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journal homepage: www.elsevier.com/locate/eehEarly-life disease exposure and occupational status: The impact of yellow fever during the 19th century[☆]

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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,

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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.

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.

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