How the Other Half Died: Immigration and Mortality in US Cities*

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Abstract

Fears of immigrants as a threat to public health have a long and sordid history. At the turn of the 20th century, when millions of immigrants crowded into dense American cities, contemporaries blamed the high urban mortality penalty on the newest arrivals. Nativist sentiments eventually led to the implementation of restrictive quota acts in the 1920s, substantially curtailing immigration. We capture the "missing immigrants" induced by the quotas to estimate the effect of immigration on mortality. We find that cities with more missing immigrants experienced sharp declines in deaths from infectious diseases from the mid-1920s until the late 1930s. The blame for these negative mortality effects lies not with the immigrants, but on the living conditions they endured. We show that mortality declines were largest in cities where immigrants resided in the most crowded and squalid conditions and where public health resources were stretched the thinnest. Though immigrants did die from infectious diseases at higher rates than the US-born, the mortality decline we find is primarily driven by crowding not changes in population composition or contagion, as we show mortality improvements for both US- and foreign-born populations in more quota-affected cities.

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1 Introduction

Fear of the outsider as an "immigrant menace" threatening domestic health with contagious diseases is deeply ingrained in the human psyche (Kraut 1988; Schaller and Neuberg 2012).[1] These sentiments have reverberated throughout American history, particularly in times of mass migration (Kraut 1988; Lee 2019a; Markel and Stern 2002). At the end of the 19th century, Eastern European Jews were regarded as especially vulnerable to tuberculosis, and Chinese residents in San Francisco were irrationally quarantined during a bubonic plague outbreak. In the 1980s, with no basis in data, Haitian migrants were labeled as a high-risk group for HIV infection (Kraut 1994, 2019). Today, many Americans hold mistaken and sharply negative views about immigrants (Alesina et al. 2018), misperceptions that may be exploited by those accusing immigrants of bringing infectious diseases across borders (Shah 2020; Belluz 2018; Rogers 2018).

In this paper, we show that these nativist fears were self-fulfilling in the early 20th century. A broken urban governance system—Steffens (1904)'s shame of the cities—allowed and even encouraged the housing of immigrants in crowded and squalid conditions in urban slums.[2] But a city is only as safe or healthy as its most vulnerable populations because infectious diseases do not respect neighborhood boundaries; ultimately, the living conditions of immigrants contributed to the urban mortality penalty. When the US curtailed immigration in the 1920s, deaths from infectious diseases declined because many American cities faced fewer congestion constraints. However, far from supporting the recurring narrative of immigrants as disease carriers, our results underscore the high stakes of well-managed public health infrastructure and the dangerous spillover effects of inequality in urban living conditions. Some contemporaries evidently understood this point; the

[1] Medicalized nativism predates both modern medicine and the nation-state. Already in 250 BC China, smallpox was known as "Hunpox" (Hopkins 2002); the Black Death unleashed a wave of deadly pogroms against Jews (Voigtländer and Voth 2012). Syphilis was known initially as French pox (to the English), the Chinese disease or the disease of the Portuguese (to the Japanese), the Polish disease (to the Germans), the German disease (to the Polish), and a disease of the Turks (in Persia) (Cohn 2012; Green et al. 2010). Absent from the American nativist's narrative on immigration and disease is the deadliest example of how destructive the germs carried by newcomers can be: the deaths of the majority of the indigenous populations of the Americas during European colonization (Diamond 1998; Cook 1998).

[2] In Chicago, immigrant neighborhoods were intentionally zoned for noxious industrial use (Shertzer et al. 2016). The demand for urban zoning had been driven, in part, by concerns over immigration (Shertzer et al. 2018). In New York, Steffens (1904) chided Tammany Hall leaders for "caus[ing] the troubles they relieve... let[ting] the Health Department neglect the tenements..." Informal barriers to better housing—racial covenants and discrimination of the type that, for example, forced Italian immigrants to live only in Little Italies—increased crowding conditions in ethnic enclaves (Wang 2001).

National Industrial Conference Board reported in 1923 that:

> [I]mmigrants as a class are frequently blamed for bad housing and living conditions over which they have no control. These conditions may be due in part to the attitude of Americans toward immigrants, and to the economic status of the immigrant working class.

America's urban centers at the turn of the 20th century were deadly places to live.[3] This was particularly the case for recent arrivals, who often clustered in the poorest, most densely populated, and unhealthiest districts. In 1890, when almost a third of the urban population was foreign-born, the urban mortality rate stood at 25 deaths per 1,000 inhabitants, about 35 percent higher than in rural areas (Haines 2001; Willcox 1906). The positive correlation between immigration and mortality was cited by many contemporaries as evidence that immigrants caused the spread of infectious diseases in cities (Higgs 1979; Kraut 1994).

Large-scale immigration and the clustering of newcomers in poverty-stricken neighborhoods also stoked xenophobia during this period. A political movement led by the Immigration Restriction League emerged in the 1890s with the aim of restricting immigration, especially from culturally distant countries (Jones 2013; Lee 2019a). The nativist movement gained momentum after World War I, scoring major political victories with the introduction of immigration quotas in the 1920s. The quota system significantly curtailed immigration to the US: compared to the 4.5 million arrivals from 1910-1914, fewer than 800,000 were admitted under the new federal law between 1925-1929. Beyond simply constraining the total number of immigrants, the quotas were designed to disproportionately limit immigration from Southern and Eastern Europe—the main sending regions at the start of the 20th century—while granting more quota slots to immigrants from Northern Europe and setting no limits on immigrants from the Western Hemisphere.

We exploit this fundamental shift in US immigration policy to estimate the causal effect of immigration on urban mortality. While the quota system was implemented nationally, the combination of differential restrictions across sending countries and the tendency of immigrants to cluster in areas with already-established migrant networks led to different repercussions for each city. We

[3]Immigration during the Age of Mass Migration (1850-1920) was an overwhelmingly urban phenomenon. Approximately 30 million Europeans migrated to the US with a peak of over a million arrivals in 1907. In 1910, almost 15 percent of the population was foreign-born, with three-fourths settling in urban locations (Abramitzky and Boustan 2017; Ward 1971).

construct a measure of "missing immigrants" to capture how intensely each city was affected by the quota policy.[4] First, for each nationality, we compute the total number of missing immigrants by comparing the expected number of arrivals under pre-quota migration—projected from historical trends—to the corresponding quota limit. Second, we allocate the missing immigrants to the cities they might have moved to based on pre-quota settlement patterns. The resulting city-level intensity of the immigration policy, which we refer to as quota exposure, can be interpreted as the local rate of missing immigrants.

We use difference-in-differences to evaluate how quota exposure affected urban mortality. We draw on annual cause-of-death mortality statistics for 348 US cities from 1900-1937. We show that mortality trends of subsequently more and less quota-affected cities evolved similarly prior to the policies. However, with the enactment of the immigration restrictions, mortality rates started to fall more rapidly in cities more affected by the quota system. This decline was driven by changes in deaths due to infectious diseases. External causes of death also fell in cities most affected by the quotas, though such causes account for a far smaller share of total deaths than infectious causes do, which were the primary causes of death at the beginning of the 20th century. Our baseline estimates suggest that a 1 percentage point increase in the rate of missing immigrants reduced the overall mortality rate by around 2 percent, the infectious-cause mortality rate by 5 percent, and the external-cause mortality rate by 6 percent. We find no effect on mortality arising from non-infectious causes, such as cancer and cardiovascular diseases.

Could our results simply reflect compositional changes in the population following the implementation of the quota system? Compared with their US-born counterparts, immigrants suffered from excess mortality across most causes and age groups at the turn of the 20th century (Dublin 1916). The excess mortality of immigrants, coupled with a reduction in their numbers under the quota system, would generate a mechanical decline in overall mortality rates even if the mortality of the US-born was not affected at all. Unfortunately, mortality data by nativity are not available for cities after 1922, which prevents us from assessing the composition channel directly. Given the data limitations, we provide two pieces of indirect evidence to demonstrate that compositional changes alone do not tell the full story. First, we show that the quota effects we estimate are too

[4]Edith Abbott (1927) first introduced the concept of "missing immigrants" at the round table discussion on the economic effects of immigration restrictions at the American Economic Association meetings.

large to be explained by just compositional changes. Second, we find that mortality rates among African Americans also fell in response to the quota policy, though less sharply compared with whites. Since nearly all African Americans were US-born in this era, these results suggest that spillover effects from immigrants to the US-born must have existed.

Beyond changing the foreign-born share, the quota system could have affected the selection of immigrants, shifting the composition of health status among immigrants. We digitized archival records on the number of immigrants denied entry by reason and year. We find that few immigrants were ever rejected for reasons related to infectious diseases before the 1920s and that these numbers did not change significantly after the introduction of quotas. Since the quota system did not reduce the odds of less healthy immigrants entering the country, we find it unlikely that such changes in the health composition of immigrants could explain our baseline results.

Rather than changing the composition of health in urban populations, we argue that the quota acts improved urban mortality because the reduced rate of immigration relaxed housing and health-care constraints. Scholars have long argued that overcrowding and poor housing conditions in immigrant neighborhoods contributed to the mortality penalty in American cities at the turn of the 20th century (Higgs 1979; Kraut 1994). New arrivals clustered in parts of cities where housing was cheap and close to their workplaces (Ward 1968); such crowded and unsanitary districts were focal points for the spread of infectious diseases (Costa 2015). We provide four pieces of evidence for the congestion channel. First, the quota policy reduced diseases associated with crowding, such as pneumonia and measles. Second, we compare the effects of the quota policy in cities where immigrants lived in more or less crowded conditions prior to the quotas. We find that internal density—the average number of people in a dwelling—and public health density—proxied with newly-collected historical data on patients per hospital or patients per hospital bed—both amplify the effect of missing immigrants on mortality rates.[5] Third, the quota policy reduced housing congestion; more quota-exposed cities experienced relatively larger declines in the number of boarders, lodgers, and people living in multifamily dwellings, measures that capture constraints in housing supply. Fourth, we turn to rural counties, which are by definition less dense and congested. We find that the most quota-exposed rural counties did not experience relative declines in mortality

[5]We do not find any strong evidence suggesting that our results are driven by preferences for redistribution since the quota shock did not increase local spending on public health in more affected cities.

rates.

Our paper contributes to the literature on the urban mortality transition. City dwellers in the late 19th century faced a substantial mortality penalty. In the decades that followed, the urban mortality rate dropped by more than half, and the urban mortality penalty largely dissipated by the 1940s (Haines 2001).[6] A sizable literature attributes this decline to improvements in public health infrastructure (Alsan and Goldin 2019; Costa 2015; Cutler and Miller 2005; Cutler et al. 2006), but two recent studies by Anderson et al. (2019a,b) have contested these conclusions, arguing instead that better nutrition and improved living conditions played more important roles. Our study highlights how drastic changes in US immigration policy also contributed to the decline in urban mortality over this period, particularly by reducing deaths from infectious diseases through relaxing housing and healthcare constraints. While we are not the first to suggest that immigration might be a factor in understanding the urban mortality penalty (e.g., Duffy 1968; Higgs 1979; Meckel 1985), our study goes beyond existing work by providing well-identified estimates of the impact of immigration on urban mortality for a large sample of US cities.

We also contribute to the long literature on the broader implications of immigration during the Age of Mass Migration; for an overview, see Abramitzky and Boustan (2017). In terms of research design, our paper is closest to studies exploiting the quota system to identify the impact of immigration on various socio-economic outcomes (Abramitzky et al. 2019; Doran and Yoon 2018; Moser and San 2020; Tabellini 2020; Xie 2017). One advantage we have compared to existing studies is that our outcome of interest—mortality—is available annually at the city level. Annual data enables us to study the short- and long-term dynamics of the quota policy and we can assess the parallel trends assumption more carefully.[7]

Our work is related—though distinct—from more contemporary studies of the "healthy immi-

[6]There is some disagreement among demographers and economic historians about the nature of the mortality transition—whether it was a steady and continuous decline during the second half of the 19th century or an abrupt and rapid decline after 1880 (Coale and Zelnik 1963; Higgs 1971; Meeker 1971). The next section provides a brief overview of this literature; see also the reviews by Cutler et al. (2006) and Costa (2015) for further details and references.

[7]Greenwood and Ward (2015), Massey (2016), and Ward (2017) examine how the immigration quotas changed migration behavior. Greenwood and Ward (2015) show that emigration rates declined significantly after the quota policy was introduced, especially from unskilled occupations and farming, which Ward (2017) argues is driven by a lower rate of unplanned return migration during the 1920s. Massey (2016) examines how the enactment of the quotas affected migrant selection, and finds that the average skill level of immigrants increased after the change in policy.

grant" effect. Though politicized narratives sometimes paint a negative picture of immigrants as unhealthy and fiscal drains on destination health systems, in reality immigrants, driven in part by selection, tend to be healthier than their native-born counterparts across the world (Borhade and Dey 2018; Aldridge et al. 2018). This patterns holds in the US today, though the health advantage erodes with time spent in the US as immigrants "assimilate" (Antecol and Bedard 2006).[8]

2 Historical Background

In this section, we describe the context for our study, focusing first on the urban mortality penalty in the US over the late 19th and early 20th centuries, then on immigration to American cities, and finally detailing the intersection of health and immigration during the Age of Mass Migration.

2.1 The Urban Mortality Penalty

Historically, the benefits of living in urban centers came with significant health costs, including a severe urban mortality penalty. From 1820-1920 in the US, the share of people living in cities increased from 7 to over 50 percent and real GDP per capita more than quadrupled. Despite substantial improvements in real wages and productivity, health indicators such as height and life expectancy worsened for the average American (Costa and Steckel 1997; Kim and Margo 2004).

Economic historians and demographers attribute a large part of the decline in health quality to the poor and unsanitary living conditions in cities, since the US started to urbanize and industrialize well before significant advances in public health were made (Duffy 1992; Higgs and Booth 1979; Melosi 2000). Greater density and crowding in cities, coupled with unsanitary conditions of living quarters, facilitated the spread of respiratory diseases such as influenza, pneumonia, and tuberculosis—the three largest single causes of death at the beginning of the 20th century (Jones et al. 2012). Poor living conditions were also conducive to the spread of food- and water-borne diseases like as diarrhea and typhoid (Condran and Crimmins 1980; Haines 2001).

Contemporary doctors and public health scholars acknowledged the detrimental consequences of poor housing conditions on health (e.g. Krieger and Higgins 2002; Stella 1908).[9] In particular,

[8]Economic historians have also studied selection of immigrants during the Age of Mass Migration (for example, from Mexico (Escamilla-Guerrero and Lopez-Alonso 2019; Kosack and Ward 2014) and from Italy (Spitzer and Zimran 2018)).

[9]Edwin Chadwick's "*Report on the Sanitary Condition of the Labouring Population of Great Britain*"

tenement buildings in the urban slums—which were characterized by overcrowding, a lack of ventilation, and inadequate toilet facilities—were viewed as incubators of diseases (Addams 1911; Duffy 1992; Kraut 1994).[10] The Danish-American social reformer Jacob Riis (1890) painted a dramatic picture of the dwelling conditions that the working-class faced at the time in his famous book *How the Other Half Lives: Studies among the Tenements of New York*.

The urban mortality penalty was substantial at the turn of the 20th century, but death rates started to decline in the following decades. Between 1900 and 1940, mortality rates fell by 40 percent in cities and the urban-rural mortality gap largely disappeared (Haines 2001) and life expectancy and health status improved significantly (Troesken 2015).[11] The leading causes of death also shifted from respiratory, food-, and water-borne diseases to non-communicable diseases (Cutler and Meara 2004; Cutler et al. 2006; Jones et al. 2012).

There is an ongoing scholarly debate about the factors contributing to the urban mortality decline. One prominent view holds that better nutrition, rising incomes, and sustained economic growth were key for the mortality transition to take place (Fogel 1997, 2004; McKeown 1976; McKeown and Record 1962). Others regard public health interventions as important drivers of the mortality decline (Cain and Rotella 2001; Costa and Kahn 2006; Troesken 2004).[12]

While poor housing conditions were correlated with higher rates of infectious disease transmission (Krieger and Higgins 2002), little is known about their causal role in the decline in deaths due to infectious diseases over the first half of the 20th century. Condran and Cheney (1982) argue that few public health activities were directed towards reducing pneumonia (a disease associated with poor housing conditions), as it was not considered to be contagious at the time. This might explain why, overall, mortality rates due to influenza and pneumonia remained fairly stable during the first two decades of the 20th century (Anderson et al. 2019b, Figure 3) before the quota policy

in 1842 associated poor housing conditions with the spread of infectious diseases. The notion of housing as a public heath issue was also emphasized by Friedrich Engels in his famous book "*The Condition of the Working Class in England*" in 1845.

[10] Unsanitary housing conditions were also common in newly-established manufacturing towns and industrial suburbs (Lopez 2012).

[11] The transition in southern cities lagged behind in the first half of the 20th century due to their larger share of African Americans, who suffered from higher risks of infectious mortality (Feigenbaum et al. 2019).

[12] For example, there is ample evidence that the roll-out of sewage and access to clean water contributed to the mortality decline at the beginning of the 20th century (Alsan and Goldin 2019; Condran and Crimmins-Gardner 1978; Cutler and Miller 2005; Ferrie and Troesken 2008). Other studies show that public health regulations related to the supply of clean milk improved the health conditions of infants by reducing deaths from diarrhea (Komisarow 2017; Lee 2007; Olmstead and Rhode 2004).

was introduced. Ours is the first comprehensive city-level study that provides rigorous empirical evidence showing that the relaxation of housing constraints reduced infectious diseases in American cities during this period in history.

2.2 Immigrants in US Cities

Mass migration from Europe contributed to both the development and overcrowding of American cities. Between 1850 and 1920, roughly 10 percent of Americans were born abroad and the annual inflow of immigrants exceeded 1 percent of the total population in several years (Abramitzky and Boustan 2017).[13] This unprecedented episode of mass migration was primarily an urban phenomenon. By 1920, more than three quarters of the foreign-born population lived in urban areas (Gibson and Lennon 1999).

Newcomers were particularly attracted to downtown districts where housing was cheap and close to their workplaces (Ward 1968), but crowded tenements also became focal points of the spread of infectious diseases (Costa 2015). A contemporary report by the National Industrial Conference Board (1923, pp. 43-44) notes that immigrants in cities have the tendency to overcrowd, to keep boarders and lodgers, and to have several families living together in small quarters, which the Board argued would foster insanitary housing conditions and the maintenance of slums. The high levels of urban mortality coupled with the concentration of immigrants in cities prompted several studies to draw a positive correlation between urban mortality and immigration, particularly before the onset of the epidemiological transition (e.g., Duffy 1968; Howard 1924; Meckel 1985).

Contemporaries noted that immigrants had higher mortality rates compared to the US-born and sometimes even relative to the mortality rates in their home countries.[14] While such disparities could partly reflect negative selection in immigration, they were primarily driven by the poor living conditions that the newcomers faced in US cities.[15] Stella (1908), for example, blamed overcrowding

[13]The first wave of immigrants mainly came from Western and Northern Europe. By the end of the 19th century, however, the center of emigration had shifted to Southern and Eastern Europe.

[14]Dublin (1916, 1922) and Dublin and Baker (1920), for example, analyzed the 1910 mortality statistics of New York and Pennsylvania and found lower life expectancies for foreign-born people compared to US-born whites of US-born parentage.

[15]Like immigrants, newly migrated African Americans in this period also faced abysmal living conditions in industrial cities. Eriksson and Niemesh (2016), for example, find a substantial increase in infant mortality among black households that moved to northern cities as part of the Great Migration. Much of the adverse health effect was due to residential location in unhealthy neighborhoods within northern cities.

and a lack of ventilation in rooms for the higher respiratory-related mortality rates of Italian children in New York. Likewise, Dr. William H. Guilfoy—the New York City Department of Health Records Registrar—identified congested housing as the main reason for Italians' seeming predisposition to pneumonia (Kraut 1994, p. 127). Higgs and Booth (1979) find that mortality rates for both the foreign- and US-born in 1890 are similar once population density and age structure are accounted for, suggesting that the living conditions of immigrants in US cities largely explain their elevated mortality rates.

2.3 Public Health and Immigration Restrictions

As immigration accelerated towards the beginning of the 20th century, many Americans feared how this would affect the different facets of their lives, from the economy to politics to culture. Concerns over public health, in particular, were frequent and visceral.[16] Nativists fueled these fears by blaming immigrants for the outbreak of diseases—Italians, for example, were held responsible for the polio outbreak in 1916 and Jews were stigmatized as carriers of tuberculosis (Kraut 1994, pp. 109-110, p. 155).

With the shift in source countries of immigration towards Southern and Eastern Europe, nativists' efforts at the national level went from tightening the rules for nationalization towards immigration restrictions (Higham 2002, pp. 97-98). Medical advancements during the 1880s played into their hands. After the germ theory of disease gained acceptance, the movement to restrict immigration saw medical inspections as a promising tool to exclude "undesirable" newcomers.[17] Medical testing of immigrants became widely accepted by the public and it appealed to politicians as a way to use medical experts to justify immigration policy (Kraut 1994; Yew 1980).

By 1891, federal immigration officials conducted health inspections at all ports of entry and immigrants were excluded if they were deemed likely to become public charges; had a criminal past; or suffered from "loathsome" or dangerous contagious diseases. Powderly (1902, p.60), the Commissioner General of Immigration, called for even stricter restrictions if the US was to avoid

[16] During the late 19th century, contemporaries attributed epidemics in large American cities to immigration, including the outbreak of smallpox in the 1870s and 1880s and the influenza outbreak from 1890 to 1892 (Higgs 1979, pp. 399-403).

[17] Kraut (1994) vividly describes how nativists combined scientific knowledge and data to claim that certain diseases were more common among newcomers in order to demonstrate their biological inferiority.

becoming the "hospital of the nations of earth." Policies moved in that direction as the list of excuses for exclusion grew larger with the passage of new immigration laws in 1903, 1907, and 1917 in Congress.

Despite increasingly stringent laws to exclude "diseased immigrants," the number that were barred from entry for medical reasons was never very high. According to Kraut (1988), those denied entry due to poor health conditions did not exceed 3 percent of the total number of immigrants in any given year between 1891 and 1924, and the average number of exclusions for medical reasons was less than 1 percent over the same period. Our data on the number of immigrants denied entry by reason and race, obtained from the *Reports of Commissioner General of Immigration*, suggest similar numbers. Focusing on rejections related to infectious diseases, we find rejection rates below 1 percent from 1900-1930 (see Section 6.4).[18]

The nativist movement eventually scored major political victories with the passage of the immigration quota acts in 1921 and 1924 (Lee 2019b). While the quota policies reflected the racial perceptions of eugenicists (Ludmerer 1972), they also spoke to popular economic concerns that unskilled labor from Southern and Eastern Europe would drive down wages and compete with blue-collar white US-born workers for their jobs (e.g. Jenks and Lauck 1911). With the enactment of the quota system, European immigration, which had remained virtually unrestricted up until the first decades of the 20th century, declined substantially from 4.5 million between 1910 and 1914 to less than 800,000 between 1925 and 1929.[19]

The so-called Emergency Quota Act of 1921 imposed quotas that were based on 3 percent of the foreign-born populations of each nationality listed in the 1910 Census, with a cap of 357,000 immigrants annually (King 2000). Since the quotas were tied to the origin composition in 1910, immigration from each source country was affected differently: the quota slots allocated to immi-

[18]While overall rates were low, the relative frequency of rejection on medical grounds did grow as a share of all rejections at the border. While only 2 percent of the barred entries in 1898 were on medical grounds, the number increased to more than 60 percent in 1916 as a result of improved diagnostic techniques (Kraut 1988; Yew 1980). We calculate lower rates of people denied entry for disease-related reasons, never exceeding 30 percent. Still, the small share of people rejected on medical grounds at a time of growing anti-immigrant animosity and panics about public health suggest that the positive correlation between immigration and urban mortality before the quotas or any effects of the quotas on mortality are very unlikely to be driven by immigrants bringing new or more virulent diseases from abroad.

[19]While a literacy test had been introduced in 1917 (known as the Literacy Act), it was perceived to be ineffective as it failed to reduce immigration on a large scale; see Goldin (1994) for a detailed discussion of the political economy behind the passage of the Literacy Act.

grants from Western and Northern Europe were fairly generous, while the law curtailed immigration from Southern and Eastern Europe. Immigrants from Canada, Mexico, and Latin America were exempted from the Act and faced no restrictions.

In 1924, the quota system was made permanent and two significant changes were introduced that would limit immigration even further. First, the quota ceiling was lowered from 3 to 2 percent of the foreign-born populations of each nationality. Second, the reference year for these population counts was pushed back from 1910 to 1890. These changes meant that immigrants from Southern and Eastern Europe were almost entirely excluded. The Immigration Act of 1924 also completely banned immigration from Asia.[20] The annual overall quota was set to a total of 150,000 immigrants in 1929. This system remained in place, apart from some minor modifications, until the quota regime was replaced by the Immigration and Naturalization Act in 1965.

Overall, the quota acts of 1921 and 1924 led to a sharp and lasting reduction in immigration to the US. The foreign-born population declined from 15 percent of the US population in the pre-quota period to a low of 5 percent in 1970, before it started to rise again (Abramitzky and Boustan 2017, Figure 1). In the following sections, we investigate how this fundamental shift in immigration policy during the 1920s affected mortality in US cities.

3 Data

Our empirical analysis draws on annual city-level mortality statistics by cause and annual migration figures to determine if mortality in American cities was affected by the quota-induced reduction in immigration. This section describes our data sources.

The Census Bureau has systematically collected and published annual city-level mortality data since 1900. These volumes record the number of deaths in total and by specific cause at the city level. We digitized the annual city-level mortality data from 1900-1937. Our analysis ends in 1937 for three reasons. First, 1937 marks the onset of modern medicine in the US (Jayachandran et al. 2010), and so past work on mortality often stops in 1936 or 1937 (e.g. Cutler and Miller 2005; Feigenbaum and Muller 2016). Second, there are significant changes in cause-of-death coding after

[20]Immigration from most parts of Asia and the Pacific Islands was already banned in 1917 by the Asiatic Barred Zone (which was a separate section of the Literacy Act), but this zone did not include Japan due to the Gentlemen's Agreement of 1907.

1937 that make it difficult to standardize the panel (Feigenbaum et al. 2019). Third, we calculate mortality rates by dividing the number of deaths by city population and control for age structure in the main analysis; both require information from the complete population censuses, which are only available up until 1940.[21]

In addition to studying overall mortality, we also group the specific causes of death into three categories: deaths due to infectious diseases, non-infectious diseases, and external causes. Some examples in the infectious disease category are pneumonia, tuberculosis, influenza, and diarrhea; the non-infectious group includes cancer and tumors, cardiovascular diseases, and diabetes; and the external causes encompass all accidents, homicides, and suicides. Appendix Table A.1 lists the specific causes in each category.[22] As with the overall mortality rates, we calculate the cause-specific mortality rates by scaling the total death counts in each category by city population.

We focus on cities with at least 30 years of observations in the 1900-1937 mortality data, which yields a baseline sample of 348 cities.[23] The main results presented in Section 5 are robust to various sample adjustments, including a fully balanced panel of cities at one extreme and a sample that includes any city with at least a year of mortality data at the other extreme.[24]

To show that the quota effects are not simply due to changes in the population composition of cities, we collect three additional types of mortality data. First, we obtain mortality statistics by race for a subset of 128 cities from 1906-1937.[25] Second, we digitize the number of deaths in each city by nativity for 1900-1922; the only years when they are available.[26] Third, we collect national-level mortality statistics by cause, age, and country of origin. These national-level country-of-origin data are only available in few years. We focus on the figures from 1910, a pre-quota year.

We rely on the annual migration statistics from 1899-1930 to construct our measure of quota

[21] We use a log-linear interpolation to estimate the population in each city during intercensal years.

[22] A specific cause of death is only included in one of the three categories if we are able to construct a consistent series for that cause throughout the sample period.

[23] The mortality records in 1931 and 1932 are much thinner, and we only observe mortality data for 231 of the 348 baseline cities.

[24] Cities can and do change their boundaries slightly in the early 20th century. While we cannot reconstruct a complete history of such changes, our results are robust to aggregating the city-level data to the county-level, as shown in Table A.10.

[25] The mortality records only begin reporting city-by-cause-by-race data in 1906. The data include just two racial categories: white and non-white. Furthermore, not all cities report race-specific mortality data. The cities reporting cause-of-death data by race tend to be the largest cities in the sample or those in the South. Nonetheless, the impact of the quota system is evident even in this subset of cities (see Table 6).

[26] Data by nativity are not available in 1912 and 1913.

exposure.[27] For the years 1899-1924, we digitized annual immigration data published in Willcox (1929); data for the years 1925-1930 are digitized from the *Statistical Abstract of the United States* (U.S. Department of Commerce 1929, Table, 106; 1931, Table 99). The annual quotas by nationality from 1922-1930 are retrieved from the same sources. We also collected annual data on the number of immigrants denied entry at the US border by cause and race for the years 1900-1930 from the *Reports of Commissioner General of Immigration.*

4 Research Strategy

In this section, we describe how we construct our measure of quota exposure; provide preliminary evidence that mortality rates did not vary systematically across cities prior to the quota acts; and present our baseline specification.

4.1 Measuring Quota Exposure

While the quotas reduced immigration inflows at the national level, the intensity of this immigration shock across cities, which we refer to as quota exposure, varied dramatically. We measure quota exposure by combining changes in the magnitude of immigration nationwide with pre-treatment settlement patterns of different immigrant groups by city. Formally, we define quota exposure for city c, *Quota exposure*$_c$, as:

$$\textit{Quota exposure}_c = \frac{100}{P_{c,1910}} \sum_{n=1}^{N} \max\left(\widehat{M}_{n,1922-1930} - Q_{n,1922-1930}, 0\right) \frac{FB_{nc,1910}}{FB_{n,1910}}. \tag{1}$$

There are two components to our measure of quota exposure. The first piece, $\max(\widehat{M}_{n,1922-1930} - Q_{n,1922-1930}, 0)$, calculates the national "shift" of immigrants from each sending country n as a function of the quota.[28] To compute this, we predict how many immigrants from country n would have arrived each year had the quota system not been enacted. The predictions are based on the

[27] The immigration data correspond to the fiscal year, which ends on June 30. For example, the immigration year of 1922 refers to immigration inflows between July 1, 1921 and June 30, 1922.

[28] The "missing immigrant" approach was first developed by Ager and Hansen (2016, 2017).

following regression, which we run separately for each sending country:[29]

$$M_{nt} = \beta_1 \ln t + \beta_2 (\ln t)^2 + \varepsilon_{nt}, \tag{2}$$

where M_{nt} is the actual inflow of migrants from country n in year t over the pre-quota period 1900-1914. We only use the annual inflows from 1900-1914 because the outbreak of World War I (WWI) interrupted immigration to the US substantially. The fitted model (2) can then be used to generate out-of-sample predictions for each nationality n over the period 1922-1930. The average of these predictions, $\widehat{M}_{n,1922-1930}$, captures the *expected* annual inflow of immigrants during the post-quota period in a counterfactual scenario with no restrictions. We interpret the difference between $\widehat{M}_{n,22-30}$ and $Q_{n,22-30}$, the latter of which denotes the average annual quota for nationality n from 1922-1930, as the average number of missing immigrants from country n each year due to the quota system. In cases where $\widehat{M}_{n,22-30} - Q_{n,22-30}$ is negative, we set the number of missing immigrants to zero; this is always the case for countries without legal quotas, such as Canada and Mexico.

The second component of equation (1), $\frac{FB_{nc,1910}}{FB_{n,1910}}$, then distributes the missing immigrants across cities based on the share of immigrants from country n living in city c in 1910.[30] This assignment rule draws on the well-documented tendency of new immigrants to settle in places with more immigrants from the same country of origin (Card 2001). We would thus expect more immigrants to be missing after the quota system was in place in cities that had larger pre-existing communities of the targeted nationalities. Summing over all immigrant nationalities and normalizing by the city population in 1910 (scaled by 100, $\frac{100}{P_{c,1910}}$) gives the annual number of missing immigrants per 100 inhabitants in city c.[31]

For concreteness, Figure 1 illustrates the computation of missing immigrants for four sending countries. Panels A and B look at Russia and Italy, two countries heavily affected by the quota system, while Panels C and D consider Ireland and Sweden, two countries with relatively generous

[29]The regression model allows for potential non-linearities in immigration flows. Alternative functional forms modelling such non-linearities yield similar results. If the predicted inflow for nationality n in year t is negative ($\widehat{M}_{nt} < 0$), it is set to zero. In Figure A.1, we show that our main results are quite robust to other sensible ways to predict counterfactual immigration inflows after the quota acts were in place.

[30]The settlement patterns of immigrants are computed from the complete counts of the 1910 census.

[31]Equation (1) is our preferred measure of quota exposure as it can be interpreted in terms of immigration rates. For robustness, Appendix A.1 details an alternative measure of exposure which calculates the "bite" of the quota system at the city level. Appendix Table A.4 presents the baseline results with this alternative.

quotas. The black solid lines refer to the actual inflows of the respective nationalities while the black dashed lines denote the predictions. Panel A shows that over 150,000 Russian immigrants are predicted to arrive in 1922, while under the Immigration Act of 1921, the annual quota for Russians was about 30,000 (solid red line).[32] This implies that about 120,000 Russian immigrants were "missing" in 1922 due to the quota system. These missing Russian immigrants are then allocated across US cities according to their settlement patterns in 1910. Figure 1 also shows that the quota system resulted in missing Italian immigrants (Panel B), but the predicted immigration flows for Irish and Swedish immigrants are lower than their quota numbers (Panels C and D), implying that there were no missing immigrants from these countries.[33]

Overall, the quota system substantially curtailed immigration in the 1920s. Figure 2 shows the total number of actual arrivals to the US from quota countries (solid line) and the number of immigrants we predict would have arrived had the quotas not been imposed (dashed line). The difference between these two lines gives the total number of missing immigrants for each post-quota year. We observe approximately 720,000 missing immigrants each year under the Immigration Act of 1921. This number rises to about 860,000 with the Immigration Act of 1924. Relative to the total US population in 1920, this translates to an *annual* average of 0.8 missing immigrants per 100 inhabitants.

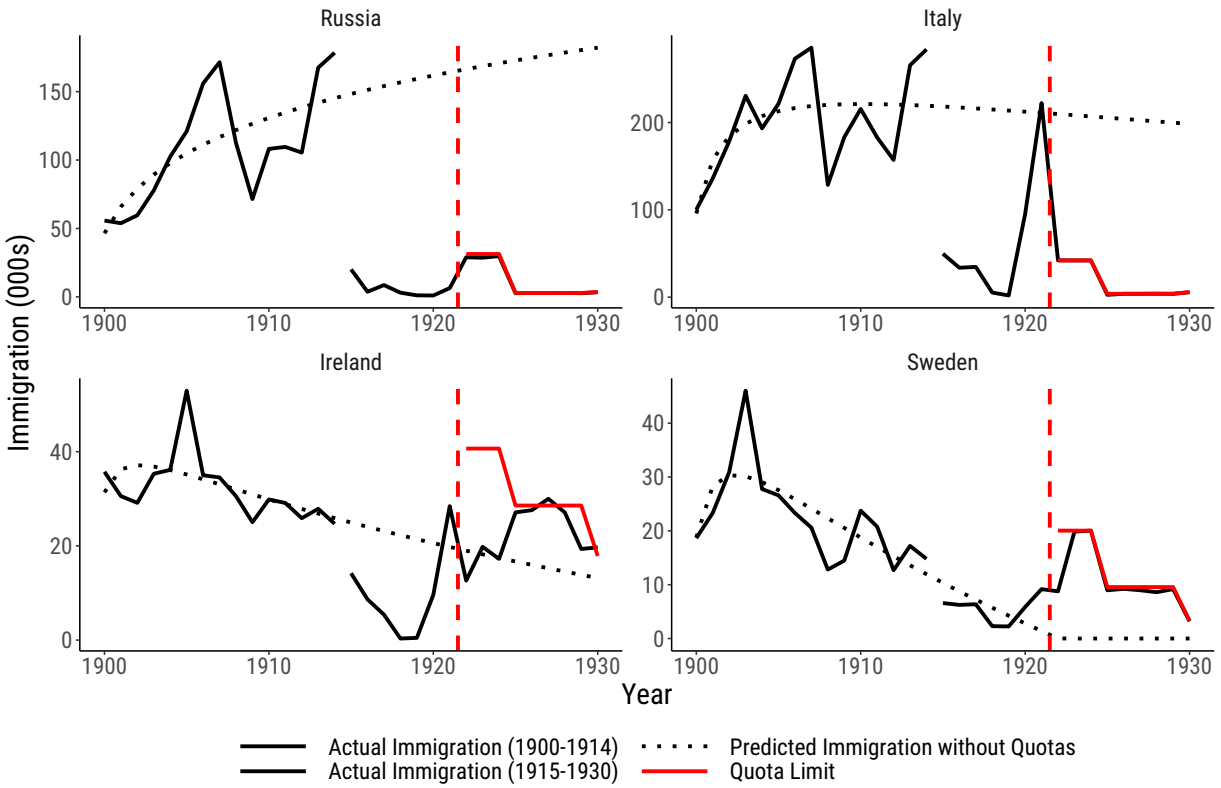
Figure 3 shows a map of log quota exposure for the cities in our baseline sample. Our cities are mostly located in the Northeast and Midwest.[34] In terms of the intensity of the immigration shock, cities in the South are generally less affected. Despite the geographic clustering of cities and treatment intensity, our results are robust to controlling for state-by-year fixed effects, suggesting that they are not driven by time-varying differences across states.

[32]During this period in history, most Russian immigrants were not ethnically Russian but Jews from the Pale of Settlement. The immigration data do not distinguish people by race or ethnicity, only by country of origin. For more on Russian and Jewish immigrants at the time, see, e.g., Aaronson et al. (2018) and Xu (2019).

[33]The prediction for Swedish migrants is set to zero for the years with negative predictions.

[34]In the appendix, we zoom in on cities located in New England and the upper Mid-Atlantic region (Figures A.4-A.7).

Figure 1: Missing Immigrants from Four European Countries: Russia, Italy, Ireland, and Sweden ('000s)



Notes: We choose these four countries to illustrate our procedure for predicting missing immigrants. We plot actual migration, country-specific migration quotas, and predicted migration in a counterfactual scenario with no quotas based on pre-WWI migration flows. For Russia and Italy, the quotas were binding and both the actual number of immigrants and the quota limit are substantially below pre-WWI levels. In contrast, Irish immigration started falling before the outbreak of WWI and the quotas were not particularly restrictive. Swedish immigration, which was also on a downward trend, seems unaffected by the quotas.

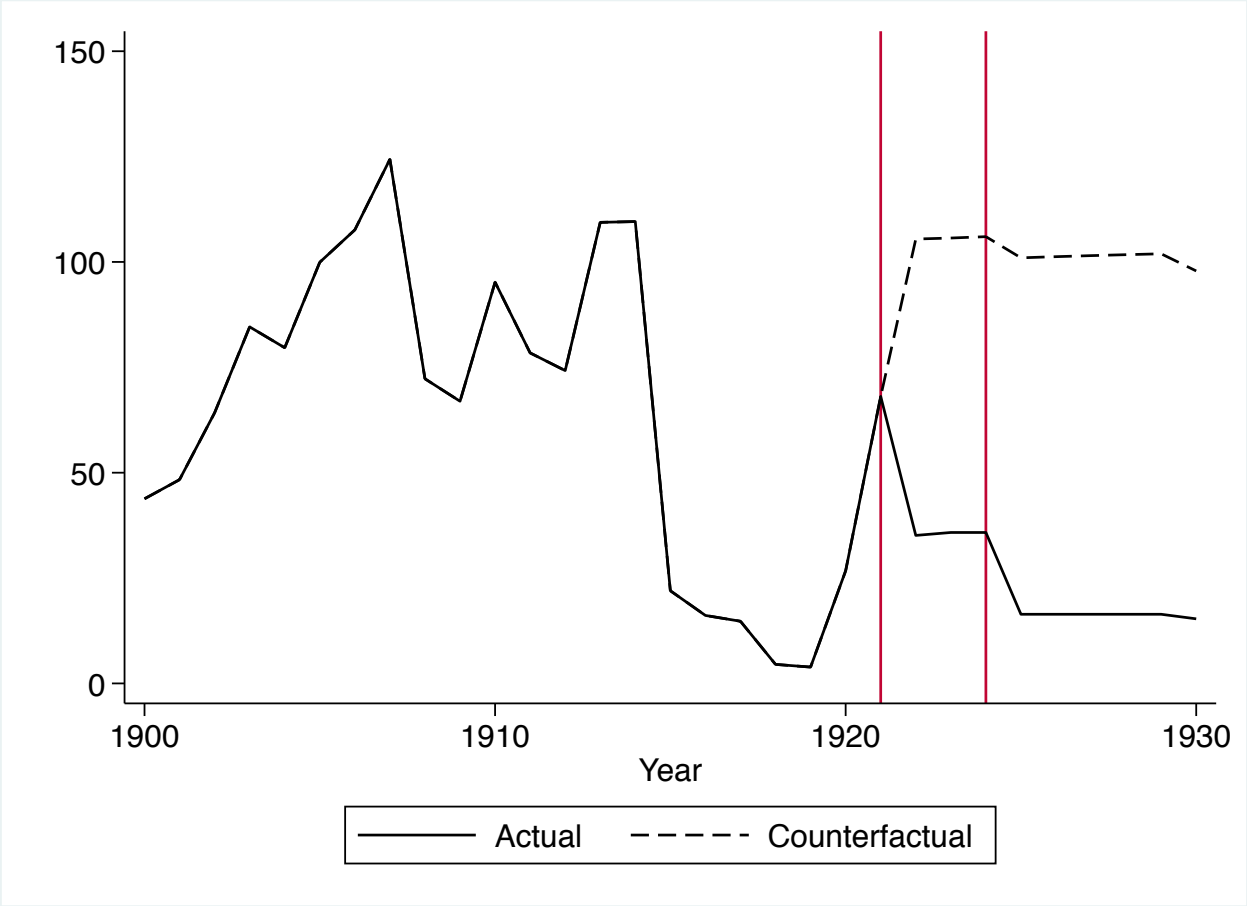
4.2 Level and Trend Balance

Before describing our estimation strategy, we show that pre-quota mortality rates were relatively balanced across cities with varying degrees of subsequent quota exposure and that pre-quota foreign-born mortality penalties do not correlate with quota treatment intensity.

First, we consider differences in both the level and trend in city mortality. While descriptive in nature, these exercises provide a first check of whether quota exposure is systematically related to changes in mortality rates before the quotas were imposed. In the absence of such associations, one may be more confident that our baseline results are not compromised by unobserved confounders.

To quantify level differences in mortality across cities during the pre-quota period, we use

Figure 2: Actual and Counterfactual Immigration from Quota Countries (10,000s)



Notes: This figure shows the actual (solid line) and counterfactual (dashed line) immigration from quota countries. The difference between the two lines gives the number of missing immigrants. For the main analysis, we calculate the number of missing immigrants by sending country rather than as a whole, and then apportion the missing immigrants across cities based on historical settlement patterns. This gives us the number of missing immigrants by city.

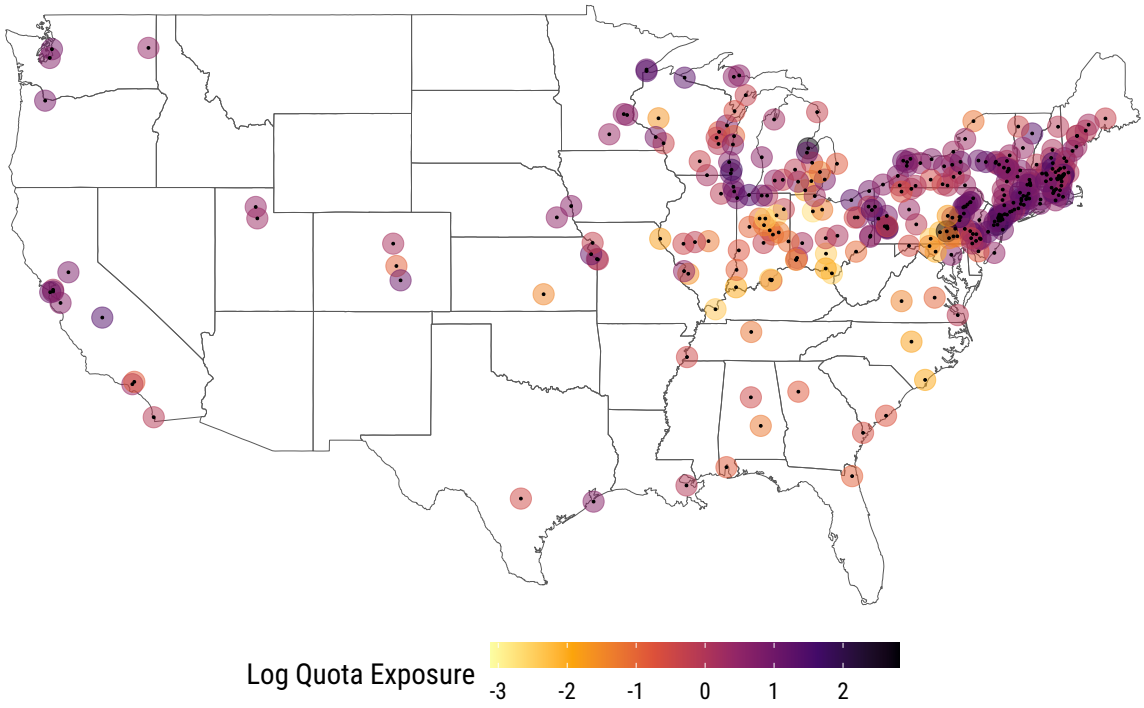
cross-sectional data from 1910 and 1920 and implement a balancing test:

$$\ln m_c^D = \alpha + \beta^D \, Quota \ exposure_c + \mathbf{AGE}_c' \Phi^D + \varepsilon_c^D, \tag{3}$$

where $\ln m_c^D$ is the log mortality rate for cause-of-death group D in city c, as measured in 1910 or 1920. We control for a city's age structure, $\mathbf{AGE}_c'$, and cluster standard errors at the city level.[35] For each outcome, β^D captures the level differences across cities that would later be differentially exposed to the quota system.

[35]Specifically, $\mathbf{AGE}_c'$ includes the share of a city's population aged 20-49 and aged 50+, with 0-19-year-olds comprising the omitted group. Including these controls is akin to standardizing mortality by age.

Figure 3: US City Map of Quota Exposure



Notes: This map displays the cities in our baseline sample and the corresponding quota exposure for each city. Darker colors denote higher values of quota exposure. We omit Key West, FL to avoid plotting what appears to be an errant point off the tip of Florida. In Figure A.4 we zoom in on the northeast. In Figures A.5, A.6, and A.7, we zoom in on Massachusetts, New Jersey, and Pennsylvania, three of the states with the most coverage of cities in our data.

In both pre-quota periods, we find that cities which subsequently faced different rates of quota exposure were relatively balanced across all four cause-of-death groups (all causes, infectious causes, non-infectious causes, and external causes). Table 1 displays the estimates from equation (3). The odd columns show the results for 1910, while the even columns are based on 1920. Reassuringly, the estimated coefficients of interest are all close to zero, substantively small, and statistically insignificant.

Moving beyond level differences, we now consider if the trends in mortality varied systematically across cities before the quota system was implemented. For simplicity, we divide cities into two groups—those that are below and above the median quota exposure—and compute the average mortality rate for each group by year. While the use of just two groups generates a relatively

Table 1: Balancing Tests for 1910 and 1920

	Dependent variable is mortality rate (in logs) for:							
	All Causes		Infectious Causes		External Causes		Non-Infectious Causes	
	1910	1920	1910	1920	1910	1920	1910	1920
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Quota Exposure	-0.002	-0.007	0.013	0.011	-0.009	-0.018	-0.005	-0.004
	(0.007)	(0.007)	(0.012)	(0.008)	(0.011)	(0.011)	(0.009)	(0.007)
Observations	348	348	348	348	348	348	348	348
R-squared	0.018	0.091	0.121	0.072	0.054	0.025	0.403	0.506

Notes: This table reports the results from a balancing test where we regress our outcomes, measured in 1910 or 1920, on future quota exposure, controlling for the age structure of a city. The outcomes are the mortality rate (in logs) for all causes (columns 1 and 2), infectious causes (columns 3 and 4), external causes (columns 5 and 6), and non-infectious causes (columns 7 and 8). Appendix Table A.1 lists the individual causes included in these categories. Robust standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

coarse comparison, it allows for a clearer visualization; the full variation in quota exposure will be exploited in the main analysis. Figure 4 depicts the trends for cities below (dashed line) and above (solid line) the median quota exposure.

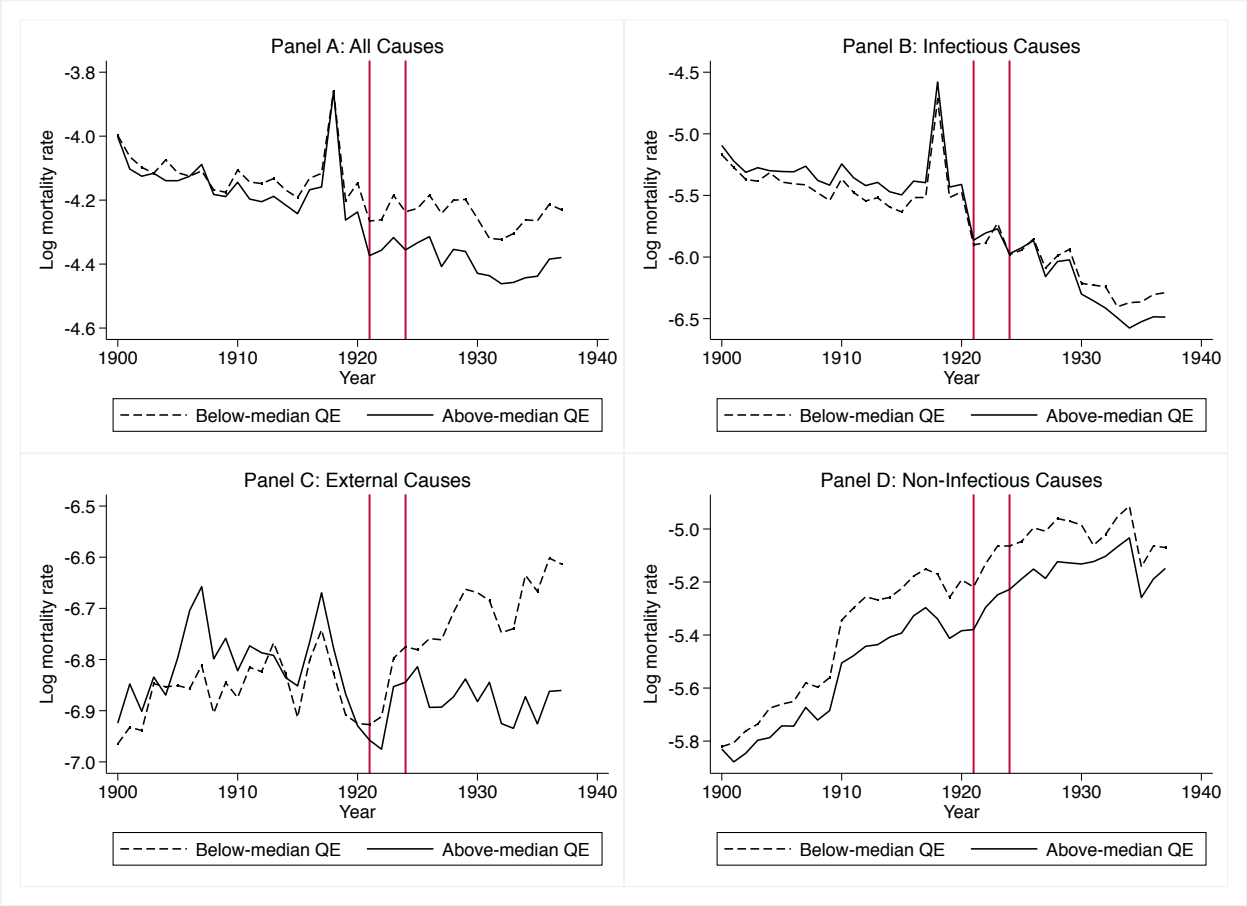
We observe a general downward trend for the all-cause and infectious-cause mortality rates (Panels A and B) until the Great Influenza Pandemic hit the US in 1918. Thereafter, the all-cause mortality rate decreased slightly more in the above-median quota exposure group. This small pre-quota gap widens in the post-quota period from 1922-1937, even as average mortality rates continue falling for both groups. A similar pattern is observed for the infectious-cause mortality rate, but there is less distinction between the two groups of cities.[36]

The patterns are slightly different for mortality associated with external and non-infectious causes. For external-cause mortality (Panel C), cities below and above the median quota exposure do not follow each other as closely prior to the quota restrictions. However, visually, there are no persistent patterns in the pre-quota differences.[37] Furthermore, both groups had similar rates of mortality just before the full implementation of the quotas. The pattern changed dramatically after 1924, where we see a clear divergence. For mortality due to non-infectious causes (Panel D), we observe a persistent upward trend both before and after the introduction of the quotas, with a fairly stable gap between the two groups of cities. This suggests that the immigration restrictions

[36]We also see that the level of mortality due to infectious causes was higher for the more exposed group prior to the quotas. This is not mirrored in the all-cause mortality rate, probably because the level of mortality due to non-infectious causes was higher for the less affected group (see Panel D).

[37]One reason for the variation in deaths due to external causes could be statistical—this is a relatively small cause-of-death group.

Figure 4: Average Log Mortality Rates by Quota Exposure



Notes: This figure shows the average log mortality rates for all causes, infectious causes, external causes, and non-infectious causes, separately for two groups of cities: The dashed lines denote cities with below-median quota exposure, while the solid lines refer to cities with above-median quota exposure.

may have had little effect on this mortality category.

The Great Influenza Pandemic of 1918-1919 could have affected all four mortality rates to some degree. While the intensity of this shock appears reasonably similar for cities above and below the median quota exposure, the pandemic could have had long-run repercussions that varied systematically across cities. To account for this possibility, in our baseline specification we will control for mortality rates in 1918-1921 interacted with a full set of year fixed effects. This eliminates all variation in mortality during these years and accounts for potential long-run effects related to the Great Influenza Pandemic.

Beyond the level and trend in mortality rates, we also find little systematic relation between the foreign-born mortality penalty prior to the implementation of the quotas and subsequent quota

exposure. We draw on mortality-by-nativity data from 1900-1922 which, as we described in the data section, only exist through 1922 and, thus, can only be used in these pre-quota specification checks, not as outcomes directly. Nonetheless, data allows us to assess if excess mortality for immigrants is systematically related to quota exposure in the pre-quota period. Figure 5 presents event-study estimates of triple interactions between quota exposure, a dummy for foreign-born, and year fixed effects from a regression that stacks the mortality data and controls for age structure, city-by-year fixed effects, foreign-born-by-year fixed effects, and city-by-foreign-born fixed effects. Generally, cities with different levels of quota exposure faced similar foreign-born mortality penalties before the introduction of quotas. The immigrant mortality penalty was slightly lower in some years for more exposed cities, but this would work against our main finding. The one year with a seemingly large correlation between future quota exposure and the foreign-born mortality penalty is 1918, during the Great Influenza Pandemic, though the difference is not statistically significant. We also see that the foreign-born mortality penalty did not differ in trends in the years before the quotas. From Figure 5, we conclude that cross-city differences in the immigrant mortality penalty are unlikely to confound our main analysis.

4.3 Estimation Approach

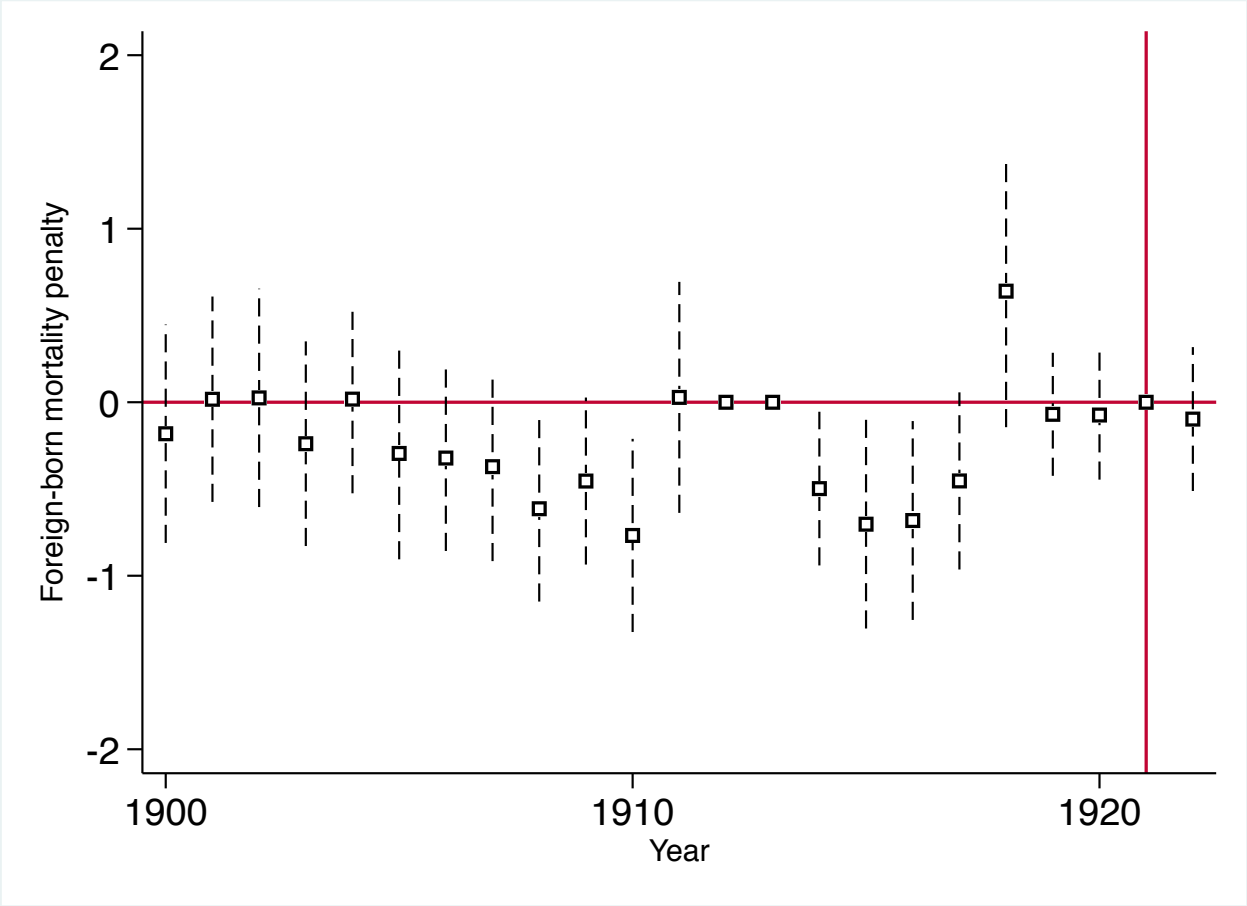
We estimate the impact of the quotas on mortality by comparing cities that were more and less affected by the quotas, before and after they were implemented (a difference-in-differences (DiD) approach). Specifically, we run the following event-study model with mortality data from 1900-1937:

$$\ln m_{ct}^{D} = \alpha + \sum_{j=1900}^{1937} \beta_j^D \ \textit{Quota exposure}_c \times I_t^j + \mu_c + \mu_t + \mathbf{AGE}_{ct}'\Phi^D + \mathbf{X}_{ct}'\Gamma^D + \varepsilon_{ct}^D, \qquad (4)$$

where m_{ct}^D is the annual mortality rate for cause-of-death group D (i.e., all causes, infectious causes, non-infectious causes, or external causes) in city c during year t.[38] Our measure of the immigration shock, $\textit{Quota exposure}_c$, is interacted with year fixed effects (I_t^j), leaving out 1921 as the omitted year. At a minimum, we always control for city fixed effects (μ_c), year fixed effects (μ_t), and the

[38] While grouping diseases into broad categories reduces noise, it implicitly forces the specific causes within a group to have the same treatment dynamics. Table 3 studies how the quota restrictions affected several specific infectious causes of deaths, such as pneumonia, influenza, and tuberculosis.

Figure 5: Differences in the Foreign-Born Mortality Penalty by Quota Exposure and Year



Notes: This figure shows the event-study estimates for the foreign-born mortality penalty based on triple interactions between quota exposure, a foreign-born dummy, and year fixed effects. The omitted reference year is 1921. The mortality data are stacked by nativity and the regression controls for city-by-year fixed effects, foreign-born-by-year fixed effects, and city-by-foreign-born fixed effects. Mortality-by-nativity data are not available in 1912 and 1913.

age structure of a city ($\mathbf{AGE}'_{ct}$). In the baseline specification, the vector of controls $\mathbf{X}'_{ct}$ includes log population in 1910 interacted with year fixed effects, and mortality in four pre-quota years ($\ln m^D_{c1918}$, $\ln m^D_{c1919}$, $\ln m^D_{c1920}$, and $\ln m^D_{c1921}$) interacted with year fixed effects to control for effects of the Great Influenza Pandemic. As a robustness check, we also add state-by-year fixed effects (μ_{st}) to estimating equation (4).[39] We cluster standard errors at the city level.

The key identifying assumption for our DiD approach is that mortality rates in high quota-exposed cities would have developed similarly to low quota-exposed cities had the quota system

[39]To account for the immigration shock due to WWI, we build a WWI control in a similar way to our measure of quota exposure. Our quota exposure effects are robust to the inclusion of this control, as shown in Appendix Table A.2.

not been in place. While not directly testable, an absence of pre-trends would suggest that the identifying assumption may be reasonable. The post-quota coefficients, β^D, can then be interpreted as the impact of quota exposure on mortality rates in a particular year.

After presenting the event-study estimates, we modify equation (4), replacing the year fixed effects with a treatment indicator for the period after 1921. This is a pure mean-shift model that quantifies the average effect of the quota system. It will also be used to test for heterogeneous treatment effects. Our baseline results are not population weighted, but our findings are robust to population weighting, as we show in Table A.9.[40]

Our DiD strategy shares similar features with the classic shift-share instrumental variables approach (Bartik 1991; Card 2001) since we rely on past settlement patterns and aggregate shocks to predict subsequent migration flows to each city. However, in contrast to the classic shift-share method, our strategy properly isolates the policy-driven variation in aggregate migration flows induced by the quota system. A typical shift-share approach exploits all migration flows (domestic and abroad) that occur over the period of interest and is thus not well-suited for studying specific policy changes.[41] In addition, using the quota system for identification offers two further advantages over the classic shift-share setup (without any major shocks). First, the quota system abruptly curtailed immigration inflows at the national level. The change in policy is thus plausibly exogenous to local economies, increasing our confidence in the validity of the research design here. Second, because the quotas affected the origin-composition of immigrant flows significantly, this allays potential concerns that our results could conflate the short- and long-run effects of immigration shocks (Jaeger et al. 2018).

[40] There are arguments for and against weighting. Solon et al. (2015) give three reasons for weighting when attempting to estimate causal effects: to increase precision by correcting for heteroskedasticity, to address non-random sampling, and to obtain average treatment effects in the presence of heterogeneity. However, they find that these motives are not always justifiable. Chodorow-Reich (2019) shows that population weighting can increase the degree of bias when samples are small. While our conclusions are not sensitive to weighting, we emphasize the unweighted results as our unit of analysis is the city.

[41] Because the classic shift-share approach exploits multiple shocks (i.e., "shifters"), it is not always possible to establish a clear zero date to test for the existence of pre-trends. In contrast, our identification strategy is based on the quota acts, which provide a clear zero date that can be exploited to validate our research design. See Goldsmith-Pinkham et al. (2018) for a discussion and comparison of the shift-share IV- and DiD-estimation methods.

5 The Impact of the Quota Policy on Urban Mortality

We begin our empirical analysis by showing the event-study estimates from equation (4), controlling for city and year fixed effects, age structure, log city population size in 1910 interacted with year fixed effects, and the 1918-1921 mortality rates interacted with year fixed effects. This specification allows us to track the evolution of the quota effects and to check if there were any differential trends before the quotas were enacted.

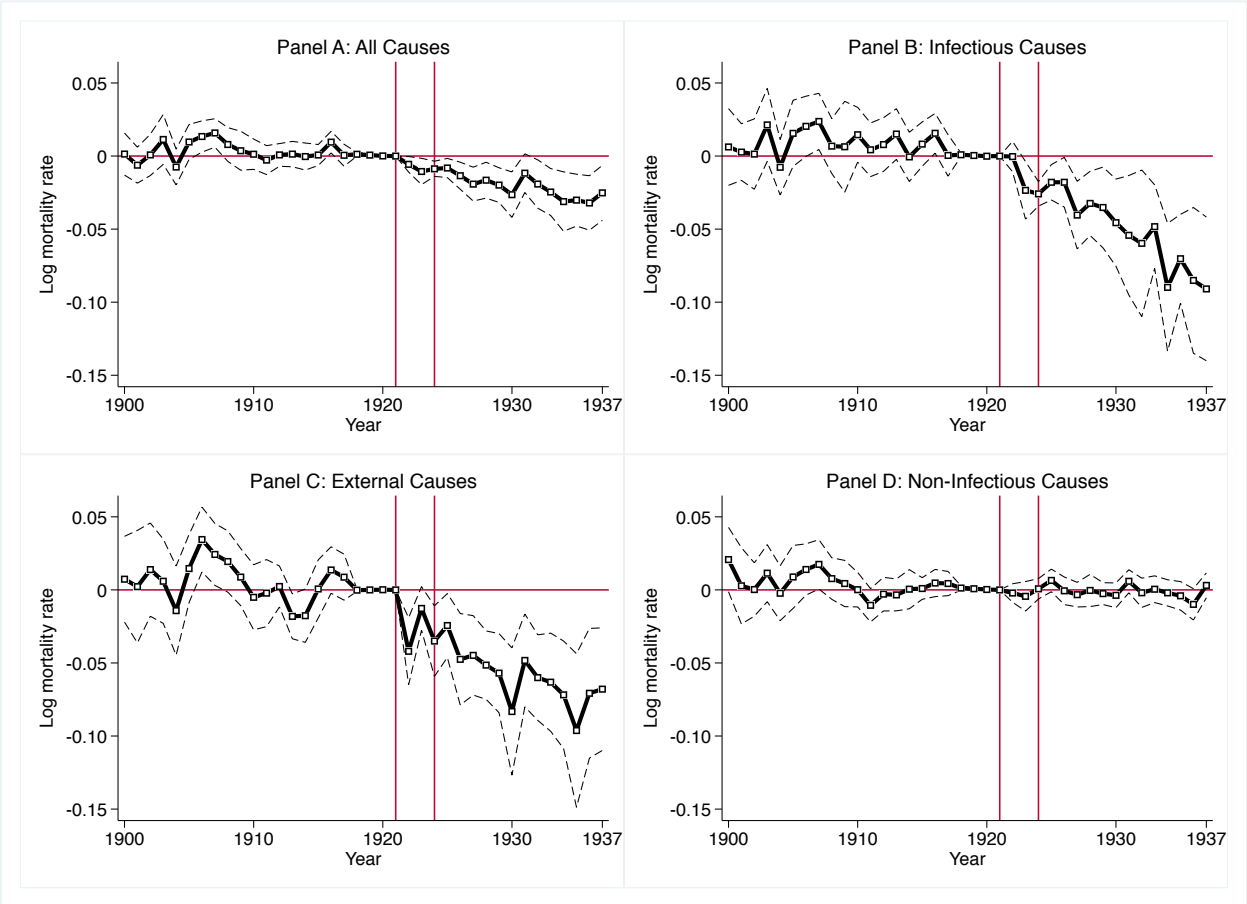
Figure 6 displays the results. For the all-cause mortality rate (Panel A), a clear pattern emerges after the policy change: more quota-exposed cities experienced sustained declines in mortality relative to less exposed cities. Using 1929 as an example, we estimate a coefficient of $\hat{\beta}_{1929} = -0.02$, which implies that losing one immigrant per 100 inhabitants causes the mortality rate to decline by 2 percent. This effect increases somewhat until 1936, when it reaches 3.2 percent. Panel A also illustrates the relatively flat pre-trends in the all-cause mortality rate from 1900 to the outbreak of the Great Influenza Pandemic (1918/19), providing empirical support for the identifying assumption.[42]

We explore which categories of death are driving the relative decline in overall mortality for cities that were more exposed to the quota restrictions in the remaining panels in Figure 6. We find that deaths due to infectious diseases (Panel B) and external causes (Panel C) declined substantially after the quota system was introduced.[43] However, Panel D reveals that there are no significant differences in non-infectious causes of death between high and low quota-exposed cities, which suggests that the declines in infectious and external causes do not simply reflect changes in the population composition. Immigrants had excess mortality in all three causes-of-death groups (see Figure A.3 in the appendix), but only mortality due to infectious and external causes respond to

[42]Recall that we mechanically force the pre-quota estimates to zero during the years of the Great Influenza Pandemic by including the 1918-1921 mortality rates. Appendix Figure A.2 shows the estimates from a specification that includes mortality rates from 1900-1921—the post-quota treatment effects are similar to those in Figure 6.

[43]External causes of death include accidents, suicides, and homicides. Since the *Mortality Statistics* only report accidents and homicides separately after 1920, it is not possible to separate out pre-quota trends between them. Nonetheless, when estimating post-quota treatment effects separately, we find that mortality due to both causes declined similarly after 1921. It could be that the relative decline in deaths due to external causes reflects the clustering of quota-affected immigrants into hazardous occupations. Section A.2 tests whether more quota-exposed cities experienced larger declines in the share of people working in dangerous jobs, and finds no evidence that this was the case.

Figure 6: Event-Study Estimates of Quota Effect by Cause-of-Death Category



Notes: This figure shows the event-study estimates of the effect of quota exposure on log mortality rates based on estimation equation (4) by general cause-of-death category: all deaths (a), infectious (b), external (c), and non-infectious (d). We include controls for city age structure, city fixed effects, year fixed effects, log city population in 1910 interacted with year fixed effects, and log city mortality rates for 1918-1921 interacted with year fixed effects. The omitted reference year is 1921.

the introduction of the quota policy.[44] In Section 6, we disentangle the mechanical composition effect from the spillover effect more formally.

Table 2: Baseline DiD Estimates of Quota Effect by Cause-of-Death Category

	Dependent variable is mortality rate (in logs) for:							
	All Causes		Infectious Causes		External Causes		Non-Infectious Causes	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Quota Exposure × Post	-0.021***	-0.021**	-0.050***	-0.042***	-0.056***	-0.050***	-0.005	0.004
	(0.008)	(0.008)	(0.016)	(0.013)	(0.017)	(0.016)	(0.006)	(0.003)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-by-Year FEs	No	Yes	No	Yes	No	Yes	No	Yes
Observations	12587	12333	12587	12333	12587	12333	12587	12333
R-squared	0.794	0.829	0.859	0.885	0.588	0.638	0.874	0.898

Notes: This table reports the baseline DiD estimates. The outcomes are log mortality rates for our four cause-of-death categories (all causes, infectious causes, external causes, and non-infectious causes). Appendix Table A.1 lists the individual causes included in each category. Quota exposure is defined in equation (1) and interacted with an indicator for years after 1921. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and controls for the city's age structure. The even-numbered columns also control for state-by-year fixed effects. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

With a more parsimonious DiD specification—we replace the year effects in the event-study with a post-treatment indicator—we continue to find that more exposed cities experienced relatively greater declines in mortality rates due to all causes, infectious causes, and external causes, but not for non-infectious causes. Table 2 reports the results from this analysis. The odd-numbered columns contain the same controls as the event-study specification, while the even-numbered columns add state-by-year fixed effects, forcing the comparison to be between cities in the same state and year but with different degrees of quota exposure. State-by-year fixed effects hold constant state specific legislation implemented in different years, such as female suffrage, which previous research has shown to improve population health (Miller 2008). Reassuringly, relatively small differences in the estimated DiD coefficients are observed between the two specifications.[45] The point estimates

[44]There are two caveats here. First, non-infectious deaths tend to occur at later ages—it would thus take more post-quota years for a mechanical (or compositional) effect to materialize. Second, although age-specific mortality rates for non-infectious causes (such as cancer) are high at later ages, relatively few immigrants end up dying from those as they are likely to succumb to infectious diseases at earlier ages.

[45]Our findings are also robust to controlling for pre-quota city-specific linear trends. In particular, we

imply that every one less immigrant per 100 inhabitants results in the all-cause mortality rate declining by 2.1 percent, the infectious-cause rate falling by 5.0 percent, and the external-cause rate dropping by 5.6 percent. We do not find any statistically significant effects on mortality due to non-infectious causes and thus focus on the other three cause-of-death groups in the subsequent analysis.

We can examine the effects of the missing immigrants by specific cause of death (Table 3). The point estimates are negative and statistically significant for pneumonia (column 1), diarrhea (column 5), measles (column 6), diphtheria (column 7), deaths in early infancy (column 9), and accidents/homicides (column 10). Other causes, such as tuberculosis (column 2), influenza (column 3), or suicide (column 11), have small and insignificant estimates. The most important single cause-of-death that the quotas reduced was pneumonia—important in that, when combined with influenza, it was the leading cause of death in the urban US in 1900—and this aligns with our crowding interpretation.

Our main conclusions are robust to different ways of implementing the analysis. First, similar results are obtained with an alternative measure of quota exposure that captures the "bite" of the quota system as illustrated in Greenwood and Ward (2015) (see Table A.4). Second, in Figure A.1, we show that our main results are quite robust to other sensible ways to predict counterfactual immigration inflows after the quota acts were in place. Third, we consider the numerator and denominator of mortality rates separately. Because mortality counts are scaled by log linearly interpolated population data in the baseline analysis, one might worry that the quota effects are driven by the population denominators.[46] To address this, Appendix Table A.5 reports the effects of quota exposure on the level of mortality and population size separately. Column 1 reveals that cities that were more exposed to the quota system experienced relative declines in population size, which in itself would increase mortality rates and work against our original results. This inverse relation is also consistent with a crowding interpretation of our initial findings (see Section 7).[47]

follow Goodman-Bacon (2016) by estimating city linear trends in the full sample of cities with all controls prior to 1921, and residualize both the pre- and post-quota mortality outcomes using these estimated trends.

[46]The recent debate over the impact of clean water supply on mortality in US cities (Anderson et al. 2019a; Cutler and Miller 2005, 2019) is a good example of how sensitive conclusions can be to the way in which outcomes are measured. See, also, the work by Arthi et al. (2019).

[47]This evidence does not necessarily contradict the finding in Abramitzky et al. (2019) where quota-missing immigrants were almost fully replaced in urban labor markets. Their analysis is based on decennial data and implemented at a more aggregate level, State Economic Areas (SEAs), compared to cities in our analysis.

Table 3: DiD Estimates of Quota Effect for Selected Single Causes of Death

	Dependent variable is mortality rate (in logs) for:										
	Pneumonia	Lung TB	Influenza	Typhoid	Diarrhea	Measles	Diphtheria & Croup	Whooping Cough	Early Infancy	Accidents & Homicides	Suicide
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
Quota Exposure x Post	-0.040***	-0.002	-0.001	-0.025	-0.133***	-0.135***	-0.070*	-0.029	-0.065***	-0.061***	-0.017
	(0.012)	(0.008)	(0.017)	(0.023)	(0.049)	(0.051)	(0.038)	(0.028)	(0.021)	(0.018)	(0.012)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 x Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates x Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	12587	12328	9590	4984	10798	2118	6057	4509	12432	12587	8642
R-squared	0.663	0.831	0.714	0.859	0.818	0.533	0.663	0.509	0.632	0.585	0.433

Notes: This table reports DiD estimates for specific causes of deaths (see top row). Quota exposure is defined in equation (1) and interacted with an indicator for years after 1921. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and controls for the city's age structure. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses. In Appendix Table A.3, we show that our results are generally robust to including state-by-year fixed effects.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Importantly, the remaining columns of Appendix Table A.5 show large negative effects on the log of total mortality due to all causes, infectious causes, and external causes. We thus obtain a similar picture even without using any population data. Fourth, Appendix Table A.6 implements the DiD specification separately for the eight nationalities that were most affected by the quotas and the remaining nationalities summed together.[48] This exercise explores which immigrant communities are driving the baseline findings. The coefficients are mostly negative but vary in magnitude. We find that the baseline effects are driven primarily by quota restrictions on migrants from Austria, Italy, and Russia, the latter two being major sources of immigration before the quotas. Finally, Appendix Table A.7 shows that our findings are robust to controlling for the roll-out of clean water and sewerage, based on the sample of 25 large US cities in Anderson et al. (2019a).[49]

6 Interpreting the Quota-Induced Decline in Mortality

Could our findings simply reflect changes in the population composition across cities after the implementation of the quotas? Excess mortality of immigrants coupled with a quota-induced reduction in their population can generate a mechanical decline in mortality rates. This would have been straightforward to assess if mortality statistics were available by nativity over the whole sample period. Unfortunately, such data only exist from 1900-1922, almost entirely before the quotas took effect. We need alternative approaches to disentangle the compositional and spillover effects. This section proceeds in four steps.

First, we provide a simple theoretical decomposition of city-level mortality rates to illustrate how excess mortality among the foreign-born could partly explain our findings. Second, we document that the foreign-born had higher mortality rates than the US-born before the quotas were implemented. Third, we present two pieces of indirect evidence suggesting that compositional changes alone are unlikely to tell the full story: the effects are too large to just be composition shifts and immigration restrictions had spillover effects on African-American mortality rates. Fourth, we

[48] The eight nationalities are: Austria, Greece, Hungary, Italy, Poland, Russia, Turkey, and Yugoslavia.

[49] Our results are also robust to controlling for medical supplies, proxied by the number of medical staff and hospitals per 1,000 people in 1910 interacted with a full set of year fixed effects as in Catillon et al. (2018). In addition, many US cities adopted zoning laws during the 1920s to regulate the construction of new buildings. Using data collected on these adoption dates for more than 500 U.S cities, we find that our results are robust to controlling for these laws.

explain why our results are not driven by a variant form of composition change—changes in the selection of immigrants.

6.1 A Simple Decomposition of Mortality Rates

We start with the decomposition exercise. Consider the mortality rate in city c during year t ($m_{ct} \equiv M_{ct}/P_{ct}$), which can be decomposed into US-born (M_{ct}^{UB}) and immigrant deaths (M_{ct}^{FB}):

$$\frac{M_{ct}}{P_{ct}} = \frac{M_{ct}^{UB}}{P_{ct}} + \frac{M_{ct}^{FB}}{P_{ct}}. \tag{5}$$

Changes in mortality can either be driven by changes in the number of US-born deaths and/or changes in the number of foreign-born deaths, both scaled by the total city population (P_{ct}). Substituting the mortality rates ($m_{ct}^{UB} \equiv M_{ct}^{UB}/P_{ct}^{UB}$ and $m_{ct}^{FB} \equiv M_{ct}^{FB}/P_{ct}^{FB}$) into this expression and rearranging the terms yields:

$$m_{ct} = m_{ct}^{UB} + \left(m_{ct}^{FB} - m_{ct}^{UB}\right) \times p_{ct}^{FB}, \tag{6}$$

where $p_{ct}^{FB} \equiv P_{ct}^{FB}/P_{ct}$ denotes the share of foreign-born people in city c during year t.[50] Notice how the excess mortality of immigrants could influence our findings: if mortality rates are higher for immigrants ($m_{ct}^{FB} - m_{ct}^{UB} > 0$), then the larger reductions in the foreign-born share p_{ct}^{FB} for cities that are more exposed to the quotas will mechanically lead to relatively greater declines in overall mortality, even if m_{ct}^{FB} and m_{ct}^{UB} remain unchanged. The third part of this section presents evidence that the quota acts also affected m_{ct}^{UB} directly.

6.2 Excess Mortality of Immigrants

For the composition channel to matter, the mortality rates of immigrants and the US-born must differ. Drawing on the mortality by nativity data that we collected from 1900-1922, we document excess mortality of immigrants in the US over time. Figure 7 plots the foreign-born-by-year fixed effects coefficients from a regression where the outcome is total mortality per 1,000 people,

[50] For simplicity, our decomposition abstracts from internal migration, which might also be affected by the quota restrictions. Incorporating this aspect introduces an additional term on the right-hand side of equation (6), consisting of the internal-migration mortality premium times the city's share of internal migrants.

controlling for the age structure of a city. Consistent with contemporary analysis (Dublin 1916) there is a substantial mortality penalty for immigrants. Apart from the spike in 1918 due to the Great Influenza Pandemic, excess mortality hovers around 5 to 7 deaths per 1,000 people over the pre-quota period.

When we compare the mortality rates of immigrants from specific countries with the US-born, we see similar patterns: at nearly all ages, immigrants have higher mortality rates. In Appendix Figure A.3, we present age-specific mortality rates of white men and women in 1910 for all causes by country of origin.[51] While we are limited to 1910—when such data were reported by country of birth—we see that for all adult ages, immigrants had a mortality penalty.[52] This reinforces the results in Figure 7.[53]

6.3 Evidence Against a Composition-Only Effect

The combination of excess mortality among immigrants and the quota-induced decline in their population could be driving our results. However, we argue that compositional shifts alone are unlikely to be the complete story. In this subsection, we present two pieces of indirect evidence pushing back against such a mechanical effect. First, our results are too large to be explained by such mechanical changes. Second, we show that the decline in mortality rates in quota-exposed cities was also large for African Americans, a group that was almost entirely US-born.

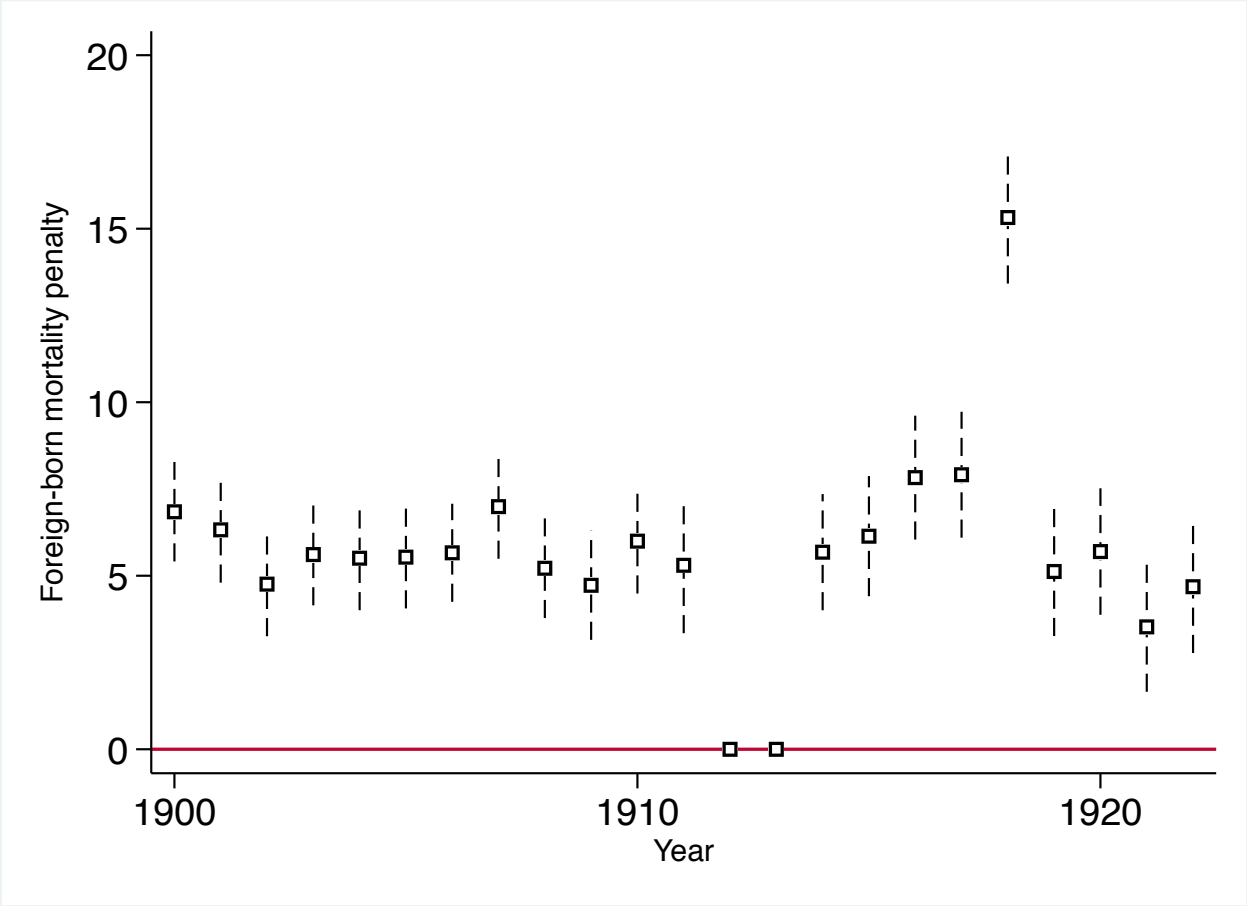
To show that our results are too large to be explained by changes in the population composition alone, we use the implementation of the quota policy to instrument the impact of the foreign-born share on mortality. The exclusion restriction is that the introduction of the quotas only influenced mortality via the share of foreign-born people. This allows us to back out an estimate of immigrants' excess mortality from the coefficient on foreign-born share ($\lambda \equiv m_c^{FB} - m_c^{UB}$). We can then compare it with the actual mortality penalties in Figure 7. A $\widehat{\lambda}$ that is much larger than what is observed

[51] Age-specific mortality rates are reported for people born in Austria, Canada, England and Wales, France, Germany, Hungary, Ireland, Italy, Russia, Scandinavia, Scotland, and the US using the following age-groups: under 10, 10-14, 15-19, 20-24, 25-34, 35-44, 45-54, and 55-64.

[52] Mortality rates are higher for US-born children compared to foreign-born children. This is true both overall and across most causes of death. This reflects the concentration of child mortality around the first year of life coupled with the underrepresentation of foreign-born infants in the under-10 age group.

[53] Irish immigrants had an especially large mortality penalty, a pattern that was also noted contemporaneously (Dublin 1916). However, the extreme excess mortality of Irish immigrants is unlikely to drive our finding of a relative decline in mortality rates for more quota-exposed cities. The quotas for the Irish were fairly generous and there are no missing immigrants from Ireland based on our measure of quota exposure.

Figure 7: Foreign-Born Mortality Penalty Over Time



Notes: This figure shows the evolution of the foreign-born mortality penalty, measured as $m_{ct}^{FB} - m_{ct}^{UB}$, in US cities from 1900-1922. The estimates show the foreign-born-by-year fixed effects coefficients from a regression where the outcome is total mortality per 1,000 people, controlling for the age structure. Mortality data by nativity data are unavailable in 1912 and 1913.

in the data would suggest that m_c^{FB} and m_c^{UB} have also changed in response to the quotas. While this would invalidate the exclusion restriction, it would lend support to our interpretation that the baseline effects are not driven simply by a reduction in the foreign-born share.

We find that $\widehat{\lambda}$ is indeed too large. Table 4 reports the results from estimating equation (6) on our main sample of cities, using $\textit{Quota exposure}_c \times I_t^{post}$ as an instrument for the foreign-born share. We include city and year fixed effects as well as controls for log population size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and the age structure of a city.[54] The first-stage indicate that cities that were more exposed

[54]City fixed effects will eliminate US-born mortality differences across cities (m_c^{UB}) in equation (6) if such differences are constant (or exogenous), which is the null-hypothesis here.

to the quota restrictions experienced larger declines in their foreign-born shares. Reassuringly, the Kleibergen-Paap F-statistic exceeds 10, allaying concerns that the instrument may be weak. Column 1 of Table 4 reports the second-stage result for the all-cause death rate. The coefficient on the foreign-born share, $\widehat{\lambda}$, is positive and statistically significant. Since $\widehat{\lambda} = 31$ with a corresponding 95% confidence interval of approximately $[15; 48]$, it exceeds the foreign-born mortality penalty displayed in Figure 7, which mostly ranges from 5 to 7 deaths per 1,000 people (or $\lambda < 7$ for the all-cause death rate). This suggests that m_c^{FB} and m_c^{UB} were also affected by the quota system. The remaining columns provide the corresponding estimates for deaths due to infectious causes, external causes, and non-infectious causes.

Table 4: Impact of the Foreign-Born Share on City Mortality

	Dependent variable is mortality rate for:			
	All Causes	Infectious Causes	External Causes	Non-Infectious Causes
	(1)	(2)	(3)	(4)
Foreign-born Share	31.37***	15.18***	6.81***	3.96
	(8.58)	(3.95)	(1.33)	(4.43)
Age Structure	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes
Observations	12587	12587	12587	12587
Kleibergen-Paap F	12.12	13.07	15.39	12.09

Notes: This table reports two-stage least squares estimates using $Quota\ exposure_c \times I_t^{post}$ as the excluded instrument for the foreign-born share. The outcomes are mortality rates for the four cause-of-death categories (all causes, infectious causes, external causes, and non-infectious causes). Quota exposure is defined in equation (1) and is interacted with a treatment dummy which equals to one for the years after 1921. All regressions include city and year fixed effects, log population size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and controls for the city's age structure. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

We can also draw on mortality data stratified by country-of-birth, cause, sex, and age to estimate $\widehat{\lambda}$s that can then be compared to the true λs for specific causes, data we previously used to document excess mortality among the foreign-born in Figure A.3. We compare the estimates to the true foreign-born mortality penalty, calculated with or without controlling for age and sex

composition as both vary dramatically between the US- and foreign-born populations. Unfortunately, these data are only available in 1910 and for a subset of causes. Nonetheless, as we see in Table 5, the estimated "effect" of foreign-born share on mortality is much too large for the primary infectious causes of death—pneumonia, tuberculosis, and typhoid—though not always significantly so. The combination of observed pre-quota mortality penalties and composition changes in the urban population are thus unlikely to be driving our baseline results.

Next, we show that mortality rates for African Americans also fell in response to the quota shock. We illustrate this with the subset of 128 cities for which mortality statistics are broken down by race from 1906-1937.[55] Since we cannot distinguish between US- and foreign-born whites in the mortality statistics, the mortality rates of African Americans, the vast majority of whom were born in the US, can serve as evidence of potential spillover effects from immigrants to the US-born in the absence of mortality data by birthplace. The dataset is stacked at the city-by-race-by-year level but we also report estimates separately by race.

African-American mortality improved with the restrictions on immigration, as we show in Table 6, though the effects are likely a bit smaller than the effects on whites. As we cannot split white mortality by country of birth, the effects on whites potentially encompass both compositional and spillover effects. Panel A shows larger declines in mortality due to all causes in more quota-exposed cities. The effect on whites is somewhat larger (the triple interaction in column 2 or a comparison of the Quota $\times$ Post terms in columns 3 and 4) but the difference is not statistically significant. Turning to deaths due to infectious causes in Panel B, we again observe larger reductions in white and black mortality in cities that were more affected by the quotas, with stronger effects for whites. Panel C displays a similar pattern for deaths arising from external causes, though here the direct effect on black mortality is not statistically significant and the relative impact on whites is twice as large.

Overall, the two pieces of evidence presented in this subsection suggest that our main results are unlikely to reflect compositional changes alone.

[55]The cities reporting mortality data by race are a mix of Southern cities of all sizes and larger non-Southern cities (Feigenbaum et al. 2019). The only two racial categories in the mortality data are white and "non-white", the latter of which primarily refers to African Americans in the context of early 20th-century urban populations.

Table 5: Comparing Estimated Effects of Foreign-Born Share on Mortality with True Foreign-Born Mortality Penalties in 1910 by Cause of Death

		Foreign-Born Mortality Penalty (λ)		
	$\widehat{\lambda}$	Raw	Age-Adjusted	Age- and Sex-Adjusted
	(1)	(2)	(3)	(4)
All Mortality	31.37	8.12	5.94	5.90
	[14.57 to 48.17]	[4.09 to 12.15]	[4.50 to 7.38]	[4.46 to 7.33]
Pneumonia	4.54	0.78	0.75	0.74
	[2.48 to 6.59]	[0.39 to 1.17]	[0.56 to 0.94]	[0.56 to 0.93]
Tuberculosis	2.38	0.82	0.51	0.50
	[0.22 to 4.54]	[0.62 to 1.02]	[0.36 to 0.66]	[0.36 to 0.65]
Typhoid	0.33	0.09	0.08	0.07
	[-0.96 to 1.61]	[0.06 to 0.12]	[0.05 to 0.11]	[0.05 to 0.10]
Death in Childbirth	0.61	0.13	0.10	0.11
	[0.23 to 1.00]	[0.06 to 0.21]	[0.03 to 0.18]	[0.05 to 0.18]
Diabetes	0.10	0.16	0.09	0.09
	[-0.39 to 0.60]	[0.10 to 0.21]	[0.06 to 0.12]	[0.06 to 0.12]
Cancer	0.21	0.93	0.53	0.54
	[-1.10 to 1.52]	[0.62 to 1.25]	[0.39 to 0.67]	[0.41 to 0.67]
Suicide	0.24	0.15	0.10	0.10
	[-0.11 to 0.59]	[0.12 to 0.19]	[0.07 to 0.13]	[0.07 to 0.13]

Notes: This table compares two-stage least squares estimates using $Quota\ exposure_c \times I_t^{post}$ as the excluded instrument for the foreign-born share for specific causes of death (column 1) with the foreign-born mortality penalties calculated using 1910 mortality data stratified by country-of-birth, cause, sex, and age (columns 2-4). All $\widehat{\lambda}$s are estimated with regressions that include city and year fixed effects, log population size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and controls for the city's age structure. The sample period is 1900-1937. Below the mortality penalty estimates, we include 95 percent confidence intervals. We construct the foreign-born mortality penalties (and corresponding confidence intervals) by regressing mortality rates by cause by sex by age group by origin country on an indicator for foreign-born country of origin in the 1910 data. In column 3, we add saturated fixed effects for age bins; in column 4, we add sex fixed effects. We use the coefficient on the foreign-born indicator as the "true" mortality penalty.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

6.4 Evidence from the US Border

In the previous subsection, we argued that mechanical composition—city populations with fewer immigrants and more US-born residents could see mortality reductions because of differential mortality rates across groups—is unlikely to explain our main results. In this subsection, we focus on

Table 6: DiD Estimates of Quota Effect by Cause-of-Death Category and Race

	Panel A. All Causes			
	White and Black		White	Black
	(1)	(2)	(3)	(4)
Quota Exposure × Post	-0.040***	-0.045***	-0.045***	-0.042**
	(0.009)	(0.015)	(0.010)	(0.016)
Quota Exposure × Post × Black		0.009		
		(0.017)		
Age Structure	Yes	Yes	Yes	Yes
Race × Year FE	Yes	Yes	Yes	Yes
Observations	5880	5880	2686	2686
R-squared	0.810	0.810	0.814	0.846
	Panel B. Infectious Causes			
	White and Black		White	Black
	(1)	(2)	(3)	(4)
Quota Exposure × Post	-0.039**	-0.058**	-0.053***	-0.037*
	(0.016)	(0.027)	(0.018)	(0.022)
Quota Exposure × Post × Black		0.035		
		(0.030)		
Age Structure	Yes	Yes	Yes	Yes
Race × Year FE	Yes	Yes	Yes	Yes
Observations	5878	5878	2686	2682
R-squared	0.847	0.848	0.865	0.834
	Panel C. External Causes			
	White and Black		White	Black
	(1)	(2)	(3)	(4)
Quota Exposure × Post	-0.067***	-0.087***	-0.086***	-0.042
	(0.009)	(0.015)	(0.014)	(0.029)
Quota Exposure × Post × Black		0.041**		
		(0.017)		
Age Structure	Yes	Yes	Yes	Yes
Race × Year FE	Yes	Yes	Yes	Yes
Observations	5820	5820	2686	2626
R-squared	0.615	0.617	0.679	0.672

Notes: This table reports DiD estimates from "stacked" specifications and separately by race (white and black). The outcomes are log mortality rates for three cause-of-death categories (all causes, infectious causes, and external causes). Quota exposure is defined in equation (1) and is interacted with a treatment dummy which equals to one for the years after 1921. Column 1 reproduces our main results from the subset of cities with mortality-by-race data, using the stacked specification. Column 2 interacts quota exposure with indicators for post-treatment and black mortality. Columns 3 and 4 run the regressions separately for white and black mortality. All regressions include controls for a city's age structure (by race), as well as race-by-year fixed effects and city fixed effects. The sample period is 1907-1937. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

a different form of compositional change within the immigrant population. If the quota system induced a change in the selection of immigrants, a reduction in the relative number of less healthy or sick immigrants arriving in the US after the quota laws could have led to reduced mortality. We

find no evidence for such an effect.

To assess whether there was a change in the health of arriving immigrants after the quotas, we collected and digitized data from the *Reports of Commissioner General of Immigration* for the years 1900-1930. These reports record the number of immigrants denied entry into the US by reason, year, and race, the latter of which resembles national origin in these data.[56] We construct the number of missing immigrants from each national origin as in our baseline setup using historical trends and compare this to the number of people arriving at the border to calculate the ratio of missing immigrants to arrivals. We refer to nationalities with a missing immigrant ratio below (above) the median as the below (above) median quota exposure group.[57]

Very few immigrants from any country were rejected at the US border for infectious diseases.[58] As we show in Panel A of Figure 8, while in some years more than 30 thousand immigrants were rejected entry to the US (1914), the number of people rejected for medical or disease-based reasons never topped out around 2600 (again in 1914). After the quotas were enacted, the total number of rejections for medical reasons peaked at only 880 in 1924 and numbered fewer than 400 in all years after 1925. The small numbers here suggest that this variant of compositional change cannot have played an important role.

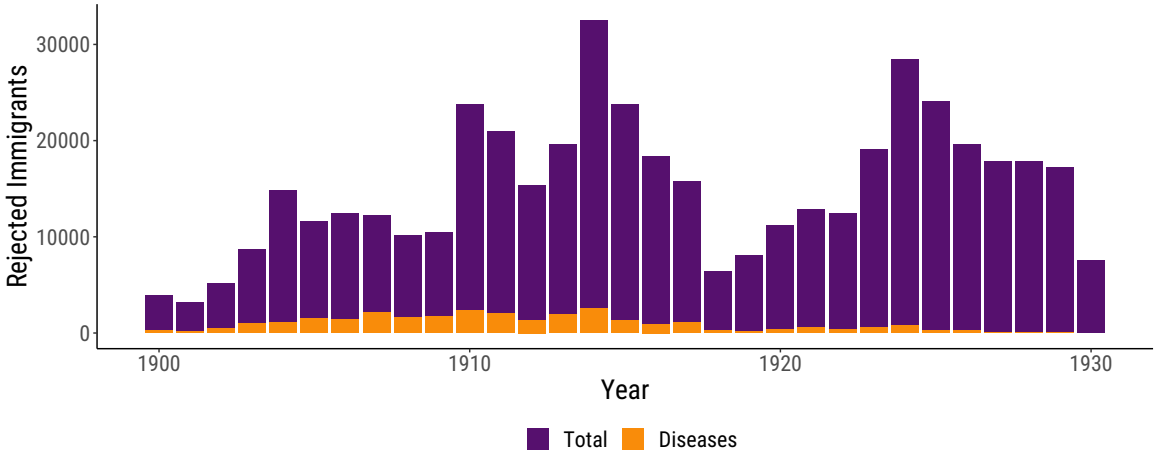
By the time the quotas were implemented, disease was not an important reason for rejection, and the quotas did little to change this, overall or differentially for more or less quota-exposed sending countries (Panel B of Figure 8). We define rejection rates as the number of people rejected at the border (for a given cause, for example, disease-related) divided by the total number of people arriving at the border. The average disease-related rejection rate was below 1 percent in almost all years, and the rates for the two groups of countries track each other in the pre-quota period. While we do see an increase in rates for the above-median group post-quota, this change is not statistically significant in an event-study analysis.

[56]Not surprisingly, given the state of eugenics and racial pseudoscience in the early 20th century, a mapping between race and nationality is relatively straightforward. A few cases, however, are less clear: the race category "English", for example, includes immigrants from both Canada and England.

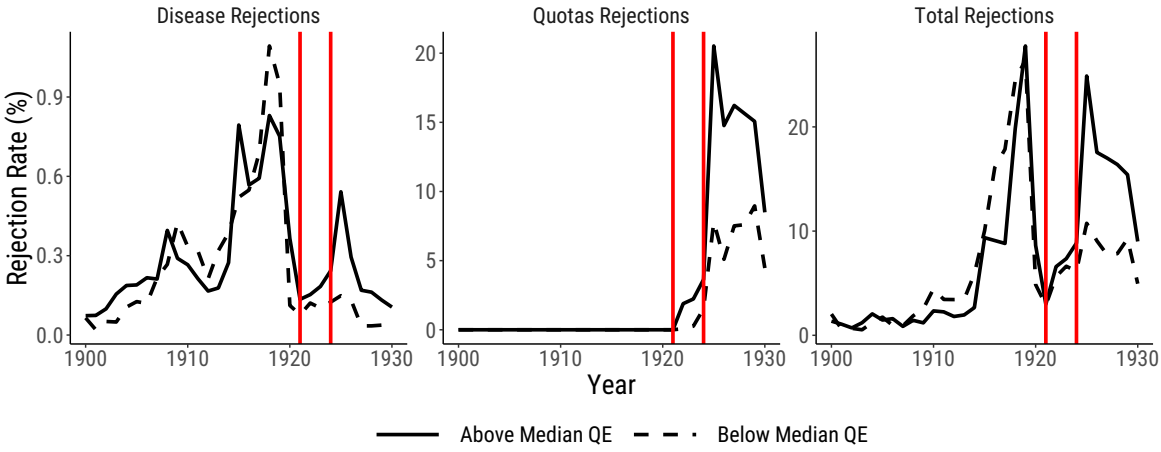
[57]Using this assignment rule, the following groups are considered to be high-quota races or nationalities: Balkans, Dutch and Flemish, Finnish, Greek, Hungarian, Italian, Portuguese, Russian, Spanish, and Turkish. The low-quota races or nationalities comprise: Central Americans, Czechoslovakia, English, French, German, Irish, Mexicans, Pacific Islander, Romanian, and Scandinavian.

[58]Rejections on the basis of infectious diseases can be consistently coded from 1900-1930. Such results are echoed in contemporary findings of the healthy immigrant or healthy migrant effect. See Antecol and Bedard (2006); Aldridge et al. (2018).

Figure 8: Rejections at the US Border by Year and Quota Exposure



(a) Total and Disease-Based Rejection Counts, 1900-1930



(b) Rejection Rates by Quota Exposure, 1900-1930

Notes: In the top panel, we plot the total number of rejected immigrants in each year, both overall and for disease-related reasons. In the bottom panel, we show the average of several metrics of US border rejections for nationalities that fell below ("Below-Median QE") and above ("Above-Median QE") the median of the missing immigrant ratio.

The relative unimportance of disease-related rejections contrasts sharply with rejections due to the quota acts. When we plot the rejection rates for reasons related to the quotas and for all reasons, we see a different story. By definition, rejection rates due to the quotas are zero before the quotas were actually implemented. Thereafter, we see a sharp increase in these rejection rates, particularly for the more exposed group. This shift is mirrored by the total rejection rate, which also shows the similarity in rates for the two groups prior to the quotas. The evidence suggests that the quota policy was not an endogenous response to disease-related rejections at the border.

In this section, we have shown that our baseline findings are unlikely to simply reflect compositional shifts within the US or in the selection of immigrants. In the next section, we document spillover effects from immigrants to the US-born.

7 Spillover Effects from Immigrants to the US-born

The improvements in urban mortality caused by the quota-driven reduction in immigration were large. In this section, we argue that a key mechanism behind these mortality effects was the relaxation of congestion constraints on both housing and access to healthcare services. We provide suggestive evidence for each type of constraint, underscoring the potential health dangers of unmanaged city growth in a setting with weak public health infrastructure.

7.1 Congestion Constraints on Housing

Contemporary observers associated overcrowding with the urban mortality penalty at the turn of the 20th century (e.g., Dublin 1916; Kraut 1994; Stella 1908). The densely populated districts in cities, where most immigrants lived, provided conducive environments for the spread of infectious diseases. In this subsection, we test if the quota policy relaxed such congestion constraints and led to stronger mortality declines in cities that were more crowded initially.

Following Higgs and Booth (1979), we differentiate between the effects of internal and external density. To capture internal density, we use the number of foreign-born people per dwelling in 1910 and standardize our measure to facilitate an interpretation of the results.[59] Our proxy for external density is city population per acre in 1910 (standardized). If overcrowded housing conditions

[59] An alternative measure of internal density is the number of US-born people per dwelling in a city. This measure yields similar results.

contributed to the spread of infectious diseases and if the quota system relaxed such congestion constraints, one would expect stronger declines in deaths from infectious diseases in initially more dense cities that were more exposed to the quotas.

To test our hypothesis, we implement an extended version of the baseline DiD model, including a triple interaction between quota exposure, a post-treatment indicator, and a measure of density. Table 7 presents the results for internal density. The odd-numbered columns include the controls from the baseline specification, while the even-numbered columns add as controls the interaction of internal density in 1910 with a full set of year fixed effects to capture any time-varying changes related to internal density that do not operate via the quota system.

We see evidence of treatment heterogeneity in the coefficients on the triple interaction terms in Table 7. Density affects the effect of the quotas on the all-cause mortality rate (columns 1 and 2), but this is primarily driven by deaths due to infectious causes (columns 3 and 4). The decline in infectious-cause mortality rates is 2.4 percentage points stronger when internal density in 1910 is one standard deviation higher (column 4). No such heterogeneity is observed for the external-cause mortality rate (column 6), while heterogeneous treatment effects on the all-cause mortality rate are significantly smaller and become statistically insignificant when controlling directly for the time varying effect of internal density (columns 1 and 2). External density, on the other hand, appears to be less important, as illustrated in Appendix Table A.8. When we interact the quota shock with the post-treatment indicator and external density in 1910, the point estimates have the same sign as the effects with internal density, but their magnitudes are significantly smaller.

Data limitations prevent us from directly assessing how the quotas affected internal density. Instead we use two alternative indicators of congested or crowded living conditions.[60] In Panel A of Table 8, we proxy for congestion using the number of people living as boarders and lodgers per 1,000 inhabitants.[61] Lodging and boarding were common in immigrant households and associated with overcrowding at the time (Abbott 1936; Park and Kemp 2006; Veiller 1913). We find that cities with higher quota exposure experienced larger declines in the number of people living as

[60] At the time of writing, there is a coding issue with the IPUMS complete census enumerations—each household is coded in a unique dwelling in 1920, 1930, and 1940. This makes it impossible to count the number of people or census households in a census dwelling for these years.

[61] Specifically, we count the number of people coded in the IPUMS complete count data with the following enumerated relationships to the household head: roomer/boarder/lodger (1201), boarder (1202), lodger (1203), roomer (1204), or tenant (1205).

Table 7: Treatment Heterogeneity by Internal Density

	Dependent variable is mortality rate (in logs) for:					
	All Causes		Infectious Causes		External Causes	
	(1)	(2)	(3)	(4)	(5)	(6)
Quota Exposure × Post	-0.021***	-0.021***	-0.053***	-0.053***	-0.053***	-0.053***
	(0.007)	(0.007)	(0.015)	(0.015)	(0.016)	(0.016)
Quota Exposure × Post × Internal Density	-0.009***	-0.008	-0.023***	-0.024**	-0.002	0.003
	(0.002)	(0.005)	(0.005)	(0.011)	(0.006)	(0.011)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes
Dwelling 1910 × Year FE	No	Yes	No	Yes	No	Yes
Observations	12155	12155	12155	12155	12155	12155
R-squared	0.792	0.792	0.858	0.859	0.590	0.591

Notes: This table reports the DiD estimates by cause-of-death category and checks for treatment heterogeneity in terms of standardized mean dwelling size for foreign-born people in 1910 (our measure of internal density). The outcomes are log mortality rates of three cause-of-death categories (all causes, infectious causes, and external causes). Quota exposure is defined in equation (1) and is interacted with a treatment dummy which equals to one for the years after 1921. The corresponding interaction term with dwelling density in 1910 captures treatment heterogeneity. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and controls for the city's age structure. The even-numbered columns also control for 1910 internal density interacted with year fixed effects. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

boarders. Each additional missing immigrant per 100 residents led to nearly four fewer boarders per 1,000 residents, compared to a base of about 70 boarders per 1,000 residents (column 1). Both the share of US- and foreign-born boarders fell, with a larger impact on the latter; albeit starting from a higher base of 101.8 foreign-born boarders per 1,000 residents versus 61 US-born boarders per 1,000 residents (columns 2 and 3). Splitting the analysis by race, we observe that the quotas had a stronger impact on African Americans compared to US-born whites (columns 4 and 5).

The quota restrictions also reduced the number of people living in multifamily households, as shown in Panel B of Table 8.[62] In line with the results for boarders and lodgers, we find that living arrangements changed more in cities that experienced larger reductions in immigration due to the quota policy. The effects are particularly large for foreign-born and black residents in the most quota-exposed cities. Taken as a whole, the evidence suggests that the quotas did relax housing

[62]Specifically, we count the number of people coded in the IPUMS complete count data with 'NFAMS' of 2 or more. According to IPUMS, "NFAMS" is a constructed variable that counts the number of families within each unit. A "family is any group of persons related by blood, adoption, or marriage. An unrelated individual is considered a separate family."

Table 8: The Impact of Quota Restrictions on Crowded Living Conditions

	Panel A. People Living as Boarders and Lodgers				
	Dependent variable: # of Boarders and Lodgers per 1000 people				
				US-Born	
	All	US-Born	Foreign-Born	White	Black
	(1)	(2)	(3)	(4)	(5)
Quota Exposure × Post	-3.65***	-1.82***	-7.66***	-1.69***	-6.50***
	(0.92)	(0.53)	(2.09)	(0.49)	(1.91)
Age Structure	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes
Outcome in 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
Dependent Mean	70.53	59.52	115.89	57.11	153.74
Observations	1627	1627	1627	1627	1569
R-squared	0.891	0.874	0.816	0.872	0.899
	Panel B. People Living in Multifamily Dwellings				
	Dependent variable: # Living in Multifamily Housing per 1000 people				
				US-Born	
	All	US-Born	Foreign-Born	White	Black
	(1)	(2)	(3)	(4)	(5)
Quota Exposure × Post	-7.79***	-4.86***	-8.00***	-4.27***	-9.43***
	(2.27)	(1.39)	(1.97)	(1.17)	(3.16)
Age Structure	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes
Outcome in 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
Dependent Mean	212.75	202.28	237.10	197.74	446.70
Observations	1627	1627	1627	1627	1569
R-squared	0.924	0.915	0.915	0.915	0.983

Notes: This table reports DiD estimates for the number of people living as boarders or lodgers per 1,000 inhabitants (Panel A) or living in multifamily households per 1,000 residents (Panel B). Each column heading indicates the group being analyzed. Quota exposure is defined in equation (1) and is interacted with a treatment dummy which equals to one for the years after 1921. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, the initial number of boarders and lodgers per 1,000 people in 1910 interacted with year fixed effects (Panel A), the initial number living in multifamily housing per 1,000 people in 1910 interacted with year fixed effects (Panel B), and controls for the city's age structure. The sample period is 1900, 1910, 1920, 1930, and 1940. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

congestion constraints, which in turn could have lowered the infectious-cause mortality rates.

7.2 Congestion Constraints on Healthcare

The quota restrictions may have also relaxed congestion constraints on public services, including access to healthcare, which is particularly relevant in the context of mortality and infectious diseases. To shed light on this, we digitized data from a report of the Department of Commerce on

benevolent institutions in 1910. This report contains city-level statistics on the number of hospitals, beds, medical staff, and patients, from which we are able to compute the number of patients relative to either the number of hospitals, beds, or medical staff (all standardized). The first two measures may be thought of as proxies for crowding in the health sector while the latter measures the quality of healthcare. We use these measures to assess how initial sickness (or morbidity) relative to the capacity of the health sector interacted with the introduction of the quotas.

We find evidence that some forms of pre-quota healthcare constraints affected the magnitude of the quota effects, as shown in Table 9. In cities where the pre-quota number of patients per hospital and patients per bed—but not the patients per medical staff—was higher, the quota effects are larger. This pattern is observed for the all-cause, infectious-cause, and external-cause mortality rates.[63] In the case of infectious-cause mortality, for example, a one standard deviation increase in the initial number of patients per bed adds 2.6 percentage points to the main quota effect (column 5). Our findings suggest that cities with initially more crowded hospitals experienced relatively faster declines in mortality rates after the quotas relaxed such congestion constraints through curtailing immigration, whereas differences in hospital quality may have been less important at that time.

7.3 Effects of Missing Immigrants on Rural Health

In this paper, we have examined the effect of immigrants on mortality in cities. We focus on cities because most immigrants lived in cities in the early 20th century and because the urban mortality transition is an important feature of American economic development. However, rural areas also attracted immigrants during the Age of Mass Migration. What effect did the quota policy changes have on health in rural counties? To summarize: very little. We see no evidence that mortality improved in the most (or least) quota-exposed rural counties. As rural American counties in the early 20th century were not crowded, these null effects are in line with our congestion and crowding mechanism.

To analyze the effects on rural mortality, we obtained annual mortality counts for rural counties from Hoehn-Velasco (2018) and scale these with population figures from the decennial censuses, as in our city analysis. We also restrict the sample to counties that were classified as rural in 1920 to avoid

[63]These estimates are based on specifications that control for the respective measures of crowding or quality interacted with year fixed effects, but similar results can be obtained without them.

Table 9: The Impact of Quota Restrictions by Hospital Crowding and Quality

	Dependent variable is mortality rates (in logs) for:								
	All Causes			Infectious Causes			External Causes		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Quota Exposure × Post	-0.019***	-0.018***	-0.013**	-0.055***	-0.054***	-0.047**	-0.043***	-0.041***	-0.036**
	(0.006)	(0.007)	(0.006)	(0.017)	(0.018)	(0.018)	(0.013)	(0.014)	(0.015)
Quota Exposure × Post × Patients per Hospital	-0.016***			-0.027***			-0.027***		
	(0.006)			(0.009)			(0.009)		
Quota Exposure × Post × Patients per Bed		-0.014*			-0.026**			-0.026**	
		(0.007)			(0.011)			(0.011)	
Quota Exposure × Post × Patients per Staff			0.005			0.009			-0.014
			(0.005)			(0.009)			(0.011)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Hospital Measure × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	10715	10715	10048	10715	10715	10048	10715	10715	10048
R-squared	0.794	0.794	0.775	0.868	0.867	0.866	0.567	0.564	0.545

Notes: This table reports DiD estimates by cause-of-death category and checks for treatment heterogeneity in terms of standardized measures of hospital crowding and quality (in 1910). These measures are the number of patients per hospital, the number of patients per bed, and the number of patients per medical staff. The outcomes are log mortality rates of three cause-of-death categories (all causes, infectious causes, and external causes). Quota exposure is defined in equation (1) and interacted with a treatment dummy which equals to one for the years after 1921. The corresponding interaction term with each measure of hospital crowding/quality in 1910 captures treatment heterogeneity. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, controls for the age structure of a city, and each measure of hospital crowding/quality interacted with year fixed effects. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses.

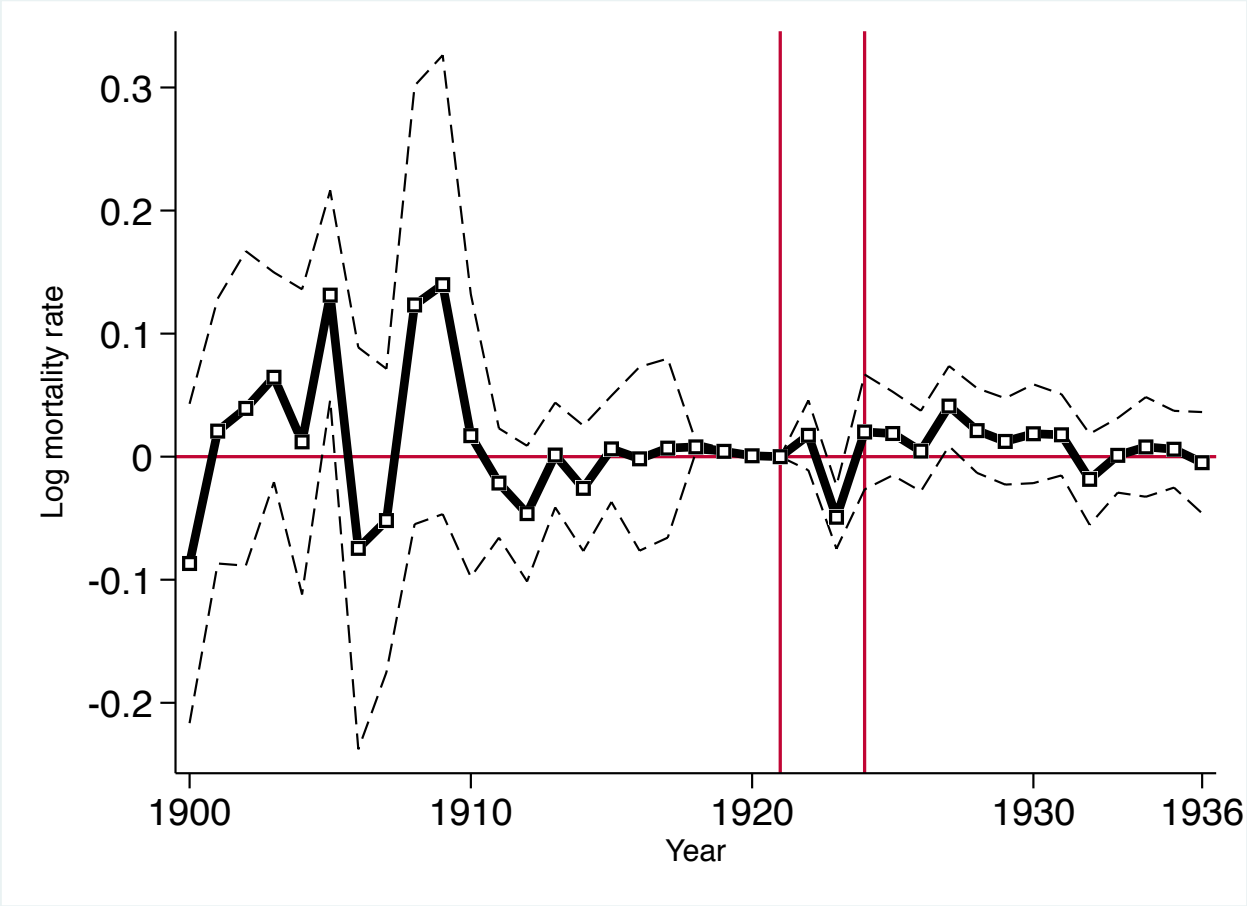
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

potential spillovers from urban areas. This leaves us with 737 rural counties. We then estimate the same event-study specification described in equation (4), but with the rural county mortality rates as the outcome and quota exposure at the county level as the main explanatory variable. Figure 9 reports the event-study estimates. While the pre-1910 estimates vary substantially, possibly due to the highly unbalanced panel in the earlier years, there are no systematic pre-quota differences. Importantly, unlike the city analysis, we do not find greater declines in mortality rates for more quota-exposed areas after the quota acts. The corresponding estimate from the parsimonious DiD specification leads to the same conclusion. This null-finding is not simply driven by rural counties not experiencing reductions in immigration, as we do find that more quota-exposed rural counties experienced larger reductions in the foreign-born share.

7.4 Public Spending and Public Health

The quota-induced reduction in immigration was a large shock that could have had many effects. One well-studied result is political: we know that US-born preferences for redistribution vary with

Figure 9: Event-Study Estimates of Quota Effect on Rural Counties



Notes: This figure shows the event-study estimates of the effect of quota exposure on log mortality rates based on estimation equation (4) for rural counties. We include controls for county age structure, county fixed effects, year fixed effects, log county population in 1910 interacted with year fixed effects, and log county mortality rates for 1918-1921 interacted with year fixed effects. The omitted reference year is 1921.

the presence of immigrants (e.g., Alesina et al. 2018; Tabellini 2020). Could this political economy mechanism explain part of our results? Specifically, cities with larger immigrant communities may have spent less on public health initially; the quota acts might then lead to increased spending on public health, improving population health outcomes.[64] We test this hypothesis using data on different dimensions of health spending per capita from Swanson and Curran (1976), which are available for a subset of cities and years. We use a specification similar in spirit to our baseline model by regressing the measure of public health spending on the interaction between quota exposure and the post-quota indicator, controlling for city age structure, as well as city and time fixed effects. We also include two baseline (1910) controls—the log of city population size and the

[64]This mechanism is not mutually exclusive with the congestion mechanism.

outcome—interacted with year fixed effects. The results, reported in Appendix Table A.11, do not provide strong support for the public spending hypothesis. We have measures of both expenses and capital outlays, separately for health, sanitation, and charity. Only the estimated effect of quota exposure on health outlays per capita is positive and statistically significant at the 10 percent level. The remaining estimates are not significant at any conventional levels, though most of them are positive.[65] While these results do not definitely rule out public spending effects, taking the weak results and the uncertain effects of public health spending in this period on actual public health, we think the crowding and congestion mechanisms are more central to our story.

8 Conclusion

US city dwellers faced a substantial mortality penalty at the start of the 20th century. Were immigrants to blame, as many contemporary nativists claimed? The urban mortality penalty was associated with immigration, in part because foreigners were observed to have higher rates of mortality, and in part because nativists fueled fears that newly arriving foreigners were carriers of germs and diseases (Dublin 1916; Kraut 1994). As the movement to restrict immigration gained ground in the 1890s, the federal government imposed stricter health inspections at the ports of entry to exclude people with "loathsome" or contagious diseases. However, we find that few people were ever denied entry for disease-related reasons, suggesting that most immigrants arrived in relatively good health. In addition, the quota acts, implemented in the 1920s, did not reduce the number of already-ill immigrants allowed to enter the country.

Instead of blaming immigrants, several public health officials regarded poor housing conditions in urban slums as the main contributor to the spread of infectious diseases and the reason for the positive correlation between immigration and mortality (Addams 1911; Krieger and Higgins 2002; Stella 1908). The efforts of social reformers like Jacob Riis increased public awareness of health-related housing concerns, but overcrowding and poor sanitation still characterized US cities when the quota acts were implemented (e.g., Abbott 1936; Park and Kemp 2006; Veiller 1921).

While the quota restrictions were primarily designed to shield US-born workers from competing migrant labor, they also contributed to the urban mortality transition in America. From the

[65] We obtain similar results with the least conservative model, which only controls for city and year fixed effects. We do not use the 1926 data from Swanson and Curran (1976) due to a coding mistake for this year.

mid-1920s until the late 1930s, more quota-exposed cities experienced relatively larger declines in mortality rates due to infectious diseases and external causes. The quota effects we estimate are not simply a mechanical byproduct arising from a combination of excess mortality for immigrants and a reduction in their population. Instead, we propose that there were also spillover effects from immigrants to the US-born that were mediated through congestion constraints in housing and healthcare. In particular, more quota-exposed cities that were initially more crowded in terms of housing or hospital facilities experienced greater declines in mortality due to infectious diseases.

In summary, the majority of immigrants arrived in good health at the beginning of the 20th century. It was their unfavorable living conditions that contributed to the urban mortality penalty at that time. In public health terms, the quota policy curtailed the number of immigrants that would have otherwise crowded into the poorest and most unsanitary districts of cities, thus accelerating the urban mortality decline in the 1920s. While the reduced inflow of immigrants relaxed some of the housing constraints, the historical narrative suggests that the quality of housing changed little during our period of study (e.g., Britten et al. 1940; Jacobs and Stevenson 1981; Krieger and Higgins 2002).

We estimate that the quota system prevented around 800,000 people annually from immigrating to the US between 1924 and 1937. Our main results imply that quotas averted up to 19,500 deaths each year (11,000 deaths from infectious causes). But this mortality reduction came at high costs, as immigration during this era had large economic benefits to local economies in the short-run (Tabellini 2020), in the longer-run (Ager and Brueckner 2013; Burchardi et al. 2016; Sequeira et al. 2017; Moser and San 2020), and to the immigrants themselves (Abramitzky et al. 2012, 2014). Could a comparable mortality reduction have been possible by other means? Without exogenous variation in other plausible policies, we cannot construct an exact comparison, but our results comparing the quota effects in cities with more or less crowded and congested living conditions suggests yes. The difference in deaths averted between cities one standard deviation less crowded—measured by internal density among immigrants—or more crowded was 24,500 deaths annually (17,700 infectious deaths), more than the total effect. Other policies—greater supply of healthcare, better public health infrastructure, better public sanitation or clean water or sewerage—could also have broken the link between immigration and urban mortality.

In all eras, low income populations are likely to crowd into dense urban settings, and sufficient

public health capacity and well-designed housing policy is needed to reduce the potential health risks. Today, substandard housing and weak public health infrastructure are major public health issues in the US and the United Nations regards improvements in housing conditions as key to limiting the spread of infectious diseases.

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A Online Appendix

A.1 Construction of the WWI Immigration Shock and the Alternative Measure of Quota Exposure

This section describes the construction of two variables: the immigration shock induced by WWI and an alternative measure of quota exposure. We define the former in a similar way to the original quota exposure variable:

$$WWI_c = \frac{100}{P_{c,1910}} \sum_{n=1}^{N} \left(\widehat{M}_{n,15-19} - WWI_{n,15-19} \right) \frac{FB_{nc,1910}}{FB_{n,1910}}, \tag{7}$$

where $\widehat{M}_{n,15-19}$ is the *predicted* average annual number of immigrants of nationality n who would have arrived in 1915-1919 had WWI not occurred.[66] We make these predictions using the same method outlined in Section 3.2. $WWI_{n,15-19}$, on the other hand, is the average number of immigrants of nationality n that actually arrived in the US annually over this period. Similar to the construction of the number of missing immigrants due to the quota system, $\widehat{M}_{n,15-19} - WWI_{n,15-19}$ denotes the number of missing immigrants as a result of WWI. These missing immigrants are then distributed according to the nationality-specific settlement patterns in 1910, $FB_{nc,1910}/FB_{n,1910}$. The total number of missing immigrants in each city is then scaled by the city population in 1910, $100/P_{c,1910}$. For the empirical analysis, we interact this cross-sectional variable with an indicator for the period after 1914.

Our alternative measure of quota exposures follows Greenwood and Ward (2015):

$$\textit{Quota exposure}_c^A = \frac{100}{P_{c,1910}} \sum_{n=1}^{N} \left(\frac{\widehat{M}_{n,22-30} - \overline{Q}_{n,22-30}}{\widehat{M}_{n,22-30}} \right) FB_{nc,1910}, \tag{8}$$

where the expression in parentheses measures the bite of the quota system for nationality n, which is then multiplied by the foreign-born share in city c in 1910, $\frac{FB_{nc,1910}}{P_{c,1910}}$. Cities in 1910 that primarily received immigrants from countries that were subsequently tightly restricted will have a "bite" that is close to 1 for most nationalities. Quota exposure would then amount to the share of foreign-born people in that city. For cities in 1910 that mainly received groups that were not restricted in the

[66] Since the immigration year ends on June, 30, the year 1915 refers to immigration from July 1, 1914 to June 30, 1915. See also footnote 27 in the main text.

1920s, quota exposure would be close to 0.

A.2 Dangerous Jobs Do Not Explain the Quota Effects on External Causes of Death

As we have shown in this paper, the quota acts of the 1920s led to a fall in mortality rates associated with external causes. One possible mechanism is a change in population composition. If immigrants were more likely to work in—and die as a result of—dangerous jobs, then the decline in the number of immigrants after the quota restrictions would mechanically pull down the rate of mortality due to external causes as well. This assumes that there were no general equilibrium effects, such as the US-born taking the jobs that were vacated or those jobs becoming safer after the quotas were imposed.

To assess this hypothesis, we construct two novel measures of the presence of dangerous jobs in each city. Our first measure is based on the 1920 *Proceedings of the Casualty Actuarial and Statistical Society of America*, which classifies industries for the purposes of group disability insurance using ratings from A to F, in order of compensation. Industries with a rating of A are the least dangerous activities. We take industries with E and F classifications to be dangerous jobs and map them to the IPUMS industry codes where possible. The share of men working in these dangerous jobs can then be computed for each city.

Our second measure is based on the 1922 Massachusetts *Annual Report of the Department of Industrial Accidents*, which records the number of deaths by industry in Massachusetts between July 1, 1920 and June 30, 1921. Again, we map these industries to the IPUMS industry codes where possible, and then use the 1920 population in Massachusetts to scale the death counts in order to determine which industries have the highest mortality rates. The top 25 industries in terms of mortality rates are taken to be dangerous jobs. We can then compute the share of men in these jobs for each city.

With our measures of dangerous jobs, we test for heterogeneity and direct effects in two ways, mirroring the analysis in Section 6. Using a DiD specification similar to the regression underlying Table 8, we find no effect of quota exposure on the share of dangerous jobs in a city. Turning instead to a triple difference specification similar to the model behind Table 7, we ask if there were heterogeneous treatment effects for cities with more or less men in dangerous jobs. We do not find

any differences on the basis of the initial prevalence of dangerous jobs.

Taken together, these results suggest that the decline in death due to external causes is not driven by a reduction in the share of people working in dangerous jobs or differences across cities in industrial accident risk.

A.3 Additional tables and figures

This section reports the appendix tables and figures.

Table A.1: Cause of Death Classifications

Infectious Causes	External Causes	Non-Infectious Causes
(1)	(2)	(3)
Pneumonia	Accidents (all types)	Cancer/tumors
TB (all types)	Homicide	Diabetes
Influenza	Suicide	Nephritis
Diarrhea		Cirrhosis of the liver
Measles		Rheumatism
Whooping cough		Diseases of the circulatory system
Typhoid fever		Diseases of the heart
Smallpox		
Menigitis		
Scarlet fever		
Diphtheria and croup		

Notes: This table shows the diseases included in our main cause-of-death categories.

Table A.2: Quota Effects Are Robust to Controlling for WWI Effects

	Dependent variable is mortality rates (in logs) of:							
	All Causes		Infectious Causes		External Causes		Non-Infectious Causes	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Quota Exposure								
× Post	-0.018***	-0.018***	-0.041***	-0.035***	-0.048***	-0.042***	0.001	0.003
	(0.006)	(0.006)	(0.013)	(0.009)	(0.013)	(0.012)	(0.005)	(0.003)
WWI Exposure								
× Post	-0.006	-0.007	-0.018	-0.016	-0.016	-0.017	-0.012	0.003
	(0.007)	(0.008)	(0.015)	(0.013)	(0.012)	(0.015)	(0.007)	(0.005)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910								
× Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates								
× Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-by-Year FEs	No	Yes	No	Yes	No	Yes	No	Yes
Observations	12587	12333	12587	12333	12587	12333	12587	12333
R-squared	0.794	0.829	0.859	0.885	0.588	0.638	0.874	0.898

Notes: This table reports the DiD estimates, controlling for the potential effects of WWI on immiration. The outcomes are log mortality rates for our four cause-of-death categories (all causes, infectious causes, external causes, and non-infectious causes). Appendix Table A.1 lists the individual causes included in each category. Quota exposure is defined in equation (1) and interacted with an indicator for years after 1921. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, controls for the city's age structure, and a control for the immigration shock from WWI (WWI exposure). The even-numbered columns also control for state-by-year fixed effects. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.3: DiD Estimates of Quota Effect for Selected Single Causes of Death, with State-by-Year Fixed Effects

	Dependent variable is mortality rates (in logs) of:										
	Pneumonia	Lung TB	Influenza	Typhoid	Diarrhea	Measles	Diptheria & Croup	Whooping Cough	Early Infancy	Accidents & Homicides	Suicide
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
Quota Exposure × Post	-0.037***	0.000	0.033*	-0.008	-0.078**	-0.111*	-0.045	-0.024	-0.059***	-0.054***	-0.004
	(0.010)	(0.010)	(0.020)	(0.028)	(0.031)	(0.061)	(0.028)	(0.020)	(0.020)	(0.017)	(0.012)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-by-Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	12333	12074	9315	4479	10535	1598	5557	3912	12178	12333	8331
R-squared	0.712	0.852	0.784	0.887	0.859	0.740	0.743	0.645	0.688	0.636	0.513

Notes: This table reports DiD estimates for specific causes of deaths (see top row). Quota exposure is defined in equation (1) and interacted with an indicator for years after 1921. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, controls for the city's age structure, and state-by-year fixed effects. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.4: Quota Effects Are Robust to An Alternative Measure of Quota Exposure

	Dependent variable is mortality rates (in logs) of:		
	All Causes	Infectious Causes	External Causes
	(1)	(2)	(3)
Quota Exposure × Post	-0.009***	-0.018***	-0.019***
	(0.001)	(0.002)	(0.003)
Age Structure	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes
Observations	12587	12587	12587
R-squared	0.798	0.863	0.597

Notes: This table reports the results from the same specifications as in Table 2 (odd-numbered columns), but using an alternative measure of quota exposure (see Appendix A.1 for its construction). Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.5: Quota Effects Are Robust to Separating Population and Total Deaths

	Dependent variable is logged			
	Population	All Causes	Infectious Causes	External Causes
	(1)	(2)	(3)	(4)
Quota Exposure × Post	-0.011**	-0.019***	-0.059***	-0.062***
	(0.005)	(0.006)	(0.016)	(0.017)
Age Structure	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes
Initial Mortality Levels × Year FE	No	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes
Observations	12587	12587	12587	12587
R-squared	0.985	0.981	0.962	0.943

Notes: This table reports the results from the same specifications as in Table 2 (odd-numbered columns), but instead of mortality rates, the outcomes are log population size (column 1) and log total deaths for the different cause-of-death categories (columns 2-4). Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.6: Quota Effects by Sending-Country Exposure

	Dependent variable is mortality rates (in logs) of:		
	All Causes	Infectious Causes	External Causes
	(1)	(2)	(3)
Quota Exposure Austria × Post	-0.242***	-0.396***	-0.375**
	(0.079)	(0.110)	(0.146)
Quota Exposure Greece × Post	-0.009	-0.068	-0.036
	(0.028)	(0.045)	(0.052)
Quota Exposure Hungary × Post	0.018	-0.029	0.041
	(0.039)	(0.029)	(0.074)
Quota Exposure Italy × Post	-0.047**	-0.117***	-0.128***
	(0.023)	(0.034)	(0.035)
Quota Exposure Poland × Post	-0.004	-0.008	-0.008
	(0.005)	(0.008)	(0.007)
Quota Exposure Russia × Post	-0.052	-0.104*	-0.176***
	(0.032)	(0.054)	(0.063)
Quota Exposure Turkey × Post	-0.026	-0.114**	0.041
	(0.034)	(0.048)	(0.056)
Quota Exposure Yugoslavia × Post	0.009	0.022*	-0.045***
	(0.008)	(0.013)	(0.015)
Quota Exposure All Other Countries × Post	-0.040**	-0.092***	-0.072
	(0.019)	(0.028)	(0.047)
Age Structure	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes
Observations	12521	12521	12521
R-squared	0.799	0.864	0.601

Notes: This table reports the results from the same specifications as in Table 2 (odd-numbered columns), but with measures of quota exposure that are based on the nationalities with the most quota exposure. For "all other countries", we sum the quota exposures across the remaining nationalities. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.7: Quota Effects Are Robust to Clean Water and Sanitation Controls

	Dependent variable is mortality rates (in logs) of:		
	All Causes	Infectious Causes	External Causes
	(1)	(2)	(3)
Quota Exposure × Post	-0.043***	-0.104***	-0.062***
	(0.011)	(0.024)	(0.021)
Filtration	0.046*	0.075*	0.069*
	(0.024)	(0.042)	(0.038)
Chlorination	0.004	0.030	0.049
	(0.015)	(0.029)	(0.039)
Clean Water	0.002	-0.020	-0.010
	(0.032)	(0.053)	(0.037)
Sewage	0.001	-0.013	-0.001
	(0.027)	(0.039)	(0.053)
Filtration × Sewage	-0.009	0.053	0.049
	(0.044)	(0.082)	(0.078)
Age Structure	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes
Observations	949	949	949
R-squared	0.957	0.961	0.836

Notes: This table reports the results from the same specifications as in Table 2 (odd-numbered columns), but for a sample of 25 large US cities for which we are able to control for the roll-out of filtration, chlorination, clean water projects, and sewage, as in Anderson et al. (2019a). Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.8: Treatment Heterogeneity by Initial City Population Size

	Dependent variable is mortality rates (in logs) of:		
	All Causes	Infectious Causes	External Causes
	(1)	(2)	(3)
Quota Exposure × Post	-0.021***	-0.051***	-0.056***
	(0.008)	(0.016)	(0.017)
Quota Exposure × Post × City Population in 1910	-0.002**	-0.005***	0.001
	(0.001)	(0.001)	(0.001)
Age Structure	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes
Observations	12587	12587	12587
R-squared	0.794	0.859	0.588

Notes: This table reports the results from the same specifications as in Table 2 (odd-numbered columns), but we also include the interaction between quota exposure and (standardized) population size in 1910 to test for heterogeneous effects by city size. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.9: DiD Estimates of Quota Effect by Cause-of-Death Category, Weighted by 1910 City Population

	Dependent variable is mortality rates (in logs) of:							
	All Causes		Infectious Causes		External Causes		Non-Infectious Causes	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Quota Exposure × Post	-0.030***	-0.025**	-0.073***	-0.043***	-0.044***	-0.038**	-0.006	0.007**
	(0.010)	(0.011)	(0.022)	(0.016)	(0.014)	(0.016)	(0.005)	(0.003)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-by-Year FEs	No	Yes	No	Yes	No	Yes	No	Yes
Observations	12587	12333	12587	12333	12587	12333	12587	12333
R-squared	0.934	0.951	0.941	0.961	0.777	0.827	0.947	0.963

Notes: This table reports the baseline DiD estimates weighted by city population sizes in 1910. The outcomes are log mortality rates for our four cause-of-death categories (all causes, infectious causes, external causes, and non-infectious causes). Appendix Table A.1 lists the individual causes included in each category. Quota exposure is defined in equation (1) and interacted with an indicator for years after 1921. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and controls for the city's age structure. The even-numbered columns also control for state-by-year fixed effects. The sample period is 1900-1937. Standard errors clustered at the city level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A.10: DiD Estimates of Quota Effect by Cause-of-Death Category, Aggregated to County-Level

	Dependent variable is mortality rates (in logs) of:							
	All Causes		Infectious Causes		External Causes		Non-Infectious Causes	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Quota Exposure × Post	-0.014*	-0.015	-0.041**	-0.037**	-0.040**	-0.038*	-0.003	0.003
	(0.008)	(0.011)	(0.019)	(0.019)	(0.020)	(0.022)	(0.006)	(0.004)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
County and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-by-Year FEs	No	Yes	No	Yes	No	Yes	No	Yes
Observations	9111	8857	9111	8857	9111	8857	9111	8857
R-squared	0.815	0.862	0.873	0.898	0.539	0.617	0.903	0.926

Notes: This table reports the DiD estimates with the data aggregated to the county level to account for city border changes during our period. The outcomes are log mortality rates for our four cause-of-death categories (all causes, infectious causes, external causes, and non-infectious causes). Appendix Table A.1 lists the individual causes included in each category. Quota exposure is defined in equation (1) and interacted with an indicator for years after 1921. All regressions include county and year fixed effects, log populations size in 1910 interacted with year fixed effects, initial outcomes in 1918-1921 interacted with year fixed effects, and controls for the county's age structure. The even-numbered columns also control for state-by-year fixed effects. The sample period is 1900-1937. Standard errors clustered at the county level are in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

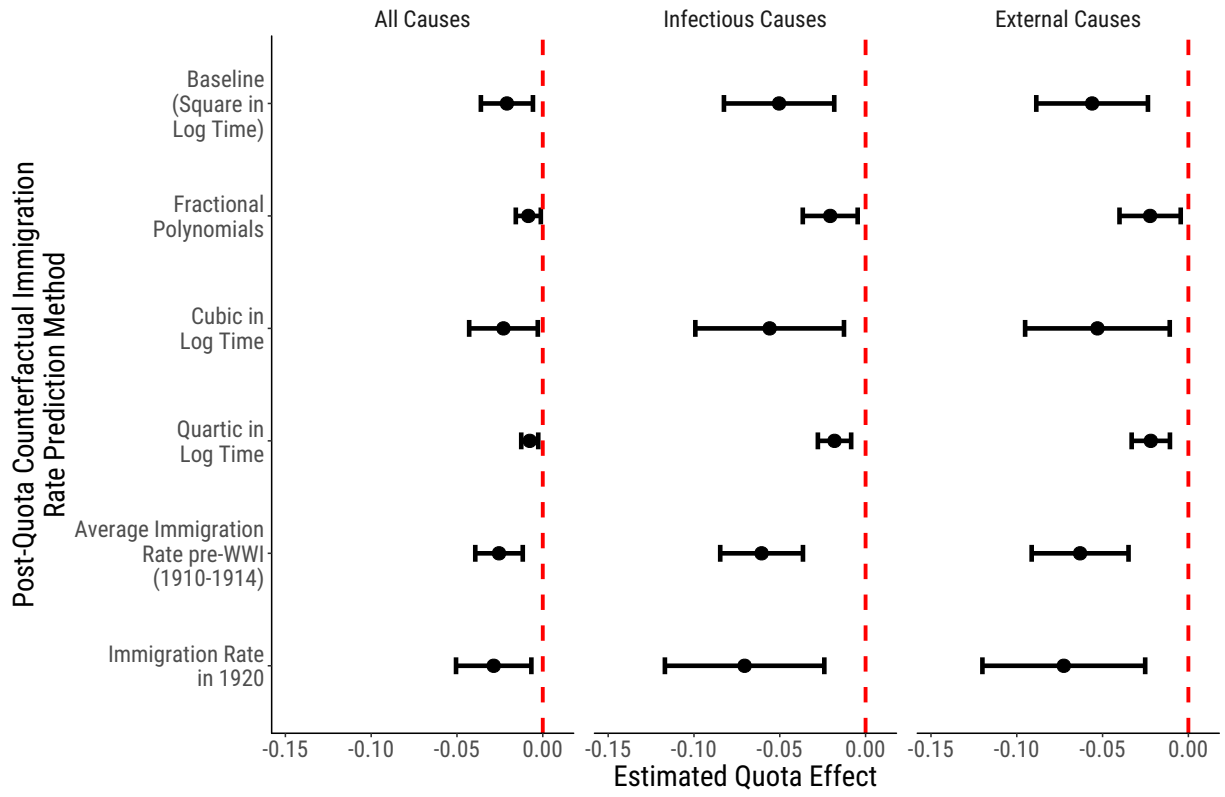
Table A.11: The Impact of Quota Restrictions on Public Spending

	Health expenses	Health outlays	Sanitation expenses	Sanitation outlays	Charity expenses	Charity outlays	All spending
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Quota Exposure × Post	0.050	0.068*	0.070	0.005	-0.024	-0.007	0.163
	(0.039)	(0.040)	(0.068)	(0.009)	(0.041)	(0.012)	(0.168)
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Outcome in 1910 × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2738	2738	2738	2738	2738	2738	2738
R-squared	0.788	0.785	0.801	0.389	0.364	0.283	0.731

Notes: This table reports DiD estimates for the various types of spending, as indicated in each column heading. Quota exposure is defined in equation (1) and is interacted with a treatment dummy which equals to one for the years after 1921. All regressions include city and year fixed effects, log populations size in 1910 interacted with year fixed effects, the initial outcome in 1910 interacted with year fixed effects, and controls for the city's age structure. Standard errors clustered at the city level are in parentheses.

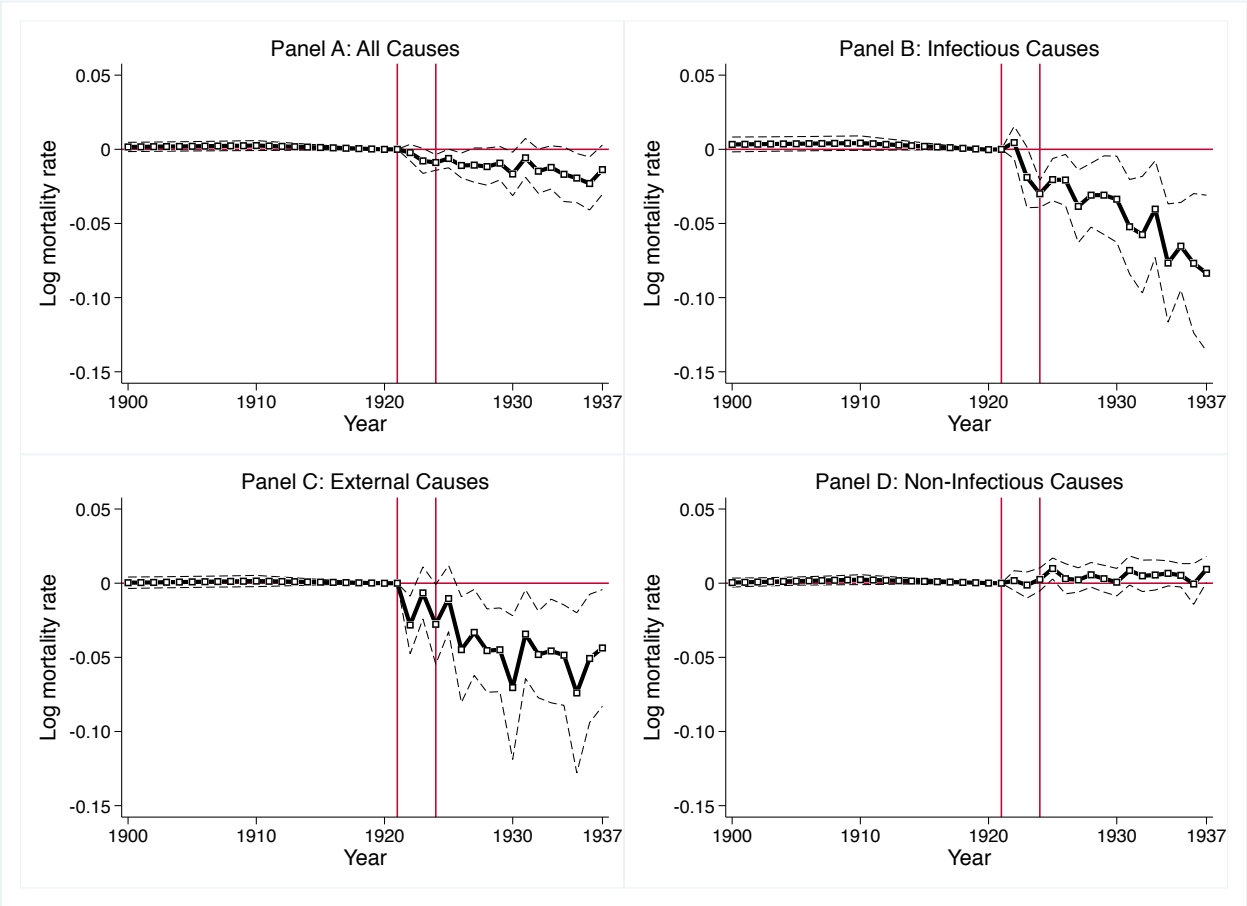
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Figure A.1: Quota Effects are Robust to Alternative Methods of Calculating the Counterfactual Immigration Rate Post-Quotas



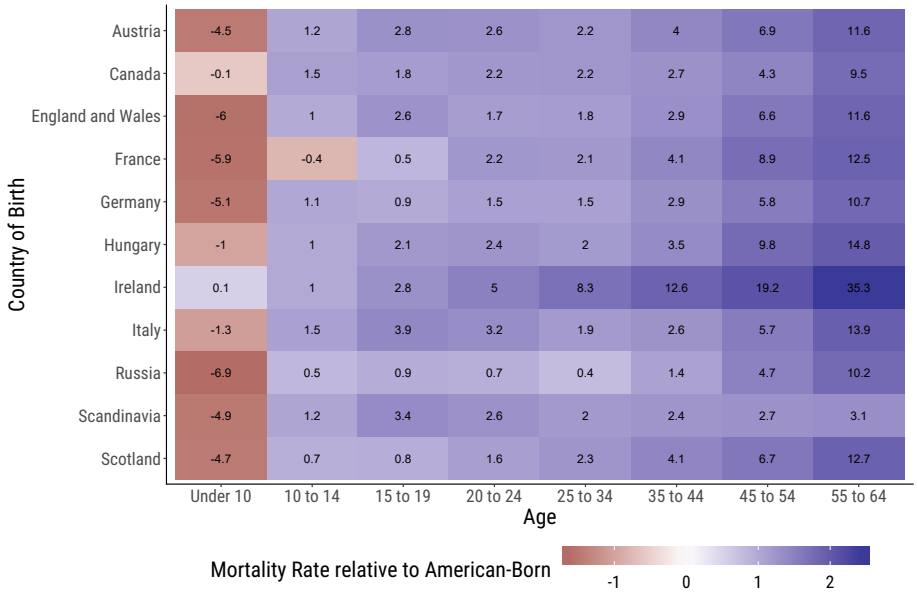
Notes: This figure plots our main DiD results with the same specification as in Table 2, but using the quota treatments based on alternative methods of predicting immigration rates after the quotas as the treatment of interest. Specifically, we use fractional polynomials, a cubic in the log of years, a quartic in the log of years, the average immigration rate before WWI (1910-1914), and the 1920 immigration rate. In all cases, our main conclusions are robust: more quota-affected cities see larger reductions in mortality rates overall, driven by declines in infectious and external causes.

Figure A.2: Event Study Estimates of Quota Effect by Cause-of-Death Category, Controlling for Initial Outcomes



Notes: This figure show the event-study estimates similar to Figure 6, but additionally controlling for the mortality outcomes from 1910-1921, which takes out all pre-quota mortality variation.

Figure A.3: Mortality Age Distribution by Country of Origin in 1910



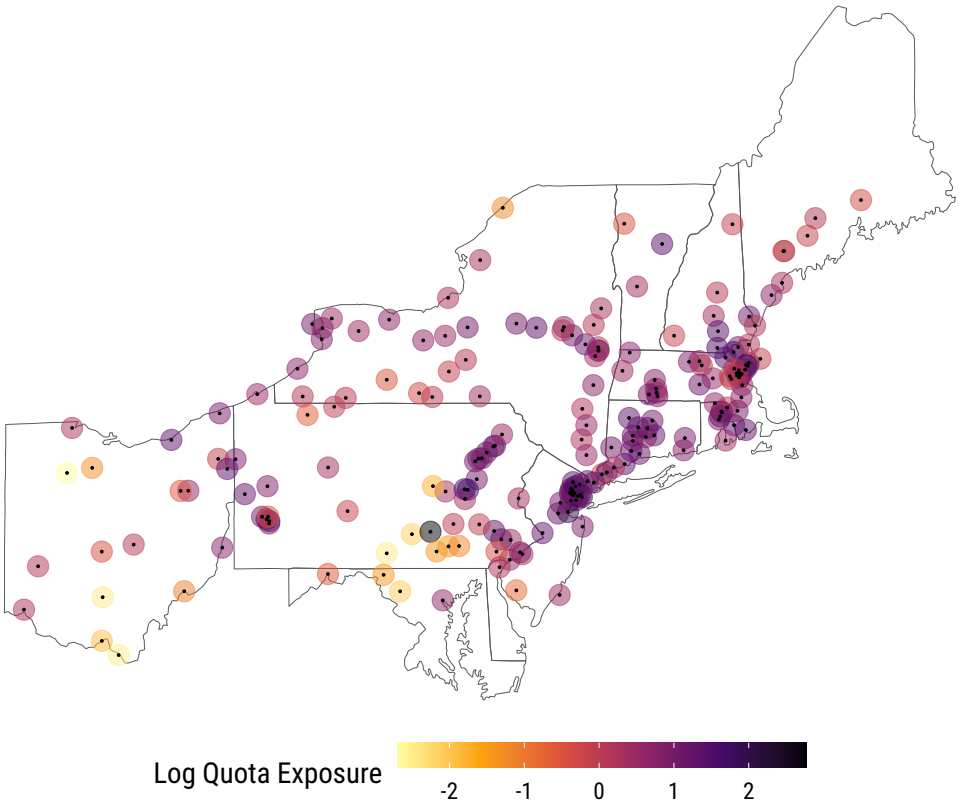
(a) Men, All Causes, Foreign-Born Death Rate Less US-Born Death Rate



(b) Women, All Causes, Foreign-Born Death Rate Less US-Born Death Rate

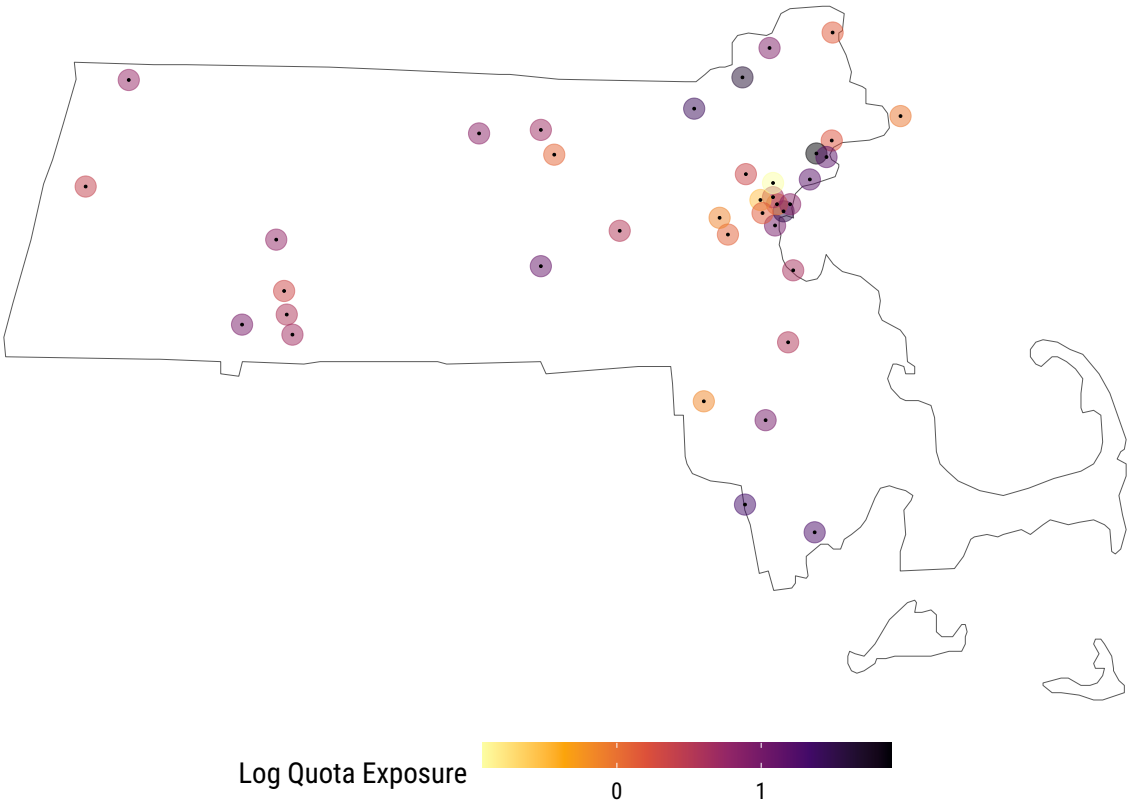
Notes: This figure shows age-specific foreign-born mortality penalties for all causes of death among white men (top panel) and white women (bottom panel). We plot the difference in deaths per 1,000, subtracting the mortality rate for immigrants from each country of birth from the corresponding US-born rates. We see that for all adult ages other than 55-to-64-year-old women from Scandinavia, there is a foreign-born mortality penalty which is often quite large.

Figure A.4: Map of Quota Exposure for Cities in Northeastern States



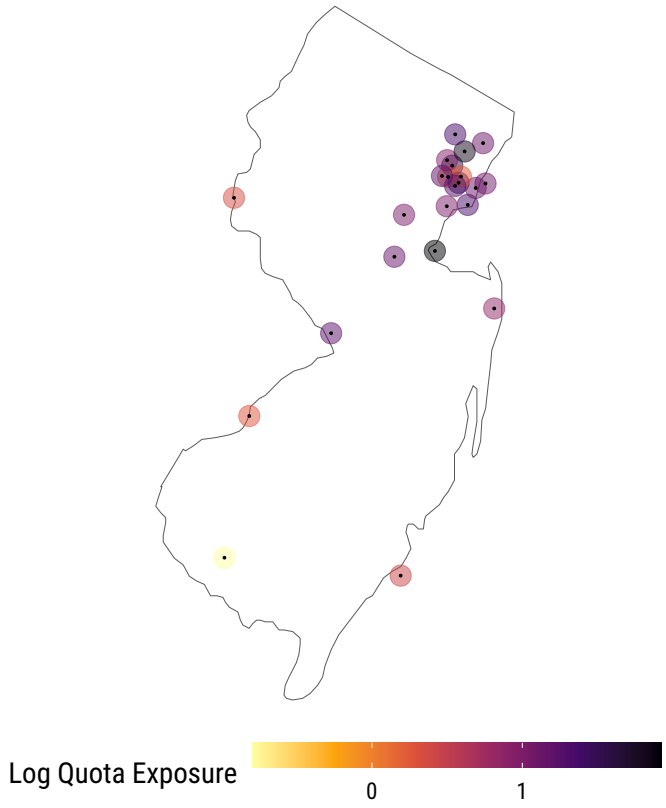
Notes: This map zooms in on quota exposure for cities located in the Northeastern states.

Figure A.5: Map of Quota Exposure for Cities in Massachusetts



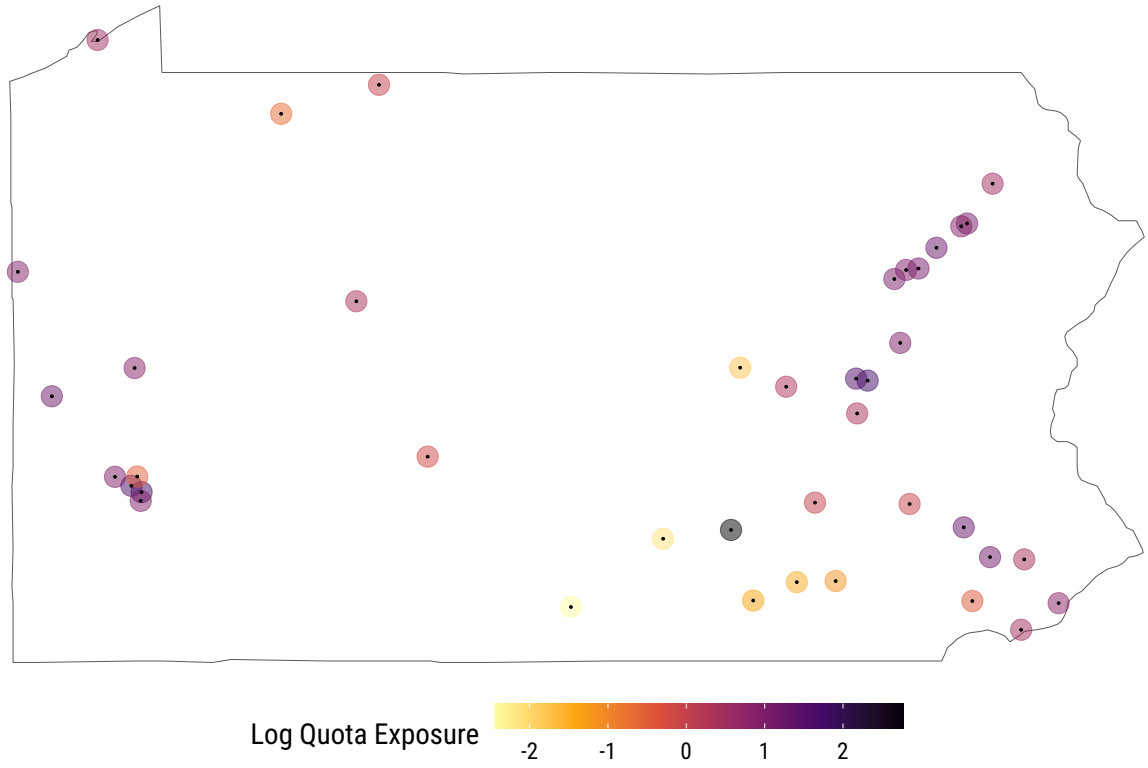
Notes: This map zooms in on quota exposure for cities located in Massachusetts.

Figure A.6: Map of Quota Exposure for Cities in New Jersey



Notes: This map zooms in on quota exposure for cities located in New Jersey.

Figure A.7: Map of Quota Exposure for Cities in Pennsylvania



Notes: This map zooms in on quota exposure for cities located in Pennsylvania.