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

Philipp Ager James J. Feigenbaum Casper Worm Hansen Hui Ren Tan†

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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 immigrants made up one-third of the population in crowded American cities, contemporaries blamed high urban mortality rates on the newest arrivals. We evaluate how the implementation of country-specific immigration quotas in the 1920s affected urban health. Cities with larger quota-induced reductions in immigration experienced a persistent decline in mortality rates, driven by a reduction in deaths from infectious diseases. The unfavorable living conditions immigrants endured explains the majority of the effect as quotas reduced residential crowding and mortality declines were largest in cities where immigrants resided in more crowded conditions and where public health resources were stretched thinnest.

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†Ager: University of Mannheim and CEPR, philipp.ager@uni-mannheim.de; Feigenbaum: Boston University and NBER, jamesf@bu.edu; Hansen: University of Copenhagen and CEPR, casper.worm.hansen@econ.ku.dk; Tan: National University of Singapore, huiren@nus.edu.sg.

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 2019; Markel and Stern 2002). Today, many Americans hold mistaken and sharply negative views about immigrants (Alesina et al. 2022), misperceptions that may be exploited by those accusing immigrants of bringing infectious diseases across borders. Blaming immigrants for surges of COVID-19 cases is one recent example.[2] In this paper, we show that the nativists' narrative of immigrants as germ and disease carriers is unwarranted. These fears are self-fulfilling: immigrants do not cause more disease directly, but instead, immigration—and the nativists' response—affects local public policies and living conditions. These policies, particularly regarding overcrowded and substandard housing, are often inefficient or completely absent and put public health at risk.

Our evidence on the link between immigration, mortality, and local public policies comes from the early 20th century during the Age of Mass Migration in the United States. We show that when the US curtailed immigration in the 1920s, deaths from infectious diseases declined. 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 poor living conditions.

Living conditions for Americans at the turn of the 20th century were poor and mortality was high, especially in cities.[3] This was particularly true for immigrants, who often clustered in the cities' 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 (Willcox 1906; Haines 2001). The positive correlation between immigration and

[1] Medicalized nativism predates both modern medicine and the nation-state. 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 germs carried by newcomers: the deaths of the majority of the indigenous populations of the Americas during European colonization (Diamond 1998; Cook 1998).

[2] See the headlines of recent articles in Online Appendix A.5.

[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).

mortality was cited by many contemporaries as evidence that immigrants caused the spread of infectious diseases in cities (Higgs 1979; Kraut 1994; Markel and Stern 2002). Against this backdrop, a number of urban reformers and public health scholars argued that the detrimental consequences of poor housing conditions on health might drive this relationship (Krieger and Higgins 2002; Veiller 1913; Stella 1908). However, rigorous quantitative evidence is lacking until now.

We argue that poorly designed (or altogether absent) local public policies in the early 20th century were driving the relationship between immigration and infectious diseases in cities. A broken urban governance system—the shame of the cities (Steffens 1904)—allowed and even encouraged the housing of immigrants in crowded and squalid urban slums.[4] But a city is only as healthy as its most vulnerable populations as infectious diseases do not respect neighborhood boundaries. Some contemporary sources noted that "immigrants as a class are frequently blamed for bad housing and living conditions over which they have no control," while underlining the root of the issue: "These conditions may be due in part to the attitude of Americans toward immigrants, and to the economic status of the immigrant working class" (National Industrial Conference Board 1923).

Large-scale immigration and the clustering of newcomers in poverty-stricken neighborhoods also stoked xenophobia during this period.[5] 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 2019). 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 thousand were admitted between 1925-1929. The quotas also went beyond simply constraining the total number of immigrants; the system was designed to disproportionately limit immigration from Southern and Eastern Europe.

Our estimation strategy exploits this fundamental shift in US immigration policy in the 1920s to obtain

[4]In New York, Steffens (1904) chided Tammany Hall leaders for "caus[ing] the troubles they relieve... let[ting] the Health Department neglect the tenements..." In Chicago, immigrant neighborhoods were intentionally zoned for noxious industrial use (Shertzer et al. 2016). Molina (2006) documents a half-century of city and county public health officials stigmatizing immigrants in Los Angeles as public health menaces, identifying their neighborhoods as "rotten spots."

[5]For example, Eastern European Jewish immigrants were regarded as "very tuberculous" (Simons 1908), and Chinese residents in San Francisco were irrationally quarantined during a bubonic plague outbreak (Kraut 1994, 2019). Such attitudes have not faded. When debating an immigration bill in congress in the 1950s, one senator used "deep-seated metaphors of disease and contagion," linking immigration to a stream and suggesting that "If that stream is healthy, the impact on our society is salutary; but if that stream is polluted our institutions and our way of life becomes infected" (Markel and Stern 2002). In the 1980s, with no basis in data, Haitian migrants were labeled as a high-risk group for HIV infection (Kraut 1994, 2019)

the causal effect of immigration restrictions on 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 construct a measure of "missing immigrants" to capture how intensely each city was affected by the quota policy.[6] For each nationality, we first compute the total number of missing immigrants by comparing the expected number of arrivals based on historical trends to the corresponding quota limit. We then allocate the missing immigrants to the cities they likely would have moved to based on pre-quota settlement patterns. The resulting city-level measure of the immigration policy, which we refer to as quota exposure, can be interpreted as the local rate of missing immigrants.

We use a difference-in-differences approach to evaluate how quota exposure affected urban mortality. Our analysis draws 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 in the pre-quota years. However, with the enactment of the immigration restrictions, all-cause mortality rates started to fall more rapidly in cities that were more affected by the quota system. This decline was mainly driven by changes in deaths due to infectious diseases, the primary causes of death at the time. 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 and the infectious-cause mortality rate by more than 4 percent. We find no effect on mortality arising from non-infectious causes, such as cancer and cardiovascular diseases.

What are the potential mechanisms through which the immigration quotas could have affected urban mortality? We first consider if changes in the population composition can fully explain the mortality decline. Compared to their US-born counterparts, immigrants had higher mortality rates across most causes and age groups in the early 20th century (Dublin 1916). This excess mortality of immigrants, coupled with a reduction in their shares under the quota restrictions, mechanically generates a decline in mortality rates. Such declines could occur even if the changes to immigration had no direct effect on the mortality of the US-born. Ultimately, we find that changes in the population composition explain only about a quarter of the total mortality effect in the median city and less than half of the total effect in nearly two-thirds of our sample of cities.

To reinforce the case against a composition-only story, we provide two pieces of evidence of spillover

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

effects from immigrants to the US-born. First, we show that the quotas reduced mortality among US-born whites, with large effects both for those with US-born parents and with foreign-born parents.[7] Second, we find large mortality declines among African-Americans in quota-exposed cities. As the vast majority of African-Americans in this era were US-born, this is strong support for the existence of spillover effects.

We then show that the main mechanism driving the link between quota restrictions and urban mortality relates to overcrowding.[8] Four pieces of evidence illustrate that this was the case. First, the quota policy reduced diseases associated with crowding, such as influenza, pneumonia, and measles. Second, we find that pre-quota measures of housing and public health crowding amplify the impact of missing immigrants on mortality rates. Third, we find that the quota policy reduced housing congestion directly. Fourth, we turn from cities to rural counties, which are by definition less crowded. We find that quota-exposure had no effect on mortality rates in rural counties. To conclude, we perform a back-of-the-envelope accounting exercise which suggests that 27 to 68 percent of the overall quota effect on mortality can be attributed to crowding.

A fertility channel is also linked to the crowding mechanism. The quotas led to a reduction in crude birth rates, a change that could be directly driven by improvements in the mortality environment due to less residential crowding (Galor 2011; Guinnane 2011). In an era of high infant and child mortality rates—driven in part by congested housing—fertility declines reduce overall mortality rates (Preston and Haines 1991). However, we show that the fertility decline accounts for at most one-tenth of our total mortality effect of the quotas.

We also evaluate and reject three alternative mechanisms. First, we consider contagion as a more direct explanation for the quota-mortality link. Most immigrants endured a harrowing passage across the Atlantic in crowded steerage conditions (Steiner 1906; Keeling 2013). After spending weeks at sea, newly arrived immigrants could be potent vectors, spreading infectious diseases of the crowd from the ship to the city. The quota system reduced the number of people disembarking fresh from such voyages. To assess this hypothesis, we digitize 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 quotas were introduced. Furthermore, our

[7]Data limitations force us to conduct this analysis at the state-level rather than at the city-level. For further details, see Section 6.1.3.

[8]Scholars have long argued that overcrowding and poor housing conditions in immigrant neighborhoods contributed to the mortality penalty in American cities (Higgs 1979; Kraut 1994). New arrivals clustered in areas where housing was cheap and close to their workplaces (Ward 1971); such crowded and unsanitary districts were focal points for the spread of infectious diseases (Costa 2015).

estimated effects of quotas on mortality remain qualitatively unchanged when we exclude port cities and their neighbors from our analysis, whereas a contagion narrative would have predicted stronger effects closer to ports. Second, we argue that effects via the labor market are unlikely to play a major role in our findings. The effect of immigration in our era on wages is unclear with estimates ranging from null (Abramitzky et al. 2019) to negative overall (Goldin 1994; Xie 2017) to positive for the US-born (Tabellini 2020). Though economic downturns were likely bad for health, the uncertainty of the effect of the quotas on the labor market as well as the persistence of our mortality effects make it unlikely that the labor market mechanism is driving the observed mortality decline. Third, we show that the quota shock had little effect on public health spending. Consequently, our results are unlikely to be caused by preferences for redistribution or spending in heterogeneous populations (Alesina et al. 1999).

This is a paper studying both immigration and crowding as the two are often inseparable, historically and today. During the Age of Mass Migration, immigrants were significantly more likely to live in overcrowded conditions—in multifamily housing, as boarders or lodgers, and in densely populated neighborhoods. Informal barriers to better housing like racial covenants and discrimination increased crowding conditions in ethnic enclaves (Wang 2001). Across the globe today, many immigrants also live in crowded quarters, conditions that risk the spread of infectious diseases. Immigrants are often poor and forced to trade off dangerous living conditions for jobs and opportunities (Marx et al. 2013), and ethnic minorities are especially likely to be discriminated against in housing arrangements (Auspurg et al. 2019; Boeri et al. 2015). Thus, we use exogenous variation in urban crowding induced by changes in immigration laws to understand the relationship between congestion and mortality, but at the same time we also learn about the health conditions of immigrants and their US-born city co-residents. In the vicious cycle linking wrong beliefs about the underlying health or "fitness" of marginalized populations (Markel and Stern 2002) and weak or lacking public policies, our results suggest that better policy—in our setting on public housing or overcrowding—could both mitigate the spread of infectious diseases in densely populated neighborhoods *and* break the connection in the nativist imagination between immigrants and poor health.

Our study relates 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).[9] A

[9]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

sizable literature attributes this decline to improvements in public health infrastructure (e.g., Cutler and Miller 2005; Alsan and Goldin 2019; Bauernschuster et al. 2020; Egedesø et al. 2020), while two recent studies by Anderson et al. (2018, 2019) have contested some of these conclusions, highlighting that better nutrition and improved living conditions could have played important roles. Our work suggests 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 (Duffy 1968; Higgs 1979; Meckel 1985), our analysis 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 literature on the broader implications of immigration during the Age of Mass Migration. Our focus on the intersection of immigration and health is novel in this literature but among the primary concerns of contemporaries, alongside well-studied questions about economic competition and cultural assimilation.[10] 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). One advantage we have compared to existing work 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 to assess the parallel trends assumption more carefully as we observe many years before treatment.

We draw on a variety of data sources in our analyses. The main results are based on annual mortality data at the cause by city level for 348 American cities from 1900 to 1937; though similar data has been used by scholars previously (including Cutler and Miller 2005; Anderson et al. 2018; Feigenbaum et al. 2019), we expand the sample and time period, standardize and categorize causes of death, and attach population denominators based on the complete count census. To study mechanisms, we also collected and digitized a number of new datasets. This includes: annual data on the number of immigrants denied entry into the US by cause and race; vital statistics by nativity at the city and state levels; annual birth data for cities in birth registration area; and city-level hospital statistics in 1910. We also constructed a

overview of this literature; see also Cutler et al. (2006) and Costa (2015) for further details and references.

[10] Our work is related—though distinct—from studies of the "healthy immigrant" effect. Though politicized narratives sometimes paint a negative picture of immigrants as unhealthy and as a fiscal drain on the healthcare systems of destination countries, 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 pattern holds in the US today, though the health advantage erodes with time spent in the US as immigrants "assimilate" (Antecol and Bedard 2006).

measure of internal density from the complete count census data, as well as measures of crowding based on boarders and lodgers and on multifamily housing. The fully assembled database should be a useful contribution to future scholarship on public health in the early twentieth century.

2 Historical Background

In this section, we describe the historical context for our study, beginning with the urban mortality penalty in the US, before discussing immigration to American cities, and then detailing the intersection of health and immigration restrictions 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. Economic historians and demographers attribute a large part of this penalty to the poor and unsanitary living conditions in cities (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 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 (Krieger and Higgins 2002; Stella 1908). In particular, tenement buildings in 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). The Danish-American social reformer Jacob Riis (1890) painted a dramatic picture of the dreadful 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*.

While the urban mortality penalty was substantial around 1900, death rates started to decline in the decades that followed. Between 1900 and 1940, mortality rates fell by 40 percent in cities and the urban-rural mortality gap largely disappeared (Haines 2001), while life expectancy and health status improved significantly (Troesken 2015). The leading causes of death also shifted from respiratory, food-, and water-borne diseases to non-communicable diseases (Cutler et al. 2006).

Poor housing conditions were correlated with higher rates of infectious disease transmission (Krieger

and Higgins 2002), but little is known about their causal effect on the decline in infectious-cause deaths over the first half of the 20th century.[11] Ours is the first comprehensive study that provides rigorous empirical evidence showing that the relaxation of housing constraints reduced infectious diseases in American cities during this period.

2.2 Immigrants in US Cities

Mass migration from Europe contributed to both the development and crowding of American cities. Between 1850 and 1920, the annual inflow of immigrants exceeded 1 percent of the total population in several years (Abramitzky and Boustan 2017). 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 1971), 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 persistence of slums. The high levels of urban mortality coupled with the concentration of immigrants in cities prompted several studies to draw a connection between urban mortality and immigration, particularly before the onset of the epidemiological transition (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 (Dublin 1916, 1922; Dublin and Baker 1920). While such disparities could partly reflect the negative selection of immigrants (Abramitzky et al. 2012), they were primarily driven by the poor living conditions that newcomers faced in US cities.[12] Stella (1908), for example, blamed overcrowding and a lack of ventilation in rooms for the higher

[11] There is an ongoing 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). Others regard public health interventions as important drivers of the mortality decline (Cain and Rotella 2001; Costa and Kahn 2006; Troesken 2004).

[12] Like immigrants, African Americans arriving from the South 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.

respiratory-related mortality rates of Italian children in New York. Likewise, the New York City health officials 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 end of the 19th century, the US-born feared how this would affect their lives, from the economy to politics to culture. Concerns over public health were frequent and visceral.[13] Nativists fueled these fears by blaming immigrants for the outbreak of diseases, such as Italians for the 1916 polio outbreak or Jews were stigmatized as carriers of tuberculosis (Kraut 1994). When the center of immigration shifted towards Southern and Eastern Europe, nativists' efforts at the national level went from tightening the rules for nationalization to immigration restrictions (Higham 2002). 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.[14] Medical testing of immigrants became widely accepted by the public and it appealed to politicians as a way of using 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 denied entry if they suffered from "loathsome" or dangerous contagious diseases. Despite the increasingly stringent laws to exclude "diseased immigrants," the number of people who were barred from entry for medical reasons between 1891 and 1924 was never very high (Kraut 1988); see also Section 6.4.

The nativist movement eventually scored major political victories with the passage of the immigration quota acts in 1921 and 1924. 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 (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

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

[14] 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.

from 4.5 million between 1910 and 1914 to less than 800 thousand between 1925 and 1929.[15]

The Emergency Quota Act of 1921 imposed quotas that were based on 3 percent of the population of each foreign-born nationality listed in the 1910 Census, with an annual cap of 357 thousand immigrants (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 immigrants 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 population of each foreign-born nationality. Second, the reference year 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.[16] In 1929, the annual overall quota was set at 150 thousand immigrants. This system remained in place, apart from some minor modifications, until the quota regime was replaced 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 (Abramitzky and Boustan 2017). 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 main data sources and we present summary statistics of our key variables in Table A.6. Other secondary datasets will be introduced in the relevant sections of the empirical analysis below.

3.1 Mortality Statistics

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. We digitize this information

[15]For more details on the implementation of the quota system in practice, see Appendix A.4. While a literacy test had been introduced in 1917 (known as the Literacy Act), it did not reduce immigration on a large scale (Goldin 1994).

[16]Immigration from most of Asia 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.

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 1936/37 (Cutler and Miller 2005; Feigenbaum and Muller 2016). Second, there are significant changes in the cause-of-death coding after 1937, making it difficult to standardize the panel (Feigenbaum et al. 2019). Third, we control for city-level age structure in the main analysis. This requires information from the complete population censuses, which are only available up until 1940.

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 influenza, pneumonia, tuberculosis, and diarrhea; the non-infectious group includes cancer and tumors, cardiovascular diseases, and diabetes; and the set of external causes encompasses all accidents, homicides, and suicides. Table A.7 lists the specific causes in each category. To obtain the overall and cause-specific mortality rates, we scale the corresponding death counts by the city population and we use log-linear interpolation to estimate the population in each city during the intercensal years.

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.[17] Our baseline results are robust to various sample adjustments, including a 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.[18]

To show that the quota effects are not simply due to changes in the population composition of cities, we collect four additional types of mortality data. First, we obtain mortality statistics by race for a subset of 132 cities from 1906-1937.[19] Second, we digitize the number of deaths in each city by nativity for 1900-1911 and 1914-1922; the only years when they are available at the city-level. Third, we collect the death counts by nativity for 1900-1911 and 1914-1932, aggregated over all urban areas in each state. These allow us to estimate the quota effects by nativity at the state-urban level. Fourth, we gather national-level mortality statistics by cause, age, sex, and country of origin in 1910, well before the quotas were implemented.

[17]The mortality records in 1931 and 1932 are much thinner, with data for 233 of the 348 baseline cities.

[18]Cities boundaries could change 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.18.

[19]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 that do tend to be the largest cities in the sample or those in the South (Feigenbaum et al. 2019). Nonetheless, the impact of the quota system is evident even in this subset of cities (see Table 4).

3.2 Immigration Statistics

We use three types of immigration data in this paper. First, we obtain annual immigration statistics from 1899-1930. For the years 1899-1924, we digitize the information in Willcox (1929). For the years 1925-1930, we digitize the figures from the *Statistical Abstract of the United States* (U.S. Department of Commerce 1929, Table, 106; 1931, Table 99). Second, we retrieve the annual quota limits by nationality over the period 1922-1930 from the same sources. Third, we collect yearly 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 our quota-exposure measure is constructed, 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 at the national level, the intensity of this immigration shock across cities, which we term quota exposure, varied substantially. We measure quota exposure by combining nationwide changes in immigration inflows with pre-quota settlement patterns of different immigrant groups in each 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 term, $\max(\widehat{M}_{n,1922-1930} - Q_{n,1922-1930}, 0)$, calculates the national change in immigrants from each sending country n as a function of the quotas. We compute this term by predicting how many immigrants from country n would have arrived each year had the quota system not been enacted. These predictions are based on the following regression, which we run separately for each sending country:

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

where M_{nt} is the actual inflow of migrants from country n in year t over the pre-quota period 1900-1914.

The fitted model from equation (2) can then be used to generate out-of-sample predictions for each nationality n over the period 1922-1930.[20] Our baseline predictions rely on annual inflows before World War I (WWI) as the war interrupted immigration to the US substantially.[21] Figure A.11 shows that our main results are robust to alternative ways of predicting post-quota counterfactual immigration.

The average of predictions produced by equation (2), $\widehat{M}_{n,1922-1930}$, captures the *expected* average annual inflow of immigrants over 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 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 quotas, such as Canada and Mexico.

For concreteness, Figure 1 illustrates the computation of missing immigrants for four sending countries. The top subfigures look at Russia and Italy, two countries heavily affected by the quota system, while the bottom subfigures consider Ireland and Sweden, two countries with generous quotas. The black solid lines refer to the actual inflows of the respective nationalities, the black dashed lines denote the predictions, and the red solid lines indicate the annual quota limits. Over 160,000 Russian immigrants are predicted to arrive in 1922, while under the Immigration Act of 1921, the annual quota for Russians was about 30,000.[22] This implies that about 140,000 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, but the predicted inflows for Irish and Swedish immigrants are lower than their corresponding quotas, implying that there were no missing immigrants from these countries.

The second term 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 (results are robust to using the 1900 or 1920 shares instead).[23] This assignment rule draws on the well-documented tendency of new

[20] If the predicted inflow for nationality n in year t is negative ($\widehat{M}_{nt} < 0$), we set it to zero.

[21] Official immigration statistics before WWI track the yearly arrivals in the administration records from Ellis Island relatively closely (Bandiera et al. 2013, Figure 3A).

[22] 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 Aaronson et al. (2020), Xu (2019), and Abramitzky et al. (2020).

[23] We compute the settlement patterns of immigrants from the complete enumerations of the 1910 census (Ruggles et al. 2021). The 1910 assignment of immigrants born in Poland and other parts of the Habsburg Empire, with the exception of Hungary, is complicated. For example, Polish speakers in 1910 were assigned either to Russia, Germany, or Austria-Hungary. However, as we show in Table A.9, our results are robust to using shares based

immigrants to settle in places with more immigrants from the same country of origin (Card 2001). We would thus expect more missing immigrants 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.

Overall, the quota system substantially curtailed immigration in the 1920s. Figure 2 shows the total number of actual arrivals 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 lines gives the total number of missing immigrants for each post-quota year (the shaded area in Figure 2). We observe nearly 700,000 missing immigrants each year under the Immigration Act of 1921. This number rises to about 850,000 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.[24]

Figure A.16 provides a map of log quota exposure for the cities in our baseline sample. These cities are primarily located in the Northeast and Midwest, where the intensity of the immigration shock also tends to be stronger. 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.

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, both in levels and trends. 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. The absence of such associations increases our confidence that the baseline results are not compromised by unobserved confounders.

To quantify level differences in mortality across cities before the quotas, we implement a balancing

on the 1920 complete counts, when the newly formed European states that emerged after WWI were allowed as responses to the birthplace question in the census. Despite these complications, we prefer to use the shares in 1910 as they predate the quotas by more than a decade and also reflect the settlement patterns before WWI. Our results are also robust to using 1900 shares, as shown in Table A.9.

[24]This calculation of 0.8 missing immigrants per 100 inhabitants shows how our measure of quota exposure has a natural scale that makes interpreting our results straightforward. To put our measure into context: during the Age of Mass Migration, foreign-born flows averaged roughly one immigrant per 100 people in the US population (Abramitzky and Boustan 2017). Among the cities in our baseline sample, the average quota exposure is 1.84 (median 1.47) with a standard deviation of 1.71. Equation (1) is thus our preferred measure of quota exposure. For robustness, Section A.2 details an alternative measure of exposure which calculates the "bite" of the quota system at the city level. Table A.11 presents the baseline results with this alternative.

test using cross-sectional data from 1910 and 1920. In both pre-quota periods, we find that cities which later 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 A.8 displays these results and the level differences are all close to zero, with only one case being statistically significant.

Moving beyond level differences, we check if the trends in mortality varied systematically across cities prior to the quota acts. 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 comparison of just two groups is relatively coarse, it allows for a clearer visualization; the full variation in quota exposure will be used in the main empirical analysis.

Figure 3 depicts the mortality 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 in 1918. Thereafter, the all-cause mortality rate decreases 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, though the distinction between the two groups of cities is less obvious.[25]

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.[26] Furthermore, both groups had similar rates of mortality just before the quotas were implemented. This 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 may have had little effect on this mortality category.

The 1918 Influenza Pandemic could have affected all four mortality rates to some degree. While the intensity of this shock appears to be reasonably similar for cities below and above the median quota exposure, it may have had long-run repercussions that vary systematically across cities. To address this

[25]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).

[26]The larger variation in external-cause mortality could reflect the relatively small number of such deaths.

concern, we will always control for mortality rates in 1918-21 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 pandemic.

4.3 Estimation Approach

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

$$\ln m_{ct}^{D} = \alpha + \sum_{j=1900}^{1937} \beta_j^D \ \textit{Quota exposure}_c \times I_t^j + \mu_c + \mu_t + \mathbf{AGE}_{ct}'\Phi^D + \mathbf{X}_{ct}'\Gamma^D + \varepsilon_{ct}^D, \tag{3}$$

where m_{ct}^D is the annual mortality rate for cause-of-death group D (all causes, infectious causes, non-infectious causes, and external causes) in city c during year t.[27] Our measure of the immigration shock, $\textit{Quota exposure}_c$, is interacted with year fixed effects (I_t^j), leaving out 1921 as the reference year. We always control for city fixed effects (μ_c), year fixed effects (μ_t), and the age structure of a city ($\mathbf{AGE}_{ct}'$).[28] In the baseline specification, the vector of controls $\mathbf{X}_{ct}'$ includes log population in 1910 and the mortality rate in four pre-quota years (1918-1921), all interacted with year fixed effects. The latter control for potential effects of the Great Influenza Pandemic. Subsequent robustness checks will also add state-by-year fixed effects.[29] We cluster standard errors at the city level.

After presenting the event-study estimates, we then modify equation (3), replacing the year fixed effects with a treatment indicator for the period after 1921. This is a pure mean-shift model that captures the average effect of the quota system. It will also be used to test for heterogeneous treatment effects. Our baseline results are not weighted by population, but doing so does not alter our findings, as shown in Table A.16.

The key identifying assumption of our DD strategy is that mortality rates in high and low quota-exposed cities would have developed similarly had the quota system not been implemented. While not directly testable, an absence of pre-trends would suggest that the identifying assumption is not violated

[27] While grouping diseases into broad categories reduces noise, it implicitly forces the treatment effect to be the same for all the specific causes within a given group. Table 2 studies how the quota restrictions affected several specific causes.

[28] Specifically, $\mathbf{AGE}_c'$ refers to the share of a city's population aged 0-19, 20-49, and 50+. Including these controls is akin to standardizing mortality by age.

[29] To account for the immigration shock due to WWI, we build a WWI control in a similar way to our measure of quota exposure. The quota exposure effects are robust to including this control, as shown in Table A.10.

(we presented such evidence with raw data in Figure 3 and will do so formally with the event-study in the next section). We emphasize that our DD approach should not be thought of as a classic shift-share instrumental variables method commonly used in the immigration literature (Card 2001), although it shares similarities. We are not using the quotas to instrument for changes in immigration flows (or anything else). Instead, we use the shares (of past settlement patterns) to determine which cities will be more or less exposed to the quota-induced shift in immigration. The threat to identification is that some other factor changed at the same time the quotas were enacted and that affected city-level mortality persistently and differentially according to quota exposure.[30] Since our identification strategy relies on a DD logic, we only uncover relative effects of the immigration restrictions on mortality, where quota exposure measures the local strength of the policy relative to a counterfactual with unregulated immigration. Therefore, our strategy cannot identify any aggregate mortality effects of the restrictions.

To make our identification assumptions clearer and probe their validity, in Appendix A.1 we follow Goldsmith-Pinkham et al. (2020). First, we calculate which sending countries weigh most heavily in our design; Italy, Russia, and Poland have the highest Rotemberg weights, all major pre-quota immigrant sources that were severely restricted by the quotas. Second, we show what city-level covariates correlate with the city-level shares from high weight countries. We either include these covariates in our standard set of controls (city population and age structure) or show our results are robust to flexibly controlling for them (by interacting pre-quota 1910 values with year fixed effects). Third, we present pretrends in our mortality outcomes, comparing cities with high and low shares of the high Rotemberg weight countries. Fourth, Table A.5 implements the DD specification separately for different sending countries. Finally, we document that the quotas affected the origin-composition of immigrant flows significantly; this helps allay concerns that our results could conflate the short- and long-run effects of immigration shocks (Jaeger et al. 2018). Together, the analysis in Appendix A.1 strengthens our confidence in the validity of our empirical design.

[30]Based on timing, two other events—national prohibition in 1920 and the Depression of 1920-21—are the likeliest threats to our identification strategy. However, the pattern of our event study results will push us to reject these alternative explanations. During prohibition, alcohol consumption fell and then increased (Dills et al. 2005). When prohibition was repealed in 1933, mortality increased on net (Jacks et al. 2021, 2020). The Depression of 1920-21 ended in July 1921. If either prohibition or the macroeconomy were driving our results, we would expect to see any of these time patterns reflected in our findings; yet we see persistent effects of the quotas on mortality in our event study through 1937 (Figure 4). Furthermore, we include year fixed effects which account for any macroeconomic swings or national policy changes.

5 The Impact of the Quota Policy on Urban Mortality

5.1 Baseline Results

We begin our empirical analysis by showing the event-study estimates based on estimating equation (3), 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 impact of the quotas over time and to assess if there were any differential trends before the quotas were enacted.

Figure 4 presents the results. For the all-cause mortality rate (Panel A), a clear pattern emerges after the change in policy: more quota-exposed cities experienced sustained declines in mortality relative to less exposed cities. Using 1929 as an example, we estimate a coefficient of $\hat{\beta}_{1929} = -0.017$, which implies that losing one immigrant per 100 inhabitants causes the mortality rate to decline by 1.7 percent. This negative effect increases somewhat until 1936, when it reaches 2.9 percent. A relatively flat pre-trend in the all-cause mortality rate is also observed from 1900 up until the outbreak of the Great Influenza Pandemic, providing empirical support for the identifying assumption.[31]

The remaining panels in Figure 4 explore which categories of death are driving the relative decline in overall mortality for cities that were more exposed to the quota restrictions. We find that deaths due to infectious diseases (Panel B) and external causes (Panel C) declined substantially after the quota system was introduced. However, no significant differences in deaths from non-infectious causes (Panel D) are observed between high and low quota-exposed cities. This suggests that the declines in infectious and external causes do not simply reflect changes in the population composition, given that immigrants had excess mortality in all three causes-of-death groups (Figure A.13).[32] In Section 6, we disentangle the mechanical composition effect from the spillover effect more formally.

Switching to a simple DD specification that replaces 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

[31] 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. Figure A.12 shows the estimates from a specification that includes mortality rates from 1900-1921 instead—the post-quota treatment effects are similar to those in Figure 4.

[32] 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.

causes. Table 1 reports the results from this analysis. Odd columns contain the same controls as the event-study specification, while even columns add state-by-year fixed effects, narrowing the comparison to 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 has previously been shown to improve population health (Miller 2008). Reassuringly, the two specifications yield similar DD coefficients.[33] The point estimates imply that every one less immigrant per 100 inhabitants results in the all-cause mortality rate declining by almost 2 percent, the infectious-cause rate falling by up to 4 percent, and the external-cause rate dropping by around 5 percent. Again, we do not find any substantive effects on non-infectious-cause mortality.

While the quota effects on infectious and external causes of mortality are similar in magnitude (Table 1), we focus our attention on infectious deaths as they were quantitatively more important at the time. In 1920, on the eve of the quotas, infectious diseases killed more than six times as many people as external causes and this ratio was even larger in the preceding decades (Table A.6).[34] The bulk of the quota effect on total mortality is thus driven by the impact on infectious-cause mortality.

5.2 Quota Effects by Specific Cause of Death

We examine the impact of missing immigrants on specific causes of death in Table 2. In the first column, we see negative and statistically significant effects on combined deaths from influenza, pneumonia, and bronchitis, which together were the leading causes of death for much of our period.[35] Figure A.9 provides the corresponding results when using an event-study framework. Negative and significant point estimates

[33]Our findings are also robust to controlling for pre-quota city-specific linear trends. In particular, we follow Goodman-Bacon (2021) by estimating linear trends for each city in the full sample with all controls prior to 1921, and residualize both the pre- and post-quota mortality outcomes using these estimated trends.

[34]External causes of death include accidents, suicides, and homicides. About 1 percent of all deaths in most years were from suicides and even fewer were due to homicides. Other accidents—occasionally detailed in the mortality reports as deaths from industrial, train, or streetcar accidents, but mostly in the catchall category of other accidents—accounted for the vast majority of external cause deaths, three-quarters in 1921, for example. While suicides are reported each year, accidents and homicides are only reported separately after 1920. It is therefore not possible to separate out the pre-quota trends between the specific external causes. Nonetheless, when estimating post-quota treatment effects separately, we find that mortality due to both accidents and homicides declined similarly after 1921. Composition could be an important mechanism for the external-cause effects as there was a substantial foreign-born penalty in accidental deaths among men (but not women). It could also be that the relative decline in deaths due to external causes reflects the clustering of immigrants in hazardous occupations. Appendix A.3 tests whether more quota-exposed cities experienced larger declines in dangerous jobs and finds no evidence that this was the case. Dangerous jobs were still done post-quotas, though we lack the data to determine if the jobs themselves became less dangerous when fewer immigrants held them.

[35]Influenza, pneumonia, and bronchitis deaths were often jointly reported during our period and have been analyzed together in past work (for example Fishback et al. 2007; Markel et al. 2007).

are also observed for diarrhea, measles, diphtheria, whooping cough, deaths in early infancy, and accidents and homicides. The impact on other causes, such as tuberculosis or suicide, are small and insignificant.

5.3 Changes in the Local Population

Did the quotas lead to a decline in city population? We find that this was the case, seemingly in contrast with Abramitzky et al. (2019) who estimate replacement for the missing immigrants in local labor markets (whites from rural areas, African Americans from the South, and immigrants from Canada and Mexico who were not subject to any quotas, were attracted to places with more missing immigrants). However, these population replacement effects are at the state economic area level and mask heterogeneity: Abramitzky et al. (2019) find central cities lose population while surrounding suburban areas grow. When we estimate the impact of the quota shock on the population of cities, we see relative population *losses* at about one fewer resident for each missing immigrant (Table A.12, column 1). On its own, the decline in city population would increase mortality rates, thus working against our results. The opposing effects—quotas reducing population size at the city level but triggering in-migration at a more aggregate geography—highlight two important features of our setting. First, by crowding into central cities, the quota-affected immigrants had very different settlement patterns from their replacements. Second, more quota-exposed cities must become less crowded as their populations fall, a key mechanism driving the mortality effects as will be discussed in greater detail below.

5.4 Robustness Checks

Our main conclusions are robust to different ways of implementing the analysis. First, we obtain similar results with an alternative measure of quota exposure that captures the "bite" of the quota system as illustrated in Greenwood and Ward (2015) (Table A.11). Second, 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.[36] To address this, Table A.12 reports the effects of quota exposure on mortality rather than mortality per capita. Even without using any population data, we observe large negative effects on the log of mortality due to all causes, infectious causes, and external causes. Third, Figure A.14 shows that the quota effects did not vary with initial residential segregation of immigrants within cities. Fourth, Table A.14 shows that

[36]The recent debate over the impact of clean water supply on mortality in US cities (Anderson et al. 2018; Cutler and Miller 2005, 2019) underscores how sensitive conclusions can be to mortality measurement.

our results 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. (2018). Finally, in Table A.15, we show that our results are robust to spatially adjusted standard errors, either using Conley (1999) standard errors or clustering at the state level.[37]

6 Mechanisms

This section explores the key mechanisms through which the immigration quotas affected mortality: changes in the population composition and overcrowding. While the composition channel can explain up to a quarter of our findings, we attribute the bulk of the quota effects to residential crowding. We make this calculation with a bounding analysis based on two well-specified reduced form estimates—the effect of quota exposure on mortality and the effect of quota exposure on crowding.[38] We further show that quota-induced changes in fertility, which could partly be driven by relaxed crowding constraints, account for at most one-tenth of the quota effect. Reduced contagion, improved labor market conditions, or increased public health spending that could be associated with the implementation of the quota system played, if anything, only a minor role.

6.1 The Composition Effect

Could our findings simply reflect changes in the population composition of cities after the quotas were implemented? Excess mortality of immigrants coupled with quota-induced reductions in their numbers can generate a mechanical decline in mortality rates. This would have been straightforward to assess if mortality statistics had been available by city and nativity over the full sample period. However, such data only exist from 1900-1922, almost entirely before the quotas took effect. Alternative approaches are thus needed to disentangle the composition and spillover effects. We proceed in six steps.

[37] Our results are also robust to controlling for medical supplies, as proxied by the number of medical staff and hospitals per 1,000 people in 1910 and 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. The demand for urban zoning had been driven, in part, by concerns over immigration (Shertzer et al. 2018). We collect data on these adoption dates for more than 500 US cities and confirm that our results remain robust even when accounting for zoning laws.

[38] The ingredients of this back of the envelope calculation are straightforward and may be applicable in other empirical settings. Our aim is to quantify what share of the total effect of our treatment (quota exposure) on our outcome (mortality) is driven by a mechanism (crowding). We are able to implement this decomposition analysis without resorting to regressions that include "bad controls." Instead, we implicitly calculate the effect of the treatment on the outcome only via the mechanism by multiplying the effect of treatment on the mechanism by the effect of the mechanism on the outcome. We then compare with (divide by) the total effect of the treatment on the outcome. In addition to our well-identified estimates of quota effects on mortality and on crowding, we do need one additional component connecting crowding and mortality. Lacking a causal estimate of this relationship, we instead use a correlation of mobility and crowding (see the details in Subsection 6.2.1). Though this correlation could be subject to upward or downward biases, we think our bounds are still informative of the general share of our total effect explained by crowding.

6.1.1 A Simple Decomposition of Mortality Rates

First, we provide a decomposition to frame the discussion. 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}):

$$\frac{M_{ct}}{P_{ct}} = \frac{M_{ct}^{UB}}{P_{ct}} + \frac{M_{ct}^{FB}}{P_{ct}}. \tag{4}$$

Changes in mortality are either 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 respective 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:

$$m_{ct} = m_{ct}^{UB} + \left(m_{ct}^{FB} - m_{ct}^{UB}\right) \times p_{ct}^{FB}, \tag{5}$$

where $p_{ct}^{FB} \equiv P_{ct}^{FB}/P_{ct}$ denotes the share of foreign-born people in city c during year t. 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 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. However, this section presents evidence that the quota acts also affected m_{ct}^{UB} directly.

6.1.2 Excess Mortality of Immigrants

Second, we show that immigrants and the US-born have different mortality rates, a necessary condition for the composition channel to matter. Drawing on our mortality-by-nativity data from 1900-1922, we document the excess mortality of immigrants over time. Figure 5 plots 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 of a city. Consistent with contemporaneous analysis (Dublin 1916), there is a substantial mortality penalty for immigrants. Except for the spike in 1918 due to the Great Influenza Pandemic, excess mortality tends to hover around 5 deaths per 1,000 people over the pre-quota period, though it falls somewhat after the pandemic.

Similar patterns can be observed when we compare the country-specific mortality rates of immigrants with the US-born: at nearly all ages, immigrants have higher mortality rates. Figure A.13 presents the

age-specific mortality rates of white men and women in 1910 for all causes by country of origin. While we are limited to 1910—when such data were reported by country of birth—we see that immigrants experienced mortality penalties across all adult ages, reinforcing the patterns in Figure 5.[39]

6.1.3 Quotas Reduce US-Born Mortality at the State Level

Third, since city-level mortality-by-nativity data only exist from 1900-1922, we turn to state-level data instead to determine if the quota restrictions affected mortality rates of the US-born.[40] From 1900-1932, consistent information on deaths by nativity is available for urban aggregations in each state. With these data, we can separately assess the impact of quota treatment on US- and foreign-born mortality, albeit at the state-level. We find that quotas reduced the all-cause mortality rate of the US-born.

To implement this analysis, we collect additional data and construct the quota shock at the state-urban level. Drawing again on the Census Bureau mortality statistics, we digitize the number of deaths in the urban areas of each state for the full US-born population, US-born whites, and the foreign-born. We pair these death counts with the population of each group based on the complete census enumerations.[41] We then calculate state-urban versions of our baseline control variables: log population in 1910, age structure, and mortality rates from 1918-1921. Finally, we recalculate the quota treatment based on state-urban foreign-born population shares in 1910 (the predictions of missing immigrants by sending country are unchanged).

We find that the immigration restrictions affected the health of the US-born population. Table 3 presents our results. Column 1 confirms our main findings from Table 1 using these more aggregate data: an additional missing immigrant per 100 people reduces total mortality by over 13 percent. We see large

[39]Three additional points: First, mortality rates are higher for US-born children compared to foreign-born children, 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. Second, Irish immigrants had especially large mortality penalties, a pattern that was also noted contemporaneously (Dublin 1916). However, the extreme excess mortality of Irish immigrants is unlikely to be driving our findings as the quotas for the Irish were fairly generous. Third, our results are robust to controlling for the baseline mortality rates in the origin countries of immigrants. Specifically, we draw on life expectancy data for origin countries in 1910 from Gapminder and construct the average life expectancy of immigrants arriving in a given city by combining the life expectancies of each immigrant group and their distribution across cities in 1910. In effect, this measures the average life expectancy of immigrants arriving in a given city. Table A.13 shows that our results are robust to including this control interacted with year fixed effects.

[40]We thank Michael Haines and Douglas Eckberg, two experts on historical census and mortality data, for confirming that post-1922 mortality-by-nativity statistics at the city level were neither published nor compiled in any other forms.

[41]We build the interpolated populations up from our city-level dataset as the urban death totals are based only on the cities that report mortality statistics. When a city begins reporting total deaths, deaths by nativity from that city also begin to be included in the state urban aggregates and we add that city's population to our denominators accordingly.

and negative effects on the US-born (column 2) and US-born whites (column 3). However, some of the US-born whites may be second generation immigrants who are likely to live in ethnic enclaves that were directly affected by the quotas (and the reduction in crowding). The state-level mortality data allow us to distinguish between deaths of the US-born by parentage. We see in columns 4 and 5 that both US-born whites with foreign-born parents and with US-born parents have lower total mortality after the quotas. Though the point estimate on second generation immigrants is larger in magnitude than the coefficient for those with US-born parents, the difference is not statistically significant and both effects are quite large and comparable to the overall effects. Since US-born mortality rates also responded to the quota shock at the state level, we find it very unlikely that the baseline effects at the city level are simply driven by compositional shifts in the population.

6.1.4 Quota Effects on African American Mortality

Fourth, as further evidence that the quota effects reflect more than just compositional changes, we show that the mortality decline in quota-exposed cities was also large for African Americans. We illustrate this with a subset of 132 cities for which mortality statistics are available by race from 1906-1937. Since we cannot distinguish between US- and foreign-born whites in the city-level mortality statistics, the mortality rates of African Americans, the majority of whom were born in the US, can shed light on potential spillover effects from immigrants to the US-born.

African-American mortality rates improved after the restrictions on immigration were imposed, as we show in Table 4, with effects of a similar magnitude as those for whites. As we cannot split white mortality by nativity, the effects on whites potentially encompass both compositional and spillover effects. The first four columns show larger declines in mortality due to all causes in more quota-exposed cities. The effects by race are statistically indistinguishable (the negative but insignificant triple interaction in column 2 or compare the Quota Exposure $\times$ Post terms in columns 3 and 4). Turning to deaths due to infectious causes in the final four columns of Table 4, we again observe larger reductions in white and black mortality in cities that were more affected by the quotas, now with marginally stronger effects for whites. Overall, the presented evidence hints at the presence of spillover effects from immigrants to the US-born.

6.1.5 Magnitudes Provide Additional Evidence Against a Composition-Only Effect

Fifth, we present indirect evidence pushing back against a composition-only narrative at the city level. Put simply, our results are too large to be explained by such mechanical changes alone. To show that our

baseline effects capture more than just compositional shifts, we estimate the "effect" of the share of foreign-born people on mortality, using quota exposure as an instrument for the foreign-born population share. The exclusion restriction here is that the quotas only influenced mortality via the foreign-born population share. This allows us to back out an estimate of immigrants' excess mortality from the coefficient on foreign-born share ($\lambda \equiv m_c^{FB} - m_c^{UB}$ in equation (5)). We can then compare it with the actual mortality penalties in Figure 5. A $\widehat{\lambda}$ that is much larger than what is observed 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 support our interpretation that the baseline effects are not driven by changes in composition alone.

We find that $\widehat{\lambda}$ is indeed too large. In column 1 of Table 5, we report the results from estimating equation (5) with our main sample of cities for both overall mortality and deaths due to influenza, pneumonia, and bronchitis, using $\textit{Quota exposure}_c \times I_t^{post}$ as an instrument for the foreign-born share.[42] Row 1 of Table 5 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 and far exceeds the foreign-born mortality penalty displayed in Figure 5 substantially. $\widehat{\lambda}$ also exceeds the penalty based on mortality data stratified by country of birth, cause, sex, and age (Figure A.13). We see that the estimated "effect" of foreign-born share on mortality is much too large for total mortality or deaths from influenza, pneumonia, and bronchitis.

6.1.6 How Large is the Composition Effect?

Finally, how much of the baseline quota effects can be attributed to the quota-induced changes in population composition? Our back-of-the-envelope calculations suggest that about one-quarter of the total quota effect in the median city is due to composition shifts and that such changes account for less than half the total effect in two-thirds of our cities.

To calculate the predicted change in mortality arising only from changes in the foreign-born share, we proceed in six steps. First, we use a simple cross-sectional city-level regression to estimate the effect of quota exposure on changes in the foreign-born share of the population from 1920-1930. We find a consistent negative relationship between the quota shock and changes in the share of foreign-born people (Table A.19). Second, we use this estimated relationship and city-level quota exposure to predict

[42] In all cases, 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. City fixed effects will eliminate US-born mortality differences across cities (m_c^{UB}) in equation (5) if such differences are constant (or exogenous), which is the null-hypothesis here. The first-stage indicates that cities which were more exposed to the quota restrictions experienced larger declines in their foreign-born shares, with Kleibergen-Paap F-statistics over 10. The latter allays concerns that the instrument may be weak.

the change in the foreign-born share for each city in our baseline sample.[43] Third, we collect mortality rates for the US- and foreign-born in each city in 1920, the decennial census immediately before the quotas. Fourth, we predict what the mortality rate in each city would have been in 1930 under the strong assumptions that mortality by nativity would be the same as they were in 1920 and that the foreign-born share of the population changed as predicted by the first two steps. Fifth, combining this counterfactual mortality rate in 1930 with the true mortality rate in 1920, we calculate the growth (decline) in mortality. This is the change in mortality accounted for by composition.

Finally, for each city, we need to compare the composition-accounted-for change in mortality rate to the total quota mortality effect. To calculate the latter, we combine our estimated treatment effects (column 1 of Table 1 or the 1930 coefficient of Figure 4) with city-level quota exposure, exploiting the log-level specification to calculate a growth rate. We divide the composition-accounted-for change in mortality by the total quota change to calculate the share of the predicted change in mortality that composition accounts for.

As we document in Table 6, composition is not the primary explanation for our main effects in most cities. Focusing on the calculations based on the DD estimates (row 1), we see that in the median city composition accounts for only 28 percent of the total quota-induced change in mortality. For more than one-quarter of cities, composition accounts for less than 10 percent of the total effect. In only 90 cities—26 percent of the sample—does composition explain more than 75 percent of the total quota effect.

Overall, the analysis in this subsection suggests that our baseline results are unlikely to reflect compositional changes alone. While the foreign-born population did suffer from high mortality penalties in this era and the quotas did shrink their share of the population, the analysis of state-level data, the clear spillovers to African-Americans, and the magnitude of our baseline effects all point toward a direct impact of the quotas on mortality rates of the US-born.

6.2 The Crowding Effect: Health Spillovers from Immigrants to the US-born

Instead of composition, we argue that the key mechanism behind the quota-induced mortality effects was the relaxation of congestion constraints on both housing and healthcare services. This subsection provides evidence for this hypothesis, underscoring the potential health dangers of unmanaged city growth in a setting with weak public health infrastructure.

[43] While our baseline sample includes 348 cities, two small cities in Massachusetts (Gardner and Westfield) do not report deaths by nativity and so the analysis in this section includes only 346 cities.

6.2.1 Congestion Constraints on Housing

Contemporary observers at the turn of the 20th century associated overcrowding with the urban mortality penalty (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. We show that the quota acts caused larger mortality declines in cities that were more crowded initially and that the introduction of the quota system relaxed such congestion constraints on housing.

Following Higgs and Booth (1979), we differentiate between internal and external density. To capture internal density, we use the number of foreign-born people per dwelling or the total population per dwelling, both measured in 1910 with Ruggles et al. (2022). We measure external density with city population per acre in 1910. If overcrowded housing conditions contributed to the spread of infectious diseases and if the quotas 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 quota shock.

To test our hypothesis, we extend our baseline DD model, including a triple interaction between quota exposure, a post-treatment indicator, and a normalized measure of density. Table 7 presents the results for internal density (Panel A uses internal density among the foreign-born; Panel B uses overall internal density). Odd columns include the controls from the baseline specification, while even columns add the interaction between internal density in 1910 and 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 rate is between 2 and 3 percentage points stronger when internal density in 1910 is one standard deviation higher (columns 3 and 4). No such heterogeneity is observed for the external-cause mortality rate (columns 5 and 6). External density, on the other hand, appears to be less important, as shown in Table A.17. 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 are an order of magnitude smaller.

Data limitations prevent us from directly assessing how the quotas affected internal density.[44] Instead,

[44]At the time of writing, there is a coding issue with the IPUMS complete counts: each household is incorrectly coded in a unique dwelling in 1920, 1930, and 1940. This makes it impossible to calculate the number of people or households per dwelling for these years.

we turn to two alternative indicators of congested or crowded living conditions. In Panel A of Table 8, we use the number of people living as boarders and lodgers per 1,000 inhabitants to proxy for congestion.[45] Boarding and lodging 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 boarders. Each additional missing immigrant per 100 residents led to roughly 3 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 105 foreign-born boarders per 1,000 residents versus 59 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).

Panel B of Table 8 considers another measure of housing congestion: the number of people living in multifamily households.[46] In line with the results for boarders and lodgers, we find that these particular living arrangements changed more in cities that experienced greater reductions in immigration due to the quota policy. The effects are largest for foreign-born residents in the most quota-exposed cities. Taken as a whole, the evidence suggests that the quotas did relax housing congestion constraints, which in turn lowered infectious-cause mortality rates.

How much of our total quota effect can be explained by the crowding mechanism? Assigning a precise share is difficult as we lack convincing causal estimates of the impact of crowding on mortality in early 20th-century American cities.[47] Nonetheless, the correlation between crowding and mortality, combined with our causal estimates of the effect of the quota shock on crowding and on mortality, can be informative. We detail our calculations in Table 9, examining all-cause mortality in Panel A and infectious-cause mortality in Panel B. First, we draw the estimated effects of quotas on our measures of crowding from Table 8, column 1. Second, we estimate the correlation between our two crowding measures and log mortality rates in 1910 and 1920, prior to the quota acts. Specifically, we regress log mortality rates on crowding with our standard

[45]We count the number of people coded by IPUMS with the following enumerated relationships to the household head: roomer/boarder/lodger (1201), boarder (1202), lodger (1203), roomer (1204), or tenant (1205).

[46]We count the number of people coded by IPUMS with "NFAMS" of two or more (see the IPUMS variable description for further details).

[47]A number of earlier studies estimate correlations between crowding or density and mortality (Higgs and Booth 1979; Crimmins and Condran 1983; Condran and Cheney 1982; Gaspari and Woolf 1985). In most cases, the authors find positive correlations between internal density (usually measured as people per dwelling) or external density (measured as people per acre) and mortality, measured for specific causes like tuberculosis or pneumonia or for all causes. However, none of these studies provide well-identified causal estimates. More, we cannot use these estimates "off-the-shelf" because of a data problem in the IPUMS coding of dwellings.

controls—log population in 1910 and age structure—and include either state or city fixed effects (see Table A.24). Third, we obtain the estimated effects of quotas on mortality from our main results in Table 1.

Our back-of-the-envelope calculations give us some sense of the role played by crowding. As we show in columns 6 and 7 of Table 9, depending on the measure of crowding and the specification, the share of the total effect that can be attributed to just one dimension of crowding that is affected by the quotas ranges from 27 to 68 percent for all-cause mortality and from 18 to 42 percent for infectious-cause mortality. While not causal, we argue that this exercise supports our claim that crowded and congested living conditions are a key mechanism driving the link between immigration restrictions and mortality.

6.2.2 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 digitize data from a Department of Commerce report on benevolent institutions in 1910. From the report, we compute the number of patients per hospital, per bed, or per 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 health sector capacity 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 10. In cities where the pre-quota number of patients per hospital and patients per bed—but not patients per medical staff—were higher, the quota effects were larger. This pattern is observed for the all-cause, infectious-cause, and external-cause mortality rates. In the case of infectious-cause mortality, for example, a one standard deviation increase in the initial number of patients per bed adds 2.5 percentage points to the main quota effect (column 5). Our results 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.

6.2.3 Effects of Missing Immigrants on Rural Health

Thus far, we have focused on cities as immigration at the time was an overwhelmingly urban phenomenon. However, immigrants were also attracted to rural areas. What effects did the quota acts have on mortality in rural counties? To summarize: very little. We see no evidence that mortality improved in the most

quota-exposed rural counties. As rural counties in the early 20th century were not crowded, these null effects are in line with our crowding mechanism.

To study the effects on rural mortality, we obtain annual rural mortality counts at the county level from Hoehn-Velasco (2018) and scale these with population figures from the decennial censuses, as in our city analysis. In this sample, we only observe total morality, not mortality by cause of death. We also exclude counties with cities larger than 2,500 people as measured in 1920 to avoid potential spillovers from urban areas. This leaves us with 737 rural counties. We then estimate the same event-study specification described by equation (3), but with rural county mortality rates as the outcome and county-level quota exposure as the main explanatory variable. Figure 6 plots the event-study estimates. While the pre-1910 estimates vary widely due to the highly unbalanced panel in earlier years, there are no systematic pre-quota differences. Importantly, unlike the results for cities, we do not find greater declines in mortality rates for more quota-exposed areas after the quota acts were implemented. This null finding does not stem from rural counties being spared from reductions in immigration, as more quota-exposed rural counties did experience larger reductions in the foreign-born population share.

6.3 The Fertility Effect

Less residential crowding improved the mortality environment in American cities, a change which might have also affected fertility. Following theoretical predictions and recent causal empirical evidence on the mortality-fertility link during the demographic transition, one might expect a fall in mortality rates to lead to a decline in gross fertility rates (Galor 2011; Ager et al. 2018). While plausible, without exogenous variation in residential crowding, we cannot test if this was the case.

However, the implementation of immigration restrictions could also lead to a change in fertility behavior independent of improvements in crowding, likely via the marriage market for the US-born (Ager and Hansen 2017; Carlana and Tabellini 2020) or foreign-born and second generation (Angrist 2002). Since infant and childhood mortality was high in cities during the early 20th century (Haines 2001), having fewer births could therefore mechanically translate into fewer (infant and child) deaths in our period. In this subsection, we show that the introduction of immigration restrictions did change fertility rates but that changes in fertility rates can explain at most one-tenth of our total mortality effects.

To assess the fertility channel, we digitized annual city-level data on births from the *Birth Registration*

Area (BRA).[48] This series starts in 1915 and we analyze it through 1937. We are only able to consistently measure total births and are thus unable to distinguish between quota effects on US- or foreign-born fertility. We apply our standard DD approach and find a decrease of approximately 0.2 births per 1,000 residents (Table A.20); an event study specification shows the same pattern of fertility reduction (Figure A.15).

How many deaths are "missing" because of the "missing" births? According to the US life table for 1920, approximately 87 percent of children survive to age 10 (Faber and Wade 1983). As the quota shock creates roughly 20 missing births per 100,000, that leads to 2.6 missing childhood deaths.[49] Combining our estimates of the total effect of the quotas on mortality from Table 1 with the sample total mortality rate in 1920 of 1,529 deaths per 100,000 (Table A.6), we calculate that the quotas led to roughly 27-29 fewer deaths per 100,000. This would imply that the fertility reduction and shift would account for less than one-tenth of our effect; likely an upper bound as improvements in the mortality environment through less residential crowding may have also contributed to the decline in fertility as well.[50]

6.4 Alternative Explanations

6.4.1 The Contagion Effect: Evidence from the US Border

Could the link between the quota acts and mortality be more direct? Trans-Atlantic travels during the Age of Mass Migration were harrowing and potentially unhealthy journeys for most immigrants, the majority of whom were packed in the steerage of ships (Keeling 1999). Writing on the "ordeal of steerage," Howe (1976) asks, "Was the Atlantic crossing really as dreadful as memoirists and legend have made it out to be? Was the food as rotten, the treatment as harsh, the steerage as sickening?" and answers "the suffering was real, it

[48] Like our mortality data, the BRA covers a growing number of cities. Fishback et al. (2007) draw on this data from 1921 on, but only 95 cities in our sample are in their dataset. From 1915-1922, we observe births by race and nativity, but only births are observed from 1923-1937, our entire post-quota period.

[49] In addition, there is a second possible path for fertility to affect total mortality, changing the composition of births; though related to our main composition exercise, this channel is not captured by our previous analysis as children born after their parents migrated to the US are, of course, US-born. Infant mortality was not just high, it appears to be unequal: nationwide among whites in 1920, there were 7.6 deaths under age 1 per 100 births to US-born mothers compared to 9.7 deaths per 100 for foreign-born mothers. However, this penalty is an artifact of national data and differential urbanization rates: when we examine infant mortality in cities, the parental nativity penalty disappears. In 1915 (the one year we have the requisite birth and infant death data for cities by parent nativity), we see 9.66 infant deaths per 100 births for US-born white parents versus 9.41 infant deaths per 100 births for foreign-born white parents, a US-born penalty. Thus, even as the quotas reduced the share of births to foreign-born mothers by 2 points in 1920 (Table A.21), without a penalty, overall mortality would be unaffected. Even using the national penalty (driven by urban-rural differences), this channel would only account for about 1 fewer death per 100,000.

[50] Drawing infant deaths from the BRA data, we can estimate the effects of quota exposure infant and non-infant mortality. We see reductions of similar magnitudes in both log infant mortality and log non-infant mortality (Table A.22).

was persistent, and it has been thoroughly documented." Trans-Atlantic ships were tinder boxes for diseases even among the healthy, as the American military learned when transporting troops to Europe during the Great Influenza Pandemic (Byerly 2010). Immigrants lived and slept in crowded shared spaces and trips took one to two weeks depending on the origin and destination (Battiston 2018). With the introduction of the quotas, fewer ships arrived in American ports and the immigrants they carried could have been healthier upon arrival, reducing contagion from travel.[51] In this subsection, we reject such a hypothesis—the quotas induced no reduction in the relative number of sick immigrants arriving in the US, no change in the selection of immigrants in terms of health, and the mortality effects were no stronger in immigration port cities.

To assess if the number of immigrants arriving in poor health changed after the quotas were in place, we digitized annual 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 national origin. We use historical trends to construct the number of missing immigrants from each origin and split nationalities into above and below median quota exposure.[52]

Very few immigrants were rejected at the US border for infectious diseases.[53] As we show in Panel A of Figure 7, while more than 30 thousand immigrants were denied entry in some years (1914), the number of people rejected for disease-related reasons never topped 2,700 (again in 1914). After the quotas were enacted, the total number of disease-related rejections peaked at only 1620 in 1924 and numbered fewer than 400 in all years after 1925.

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 7). We define rejection rates as the number of people rejected at the border divided by the total number of people arriving at the border. The average disease-related rejection rate was below 1

[51] Qualitatively, such quota effects would be inconsistent with the historical record. Historians of migration have documented that conditions in steerage improved between 1900 and WWI, including more closed berth cabins in steerage way before the quota restrictions of the 1920s (Keeling 2013). Keeling (1999) argues that such improvements were driven by non-price competition between participants in the steamship cartel.

[52] Using this assignment rule, the following groups are considered to be high-quota races or nationalities: Balkans, Dutch and Flemish, Finnish, Greeks, Hungarians, Italians, Portuguese, Russians, Spanish, and Turkish. The low- or no-quota races or nationalities comprise: Central Americans, Czechs, English, French, Germans, Irish, Mexicans, Pacific Islanders, Romanians, and Scandinavians. The original reports specify immigrant races, rather than national origin. However, given the state of eugenics and racial pseudoscience in the early 20th century, a mapping between race and nationality is relatively straightforward.

[53] The costs of returning rejected immigrants fell on the shipping companies (Battiston 2018) and so medical screenings were conducted before departure as well. Rejections on the basis of infectious diseases can be consistently coded from 1900-1930. Our results are echoed in contemporary findings of the healthy immigrant or healthy migrant effect. See, for example, Antecol and Bedard (2006) and Aldridge et al. (2018).

percent in almost all years, and the rates for above and below median quota exposure nationalities 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. The relative unimportance of disease-related rejections contrasts sharply with rejections due to the quota acts. 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. Such a shift is mirrored by the total rejection rate, where the two groups are again comparable prior to the quotas.

However, immigrants might be infected in transit but not develop any symptoms until after arriving because of incubation periods or difficult diagnoses. Such disease transmission would be unobservable to the immigration officials (and in our rejection data). If immigrants were infectious upon arriving in or near their entry port—and the contagion effect is an important mechanism—we should expect to see differences between port cities and non-port cities. To test this, we collect admittance data from the 1910 *Report of Commissioner General of Immigration*. While the majority of immigrants entered via New York (Ellis Island), the ports of Boston, Philadelphia, and Baltimore all admitted large numbers of immigrants as well. As we show in Figure A.18, our main results are robust when we exclude (1) port cities or (2) cities within 10 miles of a port or (3) cities within 10 miles of a port with greater than the median number of arrivals.

6.4.2 Improved Labor Market Conditions

Since much of the economic history literature on the Age of Mass Migration has focused on the labor market impact of immigration (Abramitzky and Boustan 2017), one might wonder if changes in labor market conditions might be an alternative channel through which the quotas affected health and mortality. We argue that this is unlikely to be an important mechanism for two reasons.

First, the effect of the quotas on local labor markets is unclear. In theory, barred immigrants could create a labor supply shock that allows remaining workers to secure economic rents (higher wages). Our city population results (Table A.12) suggest such a supply shock did occur. However, Abramitzky et al. (2019) find no such population decline when looking at more aggregated labor markets (state economic areas). Furthermore, they estimate no change in manufacturing wages in urban labor markets in response to immigration. In contrast, Xie (2017) observes wage increases using a different identification strategy. Tabellini (2020) shows that the US-born benefited from immigration and that quotas hurt employment and occupational status. Consequently, while the best historical evidence (Arthi et al. 2022; Fishback

et al. 2007) suggests that downturns were bad for health, it is hard to gauge second order effects when the first order effect of the quotas on labor markets is ambiguous.

Second, our event studies (Figure 4) show persistent effects of quota exposure on mortality through 1937. This is inconsistent with a story connecting the quotas to health via short-run business cycle effects or labor shocks. In addition, our year (and state-by-year) fixed effects should flatten any macro cycles (including the 1921 recession and the Great Depression). Even if the quotas did increase wages among the remaining city residents and workers were able to "purchase" better health, possibly through less crowded housing and better living conditions, that could still come under our crowding mechanism.[54]

6.4.3 Public Health Spending

The quota-induced reduction in immigration may have had other repercussions. One well-studied result is political: we know that US-born preferences for redistribution vary with the presence of immigrants (Alesina et al. 2022; 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. We test this hypothesis using data on health spending for 145 cities in our sample.[55] We observe expenses and capital outlays, separately for health, sanitation, and charity. Using a specification similar to our baseline model, we regress per capita 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—log city population and the initial outcome—interacted with year fixed effects. The results, reported in Table A.23, do not provide strong support for the public spending hypothesis. While most of the estimated effects of quota exposure on public spending are positive, all are small and only one is marginally statistically significant.[56] Given the weak evidence here and the uncertain effects of public health spending on actual public health during this era, we think the crowding mechanism is more central to our story.

[54]Similarly, though return migration was common (Bandiera et al. 2013), if anything the effects of the quotas on return migration would work against our baseline results. As Ward (2017) shows, return migrants were negatively selected and the quotas *reduced* unplanned return migration and *reduced* the degree of negative selection into return migration for the most restricted nationalities.

[55]We draw on data from Swanson and Curran (1976) which are based on historical Census Bureau and Commerce Department compilations, also used by Tabellini (2020). The sample includes data from 1905-1912, 1915-1919, and 1921-1930 but we do not use the 1926 data due to a coding mistake.

[56]We obtain similar results with the least conservative model, which only controls for city and year fixed effects.

7 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 mortality rates, and in part because nativists fueled fears that newly arriving foreigners were carriers of diseases (Dublin 1916; Kraut 1994). Instead of blaming immigrants, some public health officials recognized 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 US cities were still characterized by overcrowding and poor sanitation when the quota acts were implemented in the 1920s (Abbott 1936; Park and Kemp 2006; Veiller 1921).

Our results echo the views of urban reformers at the time. The majority of immigrants arrived in good health at the beginning of the 20th century and it was their unfavorable living conditions that created the apparent link between the urban mortality penalty and immigration at the beginning of the 20th century. We find that more quota-exposed cities experienced relatively larger declines in total mortality rates, largely driven by declines in deaths due to infectious diseases. 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 observe large spillover effects from immigrants to the US-born that are mediated through congestion constraints in housing and healthcare. The quotas substantially reduced residential crowding and we find that more quota-exposed cities with initially more crowded housing and hospital facilities experienced greater declines in infectious-cause mortality.

We estimate that the quota system prevented around 850,000 people from immigrating to the US annually between 1921 and 1937. Our main results imply that the quotas averted up to 18,500 deaths each year (13,300 deaths from infectious causes).[57] But this mortality reduction came at a high cost, as immigration during this era had large benefits in the long-run (Ager and Brueckner 2013; Burchardi

[57]The cities in our sample saw a decrease in infectious mortality of 59 percent from 1920-1937. Combining our estimated effect of quotas on infectious mortality with the average quota exposure, we calculate the role of immigration quotas in the overall decline in mortality. Our estimates suggest that the quotas—and ensuing reductions in crowding and changes to population composition—explain about 15 percent of the decline in infectious disease mortality between 1920 and 1937. While dwarfed by the 40 percent likely accounted for by clean water and sewerage (Cutler and Miller 2005; Alsan and Goldin 2019), our findings underscore the importance of immigration and of urban living conditions in explaining the trajectory of mortality in the early 20th century.

et al. 2019, 2021; Sequeira et al. 2020) and to immigrants themselves (Abramitzky et al. 2012). Could a comparable mortality reduction have been possible by other means? Without exogenous variation in other plausible policies, a definitive conclusion cannot be reached, but the heterogenous treatment effects we find based on how crowded cities initially were suggest yes. The difference in annual deaths averted between cities one standard deviation more or less crowded—measured by internal density among immigrants—was 42,300 (27,800 infectious deaths), more than the total effect. Other policies like improving public health infrastructure or regulating housing quality could also have broken the link between immigration and urban mortality, though the policies implemented contemporaneously by the federal government—such as inspections at ports to exclude people with "loathsome" diseases—and local governments—zoning immigrants and other poor residents into industrial-use districts—certainly did not.

Our results speak to the health challenges facing urban populations today. Substandard housing and weak public health infrastructure are still major public health issues in the US and worldwide. The United Nations regards improvements in housing conditions as key to limiting the spread of infectious diseases. Well-designed housing policies and sufficient public health capacity are crucial to reducing potential health risks.

Our findings also underscore the ever-present dangerous cycle that links nativism and health. The history of the early 20th century—which we see echos of today (see Appendix A.5)—suggests that medicalized fear of the other, particularly immigrants, often results in public policy that is either insufficient or absent; this results in health and mortality penalties for immigrants that seem to "confirm" the worst fears of demagogues and earn their extreme proposals (quotas that shut down immigration; invasive and unwarranted medical procedures at borders) more support. Perhaps better public policy—in our setting, public housing or overcrowding—might have had the dual benefits of saving lives from infectious disease in densely populated urban neighborhoods *and* breaking the presumed link between immigration and poor health.

8 Data Availability Statement

The data and code underlying this research is available on Zenodo at https://dx.doi.org/10.5281/zenodo.7506459.

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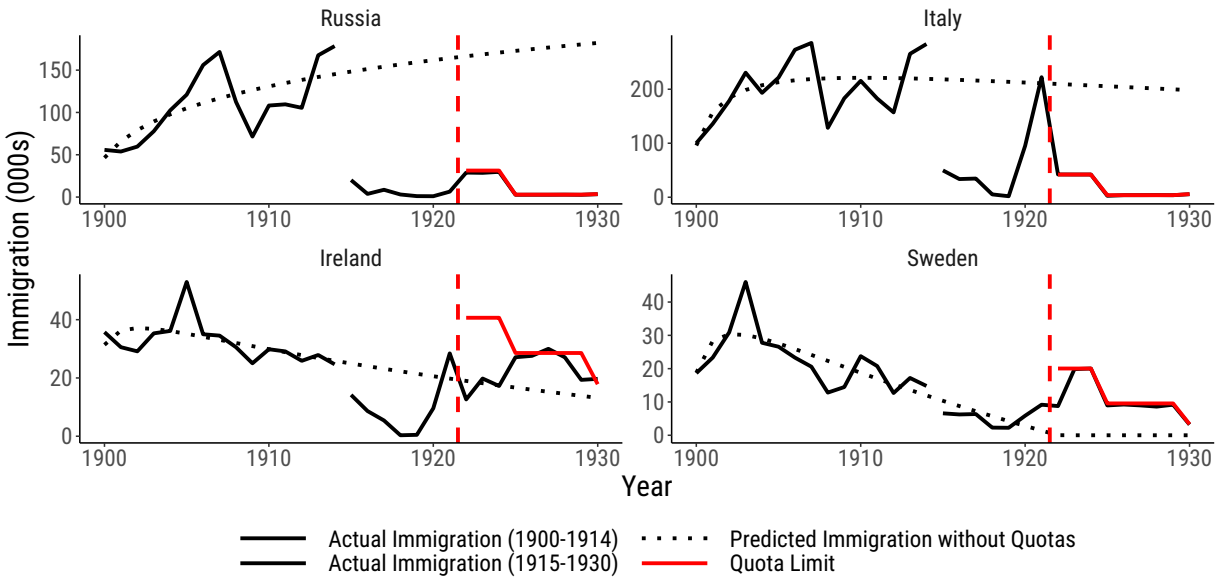
Figure 1: Missing Immigrants from Four European Countries: Russia, Italy, Ireland, and Sweden

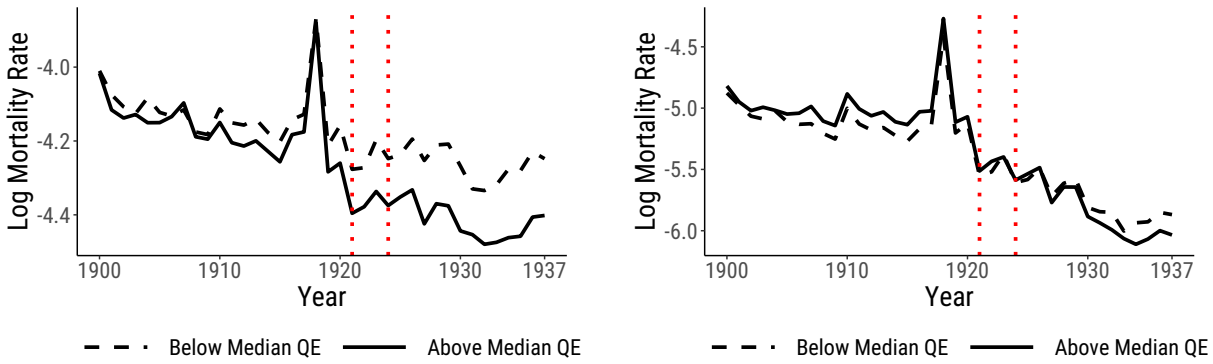


Notes: We choose these four countries to illustrate our procedure for predicting missing immigrants. We plot actual migration, predicted migration in a counterfactual scenario with no quotas based on pre-WWI migration flows, and country-specific migration quotas. For Russia and Italy, the quotas were 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, was unaffected by the quotas according to our calculations.

Figure 2: Actual and Counterfactual Immigration from Quota Countries

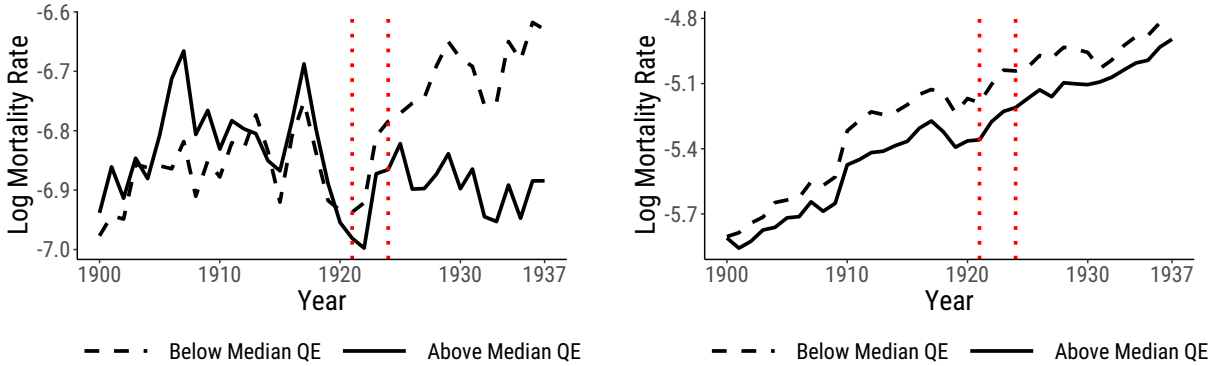
Notes: This figure shows the actual (solid line) and counterfactual (dashed line) immigration from quota countries (in 1000s). The difference gives the number of missing immigrants (shaded in gray).

Figure 3: Trends in Average Log Mortality Rates by Quota Exposure and Year



(a) All Causes

(b) Infectious Causes

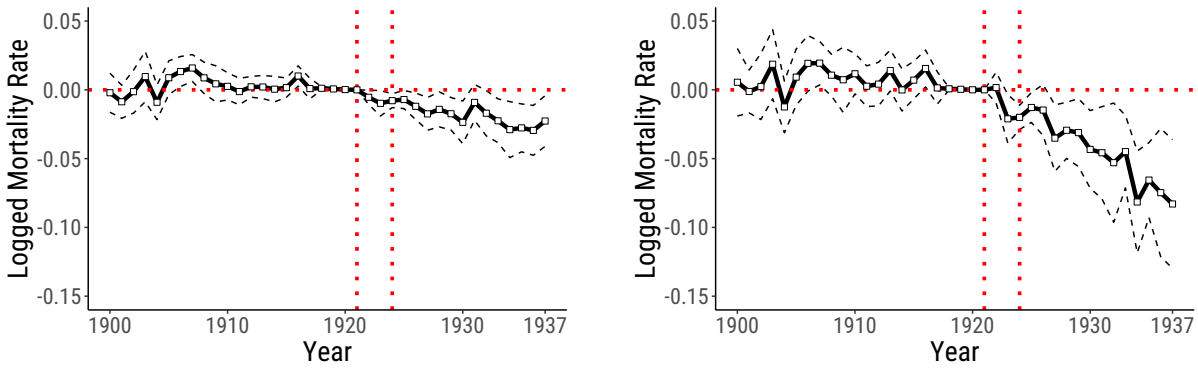


(c) External Causes

(d) Non-Infectious Causes

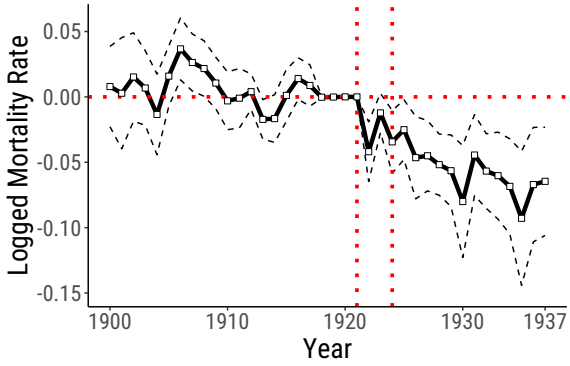
Notes: This figure shows the average log mortality rates for all causes, infectious causes, external causes, and non-infectious causes, separately for two groups of cities: The dashed lines denote cities with below-median quota exposure, while the solid lines refer to cities with above-median quota exposure.

Figure 4: Event-Study Estimates of Quota Effect by Cause-of-Death Category

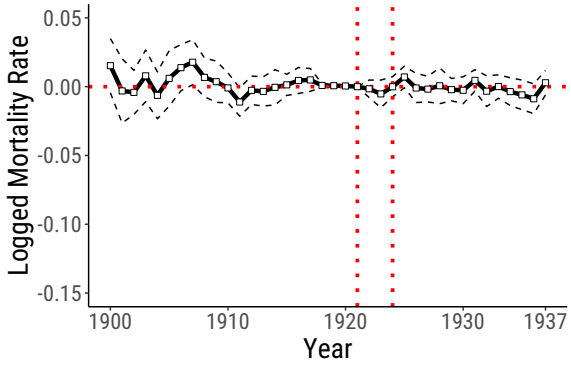


(a) All Causes

(b) Infectious Causes



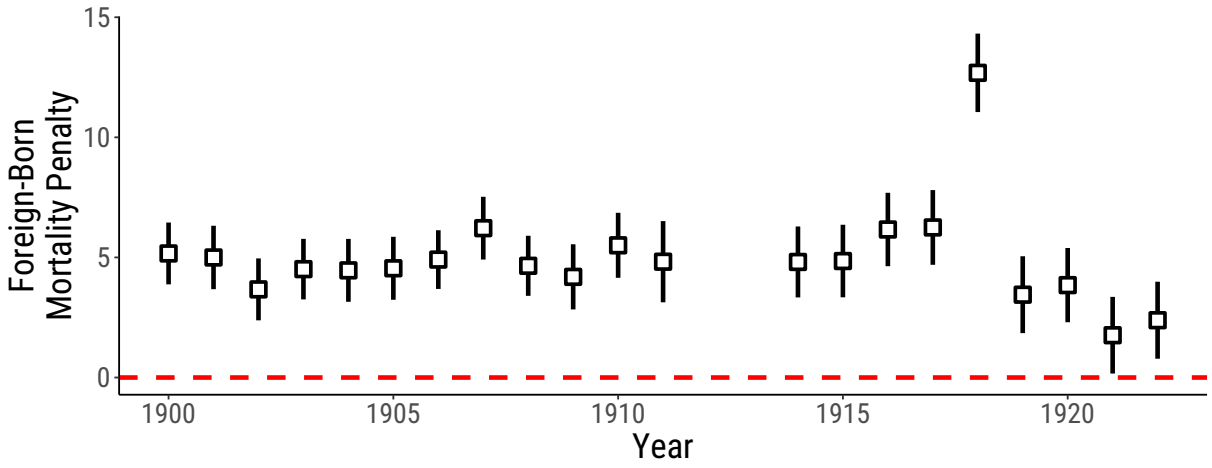
(c) External Causes



(d) Non-Infectious Causes

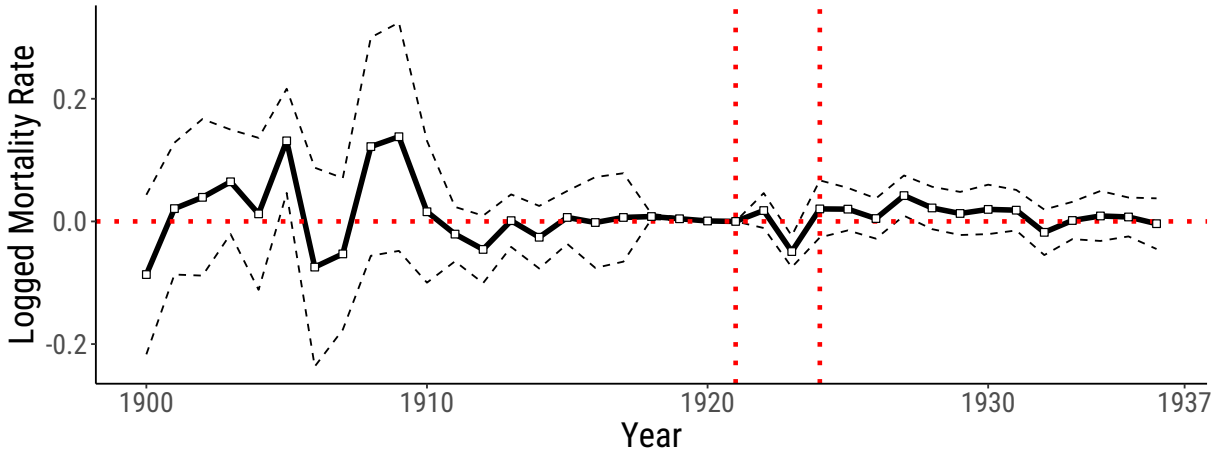
Notes: This figure shows the event-study estimates of the effect of quota exposure on log mortality rates based on estimation equation (3) by general cause-of-death category: all deaths (a), infectious (b), external (c), and non-infectious (d). We include controls for city age structure, city fixed effects, 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 reference year is 1921. The dashed lines represent 95-percent confidence intervals.

Figure 5: Foreign-Born Mortality Penalty Over Time



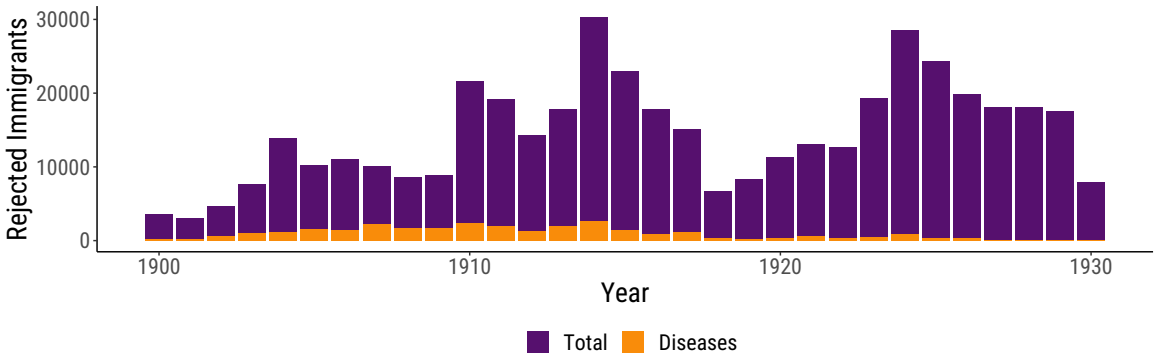
Notes: This figure shows the evolution of the foreign-born mortality penalty, measured as $m_{ct}^{FB} - m_{ct}^{UB}$, in US cities from 1900-1922. The estimates show the foreign-born-by-year fixed effects coefficients from a regression where the outcome is total mortality per 1,000 people, controlling for the city age structure. Mortality-by-nativity data are not available in 1912 and 1913.

Figure 6: Event-Study Estimates of Quota Effect on Mortality in Rural Counties

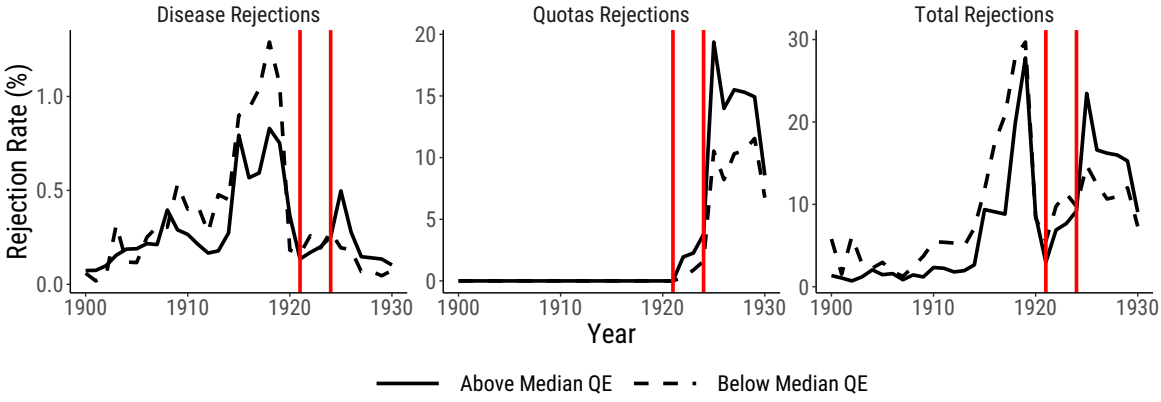


Notes: This figure shows the event-study estimates of the effect of quota exposure on log mortality rates based on estimation equation (3) for rural counties. We include controls for county age structure, county fixed effects, year fixed effects, log county population in 1910 interacted with year fixed effects, and log county mortality rates for 1918-1921 interacted with year fixed effects. The reference year is 1921. The dashed lines represent 95-percent confidence intervals.

Figure 7: Rejections at the US Border by Year and Quota Exposure



(a) Total and Disease-Related 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.

Table 1: Baseline DD 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	-0.019**	-0.018**	-0.044***	-0.040***	-0.055***	-0.048***	-0.004	0.006*
	(0.008)	(0.008)	(0.014)	(0.012)	(0.017)	(0.016)	(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
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	12,587	12,333	12,587	12,333	12,587	12,333	12,587	12,333
R^2	0.798	0.835	0.841	0.867	0.594	0.645	0.883	0.906

Notes: This table reports the baseline DD 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.7 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 and the sample size shrinks because 254 city-year observations (11 cities) are the only cities in a given state-by-year cell. 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 2: DD Estimates of Quota Effect for Selected Single Causes of Death

	Dependent variable is mortality rate (in logs) for:									
	Influenza, Pneumonia, & Bronchitis	Lung TB	Typhoid	Diarrhea	Measles	Diphtheria & Croup	Whooping Cough	Early Infancy	Accidents & Homicides	Suicide
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Quota Exposure × Post	-0.041***	-0.002	-0.008	-0.129***	-0.038***	-0.059***	-0.042**	-0.064***	-0.063***	-0.013
	(0.015)	(0.009)	(0.016)	(0.046)	(0.014)	(0.022)	(0.019)	(0.021)	(0.019)	(0.009)
Age Structure	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
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	12,587	12,558	9,799	12,043	7,430	10,141	9,352	12,547	12,587	11,589
R^2	0.735	0.828	0.790	0.810	0.480	0.601	0.489	0.740	0.578	0.396

Notes: This table reports DD estimates for specific causes of deaths. 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. The influenza and pneumonia deaths also include deaths from bronchitis. In Figure A.10, we show that our results are generally robust to including state-by-year fixed effects or using an inverse hyperbolic sine transformation. We analyze mortality data in logs since mortality rates are generally better described as log normally distributed. As there are no city-year observations with zero deaths in total or in a category (infectious, non-infectious, external), the log of death rate is always defined in our main empirical analyses. However, for some of the specific causes examined in this table, this is no longer the case. In the table above, we omit city-year observations with no deaths recorded for the given outcome cause, but using the inverse hyperbolic sine transformation of our dependent variables yields substantively similar results (the effect on diphtheria is no longer marginally significant).

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

Table 3: DD Estimates of Quota Effect by Nativity at the State Level

	Dependent variable is mortality rate (in logs) for:				
			US-Born White		
	Full Population	US-Born	All	Parents Foreign-Born	Parents US-Born
	(1)	(2)	(3)	(4)	(5)
Quota Exposure × Post	-0.135***	-0.205***	-0.169***	-0.228**	-0.177***
	(0.044)	(0.050)	(0.052)	(0.101)	(0.051)
Age Structure	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
Initial Mortality Rates × Year FE	Yes	Yes	Yes	Yes	Yes
State and Year FEs	Yes	Yes	Yes	Yes	Yes
Observations	1,137	1,137	1,137	1,106	1,137
R^2	0.753	0.762	0.589	0.616	0.711

Notes: This table reports the DD estimates in the state-urban panel. The outcome is the log mortality rate for all causes of death. Sample units are aggregations of all cities reporting death data in a given state and year. As the coverage of cities changes over time, the panel is unbalanced. Quota exposure is defined in equation (1) and interacted with an indicator for years after 1921, but calculated for state-urban areas rather than at the city level. All regressions include state 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 state's age structure. Population and age structures are all calculated for the demographic group analyzed in each column. The sample period is 1900-1932. Standard errors clustered at the state level are in parentheses.

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

Table 4: DD and DDD Estimates of Quota Effect by Cause-of-Death Category and Race

	Dependent variable is mortality rate (in logs) for:							
	All Causes				Infectious Causes			
	White and Black		White	Black	White and Black		White	Black
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Quota Exposure × Post	-0.032***	-0.035***	-0.035***	-0.039**	-0.034*	-0.049**	-0.049**	-0.040*
	(0.011)	(0.013)	(0.013)	(0.017)	(0.018)	(0.021)	(0.021)	(0.023)
Quota Exposure × Post × Black		-0.004				0.009		
		(0.016)				(0.017)		
Age Structure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Race × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	5,880	5,880	2,940	2,940	5,880	5,880	2,940	2,940
R^2	0.811	0.917	0.827	0.864	0.838	0.914	0.863	0.841

Notes: This table reports DD and DDD estimates from "stacked" specifications and separately by race (white and black). The outcomes are log mortality rates for all causes and 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. Columns 1 and 5 reproduce our main results from the subset of cities with mortality-by-race data, using the stacked specification. Columns 2 and 6 interact quota exposure with indicators for post-treatment and black mortality; we also interact all controls with a race fixed effect. Columns 3 and 4 and 7 and 8 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.

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

Table 5: Estimated Effects of Foreign-Born Share on Mortality vs. 1910 Foreign-Born Mortality Penalties

		Foreign-Born Mortality Penalty (λ)		
	$\widehat{\lambda}$	Raw	Age-Adjusted	Age- and Sex-Adjusted
	(1)	(2)	(3)	(4)
All Mortality	28.43	8.12	5.94	5.90
	[9.35 to 47.51]	[4.08 to 12.15]	[4.50 to 7.38]	[4.46 to 7.33]
Influenza, Pneumonia, and Bronchitis	6.01	0.94	0.89	0.88
	[3.01 to 9.01]	[0.42 to 1.45]	[0.65 to 1.13]	[0.64 to 1.13]

Notes: This table compares two-stage least squares estimates of the foreign-born share on all mortality and influenza, pneumonia, and bronchitis instrumented by $Quota\ exposure_c \times I_t^{post}$ (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 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 (column 2). 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.

Table 6: Share of Total Quota Effect Explained by Composition

			Cities with Composition Accounting for							
			<10%		<25%		<50%		>75%	
	$\widehat{\beta}$	Median Composition Share	N	%	N	%	N	%	N	%
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
DD	-0.019	28.0	99	28.6	161	46.5	220	63.6	90	26.0
Event-Study	-0.024	22.8	108	31.2	183	52.9	245	70.8	82	23.7

Notes: This table reports results from our back-of-the-envelope calculations of what share of the predicted decline in mortality due to the immigration quotas is accounted for directly by changes in the population composition of cities. Changes in mortality are calculated from 1920, before the quotas, to 1930, a post-quota year with census data on the foreign-born share of the population. We report the median share of deaths accounted for by composition (column 2), as well as the number of cities and share of cities where composition accounts for less than 10% (columns 3-4), less than 25% (columns 5-6), less than 50% (columns 7-8) of deaths, as well as the cities where composition changes account for 75% or more of the mortality change from 1920-1930.

Table 7: Treatment Heterogeneity by Internal Density

	Panel A. Foreign-Born 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	-0.020***	-0.020***	-0.047***	-0.048***	-0.055***	-0.055***
	(0.007)	(0.007)	(0.013)	(0.012)	(0.017)	(0.016)
Quota Exposure × Post × Internal Density	-0.010***	-0.011**	-0.020***	-0.027***	-0.001	0.000
	(0.003)	(0.005)	(0.004)	(0.010)	(0.006)	(0.012)
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
Dwelling 1910 × Year FE	No	Yes	No	Yes	No	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes
Observations	12,551	12,551	12,551	12,551	12,551	12,551
R^2	0.799	0.800	0.842	0.843	0.595	0.596
	Panel B. Total Population 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***	-0.049***	-0.050***	-0.058***	-0.058***
	(0.008)	(0.008)	(0.013)	(0.012)	(0.017)	(0.016)
Quota Exposure × Post × Internal Density	-0.009***	-0.010	-0.021***	-0.031***	-0.013	-0.015
	(0.003)	(0.007)	(0.004)	(0.010)	(0.009)	(0.014)
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
Dwelling 1910 × Year FE	No	Yes	No	Yes	No	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes	Yes
Observations	12,587	12,587	12,587	12,587	12,587	12,587
R^2	0.799	0.799	0.842	0.842	0.594	0.595

Notes: This table reports the DD 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 in Panel A) or standardized mean dwelling size for all people in 1910 (our measure of internal density in Panel B). 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.

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

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	-3.09***	-1.28***	-5.84***	-1.20***	-5.29***
	(0.85)	(0.48)	(1.48)	(0.45)	(1.82)
Age Structure	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
Outcome in 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes
Dependent Mean	69.21	59.24	104.87	56.86	152.24
Observations	1,668	1,668	1,668	1,668	1,610
R^2	0.90	0.88	0.86	0.87	0.90
	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	-6.62***	-5.50***	-7.44***	-5.08***	-7.12
	(2.13)	(1.71)	(2.14)	(1.47)	(7.35)
Age Structure	Yes	Yes	Yes	Yes	Yes
Ln Pop 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
Outcome in 1910 × Year FE	Yes	Yes	Yes	Yes	Yes
City and Year FEs	Yes	Yes	Yes	Yes	Yes
Dependent Mean	209.13	198.47	244.99	193.94	441.80
Observations	1,668	1,668	1,668	1,668	1,610
R^2	0.93	0.92	0.90	0.92	0.92

Notes: This table reports DD 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 population size in 1910 interacted with year fixed effects, baseline 1910 outcomes interacted with year fixed effects, 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.

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

Table 9: Share of Total Quota Effect Explained by Crowding

	Panel A. All Causes						
				Quota on Mortality			
	Quota on Crowding	Crowding on Mortality		$\hat{\beta}$ from Table 1			
Crowding Measure	$\hat{\beta}$ from Table 8	$\hat{\beta}$	Specification	Col 1	Col 2	Crowding %	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Boarders and Lodgers	-3.09	0.00178	State FE	-0.019	-0.018	28.8	30.2
		0.00169	City FE			27.3	28.6
Multifamily Households	-6.62	0.00130	State FE			45.2	47.3
		0.00189	City FE			65.7	68.8
	Panel B. Infectious Causes						
				Quota on Mortality			
	Quota on Crowding	Crowding on Mortality		$\hat{\beta}$ from Table 1			
Crowding Measure	$\hat{\beta}$ from Table 8	$\hat{\beta}$	Specification	Col 3	Col 4	Crowding %	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Boarders and Lodgers	-3.09	0.00279	State FE	-0.044	-0.040	19.4	21.6
		0.00253	City FE			17.6	19.6
Multifamily Households	-6.62	0.00166	State FE			24.7	27.5
		0.00251	City FE			37.4	41.7

Notes: This table reports results from our back-of-the-envelope calculations of what share of the total immigration quota effect on mortality can be accounted for by changes in crowding. To do so, we need three estimated effects: the effect of quotas on crowding, the effect of crowding on mortality, and the effect of quotas on mortality. We have causal estimates of the first and third effects and more speculative correlational evidence for the second. We have two measures of crowding that we observe in the census before and after the quota acts: the share of people in each city who are boarders and lodgers and the share of people living in multifamily households. We draw the estimated effects of quotas on these measures of crowding (both measured per 1000 people) from Table 8, column 1. We estimate the correlation between crowding and the log of mortality rates in 1910 and 1920, before the quota acts, by regressing log mortality rates on crowding with our standard controls and either state or city fixed effects. See Table A.24 for more details. We draw the estimated effects of quotas on mortality from our main results in Table 1. We calculate the crowding share by multiplying the quota on crowding effect by the crowding on mortality coefficient and dividing the product by the quota on mortality effect.