences are more homogeneous, particularly since hospitals initially developed as ethnically separate and religious institutions providing charity care.

The pandemic accelerated a transformation already underway: hospitals were shedding their association with poverty and contagion and becoming the standard site of care for middle-class patients. Ethnic and religious preferences channeled this growth into smaller institutions rather than consolidated ones, blocking the emergence of large organizations until decades later. Ironically, two global pandemics pushed American healthcare in opposite directions: the first toward the hospital, the second away from it.

These findings speak directly to debates about whether large health shocks generate lasting changes in healthcare provision, and through what mechanisms. The answer, at least in the American context, is yes, but the response flowed through private demand rather than public provision. Despite the broad nature of the health shock, we find heterogeneity in its long-term effects. In particular, our results are driven by smaller cities, which at the time of the pandemic were relatively less served by hospitals and so had more to gain from converging to the higher levels of access to hospital care already available in larger cities. Furthermore, these communities

may have been more positioned to act, with sufficient homogeneity to coordinate collective action.

Whether COVID-19 will generate a comparable legacy remains to be seen. But the historical record suggests that the durability of pandemic-induced change depends less on the severity of the shock than on whether communities are positioned to respond to it, a condition that still varies considerably across the U.S. today.

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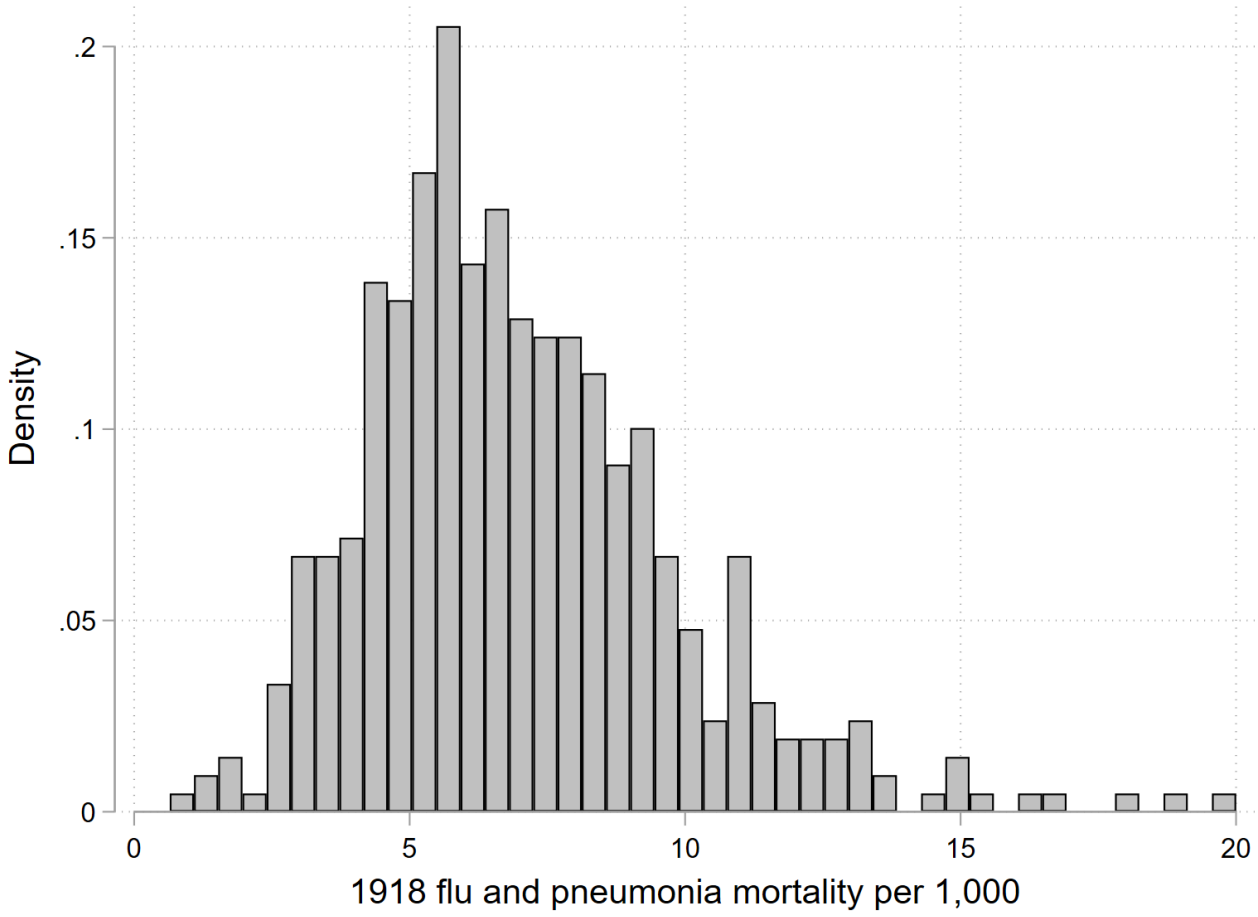
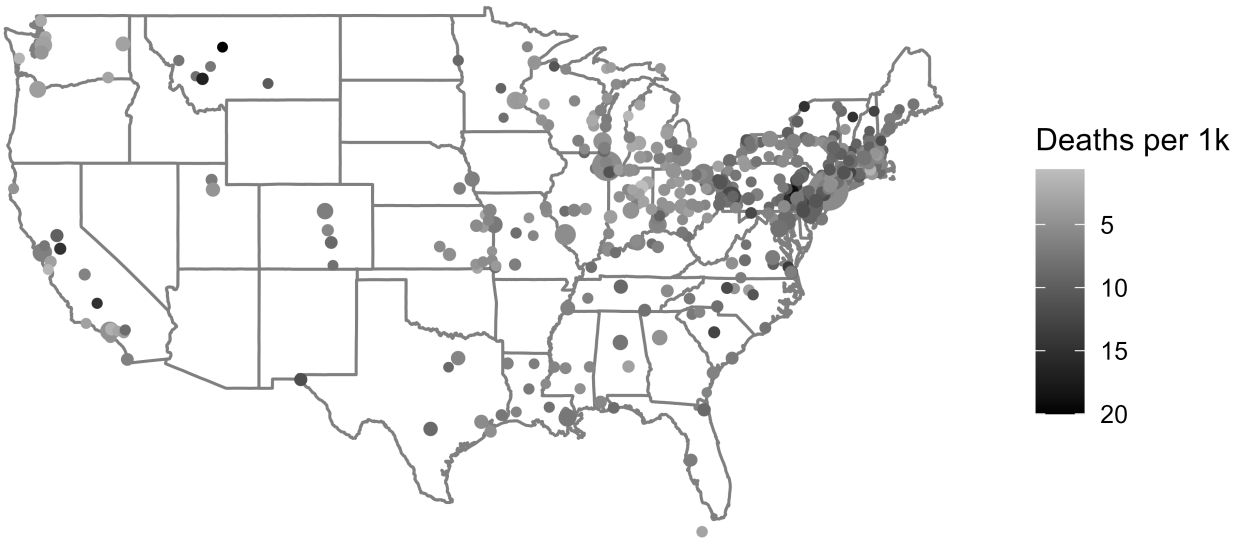
Figure 1: Distribution of pandemic mortality across cities in 1918



Notes: This figure shows the distribution of mortality for our sample of 462 U.S. cities with information on hospitals and 1918 flu mortality.

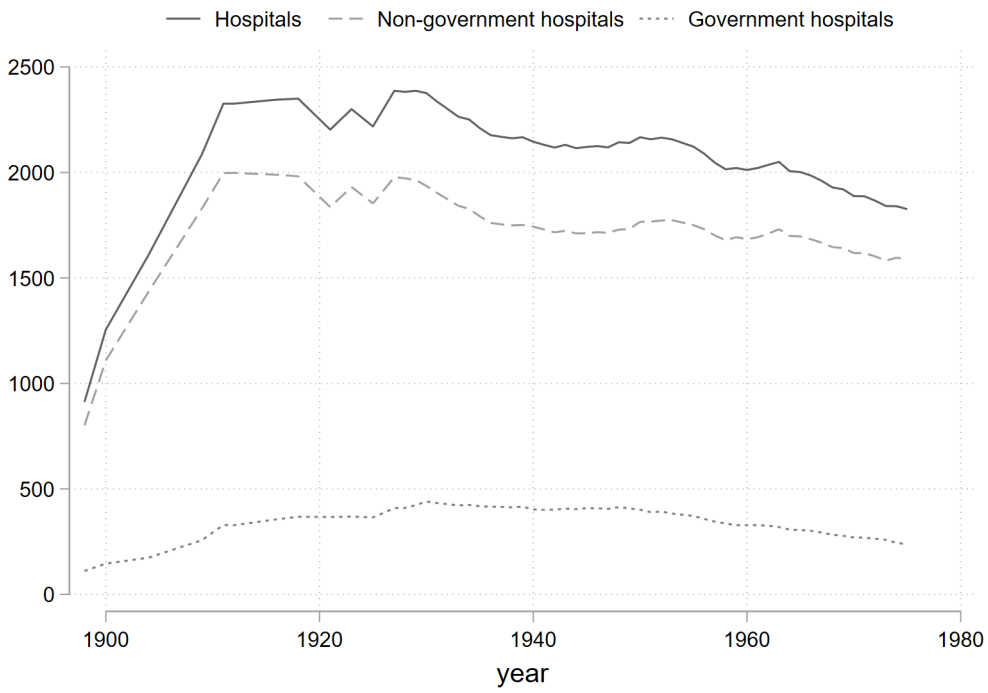
Figure 2: Geographic distribution of pandemic mortality across cities in 1918



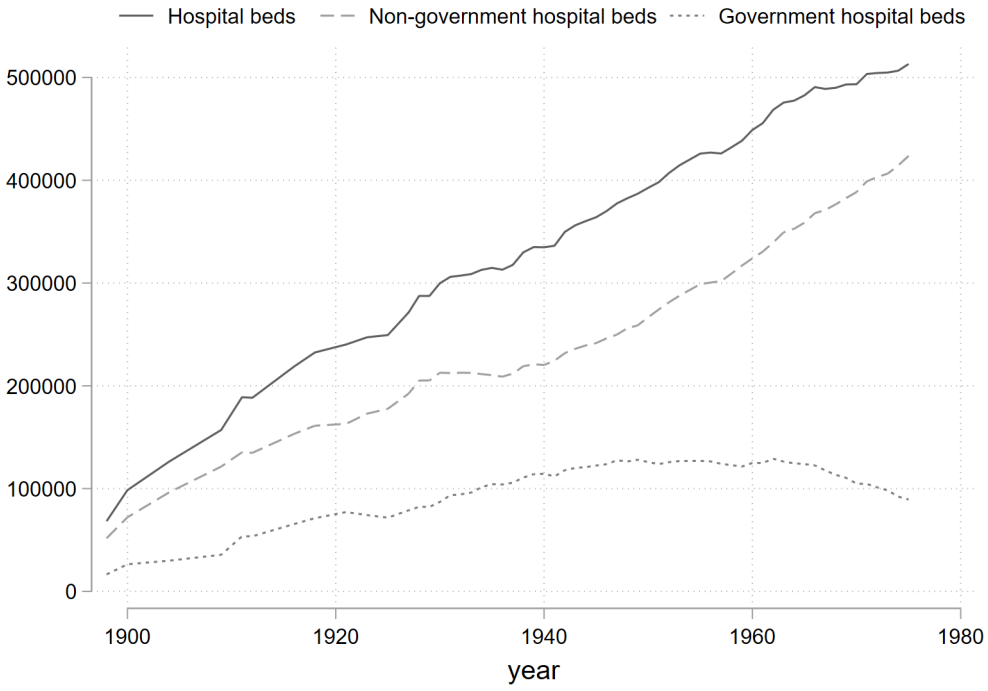
Notes: This figure shows the geographic distribution of mortality for our sample of U.S. cities with information on hospitals and 1918 flu mortality.

Figure 3: Growth of hospital provision in the United States, 1898-1975

(a) Hospitals



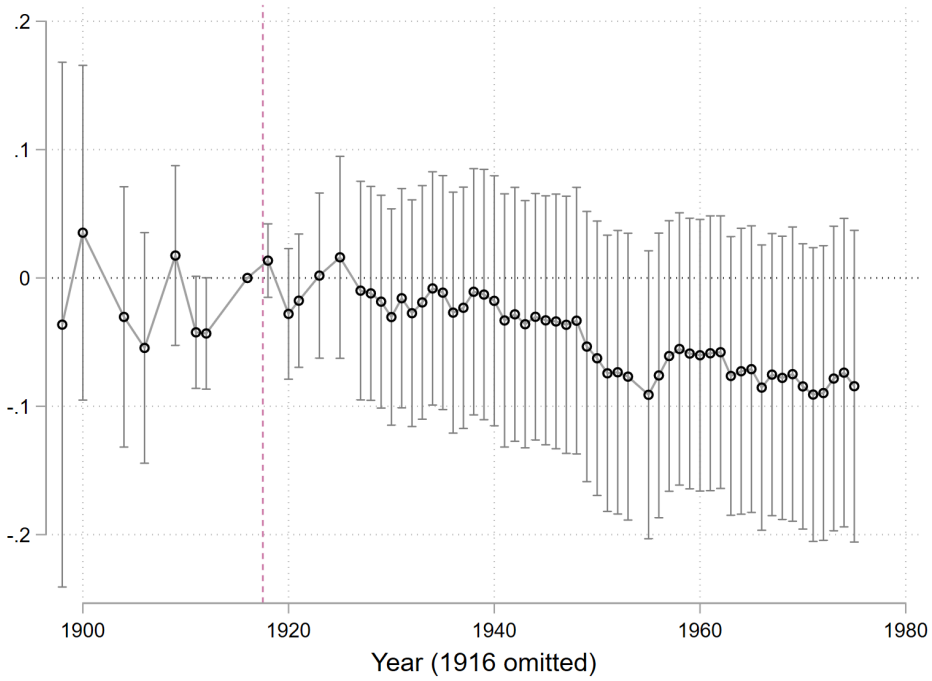
(b) Hospital beds



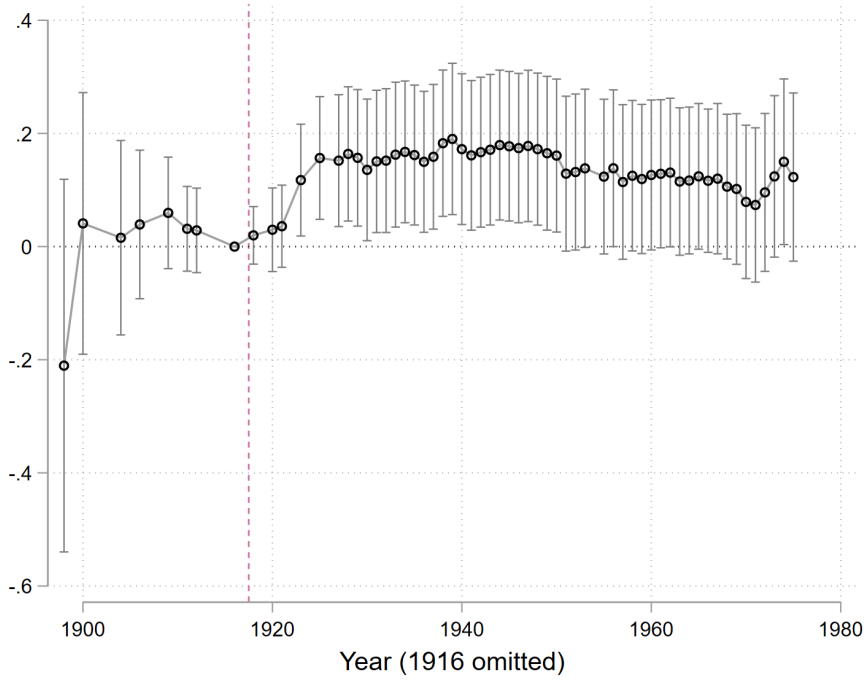
These figures show the growth of the number of hospitals (Panel a) and hospital beds (Panel b) in the United States from 1898-1975 for our subsample of cities with information on hospitals and 1918 flu mortality. The time series is limited to hospitals that provide general hospital services, and excludes specialized facilities such as mental health hospitals.

Figure 4: Effect of the 1918 flu pandemic on the Poisson count of hospitals

(a) All cities



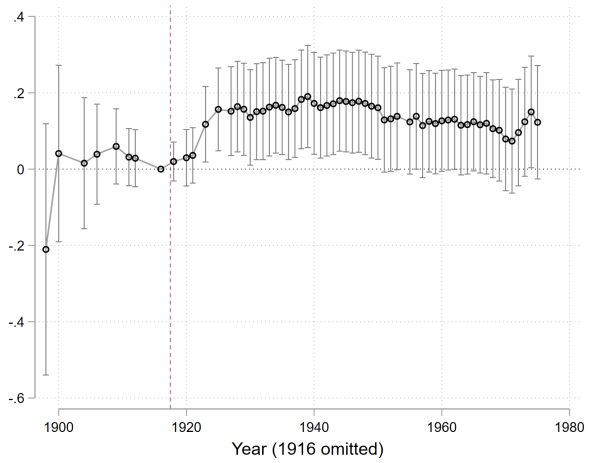
(b) Bottom 75 percentile of cities by population



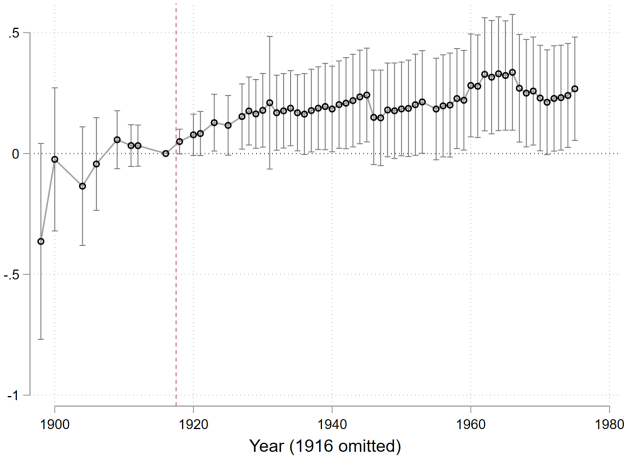
These figures show the effects of pandemic severity on the count of hospitals in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure 5: Effect of the 1918 flu pandemic on alternative hospital measures, bottom 75 percentiles of cities by population

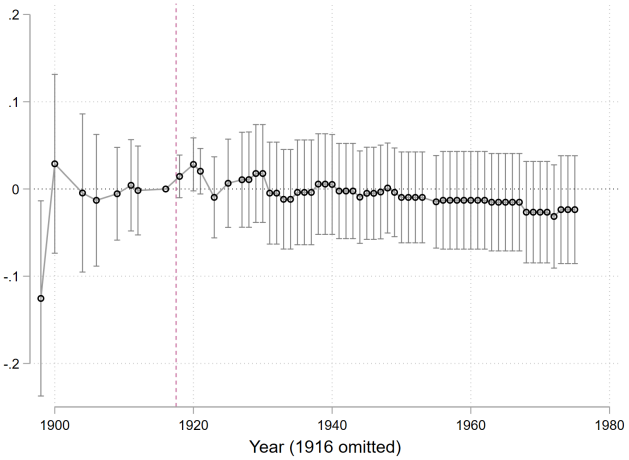
(a) Hospitals



(b) Hospital beds



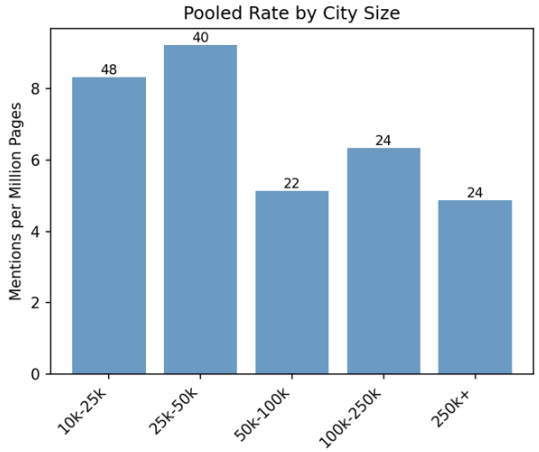
(c) Probability of having a hospital



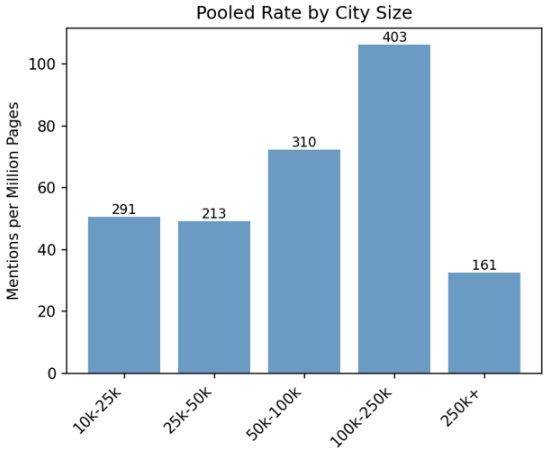
These figures show the effects of pandemic severity on alternative measures of hospitals, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A includes all hospitals as the outcome variable, while Panels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

Figure 6: Newspaper mentions of health occupation shortages, by search term and city size

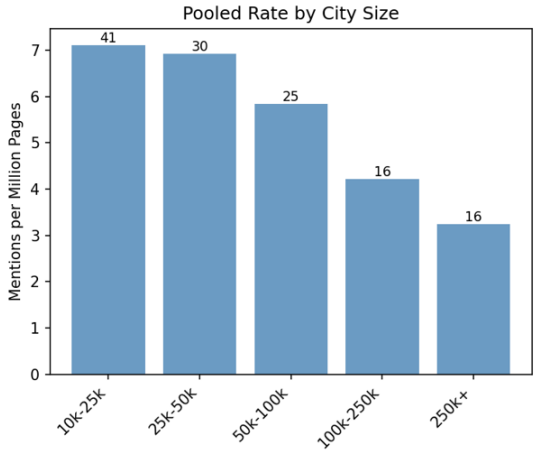
(a) "Need more doctors"



(b) "Need more nurses"



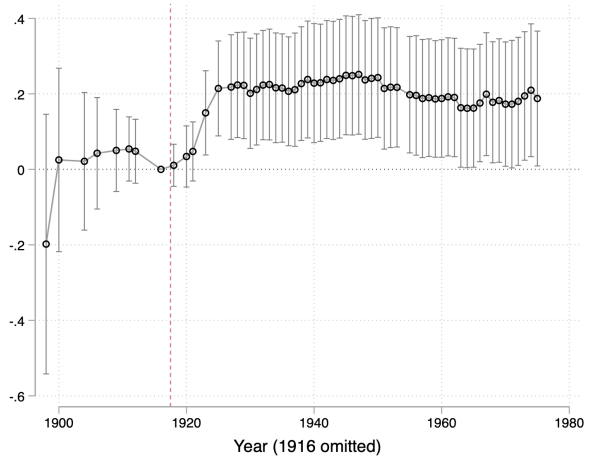
(c) "Need more nurses" + "flu"



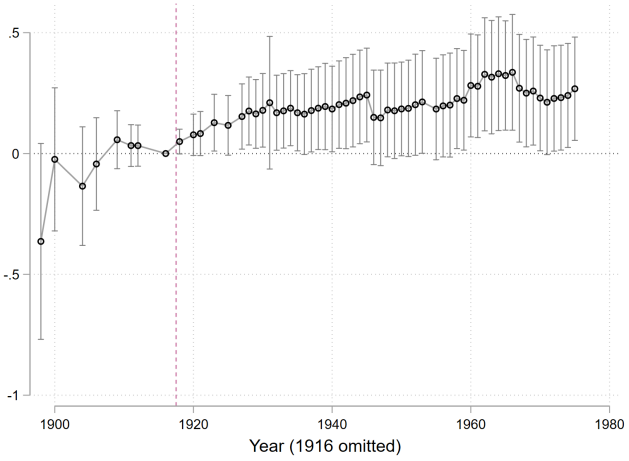
These graphs show the count of newspaper pages on Newspapers.com that include the indicated search term from 1918-1925 for cities of the indicated population size in 1920, divided by total number of newspaper pages available to be digitized for that population bin on Newspapers.com.

Figure 7: Effect of the 1918 flu pandemic on non-government hospital measures, bottom 75 percentiles of cities by population

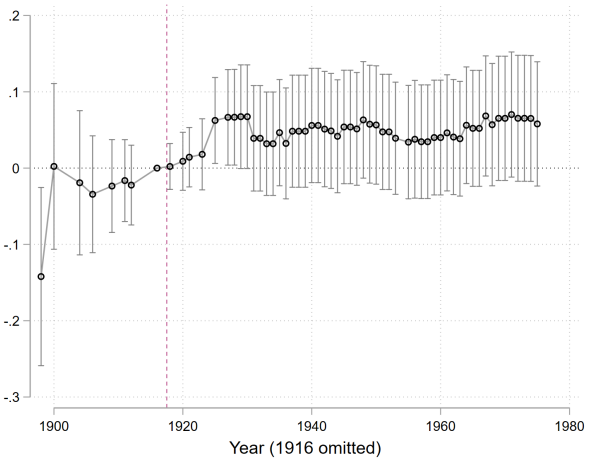
(a) Non-government hospitals



(b) Non-government hospital beds



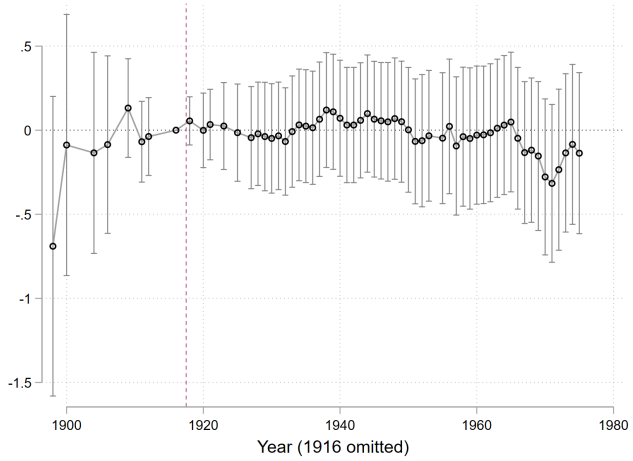
(c) Probability of having a non-government hospital



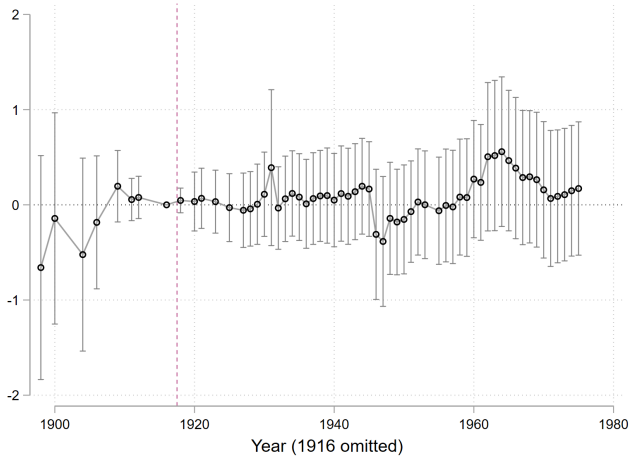
These figures show the effects of pandemic severity on alternative measures of non-government hospitals in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A includes all hospitals as the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

Figure 8: Effect of the 1918 flu pandemic on government hospital measures, bottom 75 percentiles of cities by population

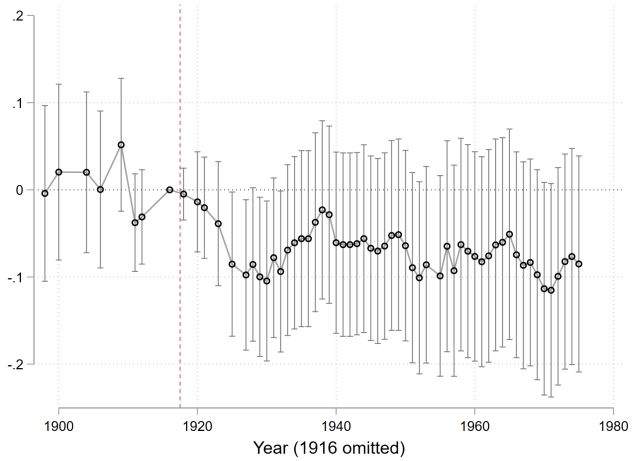
(a) Government hospitals



(b) Government hospital beds



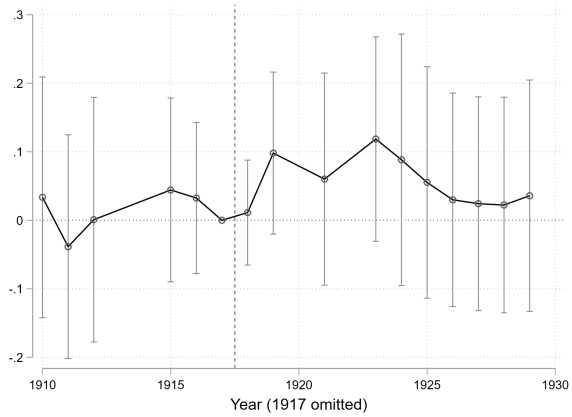
(c) Probability of having government hospital



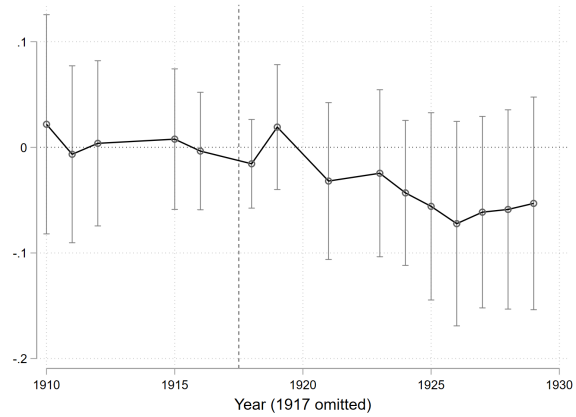
These figures show the effects of pandemic severity on alternative measures of government hospitals in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A includes all hospitals as the outcome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

Figure 9: Effect of the 1918 flu pandemic on local government health spending, full sample

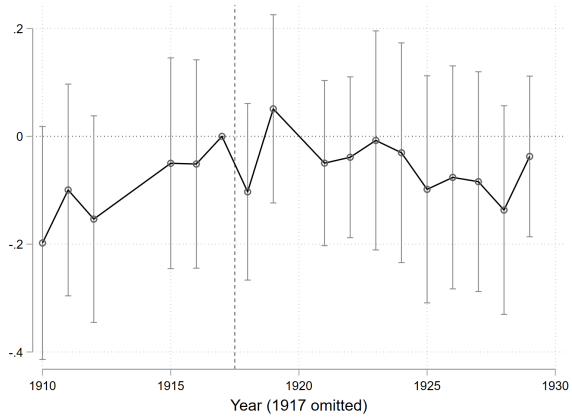
(a) Health spending, discrete treatment



(b) Health spending, continuous treatment



(c) Had health outlay, discrete treatment



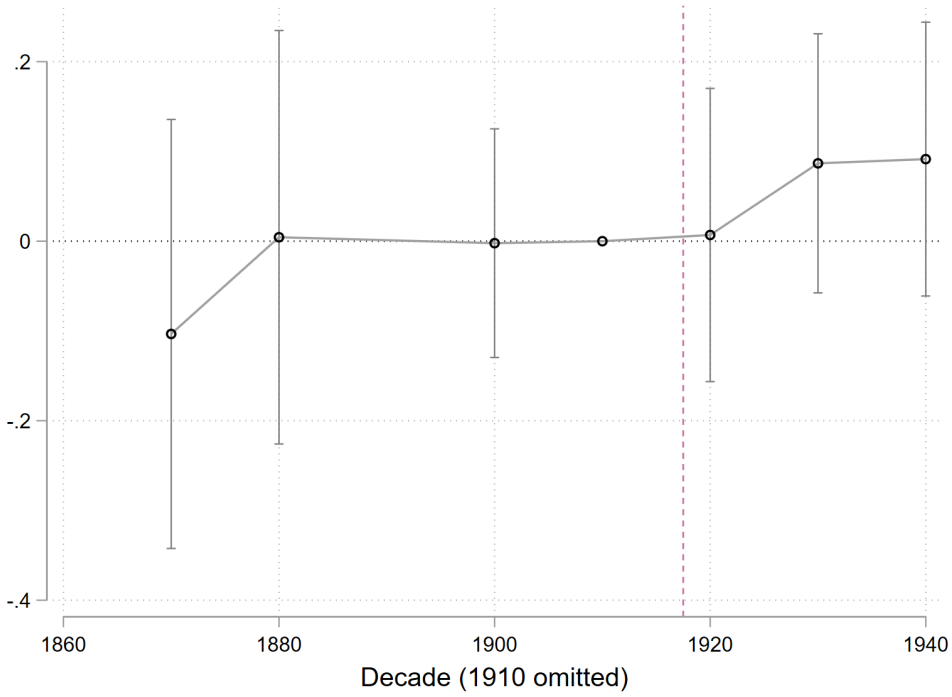
(d) Had health outlay, continuous treatment

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 labeled in each panel. The outcome variables are labeled 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.

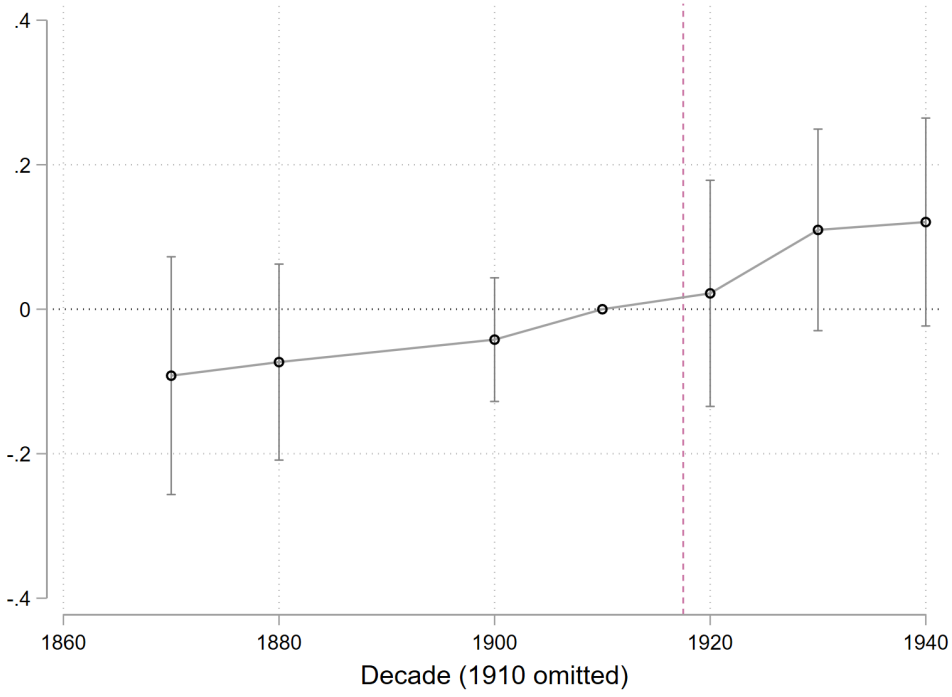
A Figures and tables for online publication

Figure A1: Effect of the pandemic on the city-level count of medical occupations

(a) Log nurses



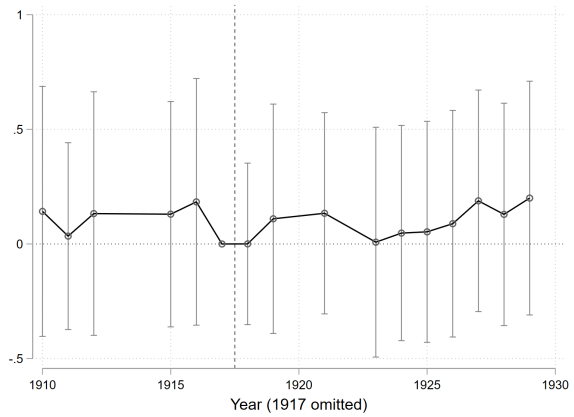
(b) Log doctors and nurses



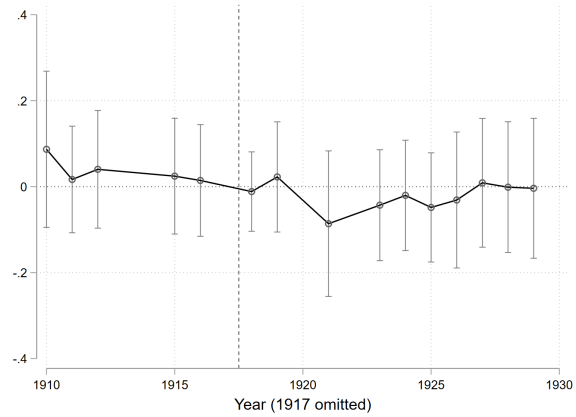
These figures show the effects of pandemic severity on decadal log occupation counts of the indicated occupations. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within the sample of cities successfully linked to the census. The sample is limited to cities in the bottom 75 percentile of the population distribution in the linked hospital-mortality data. The outcome variables are labeled in each panel. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure A2: Effect of the 1918 flu pandemic on local government health spending, bottom 75 percentile sample

(a) Health spending, discrete treatment

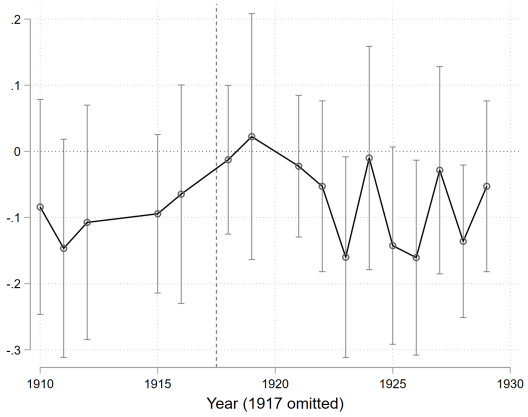


(b) Health spending, continuous treatment



(c) Had health outlay, discrete treatment

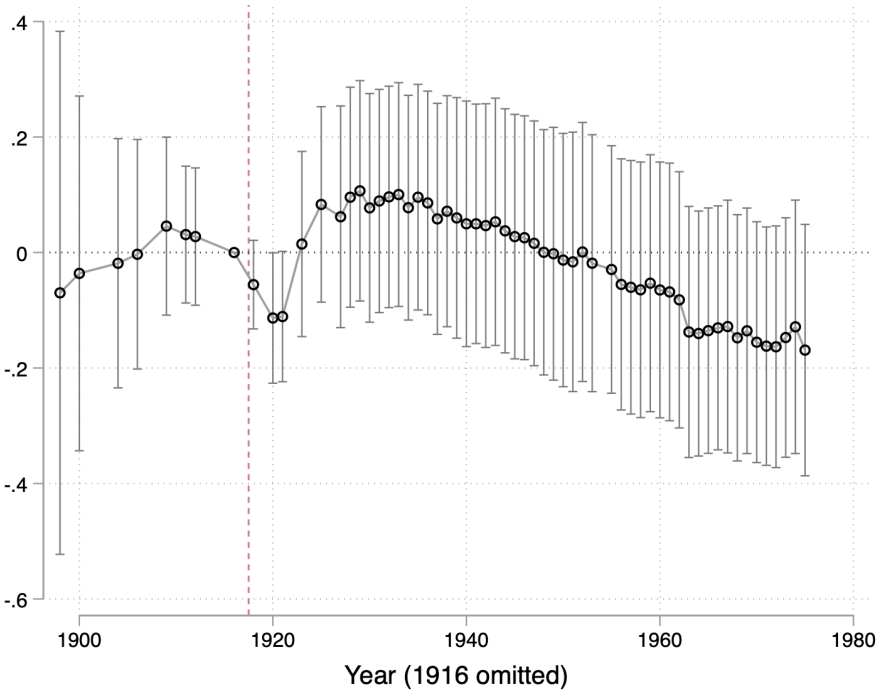
(d) Had health outlay, continuous treatment



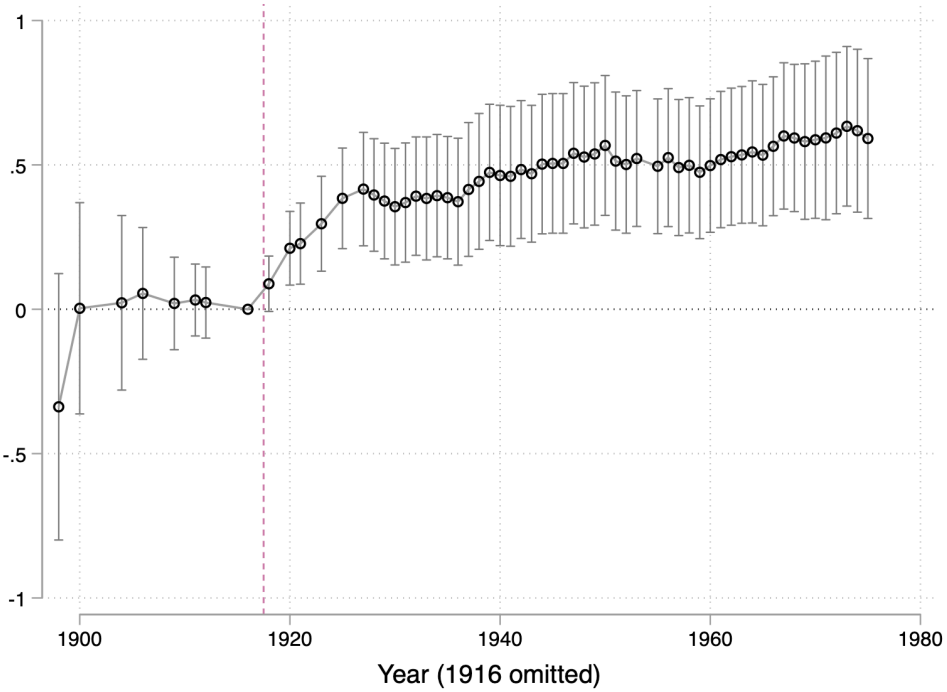
These figures show the effects of pandemic severity on alternative measures of government spending in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within this sample of cities or a continuous measure of mortality (measured in standard deviation units), as labeled in each panel. The outcome variables are labeled 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 A3: Effect of the 1918 flu pandemic on non-government hospitals (heterogeneity by city size)

(a) Above median population



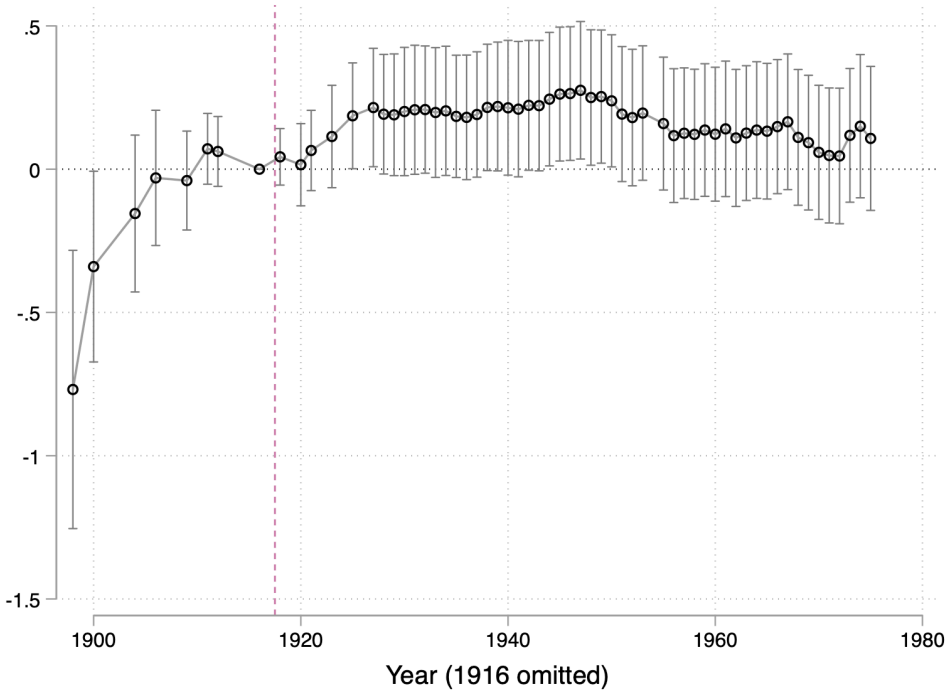
(b) Below median population



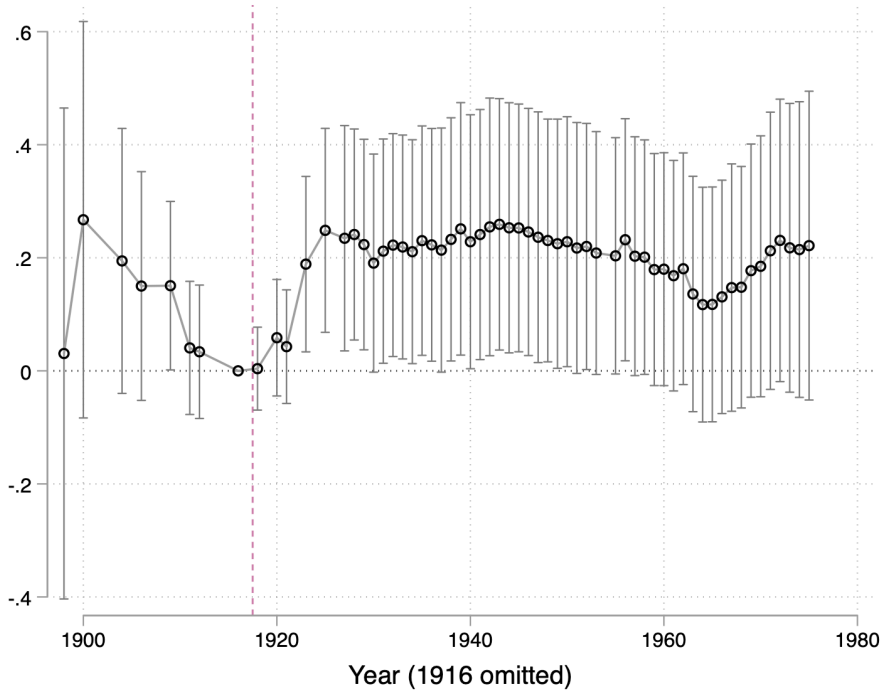
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within the overall sample of cities. The sample is limited to the bottom 75 percentile of cities and each panel shows results for the top and bottom half of the city-size distribution within that subsample. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure A4: Effect of the 1918 flu pandemic on non-government hospitals (heterogeneity by city occupation scores)

(a) Above median occupation score



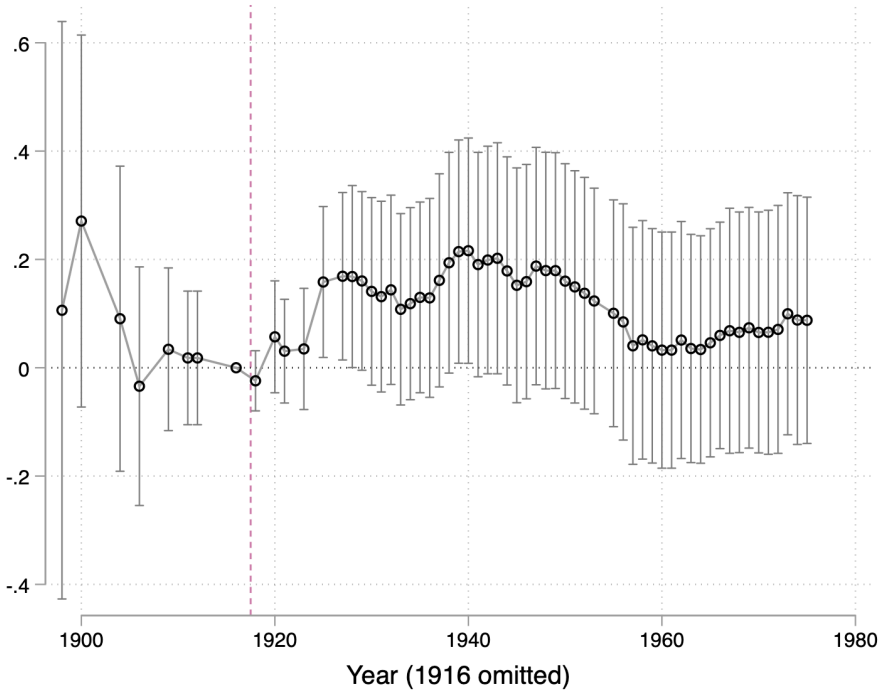
(b) Below median occupation score



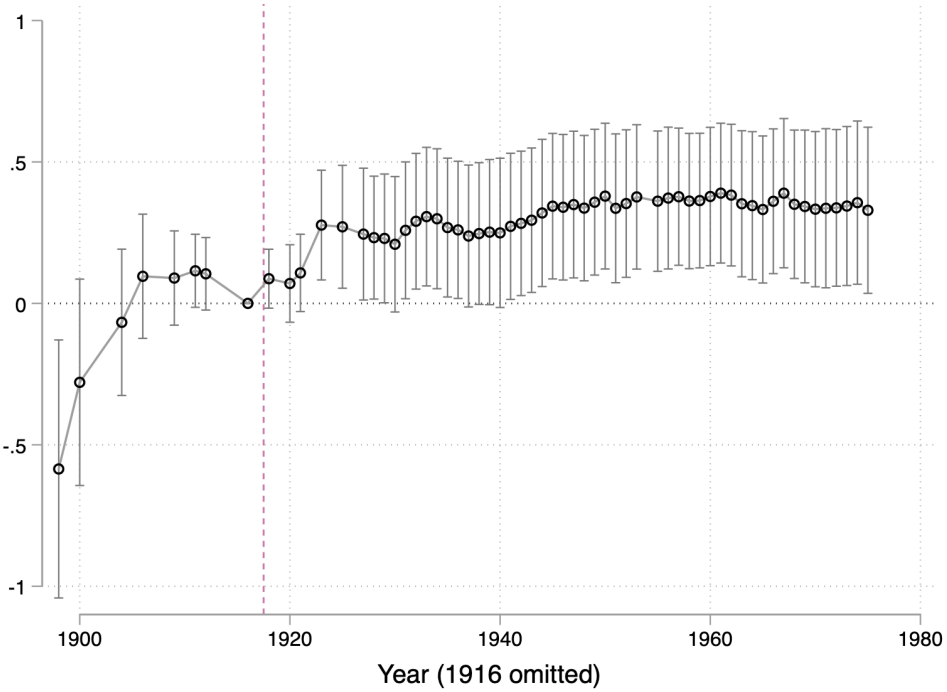
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. Panel A shows results for cities with above median occupation scores, panel B for cities with below median occupation scores. The sample is limited to the bottom 75 percentile of cities and each panel shows results for the top and bottom half of the occupation score distribution within that subsample. 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 A5: Effect of the 1918 flu pandemic on non-government hospitals (heterogeneity by city share foreign-born)

(a) Above median share foreign-born



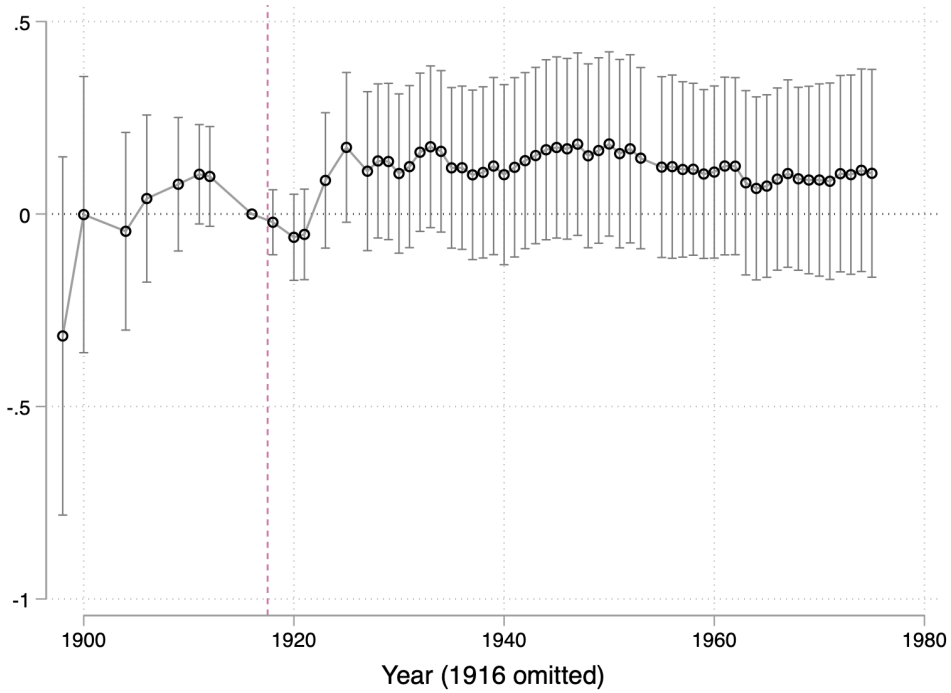
(b) Below median share foreign-born



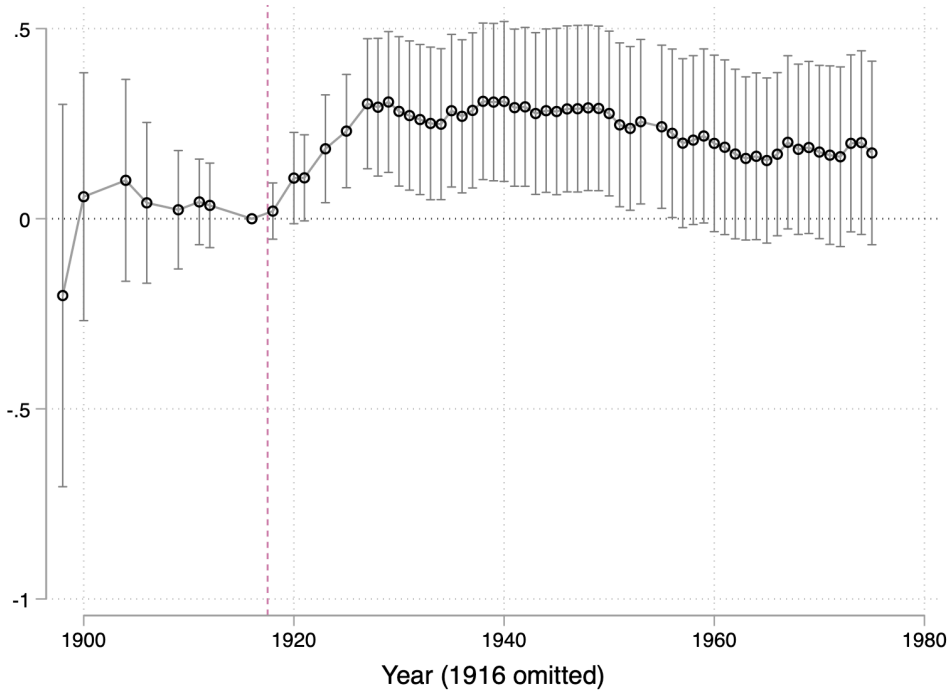
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. Panel A shows results for cities with above median share foreign-born residents, panel B for cities with below median share foreign-born residents. The sample is limited to the bottom 75 percentile of cities and each panel shows results for the top and bottom half of the foreign-born distribution within that subsample. 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 A6: Effect of the 1918 flu pandemic on non-government hospitals (heterogeneity by share population Black)

(a) Above median share Black



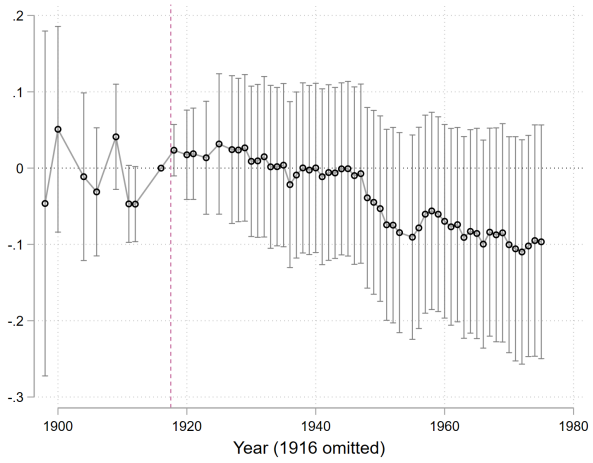
(b) Below median share Black



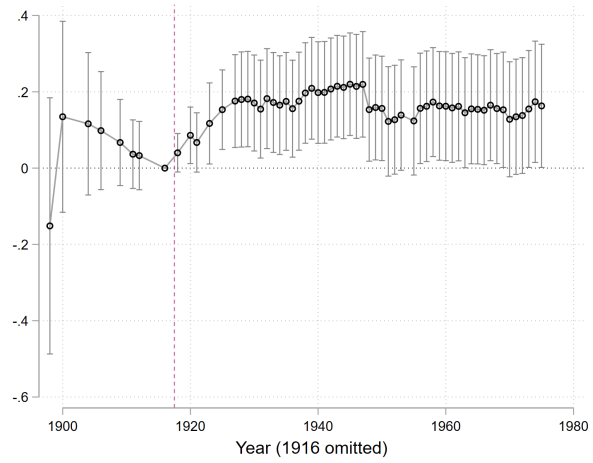
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. Panel A shows results for cities with above median share Black residents, panel B for cities with below median share Black residents. The sample is limited to the bottom 75 percentile of cities and each panel shows results for the top and bottom half of the Black resident share distribution within that subsample. 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 A7: Effect of the 1918 flu pandemic on Poisson count of hospitals, stricter hospital definition

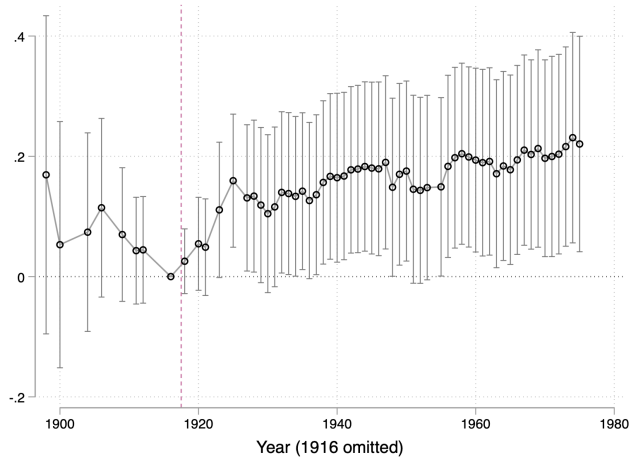
(a) All cities



(b) Bottom 75 percentiles of cities by population



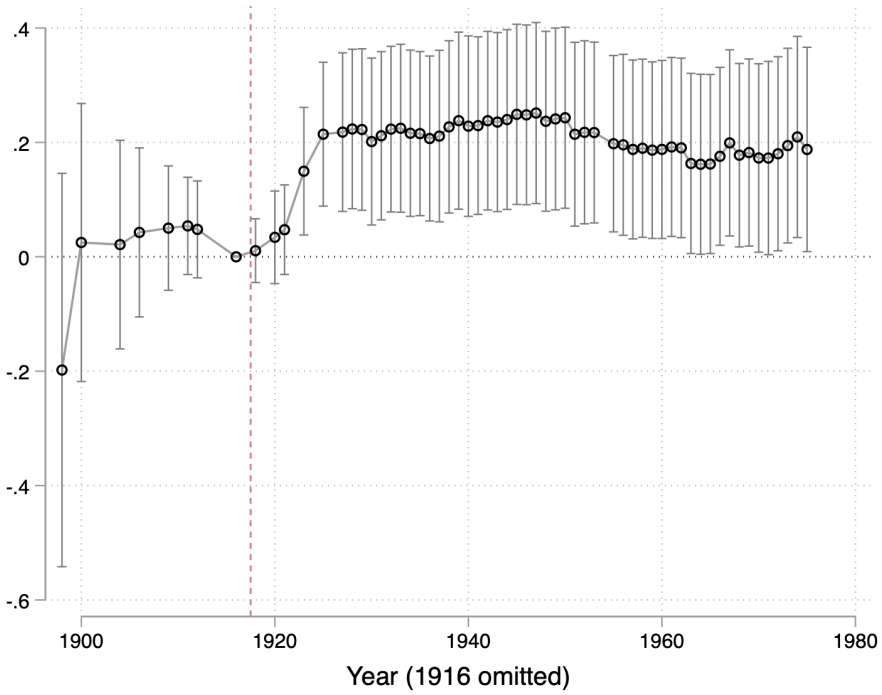
(c) Bottom 75 percentiles of cities by population, non-government hospitals



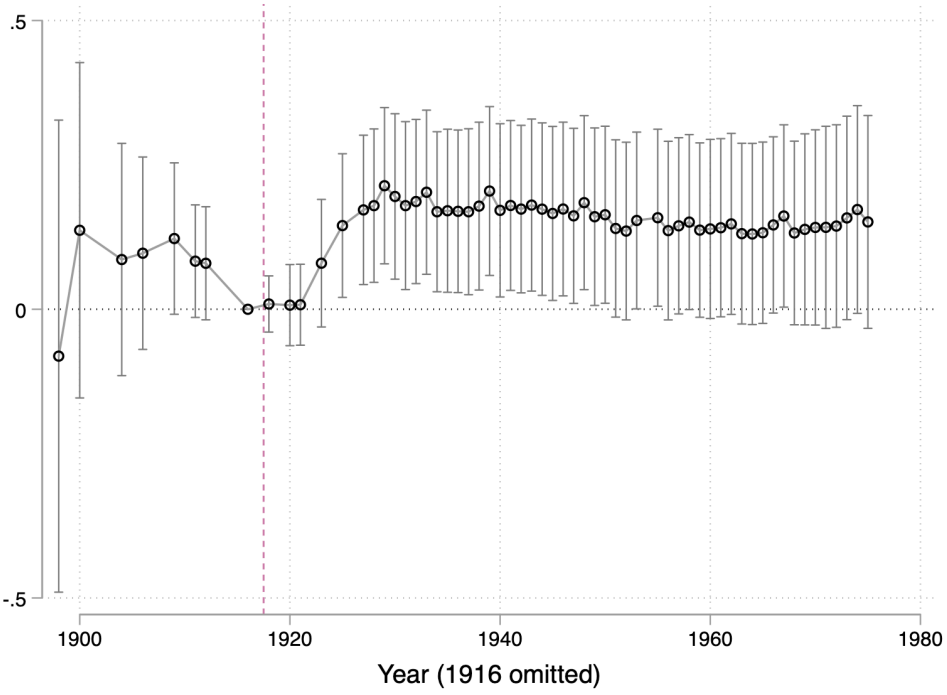
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. 95-percent confidence intervals are shown and standard errors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

Figure A8: Effect of the 1918 flu pandemic, by non-government hospital size

(a) All hospitals



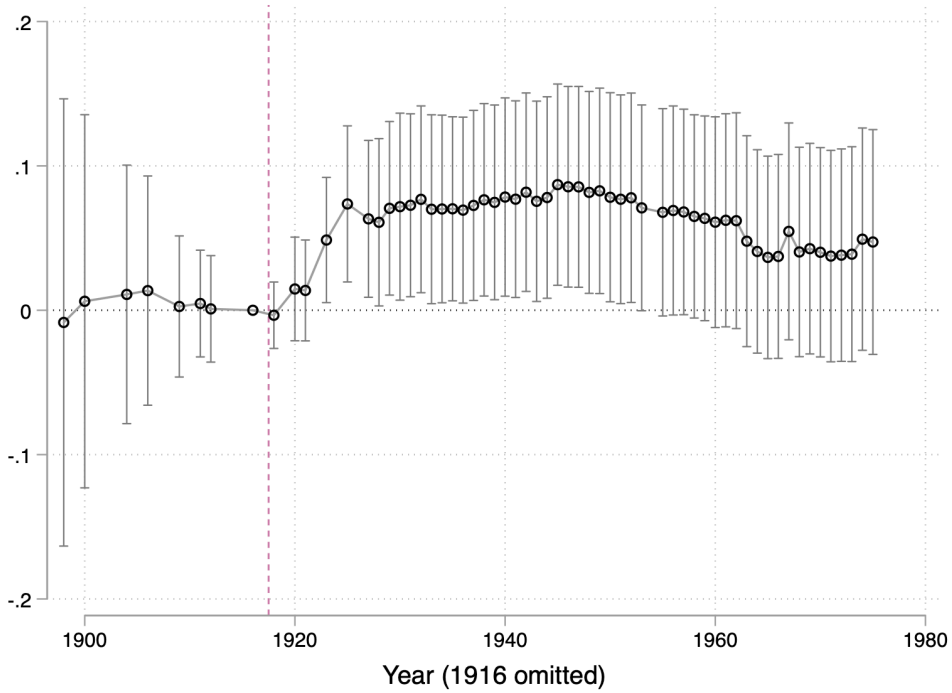
(b) Hospitals greater than 25 beds



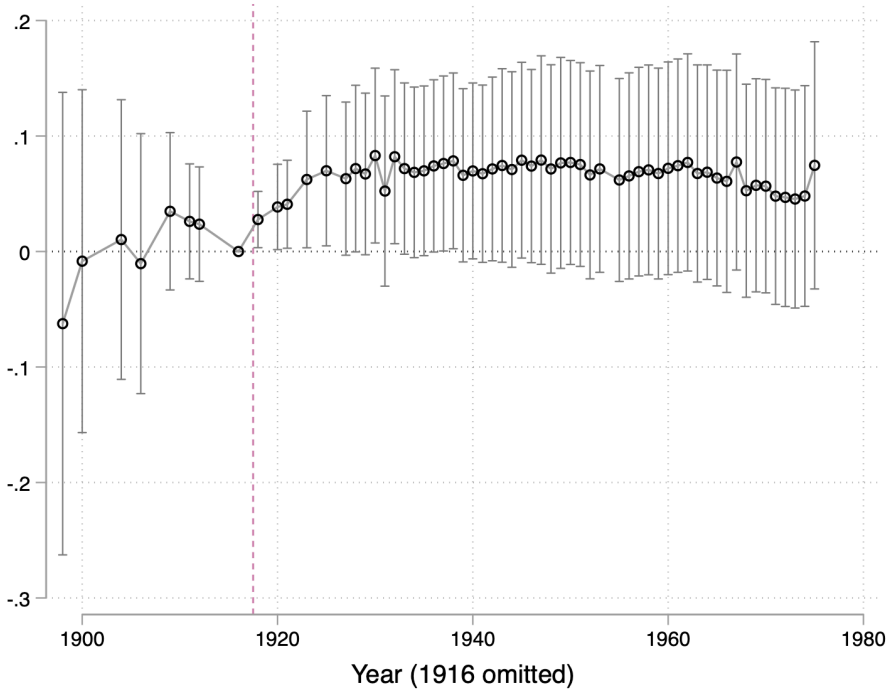
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. Panel A shows results for all hospitals, panel B for hospitals with more than 25 beds. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution within the overall sample of cities. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure A9: Effect of the 1918 flu pandemic on non-government hospitals and hospital beds (continuous treatment)

(a) Non-government hospitals



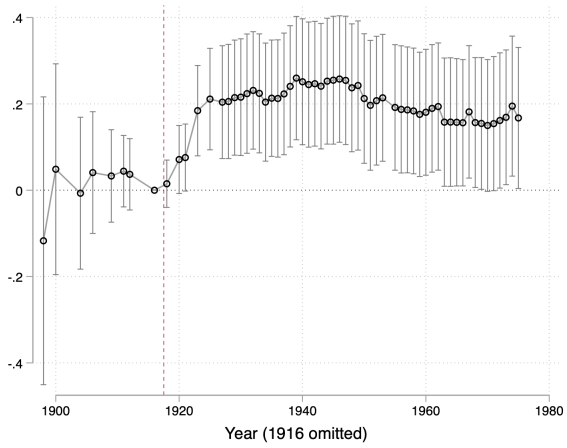
(b) Non-government hospital beds



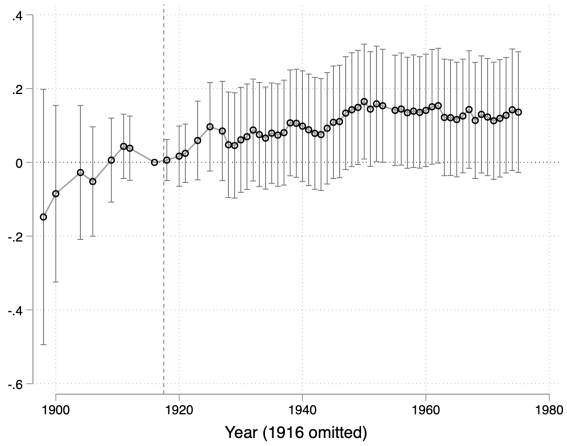
These figures show the effects of pandemic severity on hospitals and hospital beds in a given city, as estimated by the model in Equation 1. The treatment variable is a continuous measure of pandemic mortality normalized to show a 1-standard deviation increase in death rates. The outcome variables are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

Figure A10: Effect of the 1918 flu pandemic on non-government hospitals, alternative mortality measures

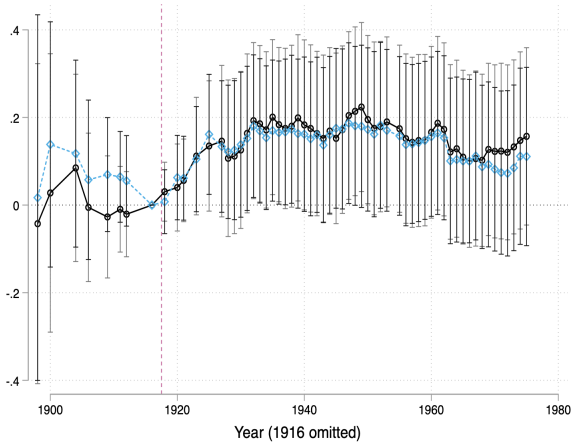
(a) Non-government hospitals, 1918-19 mortality



(b) Non-government hospitals, excess mortality



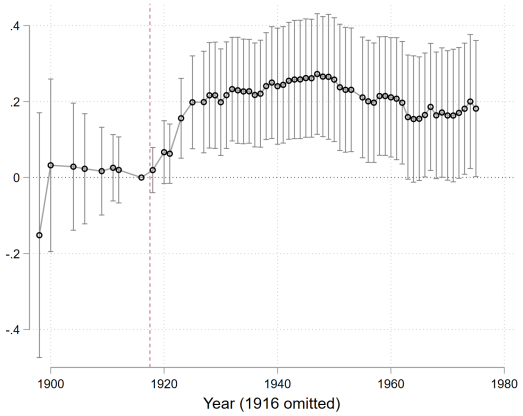
(c) Non-government hospitals, 1918 mortality thirds



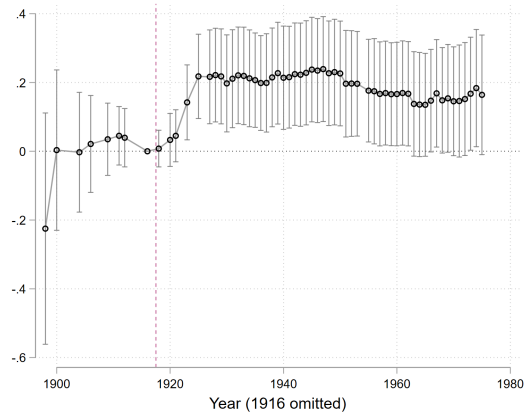
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Panel A uses 1918-19 mortality to make this calculation, Panels 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 blue, the middle third in black. Effects are relative to the omitted third and 1916, the omitted year. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure A11: Effect of the 1918 flu pandemic on non-government hospitals, specification robustness

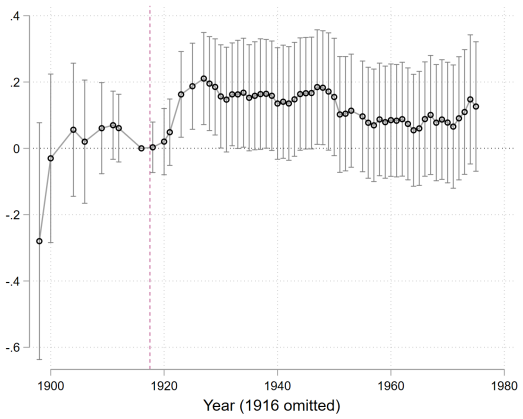
(a) Exclude interacted controls



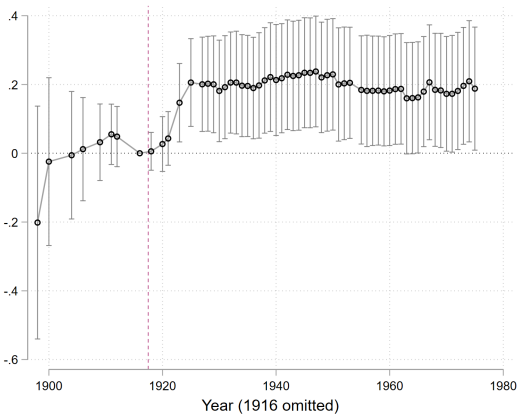
(b) Exclude interacted controls and population



(c) State-year fixed effects



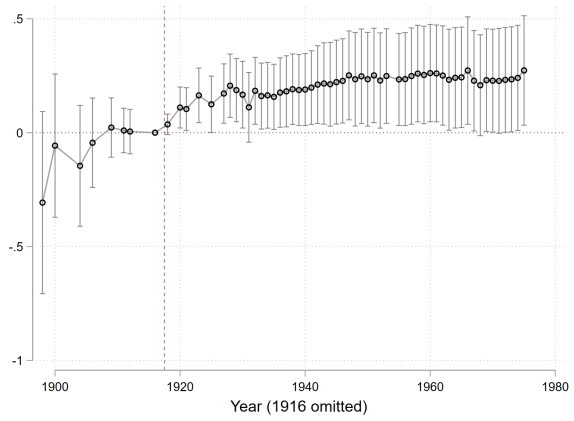
(d) Include 1930 WWI controls



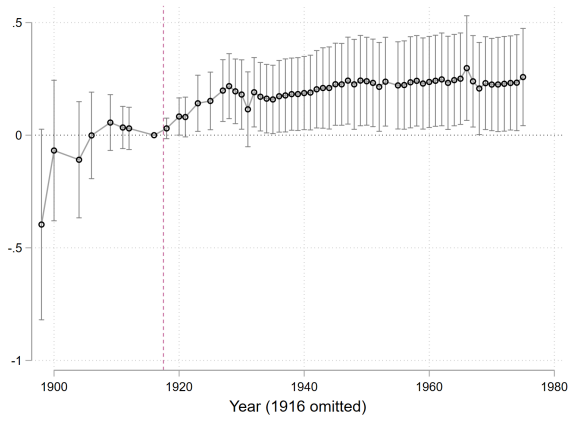
These figures show the effects of pandemic severity on the number of hospitals in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each panel makes the indicated change to the baseline regression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

Figure A12: Effect of the 1918 flu pandemic on non-government hospital beds, specification robustness

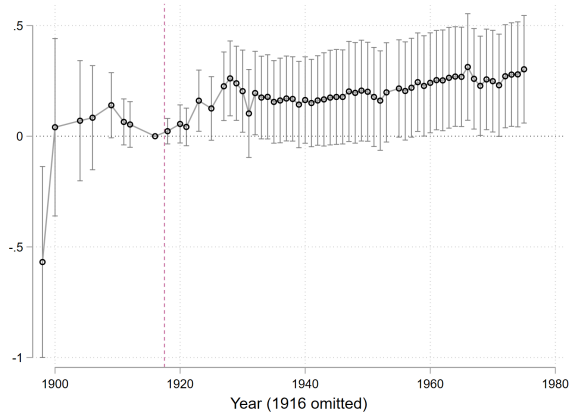
(a) Exclude interacted controls



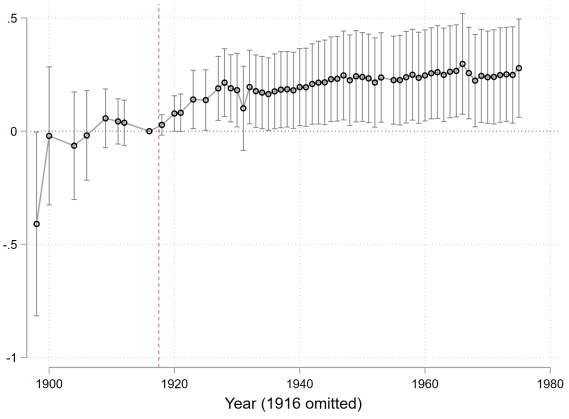
(b) Exclude interacted controls and population



(c) State-year fixed effects



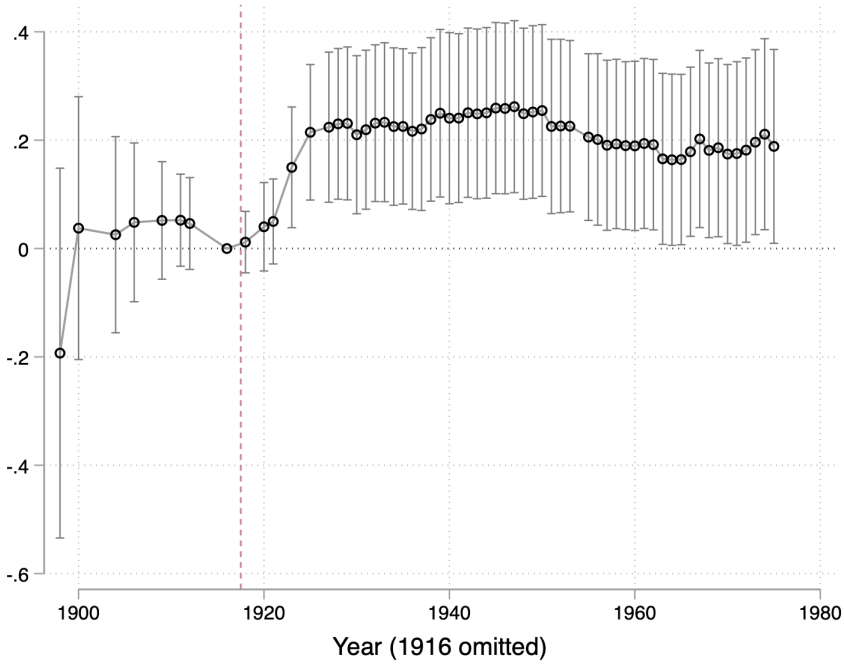
(d) Include 1930 WWI controls



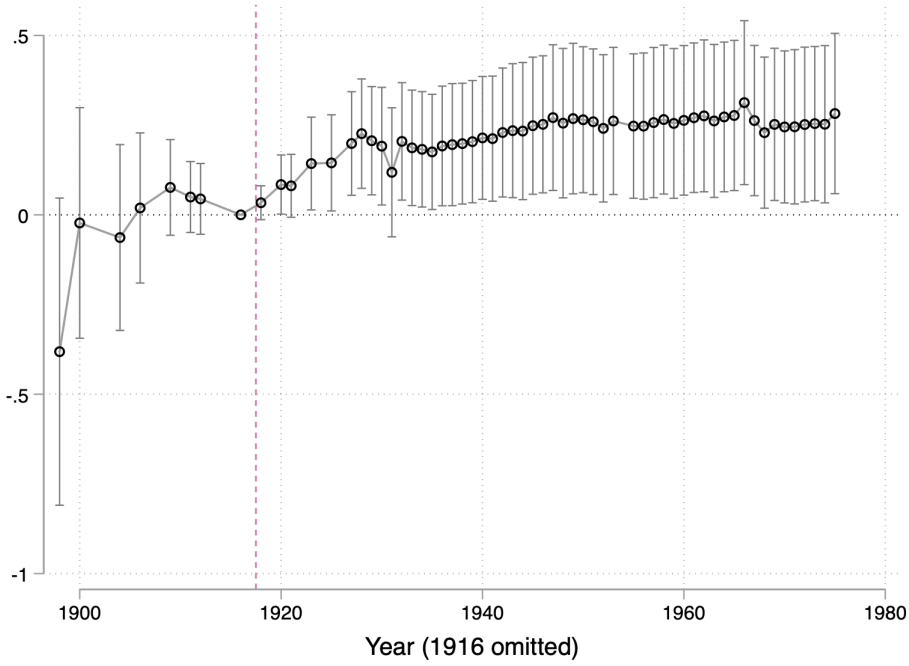
These figures show the effects of pandemic severity on the number of hospital beds in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each panel makes the indicated change to the baseline regression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

Figure A13: Effect of the 1918 flu pandemic, conditioning on Hill-Burton funding

(a) Non-government hospitals



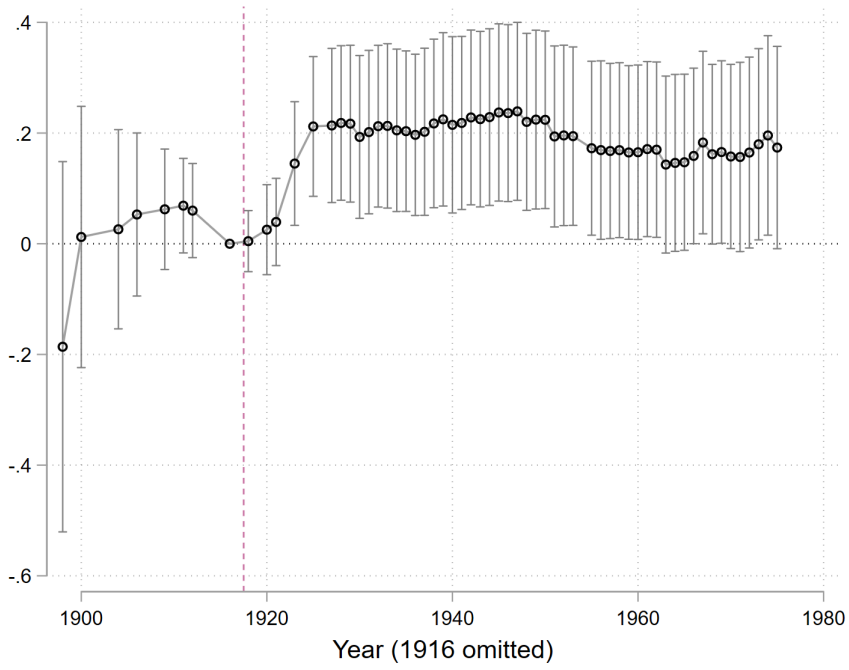
(b) Non-government hospital beds



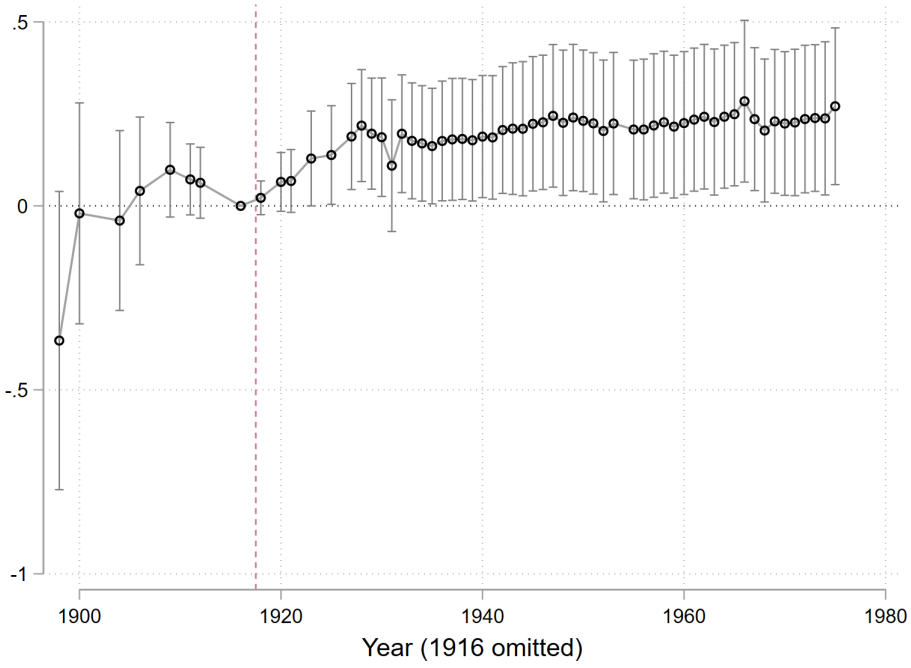
These figures show the effects of pandemic severity on the number of hospitals and hospital beds in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. For this analysis, we include logged Hill-Burton funding interacted with year fixed effects as a control variable. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure A14: Effect of the 1918 flu pandemic, controlling for log time-varying population

(a) Non-government hospitals



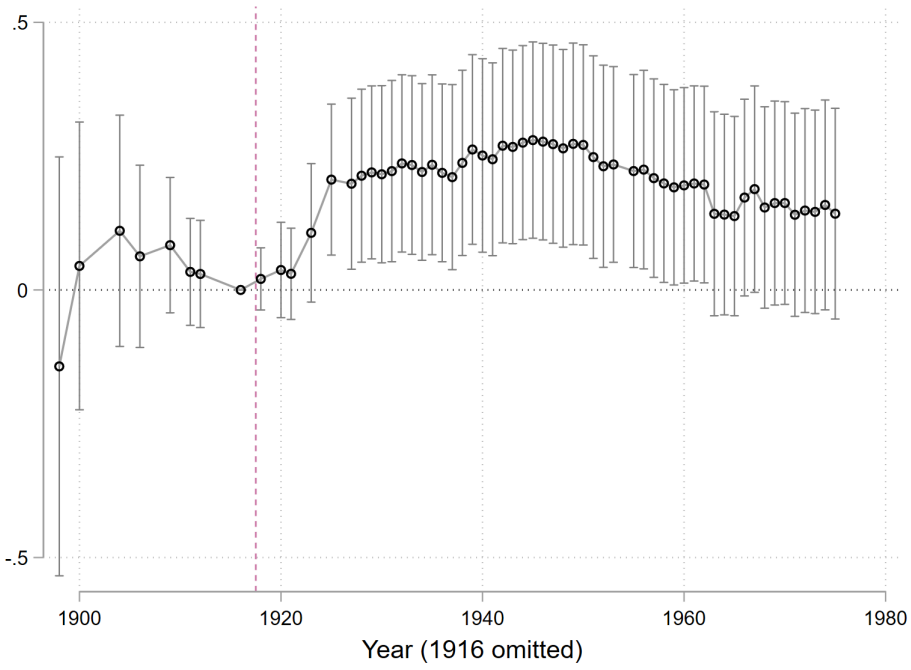
(b) Non-government hospital beds



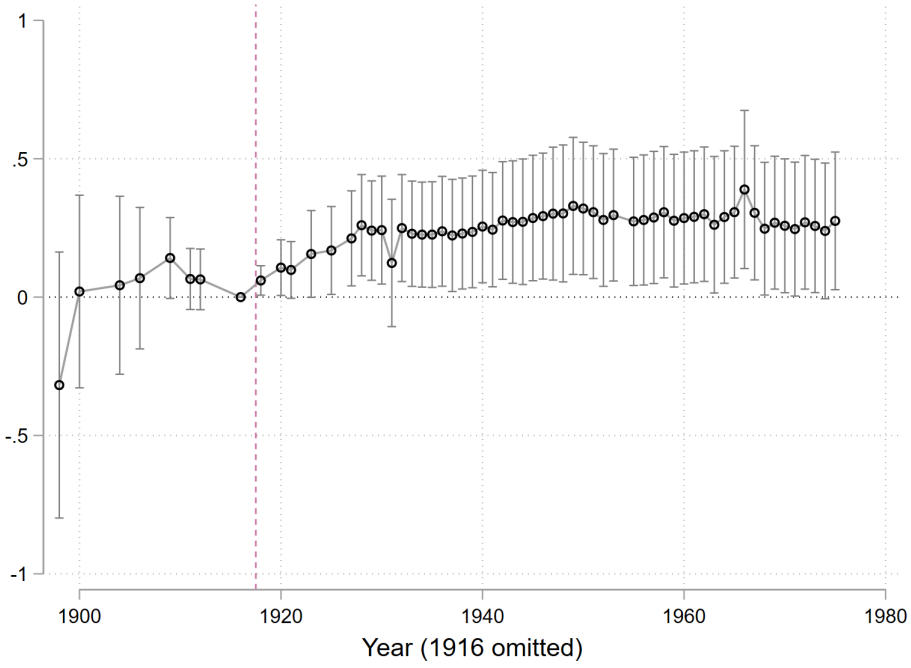
These figures show the effects of pandemic severity on the number of hospitals and hospital beds in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. For this analysis, we include logged time varying population (interpolated between census years) as a control variable. 95-percent confidence intervals are shown and standard errors are clustered by city.

Figure A15: Effect of the 1918 flu pandemic, excluding places with a pre-1923 government hospital

(a) Non-government hospitals



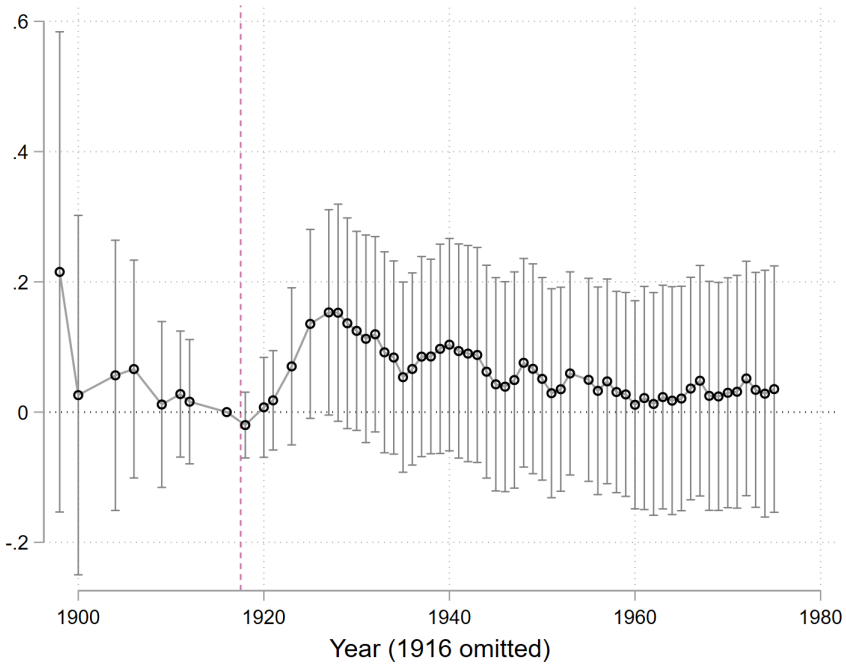
(b) Non-government hospital beds



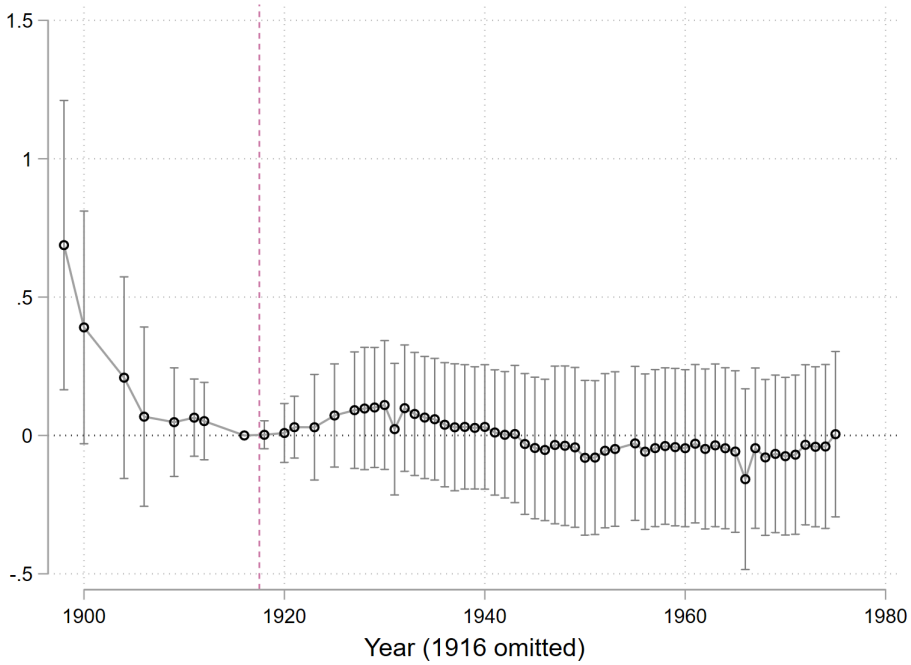
These figures show the effects of pandemic severity on the number of hospitals and hospital beds in a given city, as estimated by the model in Equation 1. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution. For this analysis, we drop all places that had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

Figure A16: Placebo test using cancer as city-level treatment

(a) Hospitals



(b) Hospital beds



These figures show the effects of pandemic severity on the number 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. 95-percent confidence intervals are shown and standard errors are clustered by city.

Table A1: Summary statistics

	Full sample		Bottom 75% by city size	
	Mean	Std. Dev.	Mean	Std. Dev.
City-year variables (N = 27,531)				
Hospitals	4.374	10.250	2.033	1.568
Government hospitals	0.734	1.533	0.396	0.671
Non-government hospitals	3.640	9.008	1.637	1.354
Hospital beds	750.115	2532.717	253.092	290.372
Government hospital beds	211.932	936.711	58.885	172.399
Non-government hospital beds	538.183	1659.151	194.207	205.386
Had a hospital	0.952	0.214	0.937	0.243
Time-invariant variables (N = 462)				
1918 flu death rate per 1000	7.049	2.814	7.124	3.011
1918 cancer death rate per 1000	0.973	0.383	0.995	0.420
1917 city population (1000s)	81.783	31.540	23.276	10.338
1910 time-invariant variables (N = 462)				
Share Black	0.068	0.125	0.065	0.125
Average OCCSCORE	24.347	1.640	24.386	1.711
Average age	28.256	2.101	28.377	2.232
Share in labor force	0.630	0.043	0.625	0.044
Share professionals	0.051	0.016	0.052	0.017
Share farmers	0.008	0.011	0.009	0.012
Share managers	0.066	0.016	0.068	0.017
Share clerical	0.062	0.020	0.059	0.020
Share sales	0.063	0.018	0.061	0.018
Share craftsmen	0.173	0.048	0.174	0.052
Share operatives	0.180	0.104	0.180	0.106
Share service	0.124	0.061	0.122	0.062
Share farm laborers	0.014	0.011	0.015	0.012
Share WW1 veterans (1930)	0.034	0.007	0.033	0.007

Notes: This table shows summary statistics for the main variables and sample used in this paper. We show separate statistics for the full sample of cities and the sample of cities in the bottom 75% of the city size distribution. 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 A2: Effect of the pandemic on hospitals, bottom 75 percentile population cities

Dependent variable and sample	Flu mortality × post	Std. error	Mean of outcome
Hospitals			
All years	0.124	(0.057)	2.032
Pre-1960 sample	0.134	(0.058)	2.083
Pre-1950 sample	0.139	(0.057)	2.080
Non-government hospitals			
All years	0.183	(0.065)	1.637
Pre-1960 sample	0.193	(0.064)	1.668
Pre-1950 sample	0.194	(0.063)	1.673
Government hospitals			
All years	0.034	(0.167)	0.396
Pre-1960 sample	0.057	(0.167)	0.416
Pre-1950 sample	0.070	(0.161)	0.409

Notes: This table shows results from our baseline model, which estimates the effect of the pandemic on the 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 and the effect is estimated using a Poisson model. Standard errors are clustered by city.

Table A3: Effect of the pandemic on non-government hospitals, alternative hospital measures, bottom 75 percentile population cities

Dependent variable and sample	Flu mortality × post	Std. error	Mean of outcome
Non-government hospitals			
Full sample	0.183	(0.065)	1.637
Pre-1960 sample	0.193	(0.064)	1.668
Pre-1950 sample	0.194	(0.063)	1.673
Non-government beds			
Full sample	0.223	(0.091)	194.207
Pre-1960 sample	0.208	(0.085)	155.482
Pre-1950 sample	0.193	(0.080)	138.166
Have a non-government hospital			
Full sample	0.078	(0.032)	0.872
Pre-1960 sample	0.075	(0.032)	0.872
Pre-1950 sample	0.077	(0.031)	0.867

Notes: This table shows results from our baseline model on alternative measures of non-governmental hospitals. The treatment variable is an indicator for being in the top 50 percent of the mortality distribution in 1918. The hospital count and bed results are estimated using a Poisson model. The probability of having a hospital result is estimated using a linear probability model. Standard errors are clustered by city.

Table A4: City-level heterogeneity and the effects of the pandemic on non-governmental hospitals, bottom 75 percentile population cities

Het. variable and sample	Flu mortality × post	Std. error
Full sample		
Size of city		
Top-half	-0.015	(0.090)
Bottom-half	0.465	(0.096)
Occupational income ranking		
Top-half	0.220	(0.087)
Bottom-half	0.130	(0.097)
Share Black population		
Top-half	0.098	(0.098)
Bottom-half	0.211	(0.086)
Share foreign-born population		
Top-half	0.078	(0.083)
Bottom-half	0.288	(0.105)
Pre-1950		
Size of city		
Top-half	0.049	(0.086)
Bottom-half	0.413	(0.093)
Occupational income ranking		
Top-half	0.256	(0.086)
Bottom-half	0.145	(0.092)
Share Black population		
Top-half	0.103	(0.095)
Bottom-half	0.242	(0.081)
Share foreign-born population		
Top-half	0.114	(0.078)
Bottom-half	0.246	(0.103)

Notes: This table shows results from our baseline model estimating the effect of the pandemic on the 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. Standard errors are clustered by city.

Table A5: Robustness checks for the pandemic's effect on healthcare provision

Specification	Flu mortality × post	Std. error
Non-government hospitals		
Baseline results	0.183	(0.065)
Stricter hospital definition	0.130	(0.067)
Exclude 1910 controls	0.198	(0.065)
With time-varying pop.	0.164	(0.065)
With 1930 controls	0.183	(0.066)
With state-year fixed effects	0.108	(0.067)
1918-1919 flu mortality treatment	0.183	(0.063)
Excess deaths mortality treatment	0.110	(0.064)
Exclude places with pre-1923 gov hosp.	0.174	(0.075)
Condition on Hill-Burton funding	0.187	(0.065)
Continuous mortality treatment (1 SD)	0.062	(0.029)
Non-government beds		
Baseline results	0.223	(0.091)
Stricter hospital definition	0.103	(0.083)
Exclude 1910 controls	0.237	(0.095)
With time-varying pop.	0.196	(0.084)
With 1930 controls	0.221	(0.090)
With state-year fixed effects	0.189	(0.100)
1918-1919 flu mortality treatment	0.147	(0.091)
Excess deaths mortality treatment	0.242	(0.092)
Exclude places with pre-1923 gov hosp.	0.240	(0.109)
Condition on Hill-Burton funding	0.228	(0.091)
Continuous mortality treatment (1 SD)	0.050	(0.037)

Notes: This table shows results from variations on our baseline model estimating the effect of the pandemic on the number of non-governmental hospitals or beds. The baseline treatment variable is an indicator for being in the top 50 percent of the mortality distribution. Each row makes the indicated change to the baseline specification. Sample is limited to the bottom 75 percentile population cities. Standard errors are clustered by city.