Pollution, Infectious Disease, and Infant Mortality: Evidence from the 1918-1919 Spanish Influenza Pandemic *

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Abstract

This paper uses the 1918 influenza pandemic to study how local environmental factors interact with an infectious disease outbreak to affect health. There were large geographic differences in pandemic-related mortality that cannot be explained by either demographic or economic factors. We examine whether local environmental factors affected pandemic severity. The empirical analysis combines newly digitized information on infant mortality, proxies for pollution from thermal power generation, and water quality for a panel of 559 American cities. We estimate a significant positive effect of local emissions on pandemic-related mortality, but find no relationship between the outbreak and local water quality. These results are consistent with the epidemiology of the influenza virus, which targeted lung function. Our estimates imply that differences in pollution levels can account for roughly 18 percent of the cross-city variation in pandemic severity. We then evaluate the impact of the pandemic on infant mortality in a counterfactual scenario in which all above-median cities reduced pollution to median levels. In this scenario, we calculate that 5,551 infant deaths would have been averted: a 13 percent decline in pandemic-related mortality.

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1 Introduction

The mortality rate of many infectious illnesses varies widely across countries and over time. A growing literature emphasizes the importance of baseline health in explaining differences in pathogen severity (see Miller et al., 2002, for example).[1] Local environmental factors, such as the availability of clean water, air quality, and access to proper nutrition may affect the stock of health capital, and hence pathogen severity. From a theoretical perspective, however, the direction of these interaction effects is ambiguous. For example, exposure to air pollution may lower baseline health, making individuals more susceptible to the consequences of a negative health shock. On the other hand, strong selection effects could raise average health capital, leading a population to be more resistant to the consequences of an infectious disease outbreak. Understanding the interrelation between these various factors and disease onset has implications for health policy and may yield important insights into the health production process (Currie, et al., 2013).

This paper examines how local environmental factors interact with an infectious disease outbreak to affect health. We study these questions in the context of the 1918-1919 Spanish influenza pandemic, an unanticipated, short-lived, and severe outbreak that killed an estimated 675,000 Americans.[2] There was wide regional variation in pandemic severity that cannot be attributed to economic development, climate or geography (Brainerd and Siegler, 2003, p.11). One environmental factor that may have influenced the severity of the "Spanish flu" was air quality. In the early 20th century, pollution levels in cities were high.[3] There were large regional differences in pollution levels driven by the local availability of coal for production and energy generation.

This setting offers a unique opportunity to study health interaction effects. First, the

[1] A related literature documents the relationship between early childhood diseases and long run health outcomes (see Costa, 2000; Case and Paxson, 2009).

[2] Worldwide 30 to 50 million people are estimated to have died from the pandemic (Crosby, 1989; Johnson and Mueller, 2002).

[3] The small amount of available data on air pollution in American cities around this period suggests that pollution levels were of the same order of magnitude to pollution in Chinese cities in the 1980s.

pandemic occurred during a period in which there was limited scope for medical interventions. Since this time period preceded the environmental movement by several decades, concerns regarding sorting across cities on the basis of health preferences are also mitigated. Second, the H1N1 strain responsible for 1918 outbreak was the result of an antigenic shift, so few individuals had developed antibodies to protect against the new virus.[4] Given that 1918 marked the first outbreak of H1N1, it is not necessary to control for previous exposure to influenza. Third, the pandemic was short-lived, limiting the scope for viral evolution.[5] We are also able to exploit historical information on the timing of pandemic onset across localities to directly control for evolution in influenza severity. Finally, there were large differences in city-level pollution exposure, allowing for a cross-city comparison in pandemic severity.

To examine the interaction effects of pollution and influenza on health, we rely on a newly digitized dataset on infant mortality for a panel of 559 American cities for the period 1915 to 1925. These data are combined with a measure of baseline city-level pollution, which is derived from a 1915 federal report that provides information on the location and capacity of steam and hydroelectric power stations in the United States. The empirical analysis evaluates the extent to which heterogeneity in infant mortality during the influenza pandemic was related to local levels of pollution using city-level variation in emissions arising from electricity generation. The time series identification comes from interacting an indicator variable for 1918, the main year of the pandemic, with the city-level steam capacity. All specifications include city and year fixed effects, and many specifications include additional controls that allow for differential trends based on geographic, demographics, and economic characteristics.

The main results suggest that local pollution exacerbated the impact of influenza pan-

[4]In contrast to the more common antigenic drift, when an existing influenza virus mutates and is no longer recognized by the immune system, antigenic shift occurs when an entirely new strand of the virus is introduced the human population. Transmission typically occurs either directly or indirect from avian strands of influenza. Antigenic shift often results in large scale pandemics, typically infecting between 15 an 40 percent of the population, and other examples of antigenic shift include the 1889, 1957, and 1968 influenza outbreaks.

[5]The pandemic lasted from September, 1918 to April, 1919, with 83% of pandemic-related mortality occurring during the first four months of the outbreak.

demic. We find a large and statistically significant relationship between local steam capacity and infant mortality in 1918, which are robust to a variety of alternative estimation strategies. On the other hand, we find no relationship between local water quality – as proxied by city-level typhoid rates – and pandemic severity. Together these results are consistent with epidemiological pathways of the disease, which targeted lung function. To interpret the magnitude of these effects, we consider a counterfactual scenario, in which all above median pollution cities reduced emission to the median level. In this setting, we calculate 5,551 pandemic-related infant deaths would have been averted, a 13 percent decline in influenza pandemic severity. The results have implications for current energy policy, and suggest that focusing solely on the direct relationship between TSP emission on health may understate the benefits of emissions abatements policies.

The paper proceeds as follows. Section 2 discusses the history of the 1918 influenza pandemic; section 3 describes the data; section 4 introduces the empirical strategy; section 5 presents the main findings; section 6 reports a variety of robustness exercises; and section 7 concludes.

2 Historical context

The influenza pandemic of 1918 was brief, but severe. The estimates of worldwide fatalities ranged from 50-100 million. In the United States, fatalities were between 675,000 and 850,000. The pandemic occurred in three waves, the second and most severe wave began in late September of 1918. The pandemic was extremely severe in October through January, and mortality remained elevated in February of and March of 1919. The 1918 pandemic lives on in the sense that nearly all influenza A cases are descendants of that virus.

Pregnant women were particularly vulnerable. They died, had stillbirths, and saw their infants die. Maternal mortality in the registration area rose from 0.66 percent to 0.92 percent. Almond (2006) found that stillbirths increased 60 percent in October 1918 and 40 percent

over the period October-December 1918. The effect of influenza on pregnant women and fetuses was not unique to this pandemic, although the effects were more severe. Influenza has long been associated with maternal and fetal death. Almond (2006) presents evidence on the adverse effects of having been in utero during the pandemic. Across a wide range of health and economic indicators, the cohort that was in utero during the pandemic did worse than adjacent cohorts.

Case fatality rates during the 1918 pandemic were greater than 2.5 percent, far higher than usual (0.1 percent), although most victims survived. Fatalities were similar across rural and urban areas. Victims often experienced hemorrhaging or rapid and severe onset of bacterial pneumonia. There were wide cross-state and cross-city differences in pandemic severity that cannot be attributed to economic, demographic, geographic or climatic factors (Crosby, 1989, p.66). One study of the pandemic concluded that excess mortality rates "appear to be randomly distributed and do not seem to be related to the level of economic development, climate, or geography (Brainerd and Siegler, 2003, p.11).

Three factors were identified as potentially important contributors to influenza severity. The first is air pollution. A relationship between air pollution and influenza has been suggested by a small number of authors (Pope and Dockery 2006, Xu et al 2013, Lee et al 2014, Liang et al 2014). The underlying mechanism is that particulate pollution and particularly environmentally persistent free radicals can some degree of immunosuppression in infants, increasing the severity of influenza.

A second is proximity to World War I bases. There is considerable qualitative evidence to suggest that movement of military personnel spread influenza from base to base and from bases to adjacent towns and cities (Morens and Fauci 2007, Erkoreka 2009, Wever and Bergen 2014). Given that the deadly second wave of the virus spread rapidly to virtually every city in America and that the medical response was ineffective, it is not obvious why cities near bases should have been differentially affected by the pandemic. On the other hand, there is qualitatitive evidence that the virus weakened by late September (Sydenstricker, 1918), so

that the first cities struck by the pandemic were exposed to a more lethal strain.

The third factor is pre-pandemic infant mortality. Some work suggests that pre-pandemic mortality is predictive of pandemic mortality (Pearl 1921, Bootsma and Ferguson 2007, Acuna-Soto et al 2011, Chowell et al 2008). This is consistent with baseline population health affecting incidence of influenza. It is likely to capture a variety of factors including population density, pollution, and health and sanitation. While there is some evidence that typhoid might be relevant for infant mortality, it is likely to be captured by pre-pandemic infant mortality.

3 Data

To examine the interaction effects of pollution and influenza on health, we combine newly digitized data on infant mortality and electricity generation with additional census data on city characteristics. Infant mortality rates were digitized for a panel of 559 American cities for the period 1915-1925 (excluding the year 1920, for which we lack information on infant deaths). This data comes from the *Mortality Statistics*, and covers 25 percent of the U.S. population and over half of the urban population.

These health outcomes are linked to a measure of city-level pollution. We rely on a 1915 federal report that provides information on the location and capacity of steam and hydroelectric power stations in the United States. We digitized information on all plants with at least 5mw of capacity in 1915, covering over half of the total capacity generated in the United States. Using GIS software, we combined these data with information on city locations to construct a measure of local exposure to pollution from steam plant emissions. Our preferred measures are total steam capacity and hydro capacity within 30 miles of each city-centroid. Additional controls include longitude, latitude, timing of the onset of the influenza pandemic, population, urban population, share white, and employment in manufacturing, and manufacturing wages. Demographic and manufacturing data are at the

county level and are from the 1910 Census of Population and 1910 Census of Manufacturing, with the exception of manufacturing wages, which is from 1900.

In addition to pollution, we assess several other explanations for cross-city heterogeneity in pandemic-related mortality. We examine whether proximity to major World War I bases affected pandemic severity. We digitize information of the locations of all major army training camps in 1918 (U.S. War Department, 1919, p.1519). For each city the our sample, we construct a measure of distance (in miles) to the distance to the nearest base. Figure 1 displays the sample of cities in our dataset and the location of army training camps. Additionally, we examine whether poor water quality may influenced pandemic severity. To assess this hypothesis, we assemble data on typhoid mortality rates at the turn of the century for 288 cities in our sample from Whipple (1908). Our measure of baseline typhoid exposure is the average number of typhoid deaths per 100,000 population for the period 1900 to 1905.

Table 1 reports the summary statistics for the main variables of interest. We report the sample means for the full sample and separately for cities above and below the median level of pollution in 1915 (24mw of steam capacity). The first row shows that overall infant mortality rates were similar across the two groups of cities. In the second row, we report the logarithm of excess mortality in 1918. This variable is constructed as the residual infant mortality rate after controlling for a city-specific linear trend. Infant mortality was 19% higher in 1918 relative to trend. The influenza pandemic was much more severe in high-pollution cities, and there was a 0.05 log point difference in excess mortality during 1918. High-pollution and low-pollution cities differed on a range of other margins. Baseline infant mortality rates were 7% higher in high-pollution cities and these cities were on average 46 miles closer to a World War I base. High-coal cities were also located in more populous counties with a greater fraction of white inhabitants. The goal of the empirical analysis is to disentangle these competing determinants of pandemic-related mortality.

4 Empirical strategy

The empirical analysis will evaluate whether heterogeneity in infant mortality during the influenza pandemic was related to local levels of pollution. We estimate the following model:

$$\begin{aligned} Log(MortRate_{ct}) = \alpha + \beta\big(Influenza_t \times Log(Pollution_c)\big) \\ + \lambda_t + \lambda_t Z_c + \lambda_t X_c + \lambda_t W_c + \eta_c + \phi_{st} + \epsilon_{ct} \end{aligned} \tag{1}$$

where the dependent variable, $Log(MortRate_{ct})$, denotes the logarithm of the infant mortality rate in city c in year t. The term η_c denotes a vector of city fixed effects, λ_t denotes a set of year fixed effects, and ϕ_{st} is a linear state trend which allows for differential trends in mortality across states.

The variable of interest, $\big(Influenza_t \times Log(Pollution_c)\big)$, identifies the interaction between baseline pollution and the year of the pandemic. The term $Influenza_t$ is a dummy variable for the year 1918, which is meant to proxy pandemic exposure.[6] The term $Log(Pollution_c)$ measures the logarithm of total steam capacity within 30 miles of the city-centroid, and is meant to proxy local pollution. The interaction term, $\big(Influenza_t \times Pollution_c\big)$, identifies the differential impact of local pollution on infant mortality during the pandemic year. A positive estimate of β would suggest that exposure to power plant emissions lowered health capital in the local population, exacerbating the impact of the influenza outbreak.[7] In particular, a positive estimate of β would suggest that exposure to power plant emissions lowered health capital in the local population, exacerbating the impact of the influenza outbreak.

The identifying assumption requires that conditional on covariates unobservable determinants infant mortality are not correlated with $\big(Influenza_t \times Log(Pollution_c)\big)$. In prac-

[6] We also estimate the interaction effect for 1919, given that 17% of pandemic-related mortality occurred between January and April, 1919.

[7] In some specifications, we also report the direct effect of the pandemic on health – the 1918 year fixed effect, $Pollution_t$ – although it cannot be readily interpreted in models that include the full set of covariates. Note that the direct effect of pollution on mortality cannot be separately identified in a model that includes city fixed effects.

tice, this condition requires that pandemic-related mortality was not systematically different across high and low coal cities for reasons unrelated to air quality. There are three primary concerns regarding this hypothesis, which we address in turn.

First, geography may have influenced both the spread and severity of the pandemic. For example, despite the rapid diffusion of the pandemic, some accounts suggest that the virulence of weakened by the end of the September (Sydenstricker, 1918). Since the outbreak occurred later western states, where coal energy production was more limited, we might identify a spurious correlation between pandemic-severity on city-level pollution. To address these concerns, equation (1) includes the term $\lambda_t Z_c$, which denotes the city-centroid longitude and latitude interacted with year. These terms allow for heterogeneity in pandemic-severity according to geography.[8]

A second concern is city-level population characteristics may have influenced the spread of the pandemic and hence the size of the infected population. This issue is particularly salient given coal pollution tended to be concentrated in larger and more densely populated cities (see Table 1). To address this concern, we interact the year fixed effects with a vector of baseline population characteristics, X_c, which include the logarithm of total population in 1910, percent urban in 1910, and the share white in 1910. These terms control for the fact that local pollution levels may be correlated with determinants of viral transmission.

A final concern is that 1918 infant mortality was elevated in high pollution cities as a result of the war. If high coal cities experienced a disproportionate increase in wartime production (and hence local pollution levels), we might observe a relative increase in infant mortality in these cities, independently of the pandemic. To address this issue, W_c controls for baseline economic conditions (log manufacturing wages in 1900 and log employment in manufacturing in 1910), which we then interact with year.[9] In addition to these issues, we explore the sensitivity of the results to a range of alternative specifications and controls.

[8]The baseline model imposes a linear functional form for longitude and latitude, although the results are robust to nonlinear specifications.

[9]A concern with this procedure is 'over-controlling', since economic conditions are likely correlated with baseline pollution levels. As a result, our preferred specification do not include these covariates.

5 Results

5.1 The overall impact of the pandemic on infant mortality

Before examining the interaction between pandemic-related mortality and local levels of pollution, we first provide evidence of the direct impact of the influenza pandemic on infant mortality rates. We estimate a series of regressions that control flexibly for trends in mortality between 1915 and 1925, and separately include a dummy variable for each year between 1915 and 1925. These year fixed effects capture residual infant mortality – annual deviations from trend – for every year in the sample period. A positive estimate on $I(Year = 1918)$, for example, would indicate that infant mortality exceeded its trend during the year 1918, consistent with pandemic-related infant mortality.

Table 2 reports these estimates. Column (1) controls for a linear year trend, column (2) allows for linear state-specific trends, and column (3) includes a linear city-specific trend.[10] Each row reports the estimates based on a different year fixed effect. In each specification, the point estimate in 1918 is large and statistically significant, implying that infant mortality rates exceeded trend by roughly 19% during the pandemic year. Assuming all excess mortality in 1918 can be attributed to the pandemic, these results imply that influenza was responsible for 10,920 infant death in our sample and 41,348 infant deaths nationwide. We cannot rule out that some fraction of the spike in mortality in 1918 was driven by alternative factors, such as U.S. entry into World War I. Nevertheless, the absolute magnitude of these results, and their size relative to estimates found for 1917, provides strong suggestive evidence of the deviation was primarily driven by influenza.

We compare these estimates to previous assessments of pandemic severity. Infant mortality accounted for 5.2% of excess mortality for the period 1918-1919.[11] Applying these estimates, we calculate that nationwide mortality attributable to the pandemic

[10]The results are robust to the inclusion of squared year trends.

[11]To derive this calculation, we calculate the change in total deaths by age group between 1917 and 1918-19 (*Vital Statistics*, 1917, 1918, 1919).

was $41,348/0.052 = 797,208$ deaths, which is comparable to previous studies that place pandemic-related deaths between 675,000 and 850,000.

The point estimates in non-pandemic years are generally small. Infant mortality rates exceeded trend by 4% in 1917, which in part may reflect U.S. entry into World War I in April 1917.[12] There was a slight decrease in mortality relative to trend between 1919 and 1922, which may reflect the relative economic prosperity during this period. Interestingly, the 5% decrease in infant mortality rates in 1919 is consistent with previous studies which have found that the vast majority of pandemic-related mortality occurred during the four month period from September, 1918 and January 1919 (see Markel et al, 2007).

5.2 Differences in pandemic-severity by local pollution

In Table 3, we examine the extent to which heterogeneity in the pandemic effect was related to local levels of pollution. Column (1)-(4) reports the estimates of β from equation (1) across several different specifications. Column (1) includes city and year fixed effects and a linear state trend; column (2) includes the longitude and latitude controls, allowing the pandemic to differentially impact mortality according to geography; in column (3), we add controls initial population characteristics; and in column (4) we add controls for initial economic conditions.

Across all four specification, the interaction effects are positive and signification, ranging from 0.012 to 0.016. Our preferred estimates imply that differences in city-level pollution can account 16% of cross-city heterogeneity in pandemic-related mortality. Comparing across above and below median pollution cities, our estimates imply the gap in pandemic mortality should be $0.0146 \times (4.90 - 1.24) = 0.05$ log points. Differences in pollution levels can thus account for the entire observed gap in excess pandemic mortality found in Table

Excess infant mortality was 21% in above-median pollution cities in 1918 (see Table 1).

[12]The war may have influenced infant rates through a variety of channels: Volutuntary food rationing may have be harmful for health, increased industrial production may have raised exposure to air pollution, and selection effects (based on the anticipated outbreak of war) may have lowered average infant health.

Given 11,044 annual births, this estimate implies that the pandemic was responsible for 46,592 additional infant deaths in 1918 – 193 per city. To interpret the magnitude of these results, we consider a counterfactual scenario in which all above-median cities reduced steam production to median capacity. This scenario would have led to a reduction 1.73 log points in individual-level exposure to pollution in these cities.[13] As a result, excess infant mortality would have been $0.21-(1.73\times0.0146) = 18.5\%$ in this counterfactual scenario, which implies that 5,551 infant deaths would have been averted, a 13% decline in pandemic severity.

5.3 Steam versus hydroelectric capacity

Local steam energy production may have influenced pandemic severity independently of air pollution. Electricity infrastructure may have been related to local economic conditions and housing quality, both of which may have influenced the severity of outbreak. To examine this issue, we re-estimate equation (1) including an interaction term of the pandemic year with both local steam and hydro capacity. Intuitively, both steam and hydro capacity should have similar effects on health through increased local electricity infrastructure, but steam capacity is also associated with higher local levels of local pollution. The comparison between hydroelectric and steam capacity provides insight into whether the estimates in Table 3 reflect the broad based effects of electricity access or those attributable specifically to pollution emissions.

Table 4 reports the estimated interaction effects of steam and hydro capacity. The point estimates for steam capacity are all statistically significant, ranging from 0.012 to 0.017. Meanwhile, the point estimates for hydro capacity are small – ranging from 0.003 to 0.007 – and statistically insignificant. These results provide confidence that interaction effect between steam capacity on pandemic-related mortality reflects local exposure to air pollution, as a opposed to some other characteristic that may have been related to local electricity infrastructure.

[13]To derive this estimate reduction, we calculate the average change in the sample of high coal cities, weighting cities by number of births.

5.4 Other determinants of pandemic severity

Table 5 assesses the importance of alternative determinants of pandemic-severity, namely baseline health and proximity to World War I bases. One of the most important determinant of infant health during the early 20th century was water quality, which differed widely across cities. We proxy water quality by the baseline city-level typhoid rates, a bacterial infection that fell sharply with the introduction of clean water technologies (Cutler and Miller, 2005). To assess whether differences in local water quality influenced pandemic-severity, we add the interaction term, I(Year=1918)× Log(baseline typhoid mortality), to the main specification in equation (1). These results are reported in column (1). The point estimates for typhoid are small and statistically insignificant. These results may reflect the fact that poor water quality typically led to gastrointestinal illnesses rather than diseases that affected lung function.

In column (3) we examine whether baseline health (more broadly) influenced pandemic-severity. We interact baseline infant mortality (average infant mortality between 1915 and 1917) with the 1918 year effect. The point estimate is positive and significant, consistent with research that argues that pre-pandemic mortality is predictive of pandemic mortality (Pearl 1921, Bootsma and Ferguson, 2007). The point estimates for air pollution remain significant despite the inclusion of this covariate, which is consistent with air quality having exacerbated the influenza outbreak primarily through its effects on morbidity rather than the selection effects associated with mortality differentials.

Given that the pandemic spread first off of World War I bases, and the first weeks of the outbreak were considered the most virulent, nearby cities may have experienced higher rates of pandemic-related deaths. To study this question we re-estimate equation (1), interacting city-centroid distance to the nearest World War I base with the 1918 fixed effect. The results are reported in column (2). The point estimate is large and statistically significant, consistent with the view that the most virulent strains spread off of army encampments. The results imply that an 80 mile decline in distance (roughly the gap between the 25th and 75th percentile) is associated with a 10% increase in excess pandemic mortality. Again, the

estimates for air pollution are robust to the inclusion of this covariate.

In column (5) we estimate a model including all four of these potential determinants of pandemic severity. The general pattern remains unchanged: air pollution, baseline mortality, and exposure to World War I bases all exacerbated the outbreak. To assess the relative importance of these mechanisms, we interact the point estimates with the standard deviations of the independent variables. Together these three factors can account for half of the cross-city variation in excess pandemic mortality. The two dominant factors were local air quality and proximity to World War I bases, which each accounted for 18% of this cross-city dispersion.

6 Robustness checks

In Tables 6 and 7, we examine the robustness of the results to several alternative specifications and controls. A major concern with the baseline findings is that evolution in the severity of the influenza strain may have been spuriously correlated with local pollution levels. Despite the fact that the pandemic was a very short-lived phenomenon, previous research suggests that the virus may have weakened in the weeks following the initial outbreak in early September (Syndenstricker, 1918). If the virus did evolve as it spread across the country, and the evolution was correlated with city-level pollution levels, the baseline results may be biased. For example, if the virus weakened as it spread west, and western cities simultaneously were less dependent on coal as a source of electricity generation, the baseline results might overestimate the relationship between air pollution and pandemic severity.

To address this issue, we construct a measure of the timing of pandemic onset. Specifically, we rely on a map constructed by Syndenstricker (1918) that identifies the week of approximate beginning of the pandemic across 376 localities (before Sept. 14th, Sept. 14th-21st, Sept. 21st-28th, Sept 28th-Oct. 5th, after Oct. 5th). We digitize this information, and assign each city in our sample to one of these five bins. We then interact this measure

with the vector of year fixed effects. By controlling for the week of pandemic onset, these models identify the impact of pollution across population exposed to similar strains of the virus. The results are reported in row 2 of Table 5. For reference, we report the baseline estimates in the first row. Controlling for the timing of pandemic onset has little effect on main findings. Across the four estimation strategies the point estimates are very similar to the baseline results. These results confirm that the interaction effect between pollution exposure and pandemic-related mortality was not driven by evolution in strain severity.

Row 3 reports the estimates for models in which the linear state trend is replaced by a city-specific trend. The results are robust to this alternative specification. Meanwhile, row 4 presents estimates for the balanced panel of cities reporting infant mortality for the full period 1915 through 1925. Again, the point estimates are very similar to the baseline results, indicating that non-random selection was not driving the original findings. In row 5, we examine the sensitivity of the results to excluding the year 1923 from the analysis. We find evidence that baseline pollution was negatively related to infant mortality in 1923 (see Table 7), which might reflect a relative slowdown in economic activity in these cities. By excluding this year from the sample, we address concerns that the main estimates overstate the positive interaction effect in 1918. The results are robust to this sample restriction.

In the final three rows, we examine the sensitivity of the estimates to alternative measures of local pollution exposure. Specifically, we estimate model based on power plant capacity within 10, 50, and 100 miles of each city-centroid. The relative magnitude of these various estimates depends on the atmospheric dispersion of power plant pollution, itself a function of the pollution density, meteorological conditions, local terrain, and smokestack height. Recent evidence from Illinois shows that 40 percent of primary $PM_{2.5}$ exposure occurs within 30 miles of a power plant, with another 30 percent occurring between 30 and 125 miles (Levy et al., 2002).[14] Historically, the dispersion was likely more limited, as plants were built with lower smoke stacks. In general, the point estimates are similar in magnitude to the baseline

[14]In contrast, secondary sulfates and nitrates disperse more widely, with over 50 percent of exposure occurring beyond 125 miles.

findings, albeit less precisely estimated. The results for steam capacity within 10 miles of the city centroid are only marginally significant, which is likely due to the fact that there is less cross-city variation in steam capacity as we restrict attention to the smaller radii. Nevertheless, the broad findings from these results support the initial evidence that local pollution levels exacerbated the impact of the influenza pandemic.

As a final test of the estimation strategy, we conduct a series of placebo exercises to examine whether steam capacity was related to city-level mortality in any of the non-pandemic years. Specifically, we estimate the interaction effect between local steam capacity and non-pandemic years, replacing the 1918 dummy variable with each year fixed effect between 1915 and 1925. This exercise addresses concerns that wartime production was directed towards cities that had large amounts of steam capacity. As a result, increased pollution associated with the spike wartime production may have led to a differential rise in 1918 mortality in these cities, independently of the pandemic. By examining the interaction effect in 1917, we can shed light on this issue.

The results are presented in Table 7. There is little evidence that annual deviations in infant mortality rates were systematically related to local levels of pollution in non-pandemic years. The one exception is the 1923, in which high steam capacity cities experienced a relative decline in infant mortality. Importantly, all but one of the point estimates for 1917 are insignificant, and much smaller than the 1918 interaction effect, suggesting that increased production associated with World War I is not the driving force between the baseline findings. Moreover, the remaining point estimates are generally smaller in magnitude, and less statistically significant than for the pandemic year, supporting the empirical strategy.

7 Conclusion

This paper exploits the 1918 Spanish influenza pandemic to examine how environmental factors interact with infectious illnesses to affect health. Combining information on infant

mortality and air pollution, we find that 17% of the cross-city variation in pandemic severity can be explained by differences in air quality. On the other hand, cross-city differences in water quality had little effect on pandemic-related mortality. Together, these findings are consistent with the epidemiology of the H1N1 influenza virus which directly targeted lung function. These results provide an example of how one particular environmental factor can interact with one particular infectious illness to affect the health of the local population, however, more research is needed to identify how various environmental inputs interact in the health production process.

Outdoor air pollution is directly responsible for 1.3 million premature deaths per year worldwide (WHO, 2009). Our results suggest that by exacerbating the impact of a disease outbreak, the full health effects of poor air pollution may actually be much larger. Given the pervasiveness of the influenza virus, which is responsible for between 250,000 and 500,000 deaths per year worldwide (WHO, 2014), there is large scope for policies aimed at improving air quality to mitigate the impact of this disease.

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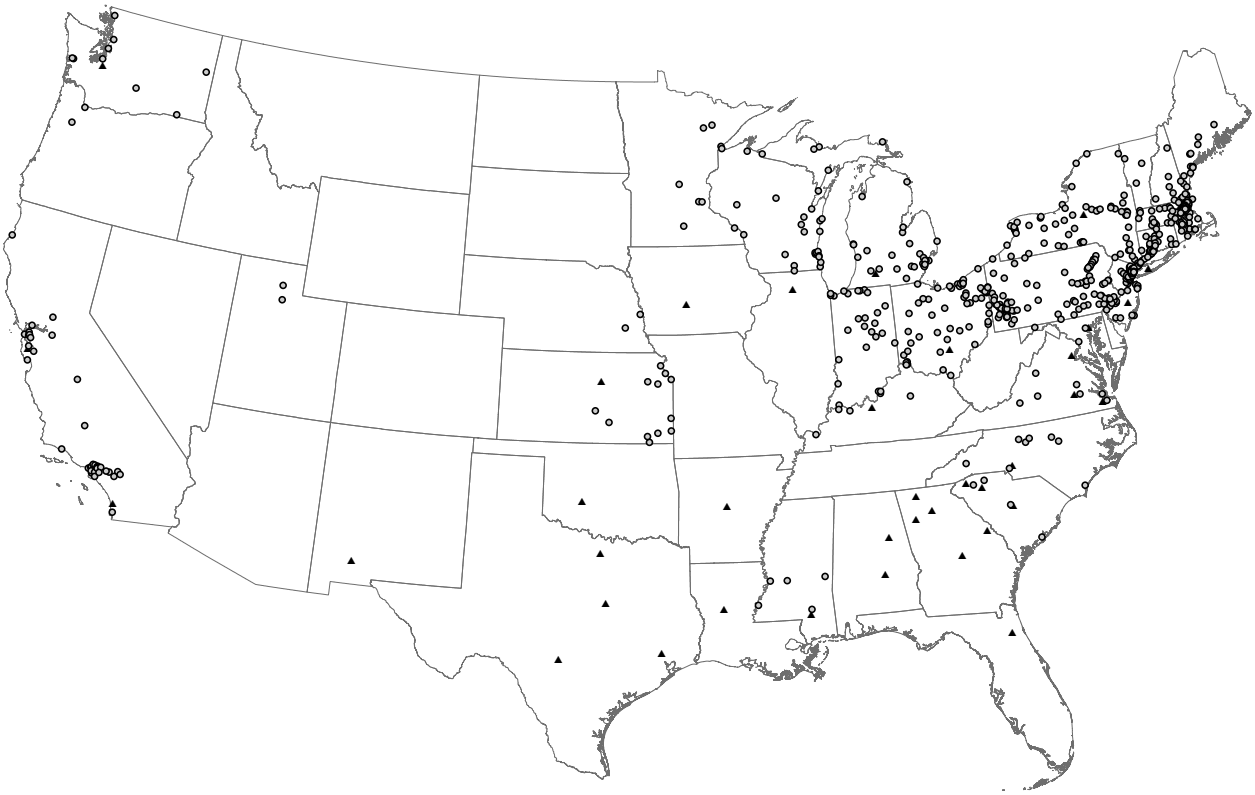
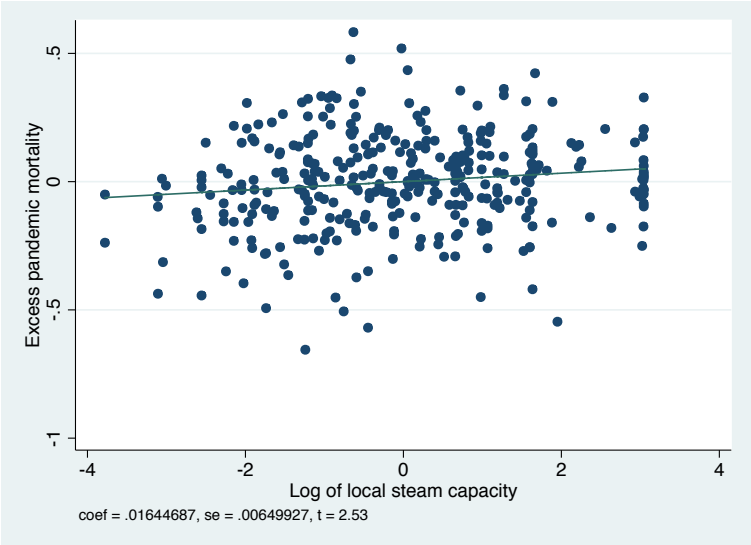
8 Figures and Tables

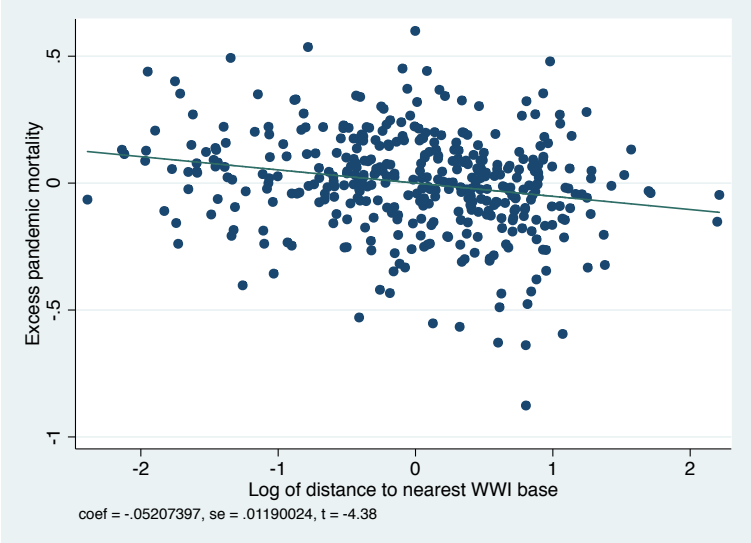
Figure 1: Sample selection and the location of army training camps in 1918



Notes: Each circle denotes to a city in the sample and each triangle denotes either a National Guard Camp, a National Army Cantonment, or other large World War I camps.
Source: U.S. War Department, 1919, p.1519.

Figure 2: Pandemic-related mortality, coal consumption, distance to WWI bases, and baseline typhoid exposure





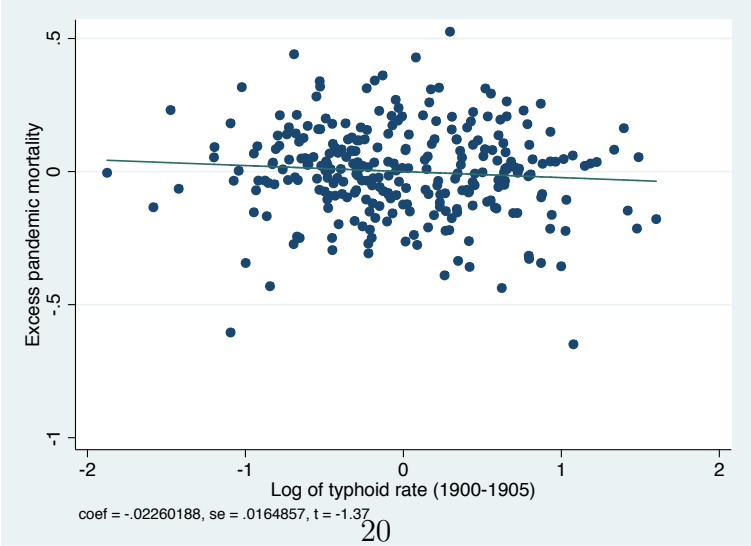
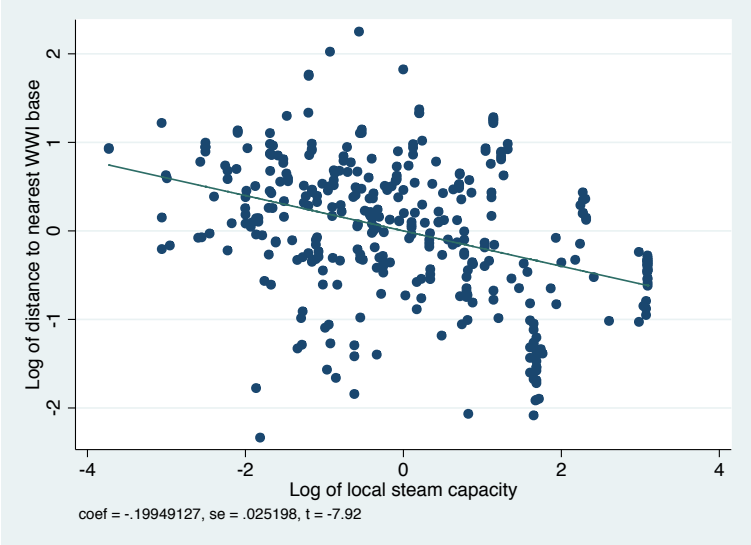
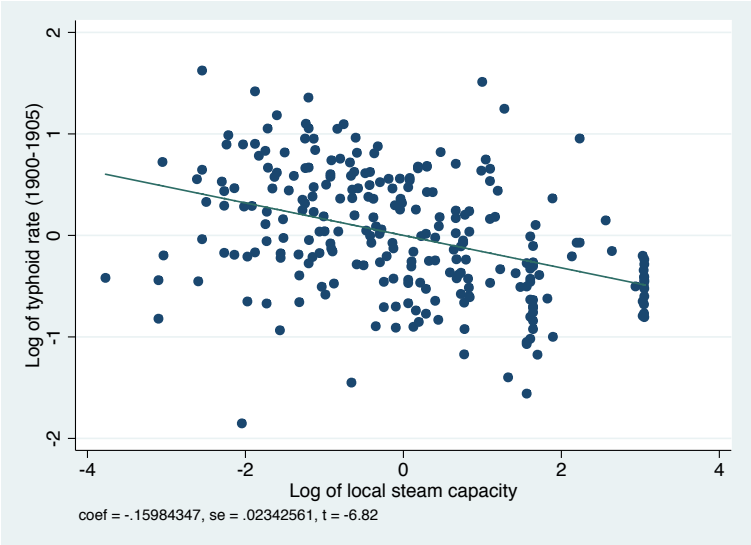


Figure 3: Correlation between coal consumption and other determinants of pandemic-severity





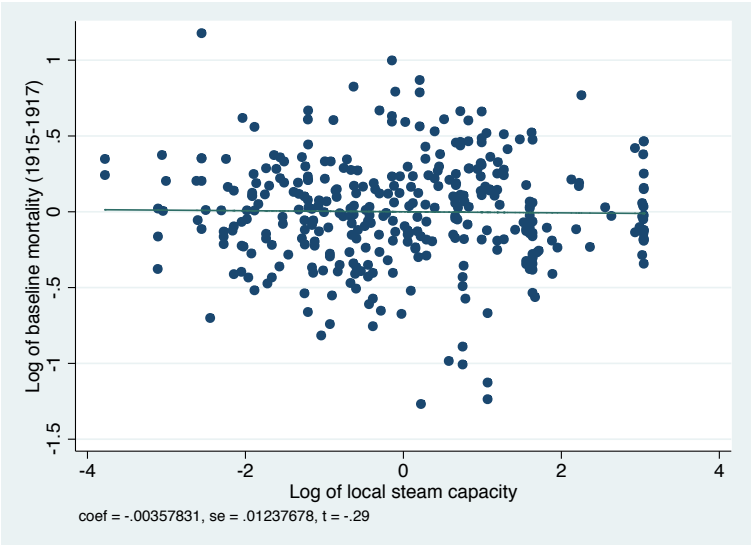


Table 1: Summary statistics

	Full sample	$\geq$ median city-level pollution	$<$ median city-level pollution
Log(infant mortality)	4.37	4.37	4.37
	[0.34]	[0.36]	[0.32]
Log(excess infant mortality in 1918)	0.19	0.21	0.16
	[0.20]	[0.18]	[0.21]
Log(steam capacity within 30 miles)	3.23	4.90	1.24
	[2.17]	[1.09]	[1.27]
Log(hydro capacity within 30 miles)	0.88	0.72	1.07
	[1.50]	[1.42]	[1.57]
Log(baseline infant mortality)	4.45	4.48	4.41
	[0.34]	[0.36]	[0.32]
Log(baseline typhoid mortality)	3.39	3.24	3.60
	[0.62]	[0.58]	[0.60]
Log(distance to nearest WWI base)	4.20	3.95	4.51
	[0.82]	[0.79]	[0.75]
Log(population in 1910)	11.62	12.20	10.93
	[1.05]	[0.99]	[0.60]
Percent urban in 1910	0.64	0.74	0.52
	[0.23]	[0.21]	[0.18]
Share white in 1910	0.95	0.97	0.93
	[0.11]	[0.05]	[0.15]
City population in 1920	68,351	98,351	32,017
	[276,828]	[369,379]	[42,639]
City births in 1920	1,642.4	2335.0	803.6
	[6,495.9]	[8,661.9]	[1,078.8]

Notes: Median city-level pollution is defined as 24.7mw of steam capacity in 1915.

Table 2: Impact of the influenza pandemic year on infant mortality

	Estimated effects		
	(1)	(2)	(3)
I(Year=1915)	-0.138***	-0.140***	-0.0854***
	[0.013]	[0.013]	[0.009]
I(Year=1916)	0.0014	0.0004	0.016*
	[0.010]	[0.010]	[0.008]
I(Year=1917)	0.035***	0.034***	0.043***
	[0.009]	[0.009]	[0.008]
I(Year=1918)	**0.188*** **	**0.187*** **	**0.188*** **
	[0.010]	**[0.0010]**	**[0.0010]**
I(Year=1919)	-0.052***	-0.050***	-0.051***
	[0.013]	[0.013]	[0.012]
I(Year=1921)	-0.020**	-0.018*	-0.020**
	[0.010]	[0.010]	[0.008]
I(Year=1922)	-0.041***	-0.040***	-0.044***
	[0.010]	[0.010]	[0.009]
I(Year=1923)	0.020**	0.021**	0.010
	[0.010]	[0.010]	[0.009]
I(Year=1924)	-0.007	-0.008	-0.020**
	[0.011]	[0.011]	[0.009]
I(Year=1925)	-0.004	-0.007	-0.021***
	[0.011]	[0.011]	[0.008]
Controls			
Year trend	Y		
State-year trend		Y	
City-year trend			Y
Obs	4,928	4,928	4,927

Notes: Each cell reports the point estimates from a different regression. The coefficient $I(Year = j)$ denotes the fixed effect for observations in the jth year. Standard errors are clustered at the city-level. ***,**,* denote significance at the 1%, 5%, and 10%, respectively.

Table 3: The effect of the pandemic on infant mortality, by local steam capacity

Dep variable: Log(infant mortality)				
	Estimate effect			
	(1)	(2)	(3)	(4)
I(Year=1918) × SteamCap30mile	0.0164***	0.0160***	0.0146**	0.0117*
	[0.0047]	[0.0049]	[0.0062]	[0.0062]
Controls				
State-specific trend	Y	Y	Y	Y
Longitude & latitude × year		Y	Y	Y
Population covariates × year			Y	Y
Manufacturing covariates × year				Y
Observations	4,927	4,901	4,901	4,868
R-squared	0.641	0.648	0.654	0.659
Number of clusters	559	555	555	551

Notes: Each cell reports the point estimates from a different regression. The variable *SteamCap*30*mile* denotes logarithm of steam capacity within 30 miles of the city-centroid. Population covariates include county-level controls for the logarithm of population, percent urban, and share white in 1910. Manufacturing covariates include the logarithm of manufacturing wages in 1900 and the logarithm of manufacturing employment in 1910. Standard errors are clustered at the city-level. ***,**,* denote significance at the 1%, 5%, and 10%, respectively.

Table 4: The effect of the pandemic on infant mortality, by local steam and hydro capacity

Dep variable: Log(infant mortality)	Estimated effects			
	(1)	(2)	(3)	(4)
I(Year=1918) × SteamCap30mile	0.0167***	0.0165***	0.0148**	0.0118*
	[0.0047]	[0.0049]	[0.0062]	[0.0062]
I(Year=1918) × HydroCap30mile	0.0058	0.0071	0.0070	0.0025
	[0.0068]	[0.0073]	[0.0075]	[0.0075]
Controls				
State-specific trend	Y	Y	Y	Y
Longitude & latitude × year		Y	Y	Y
Population covariates × year			Y	Y
Manufacturing covariates × year				Y
Observations	4,927	4,901	4,901	4,868
R-squared	0.641	0.648	0.654	0.659
Number of clusters	559	555	555	551

Notes: Each cell reports the point estimates from a different regression. The variables SteamCap30mile and HydroCap30Mile denote logarithm of steam and hydro capacity within 30 miles of the city-centroid. Population covariates include county-level controls for the logarithm of population, percent urban, and share white in 1910. Manufacturing covariates include the logarithm of manufacturing wages in 1900 and the logarithm of manufacturing employment in 1910. Standard errors are clustered at the city-level. ***,**,* denote significance at the 1%, 5%, and 10%, respectively.

Table 5: Other determinants of pandemic severity

Dep variable: Log(infant mortality)					
	Estimate effect				
	(1)	(2)	(3)	(4)	(5)
I(Year=1918) ×					
SteamCap30mile	0.0173**	0.0123*	0.0153**	0.0168**	0.0170**
	[0.00673]	[0.00649]	[0.00604]	[0.00678]	[0.00670]
Log(baseline typhoid mortality)	-0.00237			0.0138	0.0106
	[0.0230]			[0.0236]	[0.0237]
Log(distance to nearest WWI base)		-0.0507***		-0.0344*	-0.0448**
		[0.0173]		[0.0192]	[0.0195]
Log(baseline infant mortality)			0.0924**		0.0789*
			[0.0395]		[0.0475]
Observations	2,811	4,349	4,323	2,606	2,586
R-squared	0.666	0.656	0.678	0.678	0.681
Number of clusters	288	485	439	266	262

Notes: Each cell reports the point estimates from a different regression. The variable SteamCap30mile denotes logarithm of steam capacity within 30 miles of the city-centroid. All models control for state-specific trends, and geographic and population covariates. Standard errors are clustered at the city-level. ***,**,* denote significance at the 1%, 5%, and 10%, respectively.

Table 6: Robustness checks

Dep variable: Log(infant mortality)	Estimated effects			
	(1)	(2)	(3)	(4)
Baseline specification				
I(Year=1918) × SteamCap30mile	0.0164***	0.0160***	0.0146**	0.0117*
	[0.0047]	[0.0049]	[0.0062]	[0.0062]
Control for timing of influenza outbreak				
I(Year=1918) × SteamCap30mile	0.0169***	0.0160***	0.0144**	0.0125*
	[0.0054]	[0.0053]	[0.0064]	[0.0065]
Control for city-specific trends				
I(Year=1918) × SteamCap30mile	0.0144***	0.0122**	0.0136**	0.0114*
	[0.0047]	[0.0049]	[0.0060]	[0.0060]
Balanced Panel				
I(Year=1918) × SteamCap30mile	0.0155***	0.0150***	0.0139**	0.0107*
	[0.0045]	[0.0048]	[0.0061]	[0.0061]
Drop 1923 from the sample				
I(Year=1918) × SteamCap10mile	0.0156***	0.0144***	0.0130**	0.0101
	[0.00468]	[0.00492]	[0.00615]	[0.00614]
Alternative measures of local pollution				
I(Year=1918) × SteamCap10mile	0.0117**	0.0099*	0.0063	0.0055
	[0.0050]	[0.0052]	[0.0064]	[0.0062]
I(Year=1918) × SteamCap50mile	0.0163***	0.0164**	0.0125	0.0080
	[0.0054]	[0.0065]	[0.0078]	[0.0084]
I(Year=1918) × SteamCap100mile	0.0208***	0.0238**	0.0193*	0.00916
	[0.0069]	[0.0105]	[0.0107]	[0.0118]
Controls				
State-specific trend	Y	Y	Y	Y
Longitude & latitude × year FEs		Y	Y	Y
Population covariates			Y	Y
Manufacturing covariates				Y

Notes: Each cell reports the point estimates from a different regression. The variable *SteamCap*30*mile* denotes logarithm of steam capacity within 30 miles of the city-centroid. Population covariates include county-level controls for the logarithm of population, percent urban, and share white in 1910. Manufacturing covariates include the logarithm of manufacturing wages in 1900 and the logarithm of manufacturing employment in 1910. Standard errors are clustered at the city-level. ***,**,* denote significance at the 1%, 5%, and 10%, respectively.

Table 7: Pollution interaction effects, by year

Dep variable: Log(infant mortality)	Estimated effects			
	(1)	(2)	(3)	(4)
I(Year=1915) × SteamCap30mile	0.0077	0.0065	-0.0044	-0.0053
	[0.0056]	[0.0063]	[0.0081]	[0.0083]
I(Year=1916) × SteamCap30mile	0.0000	0.0052	0.0009	0.0001
	[0.0050]	[0.0061]	[0.0072]	[0.0074]
I(Year=1917) × SteamCap30mile	0.0055	0.0096*	0.0061	0.0024
	[0.0048]	[0.0054]	[0.0064]	[0.0066]
I(Year=1918) × SteamCap30mile	**0.0164*** **	**0.0160*** **	**0.0146** **	**0.0117***
	[0.0047]	**[0.0049]**	**[0.0062]**	**[0.0062]**
I(Year=1919) × SteamCap30mile	0.0064	0.0135**	0.0122	0.0129
	[0.0067]	[0.0066]	[0.0090]	[0.0086]
I(Year=1921) × SteamCap30mile	-0.0140***	-0.0116**	-0.0057	-0.0027
	[0.0045]	[0.0052]	[0.0069]	[0.0071]
I(Year=1922) × SteamCap30mile	0.0042	0.0011	0.0053	0.0060
	[0.0049]	[0.0052]	[0.0065]	[0.0067]
I(Year=1923) × SteamCap30mile	-0.0097**	-0.019***	-0.021***	-0.0208***
	[0.0048]	[0.0056]	[0.0070]	[0.0073]
I(Year=1924) × SteamCap30mile	-0.0025	-0.0019	0.00080	0.0022
	[0.0049]	[0.0056]	[0.0067]	[0.0069]
I(Year=1925) × SteamCap30mile	-0.0045	-0.0073	-0.0011	-0.00027
	[0.0053]	[0.0062]	[0.0074]	[0.0078]
Controls				
State-specific trend	Y	Y	Y	Y
Longitude & latitude × year FEs		Y	Y	Y
Population covariates			Y	Y
Manufacturing covariates				Y

Notes: Each cell reports the point estimates from a different regression. The variable *SteamCap*30*mile* denotes logarithm of steam capacity within 30 miles of the city-centroid. Population covariates include county-level controls for the logarithm of population, percent urban, and share white in 1910. Manufacturing covariates include the logarithm of manufacturing wages in 1900 and the logarithm of manufacturing employment in 1910. Standard errors are clustered at the city-level. ***,**,* denote significance at the 1%, 5%, and 10%, respectively.