Explorations in Economic History 64 (2017) 62–81

Contents lists available at ScienceDirect

Explorations in Economic History

journal homepage: www.elsevier.com/locate/eeh

Early-life disease exposure and occupational status: The impact of yellow fever during the 19th century☆

Martin Saavedra

Department of Economics, Oberlin College, Rice Hall 227, 10 N. Professor St., OH 44074, USA

ARTICLE INFO

JEL classification:
N31
I14
J24

Keywords:
Fetal origins
Early childhood
Yellow fever
Occupation
Urban mortality penalty

ABSTRACT

Using city-of-birth data from the 100% sample of the 1880 Census merged to city-level fatality counts, I estimate the relationship between early-life yellow fever exposure and adult occupational status. I find that white males with immigrant mothers were less likely to become professionals and more likely to become unskilled laborers or report occupational nonresponse if they were born during yellow fever epidemics. They also reported occupations with lower 1900 occupational income scores. The children of U.S.-born mothers (who were less susceptible to the disease) were relatively unaffected. Furthermore, I find no evidence that epidemics 3 to 4 years after birth affect adult occupational status, and the results are robust to controlling for local trade during an individual's birth year.

1. Introduction

The fetal origins hypothesis posits that *in utero* nutrition can predict heart disease in adulthood (Barker, 1995). Health economists have linked prenatal, neonatal, and postnatal disease exposure to worse labor-market outcomes (see Almond and Currie (2011), for a review of this literature). This research argues that early-life health shocks have permanent effects on human capital development. Consequently, disparities in early-life disease exposure might cause economic disparities a generation later. This research has focused mainly on the effects of influenza (Almond, 2006), malaria (Barreca, 2010), or famine-induced malnutrition (Almond et al., 2010; Neelsen and Stratmann, 2011; Chen and Zhou, 2007).

This study considers how early-life environment affects adult occupational outcomes by focusing on an epidemic disease that plagued southern port cities: yellow fever. Yellow fever caused approximately 100,000–150,000 deaths in American port cities during the nineteenth century (Patterson, 1992). The disease was responsible for the single largest city-level epidemic in American history when in 1853 8,000 New Orleanians (approximately 6% of the city) died of yellow fever. Today, there are approximately 200,000 cases of yellow fever a year leading to 30,000 deaths worldwide; the number of cases is increasing (WHO, 2014).

Beyond examining a new disease, the contribution of this study is twofold. First, native southerners often acquired a mild form of the disease during childhood and would have become immune for life. Blacks were also less prone to yellow fever.[1] Consequently, yellow fever primarily affected European immigrants, earning yellow fever the title of the “Stranger's Disease” (Pritchett and Tunali,

☆ I want to thank Erick Gong, Greg Niemesh, Seth Richards-Shubik, Raul Sanchez, Allison Shertzer, Werner Troesken, and Randall Walsh for helpful comments. I also want to thank participants at the Cliometric Society session at the 2015 ASSA meeting, the Middlebury College Economic History Workshop, the 2015 Southern Economics Association meeting, and seminar participants at the University of Pittsburgh. I also want to thank two anonymous reviewers and the Editor. Alokik Mishra provided excellent research assistance. All errors are mine.

E-mail address: Martin.Saavedra@oberlin.edu.

[1] Motulsky (1989) argues that blacks had lower fatality rates because of a genetic resistance to the disease, whereas Espinosa (2014) argues that southern blacks acquired immunity when exposed to yellow fever epidemics during childhood.

http://dx.doi.org/10.1016/j.eeh.2017.01.003
Received 25 January 2016; Received in revised form 2 January 2017; Accepted 30 January 2017
Available online 01 February 2017

1995). Most studies on the long-run effects of early-life disease exposure compare cohorts *in utero* during an epidemic to adjacent birth cohorts or to birth cohorts born in nearby cities or states. By examining how yellow fever epidemics differentially affected the children of immigrant mothers, I am able to disentangle the effects of yellow fever from other city-wide shocks that coincide with the epidemic.

Second, many studies examining how prenatal, neonatal, and postnatal disease exposure affects adult labor-market outcomes focus on epidemics from the early twentieth century. From 1880 to 1920, the U.S. economy was growing and health was generally improving. Cities installed water and sewer systems to eradicate infectious disease (Alsan and Goldin, 2015; Cutler and Miller, 2005). During the mid-nineteenth century, height and life expectancy were declining despite growing incomes (Costa and Steckel, 1997). Since the relationship between income and health may have fundamentally changed between the mid-nineteenth century and the early twentieth century, it is possible that the relationship between early-life disease exposure and adult labor market outcomes also changed. Few studies provide evidence from the mid-nineteenth century that prenatal, neonatal, and postnatal disease exposure had long-run effects on labor market outcomes.[2]

Yellow fever epidemics struck suddenly, killing many city dwellers and infecting many others. These epidemics happened unpredictably, in some years killing thousands of citizens and in other years leaving cities untouched. For example, in New Orleans, LA, yellow fever killed 17 residents in 1851, 456 in 1852, and 7,849 in 1853 (Toner, 1873). Consequently, New Orleanians born between 1851 and 1853 likely grew up in similar neighborhoods and in similar families, but they faced different disease environments during early life. The sporadic and unanticipated nature of yellow fever increases the likelihood that these epidemics were uncorrelated with unobservable variables that might affect human capital development, which would imply that the reduced form estimates take on a causal interpretation.

I identify males in the 1880 Census who were born in one of nine U.S. cities: Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. I then merge this data with city/year level fatality counts. Using an ordered probit model, I find that white males who were born to immigrant mothers during yellow fever epidemics entered lower status occupations than whites with immigrant mothers born during non-epidemic years. The results suggest that whites who were born to immigrant mothers during the 1853 yellow fever epidemic in New Orleans were 12 percentage points less likely to report a professional occupation (e.g., physician or lawyer). Furthermore, an epidemic during an individual's birth year does not predict occupational status for blacks or whites with U.S.-born mothers. I find some evidence that local yellow fever fatality rates, not only during an individual's birth year, but also epidemics one to two years after birth predict lower occupational status, whereas epidemics three to four years after an individual's birth year do not. Additionally, I estimate models in which the dependent variable is average income or average months unemployed by occupation. I find that yellow fever exposure during year of birth induced the children of immigrant mothers to enter lower-paying occupations, but they were no more likely to enter occupations with high unemployment rates. These results are robust to controlling for local trade levels during an individual's birth year.

In a seminal paper, Almond (2006) analyzed the 1918 influenza pandemic as an exogenous shock to fetal health. Almond compares cohorts who were *in utero* during the pandemic to those who were *in utero* the year before or the year after the pandemic. He uses cross-state variation in the severity of the pandemic and finds evidence that *in utero* influenza exposure reduced educational attainment and wages. Using data from Taiwan, Lin and Liu (2014) find that those *in utero* during the 1918 influenza pandemic were shorter, less educated, and sicker. Much of the fetal origins research on the 1918 influenza pandemic attributes the long-run consequences to influenza exposure. However, the increased mortality likely affected the economy as well. Karlsson et al. (2014) finds that poorhouse rates and capital returns were lower following the 1918 influenza epidemic in Sweden. By exploiting differences in yellow fever susceptibility related to nativity, I can rule out that the results are driven by city-wide economic responses affecting all inhabitants. Barreca (2010) investigates the effect of early-life malaria exposure on adult labor-market outcomes. Barreca uses historical temperature data as a source of exogenous variation in malaria death rates. Changes in temperature affect the population of mosquitoes, which are the malaria vector. He finds that *in utero* and postnatal malaria exposure worsened labor-market outcomes. Unfortunately, temperature data from southern port cities does not date back to the 1850s, precluding me from taking this approach. Case and Paxson (2005) find that disease environment during age two has the most significant effects on cognition at elderly ages. Beach et al. (2014) find that eradicating typhoid fever would have increased educational attainment by 1 month and incomes by 1%. Bleakley (2007) finds that eradicating hookworm in children increased school enrollments, school attendance, and literacy. Costa (2000) finds that the decline in infectious disease during childhood can explain part of the decline in chronic health conditions during older age among Union Army veterans. To my knowledge, this study is the first study to link yellow fever epidemics during childhood to adult outcomes.

2. Yellow fever in the United States

Yellow fever is an acute viral infection that spreads to humans through the *Aedes aegypti* mosquito. The mosquito contracts yellow fever after feeding on an infected primate and spreads the disease by later feeding on uninfected primates. Generally, human-to-human contact cannot spread yellow fever. Because mosquitoes are the yellow fever vector and are active mostly in summer, all yellow fever epidemics occurred during the summer months and ended by the first frost of the year. Symptoms of mild infections

[2] Hong (2013) finds that malaria exposure during childhood predicts whether Union Army veterans were still working in 1900, but due to data limitations this study cannot distinguish between malaria exposure between birth and age 10.

Table 1
Yellow fever outbreaks by city.

City	State	1668–1873	Post-1835	
		Number of outbreaks	Number of outbreaks	Outbreaks with more than 100 victims
Mobile	AL	28	16	6
New Haven	CT	6	0	0
Wilmington	DE	2	0	0
Pensacola	FL	22	15	0
Savannah	GA	9	4	1
New Orleans	LA	66	32	21
Baltimore	MD	14	1	0
Boston	MA	10	1	0
Natchez	MS	13	7	2
St Louis	MO	2	2	0
New York City	NY	62	14	0
Wilmington	NC	3	1	1
Cincinnati	OH	2	2	0
Philadelphia	PA	34	3	1
Providence	RI	5	0	0
Charleston	SC	52	15	7
Memphis	TN	4	4	1
Galveston	TX	10	10	9
Norfolk	VA	18	4	1

Notes Data are from Toner (1873). An outbreak includes any reported incidence of yellow fever.

include fever, headaches, nausea, and vomiting. Some of the infected enter the toxic phase of the disease. Symptoms of the toxic phase include liver damage leading to jaundice, bloody vomit, and sometimes death. Patients with mild infections tend to improve in less than one week, whereas survivors of the toxic phase of the disease recover in approximately two weeks (WHO, 2014). Children tended to get milder versions of the diseases while those during their peak childbearing years (ages 20–30) were at the greatest risk (Pritchett and Tunali, 1995; Patterson, 1992).

Carlos Finlay first hypothesized that mosquitoes were the yellow fever vector in 1881. Walter Reed confirmed Finlay's hypothesis, and in 1905, cities eradicated yellow fever by controlling the mosquito population. Yellow fever epidemics were limited to urban areas during the 19th century in the United States. The *Aedes aegypti* breed in standing freshwater located on hard surfaces, making urban cities an effective breeding ground.

After acquiring the disease, survivors were generally immune for life. The *Aedes aegypti* needed to infect previously uninfected primates to spread the disease. Consequently, cities with strong immigration experiencing economic booms and robust trade were particularly susceptible to yellow fever. The disease rarely visited the countryside. Mosquito-borne illnesses that thrived in the countryside, such as malaria, did not confer life-long immunity and consequently mosquitoes could reinfect the same hosts (Humphreys, 2001).

Yellow fever first appeared in the United States in 1693 in Boston, MA. Many port cities on the Atlantic experienced yellow fever epidemics. Boston, MA, New York, NY, Philadelphia, PA, Norfolk, VA, and Charleston, SC, experienced outbreaks during the early 1800s. These epidemics claimed hundreds or even thousands of victims. For example, yellow fever took the lives of 5,000 Philadelphians in 1793 (Toner, 1873). After 1835, trade ships were more likely to stop in southern port cities such as New Orleans, LA, Mobile, AL, Charleston, SC, and Norfolk, VA, and less likely to continue to Philadelphia, PA, New York, NY, or Boston, MA. These trade ships brought mosquitoes and yellow fever with them.

Table 1 displays the number of outbreaks by city between 1668 and 1873, the number of outbreaks between 1835 and 1873, and the number of post-1835 outbreaks that killed at least one hundred inhabitants.[3] New Orleans was the city most affected by yellow fever during the mid-nineteenth century. New Orleans had twice as many outbreaks as any other city, and more than twice as many outbreaks resulting in at least 100 deaths.

White immigrants were at the greatest risk of contracting yellow fever, whereas blacks and native whites were less prone to the disease. For example, during the 1854 epidemic in Charleston, SC, 96.1% of fatalities were white and 72.9% were immigrants (Patterson, 1992). Yellow fever took the lives of so many immigrants that it earned the name the “stranger's disease.” In 1808 in St. Mary's, GA, yellow fever took the lives of 42 of the town's 350 whites, while only taking three of the town's 150 blacks (Patterson, 1992). Epidemic yellow fever during the nineteenth century had a case fatality rate between 15 and 50% (Patterson, 1992) implying that if 8% of a city died of yellow fever, then at least another 8% were infected and survived.[4]

Because city officials did not know what caused yellow fever, cities tried various measures to stop the disease. American cities

[3] The data are from Toner (1873) and are described in more detail in the next section. An outbreak here is any reported incidence of yellow fever. Small outbreaks of a dozen or fewer fatalities were likely from the crew of a single infected ship.

[4] Patterson (1992) argues that the true number was probably closer to 15 than 50, as health boards would have been more likely to under report survivors than the dead.

created health boards with the authority to quarantine ships from infected ports and order street cleanings (Duffy, 1992). Physicians claimed that the disease only struck the "intemperate" and "imprudent." Public notices warned that excessive drinking or eating and poor personal hygiene caused the disease. Believing that immoral behavior caused pestilence, politicians frequently called for prayer, repentance, and days of fasting. Although yellow fever could kill 10% of a city, Beeson and Troesken (2006) find that yellow fever and small pox epidemics had little to no effect on long-term population growth or on trade, suggesting that any stoppage in economic activity was temporary.

The first person to my knowledge to argue that pregnant women might spread yellow fever to their fetuses was Dr. Joseph Jones in an 1894 JAMA article. Dr. Jones' evidence came from the case of a yellow fever patient at Charity Hospital in New Orleans in which a woman presented symptoms of yellow fever (nausea, vomiting, and jaundice). Shortly afterward, she gave birth to a jaundiced still-born fetus. A few days later, the woman died of yellow fever. Dr. Jones also noted several similar cases with smallpox. The modern research regarding yellow fever, pregnant women, and their fetuses has focused on reactions to the yellow fever vaccine. Nasidi et al. (1993) found that pregnant women who received the yellow fever vaccine during pregnancy had lower antibody responses relative to other women. Tsai et al. (1993) document a case in which a pregnant woman received the yellow fever vaccine and yellow fever antibodies were present in the infants blood suggesting fetal infection of the yellow fever vaccine virus. Cavalcanti et al. (2007) study 304 infants who were prenatally exposed to the yellow fever vaccine. Those exposed to the vaccine were no more likely to have major malformations, but there was a statistically significant increase in the rate of minor dysmorphisms relative to a comparison group. Several case studies suggest that lactating women who received the yellow fever vaccine transmitted the yellow fever vaccine virus to their children through breastfeeding (Center of Disease Control, 2010; Kuhn et al., 2011).

3. Data

3.1. Fatality data

Yellow fever fatality count data are from Toner (1873).[5] J.M. Toner, at the time President of the American Medical Association, pooled several sources from his medical library to document every yellow fever epidemic in the United States for which data was available. This included cities in which yellow fever "appeared and prevailed, either in epidemic form or in sporadic or imported cases since the settlement of our country." Toner constructed this data to analyze the geography in which yellow fever spreads with a particular focus on elevation (Toner argued that the disease rarely visited towns with elevations above 500 feet). Toner's data appears to be complete after the mid-1830s, and there are few subsequent epidemics with missing fatality counts. Beeson and Troesken (2006) use Toner's data to analyze the effect of yellow fever epidemics on city population growth. The Toner data is also highly correlated with other historical sources. The historian K. David Patterson compiled a list of notable yellow fever epidemics in the United States from twelve historical sources (Patterson, 1992). For Charleston, Mobile, New Orleans, Norfolk, and Savannah during the period of this study, the correlation between fatality counts in the Toner and Patterson data is 0.98. I use the Toner data instead of the Patterson data because the Toner data includes some minor outbreaks missing from the Patterson data (typically fewer than 50 fatalities). Years without recorded epidemics in the Toner data are recorded as zeros.

Without modern technology, diagnosing an individual with yellow fever would not have been without controversy. Physicians would have looked for the classic yellow fever symptoms of jaundicing of the skin and black vomit, as well as the absence of symptoms that accompany other fevers (e.g., rashes and diarrhea). However, diagnosing a city with yellow fever was an easier task for 19th-century observers. Other fevers such as malaria would not have been capable of generating the large epidemics with high case-mortality rates (Patterson, 1992).

I convert fatality counts to fatality rates under the assumption that cities grow linearly between Census years. Fig. 1 displays time-series yellow fever fatality rate data for New Orleans, LA, Charleston, SC, Mobile, AL, and Norfolk, VA. The data from Fig. 1 suggest that yellow fever appeared unexpectedly. The yellow fever fatality rate in one year does not predict the absence or presence of an epidemic in the next year. Table 2 demonstrates this fact by regressing contemporaneous yellow fever fatality rates on lagged yellow fever fatality rates for Charleston, Mobile, New Orleans, Norfolk, and Savannah using data from 1835 to 1864. The first column does not include control variables, the second column includes a dummy for each city and a linear time trend, and the third column includes a dummy for each city and set of dummy variables for each year. Lagged yellow fever fatality rates do not predict contemporaneous yellow fever fatality rates in any of the regressions. The smallest p-value is 0.23.

3.2. 1880 census

The micro occupational data are from the 100% sample of the 1880 Census available in the Integrated Public Use Microdata Series (Ruggles et al., 2010). I restrict attention to white and black males born between 1835 and 1864 because labor force participation is nearly universal for this group in 1880.[6] Normally, only state of birth is available in the IPUMS. However, in the 1880 Census the alphabetic birthplace string is available. While Census enumerators were instructed to record state of birth or territory of birth if an individual was born outside of the U.S., not all enumerators followed these instructions exactly. A subset of enumerators recorded city of birth. In the 100% sample of the U.S. Census, enough enumerators made this mistake to create a data

[5] Replication and data files are available online (Saavedra, 2017).
[6] The restriction on race drops seven Native American and seven Chinese observations from the sample.

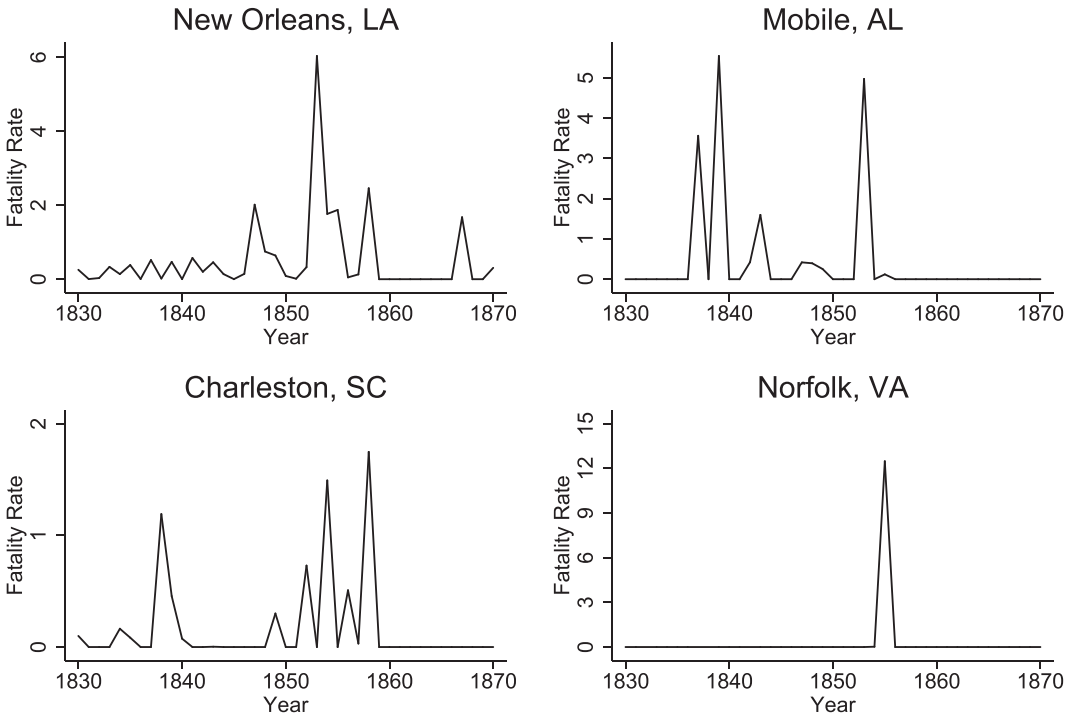

Fig. 1. Yellow fever fatality rates.**Notes:** Data are from Toner (1873). I convert fatality counts into fatality rates by assuming city population grows linearly between Census years. The fatality rates are deaths per 100 inhabitants. For example, approximately 6% of New Orleanians died of yellow fever in 1853.

Table 2
Predicting yellow fever fatality rates with lagged yellow fever fatality rates.

	(1)	(2)	(3)
Lagged yellow fever rate	−0.0101	−0.0332	−0.133
	(0.0408)	(0.0412)	(0.110)
City dummies	N	Y	Y
Linear time trends	N	Y	N
Year dummies	N	N	Y
R^2	0.0001	0.0232	0.286
N	145	145	145

Notes: Data come from Toner (1873). The sample includes New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, and Savannah, GA from 1835 to 1864. Robust standard errors are in parentheses.
* 10% significance; ** 5% significance; *** 1% significance.

set of males born in large U.S. cities.

We would expect fewer males to report being born in smaller cities, as there would have been fewer births in those cities, and smaller cities may have been less recognizable to Census enumerators. The log of the number of people reporting a particular birth city in 1880 is approximately proportional to the log of that city's population in 1850. The scatter plot in Fig. 2 displays this fact. A regression of the log of a city's 1850 population on the log of working-age males reporting being born in that city in the 1880 Census yields an R^2 of 0.81. Consequently, smaller cities with populations below 15,000 would contribute too little to the sample size to affect the results. For this reason, I limit the analysis to the 50 largest cities in 1850. This restriction includes the cities most affected by yellow fever (New Orleans, Charleston, Mobile, Norfolk, and Savannah), but eliminates some smaller ports (e.g., Galveston and Natchez).

Since yellow fever rarely visited inland cities, I further restrict the sample to cities on the Gulf Coast, Atlantic, or on major rivers or bays connecting to the Atlantic. J.M. Toner noted that yellow fever “has thus been carried to many of the cities and towns on the sea-coast, it has, fortunately, never extended far into the interior of our country, or remote from the water highways” (Toner, 1873). Lastly, the most northern outbreak resulting in at least 100 deaths during the period was Philadelphia. Consequently, I drop all cities north of Philadelphia. These sample restrictions leave nine U.S. cities: Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. I searched Census records in which the enumerator included the individual's city of birth, allowing for misspellings and variations in punctuation. Yellow fever visited Charleston, Mobile, New Orleans, Norfolk, Philadelphia, and Savannah during the sample period. Including cities free of yellow fever allows me to estimate control variables with greater precision.

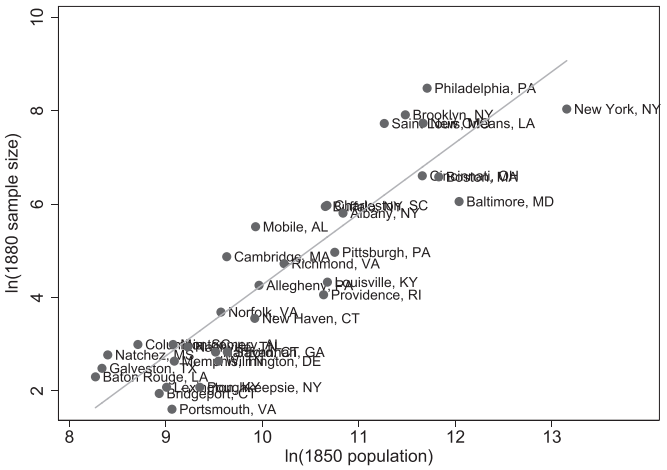

Fig. 2. 1850 city size and frequency of birth city in 1880.**Notes:** The sample consists of working-age males (ages 20–60) from the complete count of the 1880 Census who reported being born in a city.

Although a subset of enumerators may have mistakenly included city of birth, these mistakes were not limited to a narrow geographic region. To see this, the top panel of Fig. 3 maps the geographic distribution of males born between 1835 and 1864 who reported being born in New Orleans. Most individuals still reside in Louisiana in 1880. However, many moved to New York, Pennsylvania, Missouri, and California. Additionally, most states are represented, but in smaller numbers. For comparison, the bottom panel of Fig. 3 maps the geographic distribution of males who reported being born in Washington, D.C. Since Washington, D.C. is both the city of birth and state of birth, the D.C. distribution is what ideal city of birth data would resemble. For the D.C. data, most individuals stayed within the region (D.C., Maryland, and Virginia). Significant numbers moved to New York, Illinois, Pennsylvania, Missouri, and California. Additionally, most states are represented. An alternative to exploiting the mistakes of enumerators would be linking individuals across Censuses. However, the merged samples from IPUMS do not yield enough observations born during yellow fever epidemics.

Another challenge with the 1880 micro data is that enumerators recorded age as of June 1, 1880 and not birth year. IPUMS infers birth year by taking 1880 minus the reported age, which will be off by one year for some individuals. During the nineteenth century, some individuals misreported their age, rounding to the nearest age ending with a zero or five. This should introduce measurement error into the model and bias the estimates towards zero. For those with correctly reported ages, we can put bounds on

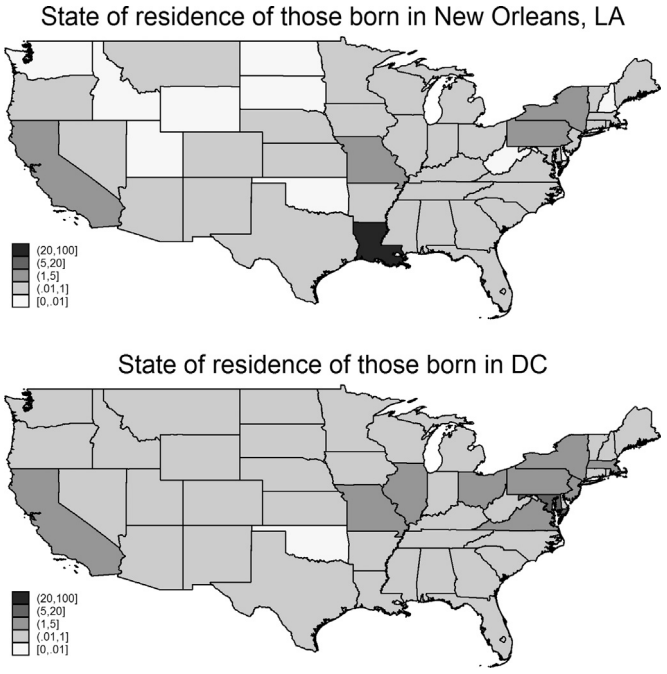

Fig. 3. State of residence in 1880 by birth city.**Notes:** Data are from the 100% sample of the U.S. Census and includes all males born between 1835 to 1864 who reported being born in either New Orleans, LA, or Washington, D.C.

Table 3
Summary statistics.

Variable	Mean	std. dev.	min.	max.
Yellow fever fatality rate	0.068	0.467	0	12.485
Immigrant mother	0.287	0.452	0	1
Immigrant father	0.314	0.464	0	1
Black	0.195	0.397	0	1
Birth year	1853.34	7.939	1835	1864
Professional	0.252	0.434	0	1
Skilled laborer	0.38	0.485	0	1
Unskilled laborer	0.267	0.442	0	1
Occupational nonresponse	0.101	0.302	0	1
N	24804			

Notes: The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864.

the age that individuals would have been exposed to yellow fever. Those born between June 2nd and December 31st would have been born one year before their IPUMs inferred birth year. Since yellow fever epidemics only occurred during the summertime and ended by the first frost of the year, such epidemics would have typically only occurred between early June and late October. Consequently, an individual with an IPUMs inferred birth year occurring during an epidemic would have been at least 0 months old (those born on June 1st and experiencing an early summer epidemic) and at most 17 months old (those born on June 2nd of the previous year and experiencing an epidemic in late October). Similarly, those with inferred birth years the year before an epidemic would have been between 12 months and 29 months during exposure to the disease. Consequently, those with a year of birth corresponding to a yellow fever epidemic were most likely exposed during the neonatal and postnatal periods.

These sample restrictions result in 24,804 observations. Summary statistics appear in Table 3. The sample includes 17,234 males born in Washington, D.C. Washington is highly represented because even enumerators who followed instructions recorded birth city. The sample also includes 4,355 males born in Philadelphia, and 2,169 born in New Orleans. No other city has more than 500 males. The average birth year is 1853.3. Individuals with a foreign-born mother and a foreign-born father comprise 29% and 31% of the sample, respectively. Twenty percent of the sample is black. The occupational categories include occupational nonresponse (10%), unskilled laborers (27%), skilled laborers (38%), and professionals (25%).

Because only Census records with city of birth are included in the sample, this sample is not necessarily representative of males born in cities. However, for this to bias the estimates of the effects of early-life disease exposure, occupational status would have to be biased in the opposite direction for those born during epidemic years and for those born during non-epidemic years. Although this kind of bias seems unlikely, I cannot rule it out.

4. Identification

Because neither income nor educational attainment are available in the 100% sample of the 1880 Census, I focus on the effect of disease exposure during year of birth on occupational choice. Occupational data is categorical; consequently, I use an ordered probit model. Suppose occupational categories are ordered from lowest to highest as follows: occupational nonresponse, unskilled laborers, skilled laborers, and professionals. Although this ordering will not be perfect (for example, the highest paid skilled laborers likely earned more than the lowest paid professionals), it should capture socioeconomic status on average. I include occupational nonresponse as the lowest category because this group would have been without any occupational earnings. Because the sample is restricted to males born after 1835, the nonresponse category will not include any women and should include few retirees. Dummies for each birth cohort will control for the fact that younger cohorts may not have completely entered the labor force yet.

Furthermore, suppose that o^*_{ibc} is a latent occupational index variable for individual i born during birth year b in city c, and is defined by

$$o^*_{ibc} = \alpha_b + \beta_c + \gamma Y_{bc} + X'_{ibc}\theta + \epsilon_i \quad (1)$$

where α_b is a set of dummy variables for each birth year and β_c is a set of dummy variables for each birth city. The explanatory variable Y_{bc} is early-life yellow fever exposure and is the yellow fever fatality rate in individual i's birth year b and birth city c. The vector X_{ibc} is a set of control variables containing dummy variables for each birthplace of individual i's mother, the population of city c during year b, and race.[7] The mother's birthplace is her state of birth if she was born in the U.S. or her country of birth if she was foreign-born. The term ϵ_i is assumed to be distributed according to the standard normal distribution. Because yellow fever epidemics occurred unpredictably, I assume that Y_{bc} is independent of ϵ_i.

Individual i enters occupational category j (which is to say $o_{ibc} = j$) if $\mu_{j-1} < o^*_{ibc} \leq \mu_j$. It follows that

[7] Population is assumed to grow linearly between Census years.

$$Pr[o_{ibc} = j] = \Phi\left(\mu_j - \alpha_b - \beta_c - \gamma Y_{bc} - X'_{ibc}\theta\right) - \Phi\left(\mu_{j-1} - \alpha_b - \beta_c - \gamma Y_{bc} - X'_{ibc}\theta\right) \quad (2)$$

where Φ is the CDF of the standard normal distribution.

One interpretation of this model is to view o^*_{ibc} as unobservable ability plus an error term. High occupational status is an increasing function of ability and the error term. Early-life disease exposure reduces ability, and consequently moves the marginal individual into lower earning occupational categories.

I also estimate several variations of Eq. (2). The first allows the effect of yellow fever for whites with foreign-born mothers, who were more susceptible to yellow fever, to be different from those with U.S.-born mothers.[8] The latent index variable becomes

$$o^*_{ibc} = \alpha_b + \beta_c + \gamma_0 Y_{bc} + \gamma_{Imm} Y_{bc} \times \mathbf{1}[\text{immigrant mother}] + \gamma_{Black} Y_{bc} \times \mathbf{1}[\text{black}] + X'_{ibc}\theta + \epsilon_i, \quad (3)$$

or similarly

$$o^*_{ibc} = \alpha_b + \beta_c + \gamma_{Imm} Y_{bc} \times \mathbf{1}[\text{immigrant mother}] + \gamma_{Black} Y_{bc} \times \mathbf{1}[\text{black}] + \gamma_{Nat} Y_{bc} \times \mathbf{1}[\text{U.S.}-\text{born mother}] + X'_{ibc}\theta + \epsilon_i \quad (4)$$

where $\mathbf{1}$[immigrant mother] is an indicator variable that is equal to 1 if the individual's mother was born outside of the United States, and $\mathbf{1}$[black] is an indicator variable that is equal to 1 if the individual is black. The indicator variable $\mathbf{1}$[U.S.−born mother] refers to whites with U.S.-born mothers. Because all observations were born in one of nine U.S. cities, there are no immigrants in the sample; however, some were born into immigrant families. The coefficient γ_{Imm} in Eq. (3) tests the null hypothesis that the children of foreign-born mothers were affected as much as the children of U.S.-born mothers, whereas γ_{Imm} in Eq. (4) tests the hypothesis that the effect of yellow fever on the children of immigrants is statistically different from zero. Similarly, the coefficient γ_{Black} tests whether blacks born during yellow fever epidemics were negatively affected. Since blacks were relatively immune to the disease, if γ_{Black} is not statistically significant, then it is unlikely that the results are driven by responses to yellow fever that negatively affected low socioeconomic status residents. It should be noted that most blacks in the sample would have been born slaves and may have been differentially affected by various human capital shocks. For this reason, I also present specifications restricting the sample to only whites. Both equations fit similar models, but have different interpretations.[9]

Another specification includes yellow fever fatality rates during an individual's year of birth, as well as the year before birth and the four years after birth. As in Eq. (4), for this specification, I interact the variables with an indicator variable equal to one if an individual is white with an immigrant mother. The coefficients in an ordered probit model do not have an easy interpretation beyond sign and significance, so I report the marginal effect on the probability of entering specific occupational categories $\frac{\partial Pr[o_{ibc}=j]}{\partial Y_{bc}}$. I focus on the probability of entering a professional occupation so that negative marginal effects can be interpreted as decreases in occupational status.

I will also analyze models in which the dependent variable is the average income or average months unemployed for each occupation in 1900. The models use the same set of regressors but with non-categorical dependent variables. The standard errors I report are robust to heteroskedasticity, but are not clustered. Standard errors clustered at the state of residence, birth city, birth year, or birth year by birth city were slightly smaller.

The identifying assumption is that anything unobservable occurring during year of birth, conditional on city, equally affected the children of immigrants and the children of U.S.-born mothers, except for the yellow fever epidemic. There are several possible threats to this identifying assumption. First, yellow fever epidemics were typically imported on trade ships. If local trade activity differentially affected immigrant families, the estimates could be biased. For this reason, I control for local trade activity as a robustness check. However, I cannot rule out that there was a temporary stoppage in economic activity that occurred through channels other than trade and that this temporary stoppage disproportionately affected immigrants. Since I find no evidence that blacks were negatively affected by yellow fever, this limits the possibility that the estimates are confounded by shocks only affecting low socioeconomic-status individuals.

Another threat to my identification strategy is that yellow fever may have occurred during seasons with particular weather patterns, and these weather patterns may have differentially affected poorer families through either nutrition or other mosquito-borne illnesses. Yellow fever may be confounded by malaria exposure, because the vectors of both diseases are mosquitoes (although not the same species). Union Army veterans from counties with the highest risk of malaria were shorter and more prone to infection (Hong, 2007). Similarly, Barreca (2010) uses temperature as an instrumental variable for malaria exposure and finds that early-life exposure to malaria decreases adult incomes. The adverse effects of childhood malaria exposure can last until old age (Hong, 2013).[10]

The city fixed effects explicitly control for the fact that yellow fever is more likely to occur in southern cities with warm summers. If a yellow fever epidemic occurred because of weather patterns that affected all cities in the Southeast/mid-Atlantic, then that would be controlled for by the year fixed effects. Unlike yellow fever, malaria rarely provided complete immunity to those previously exposed. Rather, those repeatedly exposed to malaria acquired partial immunity, lessening the course of the disease (Humphreys,

[8] The results are similar if we replace the dummy variable for having a immigrant mother with a dummy variable for having an immigrant father. The correlation between the two variables is 0.82.

[9] The models would be mathematically identical if all blacks had U.S.-born mothers. All but 36 blacks in the sample had U.S.-born mothers.

[10] Malaria and yellow fever primarily affected the same regions (the Gulf Coast and the Southeast Atlantic). I am unaware of any studies demonstrating a correlation between the timing of yellow fever and malaria epidemics within the same region. However, studies have shown that weather plays a role in predicting both diseases (Diaz and McCabe, 1999). Malaria was less sporadic than yellow fever and rarely disappeared completely (see Fig. 2 in Barreca (2010)).

2001). If the results from this paper were driven by exposure to malaria, we would expect to see some long-run adverse effects for the children of natives. Nonetheless, I cannot rule out that some of the results are confounded by malaria exposure or weather patterns.

There are several possible mechanisms through which yellow fever may have negatively impacted labor market outcomes. The children of immigrants may have been directly exposed to the virus during gestation and lactation. Alternatively, the disease may have caused stress to the mother during pregnancy, perhaps because the epidemic took the life of a family member. Persson and RossinSlater (2016) find that those who were *in utero* when their mother experienced a familial death were more likely to take medication for depression or ADHD later in life; Black et al. (2016) find that the death of the mother's parents during pregnancy decreases birth weight, but they find no evidence of long-run effects on labor market outcomes. Additionally, the children of immigrant mothers were more likely to be orphaned. I find evidence that yellow fever epidemics during the year of birth or the year after birth reduce occupational status. If the long-run effects of orphanhood are larger during the postnatal period, this could explain these results. Similarly, it is possible that sick parents were less able to care for their children during the critical window of infancy and that lack of care had long-run effects on labor market outcomes. The estimates in this study will not be able to distinguish these mechanisms, and thus represent the sum of these effects. For this reason, the estimates should not be interpreted as the effect of contracting the disease, but rather the effects of being born during a yellow fever epidemic.

5. Results

5.1. Ordered occupational category results

Estimates from Eqs. (2)–(4) appear in Table 4. The first three columns include the full sample, and the second three columns restrict the sample to whites. The displayed estimates are the marginal effect on the probability of entering a professional occupation. The main effect of yellow fever exposure during year of birth is negative but statistically insignificant for the full sample. The point estimate of the marginal effect implies that being born during a yellow fever epidemic that killed one percent of the city decreased the probability of entering a professional occupation by approximately 0.8 percentage points. Columns (2) and (3) display marginal effects from Eqs. (2) and (3), which allow for the effect of yellow fever to vary by race and nativity. The results suggest that blacks and the children of U.S.-born mothers were relatively unaffected. The estimated coefficient for those with U.S.-born mothers is close to zero and statistically insignificant. However, a yellow fever epidemic that killed one percent of the city decreased the probability of entering a professional occupation by about 2 percentage points for the children of immigrants. This estimate is statistically different from zero at the one percent level, and statistically different from the effect of yellow fever on natives at the ten percent level. The results are similar when restricting the sample to whites, except that the main effect of yellow fever is now statistically significant at the ten percent level. These results suggest that being born during a yellow fever epidemic that killed one percent of the city decreased the probability that whites entered a professional occupation by 1.3 percentage points. Columns (5) and (6) show that this effect is driven by whites with immigrant mothers, decreasing the probability that they entered professional occupations by approximately 2 percentage points.

Fig. 4 displays the predictive margins from this specification for entering each occupational category. In this case, the predictive

Table 4
The marginal effect of early-life yellow fever exposure on the probability of entering a professional occupation.

	Full sample			Whites only		
	(1)	(2)	(3)	(4)	(5)	(6)
Y_b	−0.0078	0.0028		−0.0130*	0.0002	
	(0.0054)	(0.0099)		(0.0069)	(0.0113)	
Y_b×**1** [immigrant mother]		−0.0216*	−0.0187***		−0.0217*	−0.0215***
		(0.0117)	(0.0071)		(0.0131)	(0.0078)
Y_b×**1** [black]		−0.0010	0.0019			
		(0.0117)	(0.0076)			
Y_b×**1** [U.S.-born mother]			0.0035			0.0002
			(0.0099)			(0.0113)
N	24804	24804	24804	19957	19957	19957

Notes: The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth. Each coefficient is the marginal effect of being born during a yellow fever epidemic that killed 1% of the city on the probability of entering a professional occupation. Robust standard errors are in parentheses.

* 10% significance.

** 5% significance.

*** 1% significance.

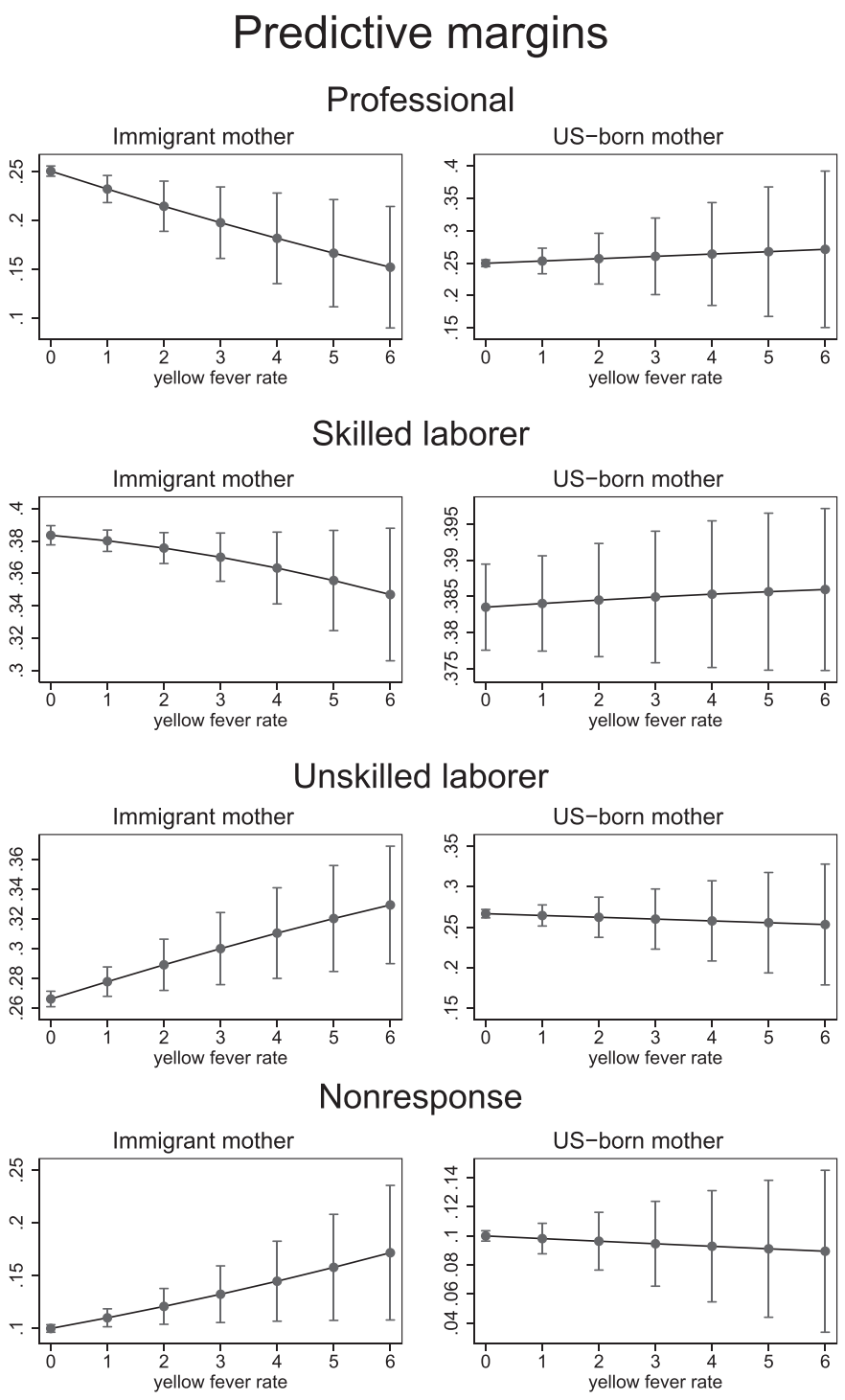

Fig. 4. The effect of yellow fever.**Notes:** Predictive margins are from the estimates in the top panel of Table 4. The predictive margins indicate the predicted probability of entering a professional occupation given a specified level of early-life yellow fever exposure holding all other covariates fixed. The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Yellow fever fatality rates are deaths per 100 people. The confidence intervals are at the 95% level.

margins indicate the predicted probability of entering a professional occupation given a specified level of early-life yellow fever exposure holding all other covariates fixed. Children born during yellow fever epidemics to immigrant mothers are less likely to be professionals, and more likely to be unskilled laborers or to not report an occupation. As the size of the epidemic increases, both the

lower and upper bounds of the 95% confidence intervals are strictly decreasing for professional occupations and strictly increasing for unskilled laborers and occupational nonresponse. However, children born during yellow fever epidemics to U.S.-born mothers are unaffected. The point estimate of the predictive margins hardly change with exposure to yellow fever during an individual's birth year. Furthermore, for the children of U.S.-born mothers, the point estimate for each occupational category when there is no epidemic (a 0% fatality rate) is contained in the 95% confidence interval for an epidemic that takes 6% of the city. The same cannot be said for the children of immigrant mothers.

5.2. 1900 occupational income score results

In the previous subsection, I assume occupations are ordered. Although this should measure socioeconomic status on average, it has some problems. For example, this approach treats professionals with high-earnings potential (e.g., lawyers and judges) the same as professionals with low-earnings potential (e.g., salesmen). Unfortunately, occupation is the only meaningful labor-market outcome in the 100% sample of the 1880 Census. An alternative approach would be to use a measure of occupational earnings potential that maps occupations to average wages or earnings within that occupation. A common example of this is the 1950 occupational income score, which is the median income of an occupation from a 3.3% sample of the 1950 U.S. Census (Ruggles et al., 2010).

It is not clear 1950 occupational income scores would be reliable given the 1880 Census occurred 70 years before the construction of the occupational index. To my knowledge, 1880 average or median income data by occupation do not exist. However, Appendix A of Preston and Haines (1991) presents average income and average months unemployed by occupation in 1900. This data was collected from three sources: Lebergott (1964), Douglas (1930), and Wright (1904).[11] Since this data follows a similar methodology to the 1950 occupational income scores available in the IPUMS (just from smaller samples and for samples 50 years earlier), I will refer to these variables as 1900 occupational income scores and occupational unemployment scores (which is a proxy for average job security by occupation). These data do not reflect the earnings or months unemployed of a worker who was exposed to yellow fever in early life; rather the index reflects the average earnings and average months unemployed of workers who entered the same occupation as a worker who was exposed to yellow fever during early life.

I code all individuals with occupational non-response as having an occupational income score of 0, since they would have had no occupational earnings.[12] The average (standard deviation) 1900 occupational income score is $552 ($304), and the average (standard deviation) occupational unemployment score is 1.5 months (1.1 months).[13] In this section, I merge this data to the 1880 Census microdata and use a Tobit model (left censored at zero) to analyze the effects of early-life yellow fever exposure on 1900 occupational income scores. I also use a linear model in which the dependent variable is occupational unemployment score.

The results are in Table 5. Each coefficient is the average marginal effect from a Tobit model. The results suggest that those born during a yellow fever epidemic that killed one percent of the city entered occupations that earned $10 less in 1900; this estimate is statistically significant at the ten percent level. The children of immigrants born during yellow fever epidemics that killed one percent of the city entered occupations that earned $14 less in 1900. This estimate is statistically different from zero at the five percent level, but is not statistically different from the coefficient on natives. When the sample is restricted to whites, those born during a yellow fever epidemic that killed one percent of the city entered occupations earning $12 less in 1900. This effect is driven by the children of immigrants, who entered occupations earning $15 less in 1900. This estimate is statistically different from zero at the five percent level.

Table 6 tests whether those born during yellow fever epidemics entered occupations with more risk of becoming unemployed. The dependent variable is the 1900 occupational unemployment score and is the average number of months unemployed for a particular occupation in 1900. Notice that the dependent variable is not a measure of whether an individual was unemployed, but rather whether they entered an occupation in which unemployment was common. None of the coefficients are economically or statistically significant. This implies that although those born during yellow fever epidemics may have entered lower earning occupations, they did not enter occupations with high unemployment risk.

5.3. Yellow fever exposure at other ages

So far, I have only considered the effect of yellow fever epidemics during an individual's year of birth. Table 7 includes the yellow fever fatality rate during an individual's year of birth, as well as the year before birth, and the four years following birth. As mentioned in the Data section, birth year is inferred using an individual's age as of June 1, 1880. For this reason, differences in coefficients for adjacent ages should be interpreted with caution. As in Eq. (4), I interact these variables with a dummy variable indicating if an individual was born to a immigrant mother. I include these years individually (as has been done in the previous sections for the individual's year of birth) and all together. To make the table more readable, I limit the sample to whites.[14] The results suggest that yellow fever during an individual's year of birth, the following year, and two years after birth predict lower

[11] The data from Lebergott (1964) come from a variety of government reports and archival records; the data from Wright (1904) come from a Bureau of Labor Statistics survey of household heads; the data from Douglas (1930) come from Bureau of Labor Statistics reports.

[12] This approach is consistent with how IPUMS constructs its 1950 occupational income score (Ruggles et al., 2010).

[13] The average (standard deviation) 1900 occupational income score is $842 ($238) for professionals, $650 ($146) for skilled laborers, and $394 ($116) for unskilled laborers.

[14] When blacks are included in the sample, the results are similar.

Table 5
The effect of early-life yellow fever on 1900 occupational income score.

	Full sample			Whites only		
	(1)	(2)	(3)	(4)	(5)	(6)
Y_b	−9.9388*	−5.1841		−11.6532*	−7.2100	
	(5.3327)	(10.0038)		(6.4521)	(10.8418)	
Y_b×**1** [immigrant mother]		−8.9110	−14.1162**		−7.4331	−14.6432**
		(11.5699)	(6.8108)		(12.2751)	(7.0372)
Y_b×**1** [black]		−2.5165	−7.7328			
		(11.2670)	(7.0108)			
Y_b×**1** [U.S.-born mother]			−5.7211			−7.2100
			(10.0471)			(10.8418)
N	22957	22957	22957	18754	18754	18754

Notes: Each coefficient is the average marginal effect from a Tobit model. The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth. The model is a Tobit model left censored at zero. Those reporting occupational non-response are assigned an occupational income score of zero. Robust standard errors are in parentheses.

* 10% significance.

** 5% significance.

*** 1% significance.

Table 6
The effect of early-life yellow fever on occupational unemployment risk.

	Full sample			Whites only		
	(1)	(2)	(3)	(4)	(5)	(6)
Y_b	0.0019	−0.0292		0.0131	0.0005	
	(0.0159)	(0.0283)		(0.0186)	(0.0303)	
Y_b×**1** [immigrant mother]		0.0464	0.0172		0.0211	0.0215
		(0.0336)	(0.0206)		(0.0352)	(0.0211)
Y_b×**1**[black]		0.0453	0.0161			
		(0.0372)	(0.0269)			
Y_b×**1** [U.S.-born mother]			−0.0304			0.0005
			(0.0283)			(0.0303)
N	22892	22892	22892	18682	18682	18682

Notes: The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth. Robust standard errors are in parentheses.
* 10% significance; ** 5% significance; *** 1% significance.

occupational status for males born to an immigrant mother when estimating the model one year at a time. When estimating the model with all years together, only year of birth and the year after birth are statistically significant. Notice that yellow fever exposure during any age is not significant for the children of U.S.-born mothers, and only the children of immigrant mothers are negatively affected. In the appendix, I reproduce the main results for two alternative measures of early-life yellow fever exposure: (1) yellow fever fatality rates during the year after birth, and (2) the average yellow fever fatality rate during the year of birth and the year after birth.

These results suggest that yellow fever exposure has long-run impacts during the neonatal and postnatal periods. Those exposed in early childhood after the postnatal period are less affected. These results are generally consistent with the extant literature. For example, Almond (2006) finds that the long-run effects of the 1918 influenza pandemic were mostly for individuals who were *in utero*. However, Barreca (2010) finds that malaria during the *in utero*, neonatal, and postnatal periods had long-run negative effects on labor market outcomes. On the other hand, Case and Paxson (2005) find that disease environment during age two has the most significant effects on cognition. Consistent with all of these studies, I find no effect at ages 3 and 4.

Table 7
The effect of yellow fever at different ages.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
$Y_{b-1} \times \mathbf{1}$[immigrant mother]	−0.0062						−0.0007
	(0.0070)						(0.0077)
$Y_{b-1} \times \mathbf{1}$[U.S.−born mother]	−0.0133						−0.0155
	(0.0106)						(0.0109)
$Y_b \times \mathbf{1}$[immigrant mother]		−0.0215***					−0.0153*
		(0.0078)					(0.0081)
$Y_b \times \mathbf{1}$[U.S.−born mother]		0.0002					0.0033
		(0.0113)					(0.0116)
$Y_{b+1} \times \mathbf{1}$[immigrant mother]			−0.0309***				−0.0256***
			(0.0088)				(0.0091)
$Y_{b+1} \times \mathbf{1}$[U.S.−born mother]			−0.0143				−0.0136
			(0.0114)				(0.0118)
$Y_{b+2} \times \mathbf{1}$[immigrant mother]				−0.0168**			−0.0122
				(0.0079)			(0.0082)
$Y_{b+2} \times \mathbf{1}$[U.S.−born mother]				0.0020			0.0007
				(0.0132)			(0.0140)
$Y_{b+3} \times \mathbf{1}$[immigrant mother]					0.0055		0.0073
					(0.0084)		(0.0090)
$Y_{b+3} \times \mathbf{1}$[U.S.−born mother]					0.0012		−0.0008
					(0.0093)		(0.0095)
$Y_{b+4} \times \mathbf{1}$[immigrant mother]						−0.0034	−0.0033
						(0.0107)	(0.0121)
$Y_{b+4} \times \mathbf{1}$[U.S.−born mother]						0.0053	0.0081
						(0.0118)	(0.0122)
N	19957	19957	19957	19957	19957	19957	19957

Notes: The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth. Each coefficient is the marginal effect of a yellow fever epidemic that killed 1% of the city on the probability of entering a professional occupation. Robust standard errors are in parentheses.

* 10% significance.
** 5% significance.
*** 1% significance.

6. Robustness tests

6.1. Local trade

The estimates from the previous sections are unbiased if (1) we have random sampling and (2) early-life yellow fever exposure is uncorrelated with other unobservable characteristics affecting human capital development. Yellow fever was associated with robust trade and booming economic times. Trade ships from the Caribbean would bring the *Aedes aegypti* mosquito and yellow fever with them, and a growing economy would then attract previously uninfected immigrants. These two forces together could result in an epidemic. If local trade and economic conditions also affect nutrition or other human capital investments, it is possible that this is confounding the effect of yellow fever on adult occupational outcomes. Similarly, for the *Aedes aegypti* to spread the disease, local weather patterns would have to be conducive to mosquito activity. Unfortunately, I am unaware of any temperature or precipitation data at the city level dating back to the 1840s and 1850s. However, several sources for local trade data exist.

In this subsection, I test whether the results remain when controlling for local trade data. Trade data come from Albion and Pope (1939), the 1879 Statistical Abstract of the United States (United States Department of the Treasury, 1879), and DeBow (1854).

Table 8
Marginal effect of early-life yellow fever on entering a professional occupation controlling for early-life trade.

	Full sample			Whites only		
	(1)	(2)	(3)	(4)	(5)	(6)
Y_b	−0.0061	0.0054		−0.0106	0.0045	
	(0.0056)	(0.0103)		(0.0072)	(0.0120)	
$Y_b \times$ **1**[immigrant mother]		−0.0228*	−0.0174**		−0.0246*	−0.0201**
		(0.0125)	(0.0075)		(0.0141)	(0.0081)
$Y_b \times$ **1**[black]		−0.0019	0.0036			
		(0.0118)	(0.0081)			
$Y_b \times$ **1**[U.S.−born mother]			0.0061			0.0045
			(0.0103)			(0.0120)
N	24791	24791	24791	19950	19950	19950

Notes: Each coefficient is the marginal effect from an ordered probit model. The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth. Robust standard errors are in parentheses.

* 10% significance.
** 5% significance.
*** 1% significance.

These sources include nominal exports, nominal imports, and total tonnage entering a city port for most years in the sample. I convert nominal trade data into real terms, and add real exports, real imports, and entering tonnage during an individual's birth year as additional controls. In addition, since local trade activity may affect immigrant families disproportionately, I also interact each of these trade measures with whether the child has an immigrant mother.

The results from this exercise are in Table 8. The ordered probit results are similar to the main estimates. The effect of yellow fever is statistically insignificant for the whole population, blacks, and whites with U.S.-born mothers. A yellow fever epidemic that took one percent of the city decreased the probability of entering a professional occupation by 1.6% points (statistically significant at the five percent level) for whites with immigrant mothers. This coefficient is statistically different from the coefficient for whites with U.S.-born mothers at the ten percent level. The results do not meaningfully change when restricting the sample to whites.

6.2. *Selection from fleeing the city*

During yellow fever epidemics, many fled to the countryside. These reactions could potentially be problematic for giving the association between early-life yellow fever and occupational status a causal interpretation. If only the wealthy could afford to leave the city, those left behind would be relatively poor. Consequently, even if yellow fever had no long-run effects on human capital formation, it is possible that those born during the epidemic would enter lower paying occupations than those born during non-epidemic years. Historically, there is no doubt that many responded to the news of a yellow fever epidemic in this way. However, it is another question of whether enough people and whether the right subset of people fled the city to explain the results documented in the previous sections.

For selection to explain the previous results, immigrants would have had to flee in larger numbers than natives. This seems unlikely, because the wealthiest citizen would typically leave the city. Natives would have been more likely to have family living in the countryside with whom they could reside until the epidemic had passed. If relatively wealthier natives fled for the countryside, then the remaining whites would have been negatively selected. This would imply that my results would be biased towards zero, and my estimates could be interpreted as lower bounds. For these reasons, selective fleeing is unlikely to be a problem.[15]

Nonetheless, we can examine whether enough people fled the city during epidemic years by observing the number of individuals that show up in the sample. A drop of 10% or less could be explained by the disease itself. However, if the number of births dropped significantly more during epidemic years, this could be reason to suspect that the number of pregnant women who fled the city were large enough to affect the regression results. Fig. 5 displays the number of individuals for each birth cohort and birth city cell. I also display a nonparametric smoothed regression line through cohorts born during non-epidemic years (years with fatality rates below 1%). Large deviations from that line during epidemic years are a sign that a substantial portion of the population fled for the countryside. Fig. 5 shows that any deviation from the trend during epidemic years was small.

[15] However, if non-immigrants fled the city, this could explain why I do not find evidence that the children of natives were negatively affected by yellow fever epidemics. Although many U.S.-born mothers may have acquired immunity during their childhoods, they were not perfectly immune as a group.

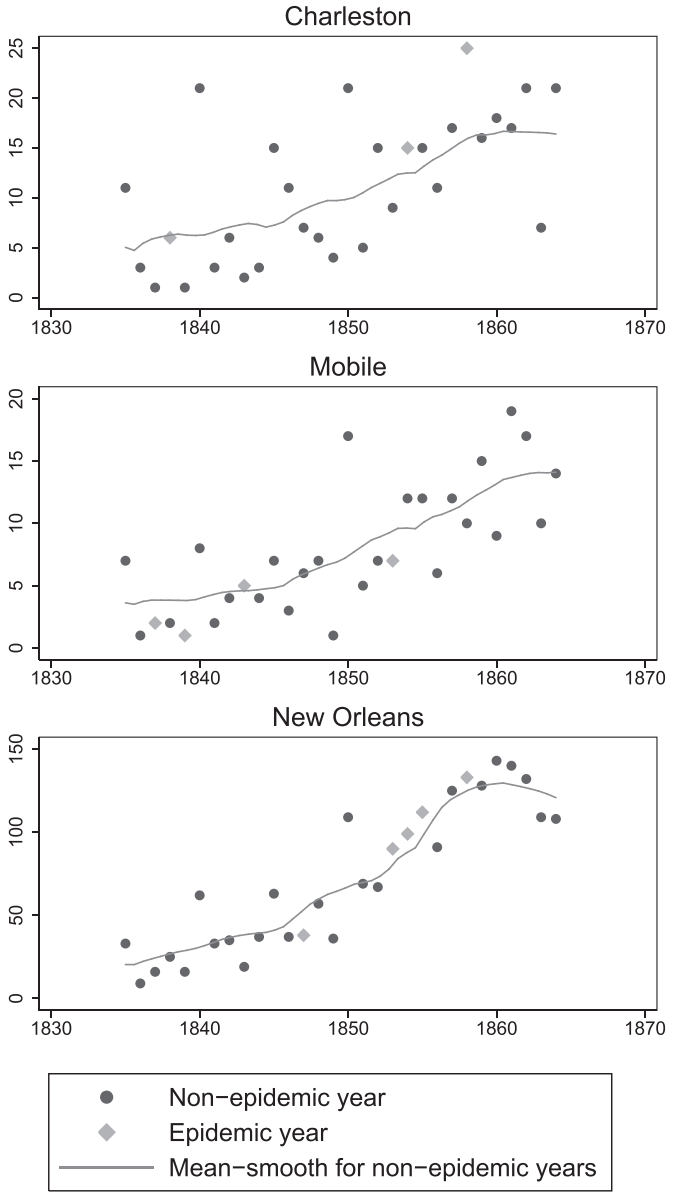

Fig. 5. The number of observations for each sample in each birth city/year cell.**Notes:** The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864.

6.3. Choice of control cities

In the previous analysis, I restrict the sample to cities that were the 50 largest U.S. cities in 1850, were southward of Philadelphia, and on the Gulf Coast, the Atlantic, or attached to the Atlantic by a major river or bay. Yellow fever rarely visited smaller cities, cities northward of Philadelphia, or cities inland. As a robustness check, I include every additional city which adds at least 100 individuals to the sample size. These cities include Albany, NY; Boston, MA; Brooklyn, NY; Buffalo, NY; Cambridge, MA; Cincinnati, OH; Louisville, KY; New York City, NY; Pittsburgh, PA; and St. Louis, MO. All of these cities were either too far north or too far inland to get yellow fever during the sample period. For this reason, they do not add additional variation to the variable of interest (yellow fever exposure). However, they will affect the estimates by giving additional variability to the controls.

Table 9 reproduces the main ordered probit estimates for this new sample. The estimates are qualitatively similar. Yellow fever during an individual's year of birth negatively predicts the highest occupational status, but is not significant at the ten percent level. Yellow fever fatality rates during year of birth interacted with a dummy for having an immigrant mother is negative and significantly different from zero. The standard errors are larger and the estimate is not statistically different from the coefficient on natives. The

Table 9
Marginal effect of early-life yellow fever on the probability of entering a professional occupation using additional control cities.

	Full sample			Whites only		
	(1)	(2)	(3)	(4)	(5)	(6)
Y_b	−0.0069	0.0024		−0.0117*	−0.0005	
	(0.0054)	(0.0101)		(0.0068)	(0.0113)	
$Y_b \times$ **1**[immigrant mother]		−0.0192	−0.0168**		−0.0184	−0.0189**
		(0.0120)	(0.0073)		(0.0133)	(0.0078)
$Y_b \times$ **1**[black]		−0.0000	0.0024			
		(0.0119)	(0.0077)			
$Y_b \times$ **1**[U.S.−born mother]			0.0030			−0.0005
			(0.0101)			(0.0113)
N	32646	32646	32646	27680	27680	27680

Notes: Each coefficient is the marginal effect of entering a professional occupation using an ordered probit model. The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). In addition, this sample includes males reporting being born in Albany, NY; Boston, MA; Brooklyn, NY; Buffalo, NY; Cambridge, MA; Cincinnati, OH; Louisville, KY; New York City, NY; Pittsburgh, PA; and St. Louis, MO. The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth. Robust standard errors are in parentheses.

* 10% significance.
** 5% significance.
*** 1% significance.

results suggest that the children of white immigrants were between 1.7 and 1.9 percentage points less likely to enter professional occupations if they were born during a yellow fever epidemic that took one percent of the city.

7. Comparisons to the 1918 influenza pandemic

Most studies examining the long-run effects of early-life disease exposure on labor market outcomes focus on epidemics occurring during the early 20th century and use data from Censuses no earlier than 1940. These Censuses have wage and education data and do not use ordered models with occupations as the dependent variable. To make the results easier to compare to the extant literature, I replicate the baseline estimates from Almond (2006) using the methodology applied in the previous sections of this study. Almond analyzes how *in utero* exposure to the 1918 influenza pandemic affected long-run labor market outcomes. Almond points out that cohort trends tend to be smooth, and because the influenza pandemic occurred suddenly, deviation from the cohort trends for those who were born in 1919 could be attributed to the pandemic. I successfully replicate the first column of Table 2 in Almond (2006), in which Almond uses data from males born between 1912 and 1922 who appear in the one percent sample of the 1960 Census. The sample is restricted to those born in the United States without imputed ages or birthplaces. Almond regresses various adult outcomes on year of birth, year of birth squared, and an indicator variable for being born in 1919. The replicated results are the first three columns in Table 10.

Those *in utero* during the 1918 influenza pandemic were 2.1 percentage points less likely to graduate from high school, had wages that were $802 less (in 2005 dollars), and total income that was $559 less. These results are remarkably close to the results in Almond (2006). Column (4) uses the ordered probit model used in the previous subsections of this paper, but with the same set of regressors and the same sample as Almond (2006). Those *in utero* during the 1918 influenza pandemic were 0.7 percentage points less likely to enter professional occupations. This estimate is significant at the ten percent level. Column (5) uses the Almond sample and interacts the 1919 dummy with a dummy variable indicating if the individual's mother was born outside of the United States and a stand-alone dummy variable if the individual's mother was born outside of the U.S. Because the interaction term is insignificant, I find no evidence that those with immigrant mothers responded more negatively to the 1918 influenza pandemic. Columns (6) and (7) repeat the yellow fever results from Table 4 for comparison. A yellow fever epidemic that killed 1% of the city had similar long-run effects on the general population as the 1918 influenza pandemic. However, the yellow fever effects were concentrated among the children of immigrants. The long-run occupational effects on the children of immigrants are between two and three times larger than the effect of the 1918 influenza pandemic on the entire U.S. population. These numbers may have been similar if the influenza results were restricted to those who were at high risk of infection. Although the yellow fever estimates may be larger, the effects are more localized and affected a smaller group, meaning the welfare loss to society may have been considerably smaller than from the 1918 influenza pandemic.

Table 10
Comparison to the 1918 influenza pandemic.

Dependent variable	HS	Wage	Income	Professional			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
1[*YOB* = 1919]	−.021***	−802.4***	−559.1*	−.007*	−.009**		
	(.005)	(257.8)	(291.6)	(.004)	(.005)		
1[*YOB* = 1919] × **1**[immigrant mother]					.013		
					(.010)		
Y_b						−.008	.003
						(.005)	(.010)
Y_b × **1**[immigrant mother]							−.0216*
							(.012)
Sample	flu	flu	flu	flu	flu	YF	YF
Model	lin prob	OLS	OLS	order	order	order	order

Notes: Columns (1)–(3) replicate Table 2 from Almond (2006); columns (4) and (5) use the ordered probit methodology from this paper on the Almond sample; columns (5) and (6) are the yellow fever results from Table 4. The Almond sample consists of males from the 1912–1922 birth cohorts from the one percent sample of the 1960 Census. Robust standard errors are in parentheses.

* 10% significance.

** 5% significance.

*** 1% significance.

8. Conclusion

The results of this paper suggest that yellow fever epidemics had profound impacts on the distribution of occupations a generation later. This implies that the economic benefits of eradicating the disease may be higher than previously thought. Furthermore, if the effects from other urban diseases such as cholera, tuberculosis, and dysentery had similar effects, then the benefits from the urban mortality transitions would be even larger.

Most studies examining the long-run effects of early-life disease exposure compare cohorts who were *in utero* during epidemics to adjacent birth cohorts, making it difficult to tease apart the effects of early-life disease exposure from city-wide responses to the epidemic. By examining a disease that differentially affected immigrants, the estimates in this paper are not confounded by city-wide responses that affected all children.

There are several caveats to this study. First, city-of-birth data is only available for a small subset of the 100% sample of the 1880 Census, and this sample may or may not be random. However, even if the sample is biased, so long as that bias is not correlated with early-life yellow fever exposure, it should not affect the results. Second, the only labor market outcome I observe is occupation. These results may not be robust to other measures of socioeconomic status; however, the lack of income, wealth, or education data in the 1880 Census precludes me from testing this hypothesis.

Another limitation is that only year of birth, and not quarter or month of birth, is available in the 1880 Census. Without at least quarter of birth, it is impossible to say whether an individual would have been *in utero* or not during an epidemic. However, because yellow fever epidemics occurred during the summertime and Census enumerators were instructed to record age as of June 1st, it likely that individuals would have been exposed during the neonatal and postnatal stages. I only find strong evidence that exposure during the year of birth and the year after birth matters, and I find no evidence that epidemics during ages 3 or 4 have long-run effects.

Although only the occupations of immigrant children were negatively affected by the disease, mechanisms other than the transmission of the disease could explain the results. It is possible that the disease took the lives of family members, and what I am observing are the long-run effects of stress rather than the effects of contracting the disease. Likewise, the long-run effects of orphanhood may disproportionately affect children still in early-life relative to immigrant children who were orphaned at later ages. For these reasons, the estimates should not be interpreted as the effect of contracting yellow fever on occupational outcomes, but rather the effect of a yellow fever epidemic on occupational outcomes.

This study examines yellow fever epidemics that occurred in southern port cities during the nineteenth century. These results may not extend to modern day yellow fever epidemics in the developing world, although many developing countries have similar life expectancy and income as nineteenth century America.

Appendix A

The results from Table 7 suggest that yellow fever epidemics during an individual's year of birth or during the year after birth may have affected the occupational outcomes of the children of immigrant mothers. Because IPUMS infers birth year from age on June 1, 1880, it will be off by one year for many individuals. For this reason, differences in the effect of yellow fever for adjacent ages should be interpreted with caution. Nonetheless, in this section I present two alternative specifications of Tables 4–6 in which the measure of early-life yellow fever exposure is either (1) yellow fever fatality rates during the year after birth or (2) the average yellow fever fatality rate during the individual's year of birth and the year after birth. Each regression also includes controls for trade activity during the individual's year of birth.

The results for the first alternative specification appear in Table 11. Males experiencing yellow fever epidemics the year after birth were less likely to become professionals, entered lower earning occupations and occupations with higher unemployment risk (although this last finding is not statistically significant at conventional levels). Because of a loss of precision, the effect for the children of immigrant mothers is not larger than the effect of yellow fever for whites with U.S.-born mothers. However, the effect for the children of immigrant mothers is statistically different from zero, whereas the effect for the children of U.S.-born mothers is not. The effect for blacks is statistically significant for one specification (the ordered probit model), but only at the ten percent level. The magnitude is approximately half of the effect for the children of immigrant mothers.

Table 12 presents the results in which the measure of early-life yellow fever exposure (*YF*) is the average yellow fever fatality rate during the individual's year of birth and the year after birth. Each coefficient presents the marginal effect of experiencing an average yellow fever fatality rate of one percent during the first two years of life. The coefficients are approximately twice as large and are estimated with greater precision. Those exposed to yellow fever epidemics during early life averaging 1% fatality rates were 1.8 percentage points less likely to enter professional occupations. These results are driven by the children of immigrant mothers, who were between 3 and 4 percentage points less likely to enter professional occupations. The coefficient for the children of immigrants is statistically different

Table 11
The effect of early-life yellow fever exposure during the year after birth.

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Probability of entering a professional occupation						
Y_{b+1}	−0.0147***	−0.0040		−0.0189**	−0.0091	
	(0.0055)	(0.0103)		(0.0075)	(0.0119)	
$Y_{b+1} \times$ **1**[immigrant mother]		−0.0186	−0.0226***		−0.0162	−0.0253***
		(0.0131)	(0.0085)		(0.0146)	(0.0091)
$Y_{b+1} \times$ **1**[black]		−0.0088	−0.0129*			
		(0.0114)	(0.0068)			
$Y_{b+1} \times$ **1**[U.S.−born mother]			−0.0046			−0.0091
			(0.0104)			(0.0119)
Panel B: 1900 Occupational Income Score						
Y_{b+1}	−12.1460*	−12.5450		−16.8676**	−13.7992	
	(6.3440)	(10.3759)		(7.7452)	(11.5824)	
$Y_{b+1} \times$ **1**[immigrant mother]		−5.1324	−17.6593*		−5.2625	−19.0616*
		(13.5047)	(9.2088)		(14.7740)	(9.8967)
$Y_{b+1} \times$ **1**[black]		11.1190	−1.4205			
		(14.5179)	(11.7027)			
$Y_{b+1} \times$ **1**[U.S.−born mother]			−13.1452			−13.7992
			(10.3977)			(11.5824)
Panel C: Average months unemployed						
Y_{b+1}	0.0195	0.0077		0.0365*	0.0437	
	(0.0178)	(0.0323)		(0.0215)	(0.0343)	
$Y_{b+1} \times$ **1**[immigrant mother]		0.0234	0.0311		−0.0125	0.0311
		(0.0401)	(0.0256)		(0.0417)	(0.0260)
$Y_{b+1} \times$ **1**[black]		0.0065	0.0143			
		(0.0382)	(0.0261)			
$Y_{b+1} \times$ **1**[U.S.−born mother]			0.0088			0.0437
			(0.0324)			(0.0343)

Notes: The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth and trade activity during their year of birth. Panel A presents the marginal effects of an ordered probit model. Panel B presents the marginal effects of a Tobit model left censored at zero. Those reporting occupational non-response are assigned an occupational income score of zero. Panel C uses OLS. Robust standard errors are in parentheses.

* 10% significance.

** 5% significance.

*** 1% significance.

Table 12
The effect of early-life yellow fever exposure averaged over year of birth and year after birth.

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Probability of entering a professional occupation						
YF	−0.0183**	0.0010		−0.0258***	−0.0035	
	(0.0076)	(0.0137)		(0.0099)	(0.0166)	
YF × **1**[immigrant mother]		−0.0356**	−0.0346***		−0.0359*	−0.0394***
		(0.0169)	(0.0106)		(0.0195)	(0.0114)
YF × **1**[black]		−0.0092	−0.0082			
		(0.0146)	(0.0103)			
YF × **1**[U.S.−born mother]			0.0011			−0.0035
			(0.0137)			(0.0166)
Panel B: 1900 Occupational Income Score						
YF	−15.0596**	−12.7611		−18.7646**	−13.8080	
	(7.4847)	(13.7328)		(9.0820)	(15.4803)	
YF × **1**[immigrant mother]		−6.5339	−19.3276*		−8.1405	−21.9485**
		(16.4375)	(9.9881)		(17.8329)	(10.2336)
YF × **1**[black]		4.2239	−8.7464			
		(14.2827)	(10.0502)			
YF × **1**[U.S.−born mother]			−13.8346			−13.8080
			(13.7834)			(15.4803)
Panel C: Average months unemployed						
YF	0.0045	−0.0367		0.0280	0.0222	
	(0.0232)	(0.0412)		(0.0275)	(0.0460)	
YF × **1**[immigrant mother]		0.0656	0.0289		0.0095	0.0317
		(0.0503)	(0.0314)		(0.0539)	(0.0318)
YF × **1**[black]		0.0465	0.0099			
		(0.0476)	(0.0352)			
YF × **1**[U.S.−born mother]			−0.0371			0.0222
			(0.0413)			(0.0460)

Notes: The sample consists of males from the complete count of the 1880 Census who reported being born in Baltimore, MD; Charleston, SC; Mobile, AL; New Orleans, LA; Norfolk, VA; Philadelphia, PA; Richmond, VA; Savannah, GA; and Washington, D.C. (including misspellings). The sample is restricted to those born between 1835 and 1864. Each regression includes a set of dummy variables for birth city, birth year, the mother's birth state/country, and race; additionally, each regression controls for the population of an individual's birth city during their year of birth and trade activity during year of birth. Panel A presents the marginal effects of an ordered probit model. Panel B presents the marginal effects of a Tobit model left censored at zero. Those reporting occupational non-response are assigned an occupational income score of zero. Panel C uses OLS. Robust standard errors are in parentheses.

* 10% significance.
** 5% significance.
*** 1% significance.

from the coefficient on white natives and statistically different from zero at the ten percent level. The children of immigrants who experienced yellow fever epidemics during the first two years of life entered lower paying occupations. This result is also statistically different from zero, but is not statistically different from the effect on the children of U.S.-born mothers. The results provide weak evidence that the children of immigrants who experienced yellow fever epidemics may have entered occupations with increased unemployment risk, but this result is small in magnitude and not statistically significant. For all three measures of occupational status, the marginal effect of early-life yellow fever exposure is statistically equal to zero for whites with U.S.-born mothers and for blacks.

Appendix A. Supplementary data

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.eeh.2017.01.003.

References

Albion, Robert Greenhalgh, Pope, Jennie Barnes, 1939. The Rise of New York Port, 1815–1860. C. Scribner's Sons, New York City, NY.
Almond, Douglas, 2006. Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 U.S. population. J. Polit. Econ. 114 (4), 672–712.
Almond, Douglas, Currie, Janet, 2011. Killing me softly:the fetal origins hypothesis. J. Econ. Perspect. 25 (3), 153–172.
Almond, Douglas, Edlund, Lena, Li, Hongbin, Zhang, Junsen, 2010. Long-term effects of early-life development: evidence from the 1959 to 1961 Chine famine. In: The Economic Consequences of Demographic Change in East Asia, vol. 19, University of Chicago Press, pp. 321–345.
Alsan, Marcella, Goldin, Claudia, June 2015. Watersheds in Infant Mortality: The Role of Effective Water and Sewerage Infrastructure, 1880–1915. Working Paper 21263, National Bureau of Economic Research.

Barker, David, 1995. The fetal origins of coronary heart disease. Br. Med. J. 311 (6998), 171–174.
Barreca, Alan, 2010. The long-term economic impact of in utero and postnatal exposure to malaria. J. Hum. Resour. 45 (4), 865–892.
Beach, Brian, Ferrie, Joseph, Saavedra, Martin, Troesken, Werner, July 2014. Typhoid Fever, Water Quality, and Human Capital Formation. Working Paper 20279, National Bureau of Economic Research.
Beeson, Patricia, Troesken, Werner, 2006. When Bioterrorism was No Big Deal. NBER Working Paper Series.
Black, Sandra E, Devereux, Paul J., Salvanes, Kjell G., 2016. Does grief transfer across generations? Bereavements during pregnancy and child outcomes. Am. Econ. J. Appl. Econ. 8 (1), 193–223.
Bleakley, Hoyt, 2007. Disease and development:evidence from Hookworm eradication in the American South. Q. J. Econ. 122 (1).
Case, Anne, Paxson, Christina, 2005. Early life health and cognition function in old age. Am. Econ. Rev.: Pap. Proc. 99 (2).
Cavalcanti, Denise P, Salomão, Maria Antonieta, Lopez-Camelo, Jorge, Pessoto, Mônica A, 2007. Early exposure to yellow fever vaccine during pregnancy. Trop. Med. Int. Health 12 (7), 833–837.
Center of Disease Control, 2010. Transmission of Yellow Fever Vaccine Virus Through Breast-Feeding – Brazil, 2009.
Chen, Yuyu, Zhou, Li-An, 2007. The long-term health and economic consequences of the 1959–1961 famine in China. J. Health Econ. 26 (4), 659–681.
Costa, Dora, Steckel, Richard H., 1997. Long-term trends in health, welfare, and economic growth in the United States. In: Health and Welfare During Industrialization, University of Chicago Press, pp. 47–90.
Costa, Dora L, 2000. Understanding the twentieth-century decline in chronic conditions among older men. Demography 37 (1), 53–72.
Cutler, David, Miller, Grant, 2005. The role of public health improvements in health advances: the twentieth-century United States. Demography 42 (1), 1–22.
DeBow, James Dunwoody Brownson, 1854. Statistical View of the United States 850. Beverly Tucker, Senate Printer, Washington, DC.
Diaz, Henry F, McCabe, Gregory J, 1999. A possible connection between the 1878 yellow fever epidemic in the southern United States and the 1877–78 El Nino episode. Bull. Am. Meteorol. Soc. 80 (1), 21.
Douglas, Paul Howard, 1930. Real Wages in the United States, 1890–1926. Houghton Mifflin Co., Boston and New York.
Duffy, John, 1992. The Sanitarians: A History of American Public Health. University of Illinois Press, Urbana and Chicago.
Espinosa, Mariola, 2014. The question of racial immunity to yellow fever in history and historiography. Soc. Sci. Hist. 38 (3–4), 437–453.
Hong, Sok Chul, 2007. The burden of early exposure to malaria in the United States, 1850–1860: malnutrition and immune disorders. J. Econ. Hist. 67 (04), 1001–1035.
Hong, Sok Chul, 2013. Malaria: an early indicator of later disease and work level. J. Health Econ. 32 (3), 612–632.
Humphreys, Margaret, 2001. Malaria: Poverty, Race, and Public Health in the United States. JHU Press, Baltimore.
Karlsson, Martin, Nilsson, Therese, Pichler, Stefan, 2014. The impact of the 1918 Spanish flu epidemic on economic performance in Sweden: an investigation into the consequences of an extraordinary mortality shock. J. Health Econ. 36, 1–19.
Kuhn, Susan, Twele-Montecinos, Loreto, MacDonald, Judy, Webster, Patricia, Law, Barbara, 2011. Case report: probable transmission of vaccine strain of yellow fever virus to an infant via breast milk. Can. Med. Assoc. J..
Lebergott, Stanley, 1964. Manpower in Economic Growth: The American Record Since 1800. McGraw-Hill, New York.
Lin, Ming-Jen, Liu, Elaine M, 2014. Does in utero exposure to Illness matter? The 1918 influenza epidemic in Taiwan as a natural experiment. J. Health Econ. 37, 152–163.
Motulsky, Arno G., 1989. Metabolic polymorphisms and the role of infectious diseases in human evolution. Hum. Biol. 61 (5/6), 835–869.
Nasidi, A., Monath, T.P., Vandenberg, J., Tomori, O., Calisher, C.H., Hurtgen, X., Munube, G.R.R., Sorungbe, A.O.O., Okafor, G.C., Wali, S., 1993. Yellow fever vaccination and pregnancy: a four-year prospective study. Trans. R. Soc. Trop. Med. Hyg. 87 (3), 337–339.
Neelsen, Sven, Stratmann, Thomas, 2011. Effects of prenatal and early life malnutrition: evidence from the Greek famine. J. Health Econ. 30 (3), 479–488.
Patterson, K.D., 1992. Yellow fever epidemics and mortality in the United States, 1693–1905. Soc. Sci. Med. 34 (8), 855–865.
Persson, Petra, Rossin-Slater, Maya, May 2016. Family Ruptures, Stress, and the Mental Health of the Next Generation. Working Paper 22229, National Bureau of Economic Research.
Preston, Samuel, Haines, Michael, 1991. Fatal Years: Child Mortality in Late Nineteenth-Century America. Princeton University Press, Princeton, NJ.
Pritchett, Jonathan, Tunali, Insan, 1995. Strangers' disease: determinants of yellow fever mortality during the New Orleans epidemic of 1853. Explor. Econ. Hist. 32 (4), 517–539.
Ruggles, Steven, Alexander, J. Trent, Genadek, Katie, Goeken, Ronald, Sobek, Matthew, Schroeder, Matthew B., 2010. Integrated Public Use Microdata Series Version 5.0, Machine-readable Database, 2010. University of Minnesota, Minneapolis.
Saavedra, Martin, 2017. Early-Life Disease Exposure and Occupational Status: The Impact of Yellow Fever during the 19th Century, [Data set]. Inter-university Consortium of Political and Social Research [distributor], Ann Arbor, MI. http://dx.doi.org/10.3886/E100385V1.
Toner, J.M., 1873. The distribution and natural history of yellow fever as it has occurred at different times in the United States. Public Health Pap. Rep., 359–384.
Tsai, T.F., Paul, Rosemary, Lynberg, M.C., Letson, G.W., 1993. Congenital yellow fever virus infection after immunization in pregnancy. J. Infect. Dis. 168 (6), 1520–1523.
United States Department of the Treasury, 1879. The Statistical Abstract of the United States.
WHO, 2014. Yellow Fever. ⟨http://www.who.int/mediacentre/factsheets/fs100/en/. (Accessed: 1-22-2016).
Wright, Carroll Davidson, 1904. Cost of Living and Retail Prices of Food 903. US Government Printing Office, Washington, DC.