ESSAYS ON CHILDHOOD CONDITIONS AND ADULT ECONOMIC AND HEALTH OUTCOMES

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This dissertation includes four essays on how childhood interventions affected adult labor market and health disparities. The first chapter investigates how early childhood environment affects longevity by examining the internment of Japanese Americans during World War II as a natural experiment. Using the roster of internees linked to death records in the Social Security Death Index, I find that those who were incarcerated within internment camps during the first four years of life died approximately two years earlier. The effect is larger for those from low socioeconomic status families and those incarcerated in colder climates. I also find that those incarcerated during early childhood were more likely to die of circulatory disease.

The second chapter of my dissertation examines how school quality within Japanese American internment camps affected adult educational attainment and labor market outcomes. Using data pooled from the 1980, 1990, and 2000 Censuses, I find that those who were incarcerated during school age were less likely to achieve collegiate or post-collegiate education, and had lower returns to education.

The third chapter examines the long-run effects of early-life yellow fever exposure on adult occupational outcomes. Using data from the 100 percent sample of the 1880 Census, I find that those born during yellow fever epidemics were less like to become professionals and more likely to become unskilled laborers. This effect is larger for the children of immigrant mothers, who were more likely to contract yellow fever than natives were.

Low birth weight children are earn less and become less educated as adults. The last chap-

ter examines whether the negative effects of low birth weight are mitigated by socioeconomic status. This chapter uses a unique data set of Korean adoptees who were quasi-randomly assigned to families and finds that neighborhood characteristics mitigate the negative effects of low birth weight, whereas family characteristics do not.

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Using War Relocation Authority records linked to the Social Security Death Index, I investigate whether the internment of Japanese Americans during WWII affected the life spans of male internees who were incarcerated during early childhood. Using un-interned Japanese Hawaiians as a control group, difference-in-differences estimates suggest that internees incarcerated within the first four years of life died approximately two years earlier. Furthermore, the internees from low socioeconomic status families and internees incarcerated in cold climates drive almost the entire effect. Additionally, NCHS cause-of-death data suggest that early childhood incarceration increased the incidence of circulatory diseases by 7 percentage points. Data on Chinese Americans suggest that the identifying assumption is satisfied.

1.1 INTRODUCTION

Over the last decade, economists have become increasingly interested in whether early childhood conditions can explain adult health and economic disparities. One way to answer this question is by finding a cohort of children who faced large and sudden changes during their early childhoods. Health economists have often turned to unique historical events, such as the 1918 influenza pandemic (Almond, 2006), hookworm eradication (Bleakley, 2006), and hospital desegregation in the American South (Chay et al., 2009). This study examines whether early childhood environments can explain variations in life spans by exploring the internment of over 100,000 Japanese Americans during World War II.

Following the bombing of Pearl Harbor by the Empire of Japan, President Roosevelt

signed Executive Order 9066, which allowed the War Department to declare any area a military zone from which it could exclude any person it deemed a threat (Ng, 2002). President Roosevelt signed the Order in February of 1942, and by March of that year, the United States Army evacuated all Japanese Americans, including children and US citizens, from the West Coast. The United States government created the War Relocation Authority (WRA) to detain all Japanese Americans residing on the West Coast, built internment camps composed of military-style barracks, and surrounded them with barbed wire and armed guards (Ng, 2002). The internees shared small rooms that ranged from 320 to 480 square feet with three to seven people and used communal showers, bathrooms and dining halls (Jensen, 1999). The internment camps were small communities consisting of hospitals, schools and housing facilities, all erected in a matter of months.

In this paper, I ask whether the internment of the Japanese Americans disproportionately affected the subsequent life spans of the youngest children within the camps. This question is relevant for two reasons. First, the World War II internment of Japanese Americans is an important chapter of American history that affected tens of thousands of children, and the long-term health effects of internment on the children are still unknown. Second, the internment of the Japanese Americans gives us an opportunity to study how large exogenous changes in early childhood conditions affect longevity. The non-voluntary, unanticipated, and near universal nature of the internment limits the possibility that families selected into better environments for their children. The health conditions within the camps were harsh, especially for young children. Nutrition was poor, dust storms caused high rates of asthma among children, and poorly insulated nurseries reached extreme temperatures during summer and winter months (Jensen, 1999). These poor health conditions negatively affected the development of the youngest children within the camps. My study will show that this event also affected their adult health outcomes decades later.

In order to analyze the effects of internment, I begin by linking records from the War Relocation Authority to death records from the Social Security Death Index (SSDI). To my knowledge, this study is the first to track the longevity of internees who have survived infancy. Then I construct a control group using death records of individuals with Japanese surnames who were likely not interned. By examining difference-in-differences estimates, I

find that incarceration within the first four years of life decreased life spans by 1.6 years or 3 percent. Furthermore, the internees from low socioeconomic status families drive almost the entire effect. Internment during early childhood decreased life spans by almost 3 years or 5 percent for the internees from low socioeconomic status families, but had no effect on the internees from high socioeconomic status families. This finding suggests that human capital investments or increased wealth might mitigate the effects of poor early childhood health. Additionally, I find that incarceration in cold climates decreased life spans more than incarceration in warm climates. Lastly, using records from the Multiple Cause of Death Data from the National Center for Health Statistics, I find that early childhood incarnation increased the probability that internees died from diseases of the circulatory system by 7 percentage points.

My findings contribute to the growing literature on how early childhood shapes adult economic and health outcomes (see Almond and Currie (2011) for a detailed survey). Health shocks that occur during gestation, infancy or the early years of childhood may permanently impact adult health and mortality risk (Case et al., 2005). For example, Dora Costa and Joanna Lahey (2005) argue that early life conditions can explain at least 16 percent of the decline in adult mortality risk from 1900 to 1960-1980. Van Den Berg, Lindeboom, and Portrait (2006) find that economic conditions during an individual's birth year predict longevity, suggesting that access to nutrients, housing, or medical care during the early stages of life have permanent health effects. Many studies in this literature exploit unexpected epidemics or the eradication of diseases as a form of exogenous variation in early health conditions. A seminal study by Douglas Almond (2006) presents evidence suggesting that the 1918 influenza pandemic lowered the incomes and educational attainment of the cohort that was in utero during the pandemic. The negative effects of epidemic exposure to influenza extend beyond those in utero, however. Anne Case and Christina Paxson (2009) argue that the cohort of children who were two years old during the pandemic had worse cognition at elderly ages.

This paper is also related to the medical literature on how adverse childhood conditions affect adult health (Dong et al, 2004; Dube et al, 2003; Danese et al, 2009). A large literature has demonstrated that children exposed to adverse conditions experience poor health,

however, few papers had demonstrated this finding using plausibly exogenous variation. Furthermore, this literature typically does not compare adverse events in early childhood to adverse conditions during the later stages of childhood.

Lastly, this paper relates to the literature on how temperature affects circulatory disease risk. Bhaskaran et al. (2010) find evidence that temperature drops increase the risk of heart attacks in the short term. Deschenes and Moretti (2009) find that extreme heat increases mortality risk during the day of extreme heat, whereas extreme cold increases mortality risk for the following 30 days. My results suggest that exposure to the cold during a critical window of early childhood might have long-term effects on circulatory disease risk. If true, policy interventions designed to protect the public from extreme cold should prioritize not only the elderly who are at an increased short-term risk of heart attacks, but also children in this critical window.

Section 2 examines the historical background of Japanese American internment and previous internment research. Section 3 discusses the construction of the sample. Section 4 outlines the econometric model, and Section 5 analyzes the results. Section 6 addresses data truncation issues, and Section 7 analyzes the how early childhood internment affected the cause of death. Section 8 concludes.

1.2 HISTORICAL BACKGROUND

After Pearl Harbor, Lieutenant General John DeWitt recommended to President Roosevelt that the military remove all Japanese Americans from the West Coast (Ng, 2002). Officially, the War Department was worried about espionage. In the event that the Empire of Japan invaded the West Coast of the United States, the War Department did not trust Japanese Americans to remain loyal. However, one month prior to Pearl Harbor, Curtis Munson submitted a report to the President and the Secretary of War indicating that there was no reason to question the loyalty of Japanese Americans to the United States. In 1988, the United States paid $20,000 in redress to every surviving internee, and in 1990, President George Bush signed an official letter of apology.

Although a small number of non-West Coast Japanese Americans were incarcerated as well, Japanese Hawaiians were not subject to mass relocation for two reasons. First, more than 100,000 Japanese Americans lived in Hawaii, and relocating them across the Pacific to remote camps in the continental United States would have been highly costly. Second, Japanese Americans comprised over one-third of the Hawaiian population, but only 2 percent of the West Coast population, and removing a third of the labor force in Hawaii would have had devastating economic consequences.

The US Army evacuated the first group of Japanese Americans on March 22, 1942. Five days later, the government prohibited voluntary evacuation from the West Coast. While the internment camps were under construction, the WRA housed the internees at the assembly centers, which consisted of fairgrounds and racetracks. The internees slept in stables that they complained still reeked of horse manure (Fiset, 1999). The assembly centers were never designed to house humans. The internees stayed within the assembly centers for several months, and by October of 1942, the WRA had moved the internees to the permanent relocation centers where internees stayed an average of 3.5 years (Chin, 2005).

Gwenn Jensen (1999) documents the harsh health environments that the internees faced. The Manzanar, Poston and Gila River camps were located in the deserts of Arizona and eastern California, and the internees frequently battled the heat. The barracks had poor insulation and cooling systems, which caused temperatures at times to exceed 104 degrees in the nurseries. Dehydration and overheating caused many of the infant deaths within the camps. The internees could only bring one suitcase of clothes per person. The internees in the northwestern camps did not bring adequate clothing and frequently complained of the cold. The water pipes in the camps were recycled from oil and gas lines, which led to frequent water contamination. The WRA rationed less than 40 cents a day for food per internee and fed the internees an imbalanced diet with little Japanese food. There were also problems with dust storms. Because dust would get inside the barracks through cracks, the internees were exposed to dust inhalation even when indoors, which led to higher rates of asthma among children.

To date, Aimee Chin (2005) is the only economist to publish a paper on Japanese American internment. Her study looks at the long-run effects of labor market withdrawal on

working-age male internees. She does not examine females because many females did not participate in the labor force during the internment period (and consequently did not withdraw from the labor market). Working-age males presumably suffered economic losses related to internment for two reasons. First, these internees had to sell, store or abandon their property before relocating, and because these sales had to happen quickly, they often received below-market prices. Second, they had to leave their careers and could not continue to build human capital through work experience. Although the internees were encouraged to work in the camps, the career possibilities were limited, and many internees did not gain as many technical skills as they would have outside of the camps. Chin uses the fact that the internees who were children and adolescents during the time of internment did not suffer an interruption in their labor market experience and employs a difference-in-differences approach similar to the one used in this study. Chin estimates that internment decreased the 1970 earnings of adult male internees by 9 to 13 percent.

1.3 DATA

The strategy of this paper is to take records from the War Relocation Authority and link them to the death records that appear in the Social Security Death Index. Linked death records compose the treatment group, and unlinked Japanese Hawaiians compose the control group. Subsections 3.1 to 3.3 describe this process in detail.

1.3.1 The WRA Records Sample

As part of the internment process, the War Relocation Authority collected the personal information of every internee who entered each of the ten WRA camps. They also collected information for all of the births within the internment camps that occurred in 1942. The Bancroft Library of the University of California at Berkeley converted these files into electronic form in the 1960s, and now most of the variables in the files are publicly available through the National Archives. The data include the following information concerning the

internees: first, middle and surnames; gender; assigned assembly and relocation centers; previous town, county and state; birth year; parents' birth countries; and father's occupation. The data also include information that in most cases is not relevant for interned children, such as occupation, marital status, and academic degrees.

I restrict the sample to male internees with non-missing names because females were more likely to change their surnames and are therefore difficult to link to their death records. The remaining WRA records contain 8,651 male internees born between 1932 and 1942, with a mean birth year of 1937.2. Of these internees, 85.80 percent are from California, 9.33 percent are from Washington, 2.73 percent are from Oregon, and 1.73 percent are from Hawaii. Each internment camp housed between 585 (Minidoka) and 1,519 (Colorado River) children in the sample. The internees came from cities and towns with a range of population densities. For example, 20.29 percent previously resided in towns with a population of less than 2,500, whereas 22.48 percent came from a city with a population of one million or more (almost all from Los Angeles). The data contains nine occupational categories for the father. Approximately half of the observations had a father who worked in farming (33 percent as a manager/operator and 16 percent as laborers), 20 percent of the observations had a father working as a non-farming manager or official, 10.5 percent had a father who was a skilled craftsmen or semi-professional. No other category comprises more than 10 percent of the sample.

1.3.2 The Social Security Death Index Sample

The Social Security Death Index (SSDI) contains information on all deaths reported to the Social Security Administration since 1962. Although the Death Master File is not publicly available for download, the SSDI can be searched from several genealogy websites. Each death record includes an individual's name, age at death, birth date, death date, state in which his or her Social Security card was issued, address in which the last social security benefit was received (if the individual received Social Security benefits), and Social Security Number. The Death Master File does not contain gender or race. In terms of completeness, the SSDI contains between 93 and 96 percent of all deaths in the United States of individuals

who were 65 or over (Mark Hill and Rosenwaike, 2001).

Using the SSDI, I construct a sample of death records with Japanese American surnames. Ideally, I would have identified death records of all Japanese Americans. However, because race is not available in the death records, I instead use the fact that Japanese Americans have surnames that are unique relative to those of other Americans. I searched the SSDI (accessed through www.rootsweb.ancestry.com in August 2011) for all individuals born between 1932 and 1942 with a surname appearing in the WRA records sample, as described in the previous subsection. I exclude a small number of surnames that appear in the WRA records (such as Torres and Williams) because they are most likely from multiracial families. This sample gives us 12,952 death records.

Because the SSDI does not have gender, I infer the gender of each observation in the SSDI based on the first name. I label an observation as male if at least 90 percent of the observations in the WRA records with that first name are male. I then restrict the SSDI sample to (inferred) males resulting in 6,161 death records with a mean life span of 64.22 years. Because the SSDI includes date of birth and date of death, I calculate life span as a fraction of a year. For example, I code an observation who was born on January 1, 1937 and who died on March 16, 2001 as having a life span of 64.2026 years ($64.2026 = 64 + \frac{74}{365.25}$).

1.3.3 Record Linkage

I would like to link records from the WRA to death records in the SSDI. Unfortunately, unique identifiers, such as Social Security Numbers, do not appear in the publicly available WRA records. I can link records by using first name, middle initial, surname, and birth year. Small variations in spelling and typographical errors might prevent two records that are true matches from matching exactly with respect to all four variables. For example, if a Billy K. Ogawa born in 1937 appears in the WRA records, I can reasonably link this record to a Bill K. Ogawa born in 1937 in the SSDI. To link records that I can be reasonably sure are true matches, I use the following 7-step deterministic record linkage algorithm:

1. Link any two records that match perfectly with respect to first name, middle name, surname, and birth year. This step results in 735 links. Set these links aside and

continue to step 2.

2. Link any two records that match perfectly with respect to first name, surname, and birth year so long as the middle initial does not conflict. This step applies if the middle initial is missing in one of the data sets. This step results in 197 links. Set these links aside and continue to step 3.
3. Phonetically code first names using the Soundex algorithm.[1] Link records that match with respect to middle initial, surname, birth year, and phonetically coded first name. This step results in 37 links. Set these links aside and continue to step 4.
4. Link records that match exactly with respect to first name, middle initial, and surname so long as the birth year differs by at most one year. This step results in 32 links. Set these links aside and continue to step 5.
5. Repeat step 4 allowing for non-conflicting middle initials. This step results in 32 links. Set these links aside and continue to step 6.
6. Recode short first names into a common long name (Bill or Billy to William). Link any two records that match exactly with respect to middle initial, surname, birth year, and recoded first name. This step results in 24 links. Set these links aside and continue to step 7.
7. Repeat step 6 while allowing for non-conflicting middle initials. This step results in 9 links.

This algorithm results in 1,066 links, which comprise the Treatment Group. Historical studies that use deterministic record linkage algorithms and have similar linkage rates include Ferrie (1996) and Long (2005).

The record linkage process links some internees from the WRA records and does not link other internees. We might wonder whether the linked sample is a random sample of internees or whether the record linkage algorithm introduces bias. To test for such bias, Table 1 compares the linked and unlinked WRA sample for three variables: (1) the proportion with a high socioeconomic status (HSES) father (determined by the father's occupation), (2) the proportion from a city with a population of at least 25,000, and (3) the proportion born in California. The differences in the sample proportions between the linked and unlinked

[1] See Herzog, Scheuren and Winkler (2007) for details on the Soundex algorithm.

sample are never statistically significant. For example, 36.5 percent of the linked sample have a HSES father, whereas 36.4 percent of the unlinked sample have a HSES father. Testing the null hypothesis that the proportions are equal yields a p-value of 0.915. The differences never have a p-value smaller than 0.3. Because this paper compares the internees who entered the camps in their early childhoods with those who entered as older children, I must also rule out that the record linkage process introduces bias for only either the older internees or the younger internees. Table 1 also tests whether there are differences between the linked and unlinked WRA records for the older (born between January of 1932 and December of 1937) and younger (born between January of 1938 and December of 1942) cohorts. Again, the differences are never significant, and the p-values are large.

After linking the internees to the death records, I must also construct a Control Group. We could use all of the unlinked death records. However, that sample would likely contain internees who were not linked because of variations in names and non-Japanese Americans who happened to have Japanese surnames. Furthermore, most of the un-interned Japanese Americans were in Hawaii and few Japanese Hawaiians were incarcerated (Chin, 2005). For these reasons, I restrict the control group to the unlinked Japanese Americans with Social Security Cards issued in Hawaii. This decision raises the question of whether Japanese Hawaiians are a good control group for West Coast Japanese Americans. I provide graphical evidence answering this question using data from Chinese Americans in Section 5.1, and formally test this assumption in the Appendix.

1.4 THE ECONOMETRIC MODEL

1.4.1 Differences-in-Differences Estimation

The ideal experiment to find the long-run effects of early childhood internment on mortality would be if only a subset of Japanese Americans were interned, and if internment were randomly assigned. Without such an experiment, we could compare the average age at death of the youngest cohort of internees with that of the non-internees from the same

cohort. This estimate might be biased because most internees were from the West Coast and the longevity of West Coast Japanese Americans might have differed from that of non-West Coast Japanese Americans even in the absence of internment. Because West Coast Japanese Americans normally migrated from Hawaii to the more racially hostile West Coast to pursue greater economic opportunities, we might expect systematic differences between those who chose to migrate and those who did not. Alternatively, we could compare the distribution of life spans for those who were interned in early childhood with the distribution for those who were interned at later ages, but this estimate might be biased if life spans are trending over time. To overcome these challenges, I follow a difference-in-differences strategy that will allow me to subtract out a fixed level difference between the treatment and control groups and a secular cohort trend.[2] Difference in differences across cohorts (instead of across time) is used by Esther Duflo (2001) to analyze the effects of a school construction program in Indonesia and by Aimee Chin (2005) to analyze the long-run effects of withdrawing from the labor market during Japanese American internment.

A difference-in-differences strategy suggests estimating the equation:

$$y_{ics} = \alpha + \beta_c + \gamma_s + \delta L_i + \theta L_i \times \mathbf{1}\left[c \geq 1938\right] + \epsilon_i \tag{1.1}$$

where y_{ics} is the life span of individual i born in cohort c and state s. The parameters β_c and γ_s are birth cohort and state fixed effects, respectively. The regressor $L_i = 1$ if the death record of individual i is linked to a WRA record. Otherwise, $L_i = 0$. By construction of the sample, if $L_i = 0$, then individual i's death record was unlinked to the WRA records and had a Social Security card that was issued in Hawaii. If the interaction term $L_i \times \mathbf{1}\left[c \geq 1938\right] = 1$, then individual i was incarcerated during early childhood. The coefficient θ is the difference-in-differences parameter of interest. The error term is ϵ_i.

We would also like to know to what extent high socioeconomic status (HSES) may have mitigated the long-run effects of early childhood internment on mortality. To test this

[2]The results do not change when I use the more flexible changes-in-changes model from Athey and Imbens (2006). I do not use a survival analysis model because I observe only failures, and do not observe the denominator population. However, using a survival analysis model limiting the sample to observations that have died by the age of 69 (where the data is truncated for the youngest cohort) yields very similar results.

hypothesis, I estimate the following variation of equation (1):

$$\begin{aligned} y_{icsj} &= \alpha + \beta_c + \gamma_s + \delta_j L_i + \theta_L L_i \times \mathbf{1}\left[c \geq 1938\right] \times \mathbf{1}\left[\text{LSES}\right] \\ &\quad + \theta_H L_i \times \mathbf{1}\left[c \geq 1938\right] \times \mathbf{1}\left[\text{HSES}\right] + \epsilon_i \end{aligned} \tag{1.2}$$

where j indexes the occupation of individual i's father. The coefficient on L_i is δ_j, which varies with the father's occupation. The indicator variables $\mathbf{1}\left[\text{LSES}\right]$ and $\mathbf{1}\left[\text{HSES}\right]$ equal one if the individual's father has a LSES or HSES occupation, respectively. HSES jobs include professionals, semi-professionals, skilled craftsmen, managers (except farm mangers), and official occupations. LSES occupations include farm workers, fishermen, service workers, unskilled laborers, salesmen, and clerical occupations. The treatment effect is θ_L for LSES internees and θ_H for HSES internees.

The identifying assumption is that in the absence of internment, the cross-cohort life spans of the internees would have been parallel to the cross-cohort life spans of the un-interned Japanese Hawaiians. This assumption does not imply that there are not fixed level differences between West Coast Japanese Americans and Japanese Hawaiians. Formally, the assumption is

$$\begin{aligned} y_{ics}^N &= \alpha_c + \beta_s + \epsilon_i \\ E\left[\epsilon_i | c, s\right] &= 0 \end{aligned} \tag{1.3}$$

where y_{ics}^N is the life spans for individual i in cohort c born in state s in the absence of internment. The parameter α_c is a cohort-specific fixed effect and β_s is a state-specific fixed effect. The second condition implies that in the absence of internment, there are no interactions between cohort and state effects.

Of course, we cannot test this assumption empirically because all internees were by definition interned. However, we can test whether the assumption is plausible by testing whether the cross-cohort life-span trend of Chinese Americans in California, Oregon, and Washington is parallel to the cross-cohort life-span trend of Chinese Hawaiians. I provide graphical evidence for this assumption in the Results section, and formally test it in the Appendix.

It is tempting to equate the terms internment effect and treatment effect in this context, but to do so is incorrect. We are comparing the differences between the internees who were younger children and the internees who were older children during their incarceration (the first difference) and subtracting out a secular cohort trend identified by the difference in life spans between the younger and older cohorts of Japanese Hawaiians (the second difference). Therefore, the treatment effect is not the effect of internment on life spans but rather the effect of early childhood internment on life spans relative to the effect of internment at an older age. We can think of age during internment as a measure of treatment intensity. The youngest cohorts are at a plausibly greater risk of suffering health shocks and are exposed consequently to high intensity of treatment. Health shocks may have affected the older cohorts at a lower intensity. In this paper, the identified treatment effect is the difference between the high intensity treatment and the low intensity treatment. I formalize this concept and relate the econometric model to a health production function in the following subsection.

1.4.2 Relating the Model to a Health Production Function

In this subsection, I present how the difference-in-differences estimates relate to a health production function similar to the model in Almond and Currie (2011). Suppose the health production function is

$$E\left[h_{c,w}\right] = A\left[\gamma I^1_{c,w} + (1-\gamma) I^2_{c,w}\right] \tag{1.4}$$

where $h_{c,w}$ is the final health for cohort c in birth place w. The index $c = 1$ for the 1938-1942 birth cohort, and $c = 0$ for the 1932-1937 birth cohort; $w = 1$ for those on the West Coast, and $w = 0$ for Hawaii. The variable $I^1_{c,w}$ measures health investments or health conditions during the early childhood, and $I^2_{c,w}$ measures health investment or health conditions during later childhood. If $\gamma > 0$, then early childhood conditions "matter" in determining adult mortality risk. If $\gamma > \frac{1}{2}$, then early childhood conditions matter more than later childhood conditions. In this model, investments in early childhood are perfect substitutes for investments in later childhood. Using a CES production function as in Cunha and Heckman (2007) would allow for dynamic complementarity. Also note that health capital does not depreciate in the way

suggested by Grossman (1972), but instead early life shocks have a persistent effect.

Suppose that in the absence of internment, childhood health conditions are an additively separable function so that

$$E\left[I_{c,w}^{j}\right] = \beta_0 + \beta_1 \mathbf{1}\left[c = 1\right] + \beta_2 \mathbf{1}\left[w = 1\right] + \beta_3 \mathbf{1}\left[j = 1\right] \tag{1.5}$$

Equation (5) is the parallel trends assumption in this model. Now suppose that internment decreases childhood conditions during the period of internment by $\mu > 0$. Then the health production function becomes:

$$\begin{aligned}
E\left[h_{1,1}\right] &= A\left[\gamma(I_{1,1}^{1} - \mu) + (1-\gamma)I_{1,1}^{2}\right] && (1.6)\\
E\left[h_{0,1}\right] &= A\left[\gamma I_{0,1}^{1} + (1-\gamma)(I_{0,1}^{2} - \mu)\right] && (1.7)\\
E\left[h_{1,0}\right] &= A\left[\gamma I_{1,0}^{1} + (1-\gamma)I_{1,0}^{2}\right] && (1.8)\\
E\left[h_{0,0}\right] &= A\left[\gamma I_{0,0}^{1} + (1-\gamma)I_{0,0}^{2}\right] && (1.9)
\end{aligned}$$

The difference-in-differences estimates from the results section are

$$\tau_{DD} = E\left[h_{1,1}\right] - E\left[h_{0,1}\right] - E\left[h_{1,0}\right] + E\left[h_{0,0}\right] \tag{1.10}$$

Plugging, equation (5) into equations (6)-(9), and plugging that into equation (10) and doing some simple algebra, yields that

$$\tau_{DD} = A\mu(1 - 2\gamma). \tag{1.11}$$

From equation (11), if $\tau_{DD} < 0$, then $\gamma > \frac{1}{2}$. This implies that if the difference-in-differences estimate is negative, then early childhood shocks are more important for determining mortality risk than later childhood shocks.

1.5 RESULTS

1.5.1 Nonparametric and graphical evidence

Figure 1 nonparametrically graphs the relationship between age at death and birth year for both the internees linked to the Social Security Death Index and the un-interned Japanese Hawaiians. Each nonparametric regression line in Figure 1 is a locally linear smooth with a bandwidth of 1.5. Bandwidths between one and three do not qualitatively change Figure 1. The nonparametric regression lines in Figure 1 are downward sloping only because the death records were collected at the same time. The youngest cohort was only 69 years old when the SSDI death records were extracted. Consequently, we do not observe any deaths that occurred after that age. In contrast, because the 1932 cohort was 79 years old, we observe the deaths of septuagenarians.

This figure provides the first graphical evidence suggesting that internment during early childhood decreased life spans relative to the life spans of the older internees. For the 1932 to 1936 cohorts, the internees mean age at death closely tracks that of the Japanese Hawaiians across cohorts. The internees from these cohorts were between the ages of 6 and 10 when they entered the internment camps. For the cohorts born after 1936, the regression line for the internees dips below the regression line for the un-interned Japanese Hawaiians. This gap reaches approximately two years for the 1938 cohort (who were four years old when they entered the camps) and increases for the cohorts who entered the internment camps at younger ages.

Figure 1 raises the question of whether Japanese Hawaiians are a good control group for Japanese internees who were almost exclusively from the West Coast. The identifying assumption is that in the absence of internment, the cross-cohort trend of life spans for West Coast Japanese Americans would be parallel to that of Japanese Hawaiians. Because this assumption is counterfactual, we cannot test it directly. Instead, I test whether the cross-cohort life-span trend of West Coast Chinese Americans is parallel to the cross-cohort life-span trend of Chinese Hawaiians.

Because the SSDI does not have race, I identify Chinese death records by using surnames.

Lauderdale and Kestenbaum (2000) list the 50 most common Chinese surnames. I extracted the death records from the SSDI with Social Security cards issued in California, Hawaii, Oregon, and Washington, and with one of the 50 Chinese surnames listed in Lauderdale and Kestenbaum (2000). Figure 2 uses these data and graphs nonparametric regression lines for Chinese Americans that are analogous to the regression lines in Figure 1. Two features of Figure 2 are notable. First, the lines appear to be parallel, suggesting that the difference-in-differences assumption is satisfied. Second, Figure 2 indicates that West Coast Chinese Americans lived longer than Chinese Hawaiians, whereas Figure 1 suggests that West Coast Japanese Americans died at younger ages than Japanese Hawaiians. The latter feature of Figure 2 suggests that, if anything, using Japanese Hawaiians as a control underestimates the effect of internment on mortality in adulthood. One way to interpret this result is that there was a secular shock that affected all internees plus an additional shock that affected just young internees. The difference-in-differences estimates identifies this additional shock.

Because the data were collected at the same time and the cross-cohort trends are downward sloping, the older cohorts are not entirely comparable with the younger cohorts. We might wonder whether this issue introduces any type of bias. Figures 3-5 address this concern. Figure 3 displays the same local linear smooth line depicted in Figure 1 but with only the deaths that occurred by age 69 to make all cohorts comparable. Again, the nonparametric regression lines for the internees and un-interned Japanese Hawaiians from the 1932 to 1936 cohorts match closely with one another. After these cohorts, however, the nonparametric regression line of the internees dips below that of the Japanese Hawaiians by approximately 2 years, with the gap widening for the youngest cohorts. Figure 4 is analogous to Figure 3 but uses Chinese death records. The same partners that appear in Figure 2 show up in Figure 4. The cross-cohort trends are parallel, and West Coast Chinese Americans lived longer than Chinese Hawaiians possibly because most Asians who migrated from Hawaii to the West Coast did so for greater economic opportunity.

Whereas Figures 1 and 3 only examine the local means, Figure 5 provides evidence of how early childhood conditions affected the entire distribution of age at death. Figure 5 graphs kernel density estimates of the age at death for the internees and Japanese Hawaiians from the 1932 to 1937 birth cohort and the 1938 to 1942 birth cohort. Because the youngest birth

cohort was 69 years old when the death records were collected, I only graph the density for the deaths that occurred between the ages of 30 and 69 (I do not graph the densities below 30 only because they are trivially close to zero, but these deaths do appear in the regression analysis). Again, the densities for both internees and un-interned Japanese Hawaiians match closely with one another for the older birth cohort. I can formally test the null hypothesis that the two samples were drawn from the same distribution by using the Kolmogorov-Smirnov test. This test gives us a p-value of 0.980. Consequently, we cannot reject the null hypothesis. For the younger cohort, however, the probability of death occurring between the ages of 30 and 55 is uniformly higher for the internees than for Japanese Hawaiians. Because the Kolmogorov-Smirnov test for the younger cohort yields a p-value of 0.002, we can reject the null hypothesis that the samples were drawn from the same distribution for all conventional significance levels.

1.5.2 Differences in Differences Results

A simple 2×2 table demonstrates the concept of difference in differences in this setting. Table 2 displays the mean age at death for the internees (the treatment group) and un-interned Japanese Hawaiians (the control group) for the younger and older cohorts. The older cohort in the control group lived an average of 65.89 years, whereas the younger cohort in the control group lived an average of 60.97 years. Because neither of these groups were incarcerated, I interpret the difference of 4.92 years in these means as the secular cohort trend. The older cohort in the treatment group lived an average of 65.8 years, whereas the younger cohort in the treatment group lived an average of 59.05 years–a difference of 6.75 years. Subtracting out the fixed cohort trend, we obtain our difference-in-differences estimate of -1.83 years. The cohort trends are negative because the cohorts are truncated at the same year but at different ages. In the regression analysis, cohort fixed effects will account for that trend. Reproducing Table 2 with only the deaths that occurred before the age of 69 decreases the secular cohort trend (as expected), but the difference-in-differences estimate is hardly changed. For the remainder of the analysis, I leave the deaths that occurred after the age of 69 in the sample, because they increase the sample size and allow me to estimate

the difference between the older internees and the older Japanese Hawaiians more precisely.

I can add controls, cohort fixed effects, and state fixed effects in a regression framework by estimating equations (1) and (2). The estimates from these regressions appear in Table 3. Each column is a separate regression with state and birth cohort fixed effects. Column (1) of Table 3 presents the difference-in-differences estimate of the early childhood internment effect. I estimate that internment during early childhood decreased life spans by -1.63 years. The estimate is significant at the five percent level and standard errors that are robust to heteroskedasticity are in parentheses. Clustering at the state/year level or bootstrapping does not change the significance of these results. Column (3) presents the same regression depicted in Column (1) but with the natural log of age at death as the dependent variable. The results suggest that internment during early childhood decreased life spans by 3 percent. The estimate is significant at the 5 percent level.

Columns (2) and (4) of Table 3 present estimates of equation (2), which allows for different treatment effects for the internees from high socioeconomic status families and low socioeconomic status families. The estimates suggest that early childhood internment decreased life spans by 2.6 years, or 4.6 percent, for the internees from LSES families and that the children from HSES families were unaffected. The coefficient on the LSES treatment effect is significant at the 5 percent level, whereas the coefficient on the HSES treatment effect is statistically equal to zero. Although the coefficient is only significant for the LSES internees, this finding does not mean that only a small group was affected–over 60 percent of the internees had a father with a LSES occupation. Several plausible reasons can explain why internment may have had a greater effect on the children from LSES families. Internees could make purchases within the camps through mail order catalogs. Many internees who were accustomed to the southern California climate were unprepared for the northwestern United States winter, and internees frequently complained about the cold. It is possible that the HSES families could more easily purchase winter clothing and blankets. Similarly, the HSES families may have purchased additional fans to stay cool during the summer months. Another possible explanation is that after the camps closed, the HSES families may have been able to recover from the financial burdens of internment more quickly. Lastly, the HSES families may have increased their human capital investments in children who spent

their early childhoods within the camps and thereby offset any negative early childhood effects.

Instead of analyzing age at death as a continuous variable, another way to analyze the data is to examine the probability of early death. Table 4 presents the marginal effects of probit regressions for an indicator variable equal to one if the observation died by a given age on the same set of regressors presented in equation (2). Each column is a separate probit regression, and includes state and birth year dummies. The results suggest that early childhood internment increased the probability of dying by the ages of 50, 55 and 60 by 6, 10 and 9 percentage points for the LSES internees, respectively. The coefficients for the HSES internees are never statistically different from zero. The fact that the marginal effects increase from 4 to 10 percentage points between 45 and 55 suggests that many of these premature deaths occurred during their late 40s and early 50s.

1.5.3 The Effect of Temperature

If early childhood environments alter adult mortality risk, then variations in the camp conditions should predict the average treatment effect by camp. The camps were standardized along many dimensions. For example, the WRA built bathrooms, barracks, and mess halls at each camp to service the same number of internees. However, the camps did differ in geography. Although most internees were originally from California, they were relocated to cold climates in Colorado, Idaho, Utah, and Wyoming, hot and dry climates in Arizona and southern California, and warm and wet climates in Arkansas.

To analyze the effect of camp climate on the average treatment effect, I estimate the following regression:

$$\begin{aligned} y_{icsr} &= \alpha + \beta_c + \gamma_s + \delta_r L_i + \phi L_i \times \mathbf{1}\left[c \geq 1938\right] \\ &\quad + \omega L_i \times \mathbf{1}\left[c \geq 1938\right] \times \text{TEMP}_r + \epsilon_i, \end{aligned} \tag{1.12}$$

where y_{icsr} is the life span of individual i, born into birth cohort c, with a SSN issued in state s, and assigned to relocation center r. The parameter δ_r varies by relocation center, and

picks up any fixed level camp differences unrelated to birth cohorts. The variable TEMP_r is the historical mean temperature in January in the town closest to relocation center r.

The results from this regression are in Table 5. In the first column, the dependent variable is life span; in the second column, the dependent variable is the log of life span. The regression results suggest that incarceration during early childhood decreased life spans (significant at the 5 percent level for both regressions), and that the effect is larger for internees incarcerated in relocation centers located in colder climates (the p-values are 0.064 and 0.053, respectively). The average mean January camp temperature was 39.7 for internees incarcerated in early life. This implies that the average treatment effect decreased life spans by 1.7 years or 3.1 percent. Figure 6 displays a scatter plot of the average treatment effect by camp against the mean January camp temperature. The average treatment effect is more negative for internees incarcerated in cold climate camps, and the treatment effect is approximately zero for the warmest camps. For example, early childhood incarceration decreased life spans more for internees incarcerated at Heart Mountain, WY, or Granada, CO, than for internees incarcerated at Gila River, AZ. The mechanism behind these results could be exposure to the cold or something correlated with colder climates, such as incidence of cold weather diseases.

1.5.4 Selection and Endogenous Fertility

Before January of 1942, West Coast Japanese Americans had no reason to believe that they would be relocated to internment camps while other Japanese Americans would be allowed to continue living their lives. However, internees may have changed their fertility decisions in response to learning about their future incarceration. If true, this response could explain a negative estimated treatment effect even in the absence of a true treatment effect. For example, suppose that high socioeconomic status parents postponed having children because they did not want them to grow up in a harsh environment. Then the children born in the camps during 1942 may have had lower life spans even in the absence of an early childhood internment effect because they came from lower socioeconomic status families.

One could argue that selection on fertility may have occurred earlier, perhaps after Pearl

Harbor. However, for selection following Pearl Harbor to explain the results, it must have affected West Coast Japanese Americans more than Japanese Hawaiians. Given the location of Pearl Harbor on the Hawaiian Islands and the strategic geographic importance of Hawaii to the War, this explanation seems highly unlikely.

To test whether selection on fertility can explain the results, I re-estimate equations (1) and (2) by dropping the observations born in 1942. This restriction implies that every individual in the sample was conceived by the first quarter of 1941 and eliminates the possibility that parents made their fertility decisions in response to the news of internment or Pearl Harbor. These estimates appear in Table 6. The estimates are similar to the estimates in Table 3. The estimates suggest that internment during early childhood decreases life expectancy by 1.9 years and 3.6 percent on average, both of which are significant at the 5 percent level. Furthermore, early childhood internment affects the internees from low socioeconomic status families more, decreasing their life expectancies by 2.7 years and 5.2 percent.

1.6 DATA TRUNCATION

Because this study uses death records collected in 2011, the data is truncated. I do not observe deaths that occurred after 2011. Furthermore, the Social Security Administration did not collect death records before 1962, and consequently, the SSDI contains few deaths that occurred before then. If there was higher infant mortality in the camps, then my estimates might understate how much early life incarceration decreased life spans. Right truncation could potentially bias my results in the other direction. For example, suppose that the internees that suffered permanent health effects from internment have already died, and that the surviving internees will live normal life spans. In this case, the true effect will be some weighted average of my estimate and zero.

I address right truncation using life expectancy data from the Social Security Administration (SSA). From the SSA life tables, I know approximately what percent of individuals live beyond a certain age, and what their life expectancies are from that age. For example, approximately 44 percent of individuals from the 1932 birth cohort would have still be alive

in 2011, with a life expectancy of 8.54 additional years. Table 7 uses this data to address what the average treatment effect would be if every internee who was negatively affected by treatment had already died by 2011, and if the surviving internees were to live their full life expectancies. I do this using the following formula:

$$\begin{aligned} E[y] &= Pr(\text{died by 2011})E[y|\text{died by 2011}] \\ &\quad + Pr(\text{dies after 2011})E[y|\text{dies after 2011}] \end{aligned} \tag{1.13}$$

I observe $E[y|\text{died by 2011}]$ in the SSDI data. Approximate values for the other terms are in the SSA life tables for the 1940 birth cohort. Table 7 uses this data to estimates the average treatment effect using differences in differences under the assumption that all internees who had their lives shortened from early childhood incarceration have already died. The results suggest that even if the surviving internees are unaffected by treatment, then internment during early childhood still decreased life spans by an average of 0.64 years.

I can also explore how long beyond their life expectancies internees incarcerated during early childhood would have to live to make the average treatment effect zero. Assuming all three other groups do not live beyond their life expectancies (internees from the 1932-1937 cohort, Japanese Hawaiians from the 1932-1937 cohorts, and Japanese Hawaiians from the 1938-1942 cohort), then those incarcerated during early childhood who are still living would have to live 1.05 years beyond their life expectancies to make the average treatment effect zero.

1.7 CAUSE OF DEATH

Cause of death is not available in the Social Security Death Index. To analyze how early childhood incarceration affects cause of death, I turn to the Multiple Cause of Death Mortality Data from the National Center of Health Statistics. This data set contains the primary cause of death classified according to the ninth and tenth editions of the International Classification of Disease. Unfortunately, this data do not contain personal identifies such as name,

birth date, or Social Security Number. Consequently, I cannot use the same name-linking strategy from the previous sections. Instead, I exploit the fact that 97 percent of Japanese American on the West Coast (Arizona, California, Oregon, and Washington) were incarcerated within internment camps, whereas only 1 percent of Japanese Americans in Hawaii were incarcerated. Consequently, race together with state of birth approximately determine internment status. However, geographic identifiers such as a state of birth are only available in the Multiple Cause of Death Mortality data from 1979 to 2004. I further restrict the sample to males.

Table 8 displays the frequencies of disease categories for this sample. The most common causes of death are diseases of the circulatory system (38.6 percent), neoplasms (36.3 percent), and diseases of the respiratory system (5.6 percent). No other disease category accounts for more than five percent of deaths in the sample.

I estimate the following multinomial logit model:

$$p_{ij} = \frac{e^{\alpha_{cj}+\beta_j \mathbf{1}[\text{West Coast}]+\gamma_j \mathbf{1}[\text{West Coast}]\times \mathbf{1}[c\geq 1938]}}{\sum_{k=1}^{15} e^{\alpha_{ck}+\beta_k \mathbf{1}[\text{West Coast}]+\gamma_k \mathbf{1}[\text{West Coast}]\times \mathbf{1}[c\geq 1938]}} \tag{1.14}$$

where p_{ij} is the probability that individual i dies from cause j. The parameter α_{cj} is a dummy variable for each birth cohort c, $\mathbf{1}$ [West Coast] is an indicator variable equal to one if individual i was born in Arizona, California, Oregon, or Washington, and $\mathbf{1}[c \geq 1938]$ equals one if individual i was born during or after 1938. The parameter γ_j indicates whether early childhood incarceration increases or decreases the probability of dying from cause j.

The marginal effect of γ_j for each cause of death is in Table 9. The results indicate that incarceration during early childhood increased the probability that internees died by disease of the circulatory system by 7 percentage points. This estimate is significant at the five percent level. No other cause of death is significant at conventional levels. Because each death is classified in one of the fifteen disease categories, the marginal effects sum to zero.

Taken together with the previous section, these results suggest that early childhood climate could increase the incidence of circulatory diseases. Bhaskaran et al. (2010) found evidence that extreme cold increased the risk of heart attacks in the short term. My results suggest that extreme cold during early childhood might increase the risk of heart attacks in the long run. Barker (1995) argued that fetal malnutrition increases the incidence of heart

disease. However, that work did not relate to early life temperature. More research is needed to answer this question conclusively.

1.8 DISCUSSION AND CONCLUSION

This paper provides evidence that early childhood conditions in Japanese internment camps had profound effects on life spans. I estimate that the male internees who entered the camps within the first four years of life on average died approximately two years earlier. Furthermore, internees from low socioeconomic status families drive almost the entire effect. I find that internment during early childhood decreased life spans by almost three years for LSES children. These results suggest that either financial means helped mitigate the hardships of internment or investments after relocation offset the negative effects of internment. Furthermore, the effect is larger for internees incarcerated in relocation camps with colder climates. Cause of death data from the National Center for Health Statistics suggest that male internees incarcerated during the first four years of life were 7 percentage points more likely to die from diseases of the circulatory system.

A back-of-the-envelope calculation can address whether the $20,000 compensation that the internees received in 1990 was adequate. Kevin Murphy and Robert Topel (2006) estimate that the statistical value of a life-year is between $100,000 and $350,000. This estimate suggests that the internees who were incarcerated during early childhood should have been compensated at least $200,000 for their increased mortality risk alone. Ignoring the financial hardships caused by internment, the internees should have received at least ten times the compensation they did.

Several limitations of these results are noteworthy. Most internees from the 1932 to 1942 birth cohort are still alive, and it is impossible to obtain a full picture of their life spans until this cohort passes away. This fact is the largest reason for the small linkage rate. As more internees die, it will be possible to increase the number of links and to obtain a fuller picture of the effects of internment. However, even if the surviving internees live their full life expectancies, then internment during early childhood would still have decreased life spans

by an average of 0.64 years.

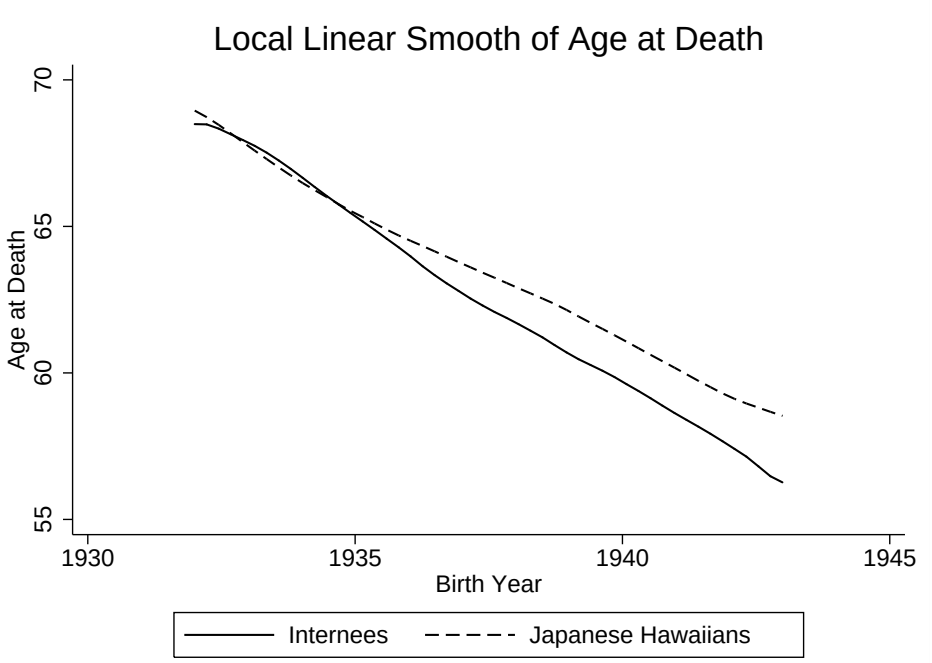
Additionally, this study does not attempt to link the internees from older cohorts (born before or during 1931) to their death records. The data to do so are publicly available, and linking these databases may answer many interesting historical questions, but is outside of the scope of this study. Lastly, this paper does not examine labor market outcomes. Although this question cannot be answered using the same name-linking strategy, using Census data and an identification strategy similar to Chin (2005) may yield insights.

1.9 APPENDIX

The identifying assumption for difference-in-differences estimation is that in the absence of incarceration the cross-cohort life span trends for the West Coast Japanese Americans would have been parallel to that of Japanese Hawaiians. Formally, this assumption can be written as in equation (3). Because this assumption is counterfactual, I cannot test it using data from Japanese Americans. However, I can test whether this assumption is plausible using data on Chinese Americans. Figures 2 and 4 provide graphical evidence that this assumption is satisfied for Chinese Americans. I discuss these figures in detail in the Result section. Here, I formally test the identifying assumption using Chinese data.

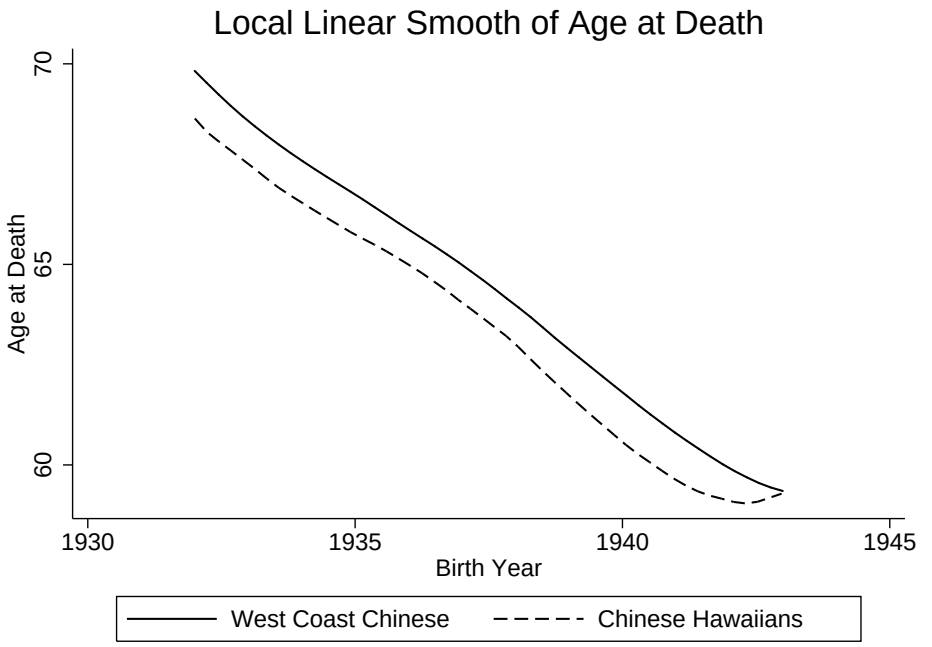
Using data from Chinese Americans that appear in the SSDI with Social Security Cards issued in California, Hawaii, Oregon, and Washington, I regress life spans on a set of dummy variables for state and for each birth cohort. If the cross-cohort trends are parallel, then the residual for each state/cohort combination should be statistically equal to zero. The residuals from this exercise appear in Table 10. For all 22 combinations (11 birth cohorts from the West Coast and 11 birth cohorts from Hawaii), the residuals are never statistically different from zero at the 10 percent significance level.

Figure 1: Age at Death of Japanese Americans by Birth Year



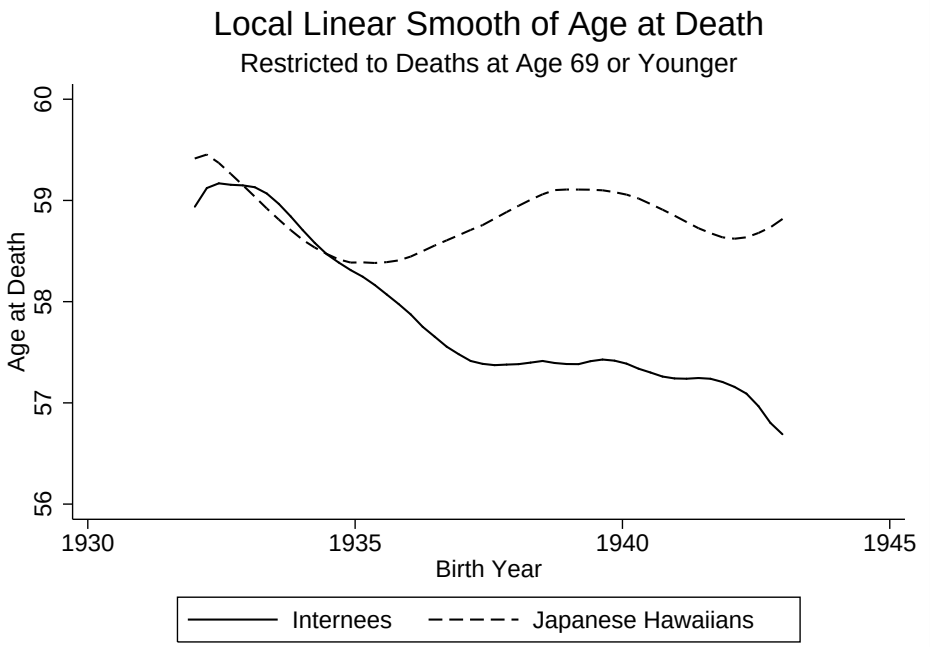
Notes: Internees data are from the WRA records linked to the SSDI. Japanese Hawaiians are the unlinked death records with Japanese surnames and social security cards issued in Hawaii. The bandwidths are 1.5 for both lines. The kernel function is the Epanechnikov. The data are restricted to males who have died by August 2011.

Figure 2: Age at Death of Chinese Americans by Birth Year



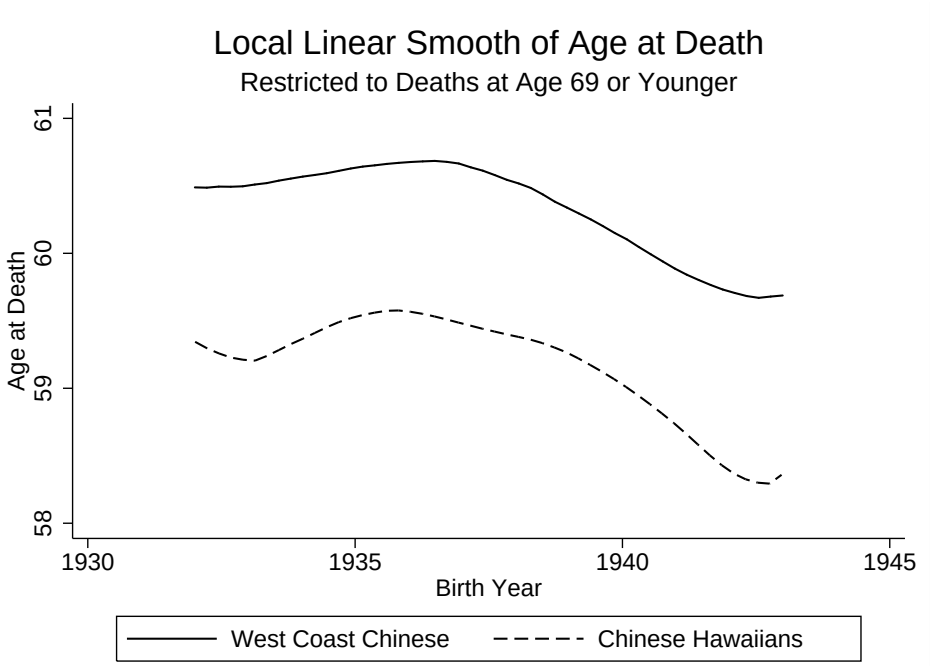
Notes: Data come from the SSDI death records with one of the 50 most common Chinese surnames according to Lauderdale and Kestenbaum (2000). The West Coast includes death records with Social Security Cards issued in California, Oregon and Washington; Chinese Hawaiians are the death records with Social Security Cards issued in Hawaii. The deaths may have occurred anywhere in the United States. The bandwidths are 1.5 for both lines. The kernel function is the Epanechnikov.

Figure 3: Age at Death of Japanese Americans (Restricted Sample)



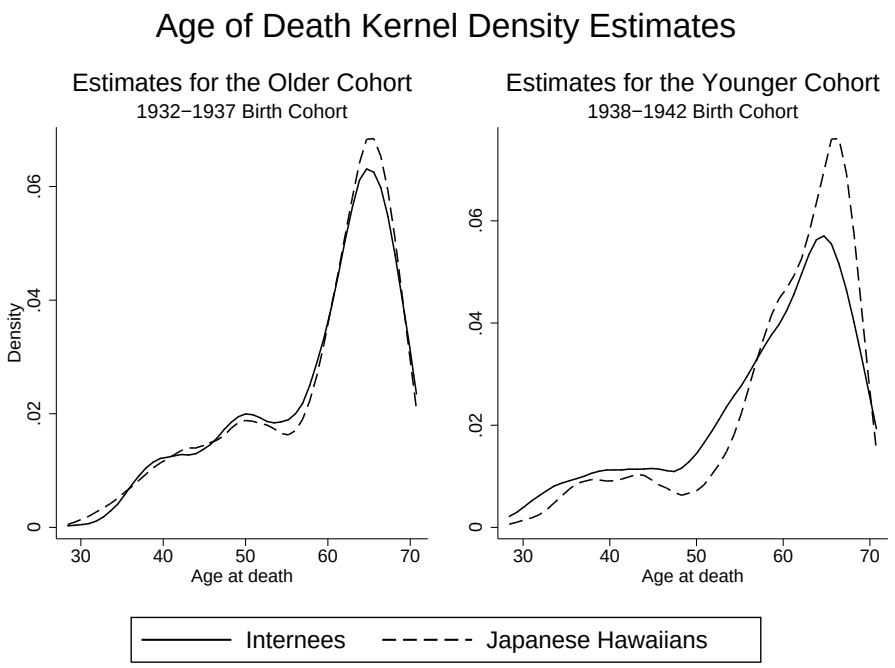
Notes: Internees data are from the WRA records linked to the SSDI. Japanese Hawaiians are the unlinked death records with Japanese surnames and Social Security Cards issued in Hawaii. The bandwidths are 1.5 for both lines. The kernel function is the Epanechnikov. The data are restricted to males who have died by August 2011.

Figure 4: Age of Death of Chinese Americans (Restricted Sample)



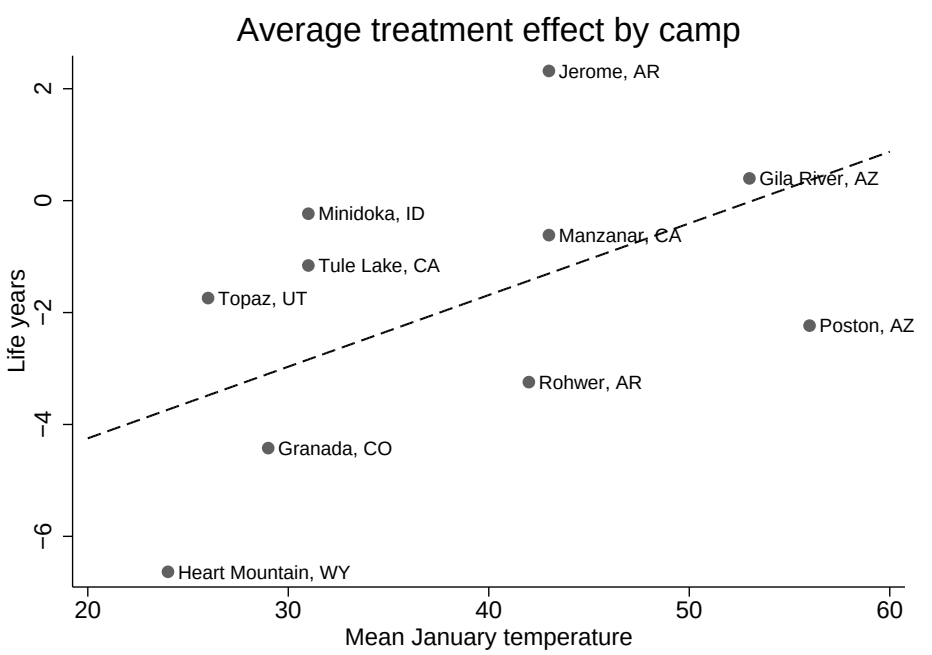
Notes: Data come from the SSDI death records with one of the 50 most common Chinese surnames according to Lauderdale and Kestenbaum (2000). The West Coast includes death records with Social Security Cards issued in California, Oregon and Washington; Chinese Hawaiians are the death records with Social Security Cards issued in Hawaii. The deaths may have occurred anywhere in the United States. The bandwidths are 1.5 for West Coast Chinese, and 2 for Chinese Hawaiians (because of a smaller sample size). The kernel function is the Epanechnikov.

Figure 5: Age at Death Density



Notes: Internees data are from the WRA records linked to the SSDI. Japanese Hawaiians are the unlinked death records with Japanese surnames and Social Security Cards issued in Hawaii. The bandwidths are determined using Silverman's rule of thumb. The kernel function is the Epanechnikov. The data are restricted to male deaths that occurred between ages of 30 and 69.

Figure 6: The Effect of Temperature



Notes: The data are from internees linked to the SSDI and unlinked Japanese Hawaiians. The sample is restricted to males. The slope of the line is from a regression estimating equation (12).

Table 1: WRA Record Summary Statistics for Linked and Unlinked Internees

Full Sample	Linked Mean	Unlinked Mean	Difference	p-value
HSES	0.365	0.364	0.002	0.915
	(0.015)	(0.006)	(0.016)	
City population at least 25,000	0.510	0.526	-0.017	0.318
	(0.016)	(0.006)	(0.017)	
Born in California	0.825	0.832	-0.007	0.557
	(0.012)	(0.004)	(0.012)	

Older Cohort	Linked Mean	Unlinked Mean	Difference	p-value
HSES	0.353	0.350	0.003	0.884
	(0.019)	(0.008)	(0.021)	
City population at least 25,000	0.501	0.509	-0.008	0.710
	(0.019)	(0.008)	(0.021)	
Born in California	0.833	0.842	-0.009	0.566
	(0.014)	(0.006)	(0.016)	

Younger Cohort	Linked Mean	Unlinked Mean	Difference	p-value
HSES	0.385	0.376	0.009	0.731
	(0.025)	(0.008)	(0.027)	
City population at least 25,000	0.525	0.545	-0.020	0.465
	(0.026)	(0.009)	(0.028)	
Born in California	0.810	0.822	-0.012	0.559
	(0.020)	(0.006)	(0.021)	

Notes: The fathers occupation determines socioeconomic status. HSES jobs include professionals, semi-professionals, skilled craftsmen, managers (except farm managers) and official occupations. LSES occupations include farm workers, fishermen, service workers, unskilled laborers, salesmen and clerical occupations. The older cohort includes internees born between January of 1932 and December of 1937, whereas the younger cohort includes internees born between January of 1938 and December of 1942. The sample is restricted to males.

$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 2: Mean Age at Death

	Japanese Hawaiians	Internees	Difference
Older Cohort	65.890	65.795	0.095
	(0.247), N=1753	(0.402), N=671	(0.470)
Younger Cohort	60.969	59.048	1.921
	(0.328), N=913	(0.556), N=395	(0.618)
Difference	4.921	6.747	-1.826
	(0.416)	(0.675)	(0.779)

Notes: Internees are the WRA records linked to the SSDI. Japanese Hawaiians are the unlinked SSDI death records with Japanese surnames and Social Security Cards issued in Hawaii. The sample is restricted to males. The older cohort includes individuals born between January of 1932 and December of 1937, whereas the younger cohort includes individuals born between January of 1938 and December of 1942.

Table 3: The Early Childhood Internment Effect on Life Expectancy

Dependent Variable	Age at Death		Log of Age at Death	
	(1)	(2)	(3)	(4)
Treatment Effect	-1.629^{**}		-0.029^{**}	
	(0.793)		(0.015)	
LSES Treatment Effect		-2.620^{**}		-0.046^{**}
		(1.027)		(0.019)
HSES Treatment Effect		0.208		0.003
		(1.126)		(0.021)
N	3732	3732	3730	3730
R^2	0.095	0.095	0.078	0.077

Notes: Data are from the WRA records linked to the SSDI, and unlinked death records with Japanese surnames that were issued in Hawaii. All regressions include birth-year fixed effects, and state-of-birth fixed effects. The father's occupation determines socioeconomic status. HSES jobs include professionals, semi-professionals, skilled craftsmen, managers (except farm managers) and official occupations. LSES occupations include farm workers, fishermen, service workers, unskilled laborers, salesmen and clerical occupations. The sample is restricted to males. Heteroskedasticity-robust errors are in parentheses.
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 4: Probit Model Marginal Effects for Probability of Early Death

Dependent Variable:						
Indicator if Age at Death is	≤ 40	≤ 45	≤ 50	≤ 55	≤ 60	≤ 65
	(1)	(2)	(3)	(4)	(5)	(6)
LSES Treatment Effect	0.020	0.038*	0.064**	0.104***	0.089**	0.082*
	(0.016)	(0.023)	(0.029)	(0.033)	(0.039)	(0.047)
HSES Treatment Effect	0.011	0.002	0.003	-0.017	-0.023	0.018
	(0.024)	(0.031)	(0.037)	(0.044)	(0.049)	(0.058)
N	3732	3732	3732	3732	3732	3732
Pseudo R^2	0.044	0.037	0.020	0.020	0.035	0.055

Notes: Data come from the WRA records linked to the SSDI, and the unlinked Japanese Hawaiians. Marginal effects $\frac{\partial y}{\partial x}$ are reported with delta-method standard errors in parentheses. All regressions include state and birth-year dummies and a dummy variable for each of the nine occupational categories for the internees father.
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 5: The effect of camp temperature on the ATE

	(1)	(2)
Treatment indicator	-6.809^{**}	-0.142^{**}
	(2.94)	(0.062)
Treatment indicator interacted with mean January camp temperature	0.128^{*}	0.0027^{*}
	(0.069)	(0.0014)
N	3,732	3,730
R^2	0.098	0.08

Notes: Data come from the WRA records linked to the SSDI, and the unlinked Japanese Hawaiians. Robust standard errors are in parentheses. All regressions include state and birth-year dummies and a dummy variable for each relocation center.
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 6: The Early Childhood Internment Effect on Life Expectancy Excluding the 1942 Birth Cohort

Dependent Variable	Age at Death		Log of Age at Death	
	(1)	(2)	(3)	(4)
Treatment Effect	-1.850^{**}		-0.036^{**}	
	(0.837)		(0.016)	
LSES Treatment Effect		-2.719^{**}	-0.052^{**}	
		(1.069)		(0.020)
HSES Treatment Effect		-0.041		-0.002
		(1.208)		(0.023)
N	3497	3497	3496	3496
R^2	0.083	0.081	0.068	0.067

Notes: Data are from the WRA records linked to the SSDI and unlinked death records with Japanese surnames that were issued in Hawaii. All regressions include birth-year fixed effects and state-of-birth dummies for California, Hawaii, Oregon and Washington. The fathers occupation determines socioeconomic status. HSES jobs include professionals, semi-professionals, skilled craftsmen, managers (except farm managers) and official occupations. LSES occupations include farm workers, fishermen, service workers, unskilled laborers, salesmen and clerical occupations. The sample is restricted to males. Heteroskedasticity-robust standard errors are in parentheses.

$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 7: The average treatment effect if those affected have already passed

cohort	truncation age	proportion living	life expectancy from truncation age	Life span in truncated data: linked internees	Hawaiians	difference	Life spans from equation (13): internees	Hawaiians	difference
1942	69	0.64362	14.24	57.597	58.264	-0.667	74.101	74.339	-0.238
1941	70	0.63469	13.63	57.153	60.103	-2.951	73.958	75.035	-1.078
1940	71	0.61621	13.02	60.164	60.605	-0.440	74.864	75.033	-0.169
1939	72	0.597	12.42	59.224	61.789	-2.565	74.266	75.300	-1.034
1938	73	0.57705	11.84	61.108	63.185	-2.078	74.802	75.681	-0.879
1937	74	0.55637	11.26	62.853	63.138	-0.285	75.320	75.446	-0.126
1936	75	0.53494	10.69	62.082	63.992	-1.910	74.711	75.599	-0.888
1935	76	0.51267	10.13	65.278	64.295	0.984	75.968	75.489	0.479
1934	77	0.48949	9.59	65.750	66.292	-0.542	75.951	76.228	-0.277
1933	78	0.46537	9.06	68.012	66.906	1.105	76.876	76.285	0.591
1932	79	0.44032	8.54	68.395	68.437	-0.043	76.825	76.849	-0.024
1932-1937						-1.740			-0.679
1938-1942						-0.115			-0.041
DD						-1.625			-0.639

Notes: The proportion living data and life expectancy from truncation age data are from Bell and Miller (2002) for the 1940 birth cohort of males. The age at death conditional on death by 2011 come from SSDI data linked to the roster of internees and un-linked Japanese Hawaiians (see previous sections). The last three columns are from equation (13).
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 8: Multiple cause of death data frequencies

Disease categories	frequency	percent
Infectious and parasitic diseases	99	2.02
Neoplasms	1,783	36.32
Blood, endocrine, nutritional and metabolic diseases	189	3.85
Mental and behavioral disorders	22	0.45
Diseases of the nervous system	88	1.79
Diseases of the circulatory system	1,894	38.58
Diseases of the respiratory system	273	5.56
Diseases of the digestive system	155	3.16
Diseases of the skin and subcutaneous tissue	3	0.06
Diseases of the musculoskeletal system and connective tissue	17	0.35
Diseases of the genitourinary system	56	1.14
Congenital malformations, deformations and chromosomal abnormalities	11	0.22
Symptoms, signs and abnormal findings, not elsewhere classified	33	0.67
Injury, poisoning and certain other consequences of external causes	206	4.2
External causes of morbidity and mortality	80	1.63
Total	4,909	100.00

Notes: Data come from the Multiple Cause of Death Data from the National Center of Health Statistics. The sample is restricted to male Japanese Americans born in Arizona, California, Hawaii, Oregon, and Washington between 1930 and 1942. The sample only includes deaths during the years 1979 to 2004, because geographic identifies such as state of birth are not available for other years.

Table 9: Marginal effect of treatment for the cause of death

Disease category	marginal effect	s.e.	p-value
Infectious and parasitic diseases	-0.0036	0.0103	0.728
Neoplasms	-0.0462	0.0350	0.187
Blood, endocrine, nutritional and metabolic diseases	-0.0195	0.0135	0.149
Mental and behavioral disorders	0.0078	0.0050	0.113
Diseases of the nervous system	0.0045	0.0090	0.617
Diseases of the circulatory system	0.0704**	0.0345	0.041
Diseases of the respiratory system	-0.0219	0.0199	0.271
Diseases of the digestive system	0.0105	0.0123	0.396
Diseases of the skin and subcutaneous tissue	0.0092	0.0057	0.103
Diseases of the musculoskeletal system and connective tissue	-0.0019	0.0036	0.593
Diseases of the genitourinary system	0.0065	0.0075	0.388
Congenital malformations, deformations and chromosomal abnormalities	0.0005	0.0028	0.861
Symptoms, signs and abnormal findings, not elsewhere classified	0.0026	0.0050	0.601
Injury, poisoning and certain other consequences of external causes	-0.0124	0.0141	0.378
External causes of morbidity and mortality	-0.0078	0.0089	0.382
N	4,909		
Pseudo R^2	0.0140		

Notes: The marginal effects are for a multinomial logit model. The model includes a dummy for every birth year and birth state. Data come from the Multiple Cause of Death Data from the National Center of Health Statistics. The sample is restricted to male Japanese Americans born in Arizona, California, Hawaii, Oregon, and Washington between 1930 and 1942. The sample only includes deaths during the years 1979 to 2004, because geographic identifies such as state of birth are not available for other years.
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 10: Testing the Identifying Assumption

Birth cohort	West coast	Hawaii
1932	0.105	-0.813
	(0.306)	(0.976)
1933	-0.078	0.617
	(0.323)	(1.000)
1934	-0.049	0.392
	(0.327)	(1.025)
1935	0.060	-0.501
	(0.324)	(1.022)
1936	-0.122	0.982
	(0.326)	(1.018)
1937	0.097	-0.757
	(0.336)	(1.003)
1938	-0.063	0.483
	(0.353)	(1.149)
1939	-0.041	0.331
	(0.414)	(1.451)
1940	0.016	-0.122
	(0.418)	(1.337)
1941	0.168	-1.491
	(0.426)	(1.697)
1942	-0.063	0.441
	(0.449)	(1.410)

Notes: Each cell is the average residual from a regression estimating the state and birth cohort fixed effects in equation (3) using data from Chinese Americans that appear in the SSDI. Standard errors are in parenthesis, and no residual is statistically different from zero.
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

2.0 SCHOOL QUALITY AND LABOR MARKET OUTCOMES: JAPANESE AMERICAN INTERNMENT AS A NATURAL EXPERIMENT

In 1942, the United States incarcerated all Japanese Americans on the West Coast, including children, in internment camps. Using non-West Coast Japanese Americans and non-Japanese Asians as control groups, I estimate the effect of attending a War Relocation Authority school on labor market outcomes. Non-linear difference-in-differences estimates suggest that attending school within the internment camps decreased the probability of receiving a post-collegiate education by approximately 4 percentage points and decreased the probability of receiving a college degree by between 2 and 5 percentage points. Furthermore, I estimate that attending a WRA school decreased the returns to a year of schooling by between 1.1 and 1.4 percentage points.

2.1 INTRODUCTION

A central question in education economics is to what extent school quality affects final educational attainment and wages. If the returns to school quality are high, then investments into low performing schools may decrease adult economic disparities a generation later. If the returns to school quality are low, then investments to increase school quality might be better spent elsewhere, for example, on programs intended to improve the health of children. Most research on this question uses cross-sectional or panel data and measures school quality by pupil/teach ratios or teacher pay. The problem with this approach is that school quality is not exogenously determined. The parents most likely to raise successful children may sort into school districts with better measures of school quality. Although conditioning on family

income, parental education, and other measures of socioeconomic status does decrease this problem, it may not eliminate it. Experiments randomly assigning children to class sizes suffer from selection into the experiment. Parents may withdraw students assigned to larger classes and may enroll them in private schools.

This paper analyzes how school quality affects educational attainment and the returns to education by examining the internment of over 100,000 Japanese Americans during World War II. Following Pearl Harbor, the US incarcerated nearly all Japanese Americans, including children, on the West Coast in internment camps for an average of 3.5 years (Ng, 2002). These camps were small communities consisting of rudimentary housing facilities, churches, hospitals, and schools. Incarceration within internment camps unexpectedly and drastically changed the schooling conditions that school age internees faced. For example, the War Relocation Authority (WRA) paid teachers $16 per month to teach overcrowded classes with few books or desks (Ng, 2002). The non-voluntary nature of internment and the standardization of schools within the ten internment camps limits the possibility that parents sort into higher quality schools.

I analyze internment as a natural experiment that plausibly shocked school quality exogenously. Following Aimee Chin (2005), I exploit the fact that 97 percent of Japanese Americans on the West Coast were incarcerated in one of the ten relocation centers, whereas only one percent of Japanese Americans outside of the West Coast were incarcerated in internment camps. To account for the effects of incarceration unrelated to school quality, I compare internees who were of school age in 1942 to internees who had not yet reached school age. I then use data on non-West Coast born Japanese Americans (who were likely not incarcerated) to examine what differences I should have expected in the absence of internment. I then present non-linear triple difference estimates using data from non-Japanese Asians both on and outside of the West Coast. Using data pooled from the 1980, 1990, and 2000 Censuses, non-linear difference-in-differences and triple difference estimates suggest that internees who attended WRA schools were between 4.2 and 4.5 percentage points less likely to attain any post-collegiate education and were between 1.9 and 4.7 percentage points less likely to attain a college degree. Placebo tests using data from Hispanics suggests the difference-in-differences assumption is satisfied. Lastly, using data from the 1980 Census,

I find that treatment decreased the returns to a year of schooling by 1.3 percentage points.

An early paper on school quality is by Card and Krueger (1992) and analyzes the effects of school quality using Census and state-level school quality data. They find that men from states with higher quality schools received a higher return to education. Julian Betts (1995) uses data from the NLSY and finds that high schools matter in the sense that earning are not independent of the high school attended, but that standard measures of school quality such as pupil/teacher ratios do not account for these differences, suggesting that unobservable school characteristics matter more. Using panel data from the British National Development Survey, Dearden, Ferri, and Meghir (2002) find that pupil/teacher ratio does not affect educational attainment, but does have some effect on wages, especially for low ability women at the age of 33. Most of these studies examine small variations in school quality. For example, in Dearden, Ferri, and Meghir (2002), the mean pupil/teacher ratio for males is 17.1 with a standard deviation of 1.9. During the 1940s, the pupil/teacher ratio in California was 27.2 (Card and Krueger, 1992). Internees likely attended schools with similar pupil/teacher ratios before entering the camps. However, within the camps, internees recall pupil/teacher ratios that were at times over 60 (Hirabayashi, 1991).

This paper also relates to work on Project STAR. Chetty et al (2011) find that children randomly assigned to smaller K-3 classes were less likely to be enrolled in college. A problem with the STAR experiment is that families may have selected into and out of the experiments, particularly if high socioeconomic status families were assigned to large classes. The near universal and non-voluntary nature of Japanese American incarceration limits that possibility in this paper.

Section 2 provides further historical background and discusses previous research on the internment of Japanese Americans; Section 3 summarizes the data, and Section 4 presents the non-linear difference-in-differences model in this context; Section 5 discusses the results, and Section 6 concludes.

2.2 HISTORICAL BACKGROUND

On December 7, 1941, the Imperial Japanese Navy bombed Pearl Harbor killing 2,402 Americans. In response, the United States formally entered World War II, declaring war on Japan, Germany, and Italy. Upon entering the War, advisers to President Roosevelt recommended that the government remove all Japanese Americans from the West Coast of the United States. Government officials questioned whether Japanese Americans would remain loyal. They worried that Japanese Americans might spy on the movements of the US military if Imperial Japanese forces invaded the West Coast. In February 1942, President Roosevelt sign Executive Order 9066, which gave the War Department the authority to declare any region an exclusion zone from which it could remove any person without cause. By March 1942, the US Army forcibly evacuated Japanese Americans from Arizona, California, Oregon, and Washington, and the government illegalized voluntary migration from the West Coast.

Despite the strategic location of Hawaii, the government did not relocate non-West Coast Japanese Americans (most of whom were in Hawaii) unless they had connections to the Japanese government or were leaders in the Japanese community (Ng, 2002). Japanese Hawaiians made up 30 percent of the Hawaiian population and relocating them to internment camps would have been devastating to the Hawaiian economy, especially since the War increased manufacturing demand and decreased labor supply. All Japanese Americans were required to surrender any firearms and cameras, however. There was no indication before Pearl Harbor that West Coast Japanese Americans would be incarcerated while non-West Coast Japanese Americans would remain free.

The government established the War Relocation Authority (WRA) to house the internees. The Army first relocated internees to the assembly centers, which were fairgrounds and racetracks hastily converted to house people (Ng, 2002). Internees stayed at the assembly centers while the government constructed the more permanent relocation centers (or internment camps). The internment camps were small communities surrounded by barbed wire.

The WRA assigned large families a single 500 square feet room, whereas smaller families

shared a 320 square foot room; newly married couples sometimes shared rooms with other couples (Ng, 2002). The rooms had no bathroom, kitchen, or running water. Internees used communal bathrooms and dining halls. Communal eating facilities designed to feed 300 people were used by 600 to 900 (Ng, 2002). The WRA rationed only 50 cents for food per day per internee (Jensen, 1999), and internees frequently complained of poor food quality and lack of variety.

By most historical accounts, the WRA schools were lower quality than the schools internees had previously attended. For example, the classrooms lacked adequate heating during the winter. Hirabayashi (1991) quotes a teacher from the Poston War Relocation Center in Arizona as stating, "The weather was so cold it was often difficult to talk to the class. The students found it hard to take notes because their hands would get numb ... before class most of the students gathered around a bonfire."

The WRA schools had difficulty obtaining books, equipment, school supplies, and adequate facilities (Hirabayashi, 1991). Estelle Ishigo, a Caucasian American artist and writer who voluntary entered the internment camps to be with her Japanese husband, wrote:

> There were as many 60 children in a classroom 16 by 20 feet and there were very few books... At first there were no desks; some sat on benches, some on the floor and some stood leaning against the walls to write their lessons (Hirabayashi, 1991).

The lack of adequate schools within the camps inevitably affected the schoolchildren's attitude towards education and possibly steered them to careers that did not require as much education. One school age internee wrote, "Before I was going to be an engineer, but now I decided to be a farmer because of the lack of educational facilities" (Hirabayashi, 1991).

Some researchers have claimed that internment could not have negatively affected the educational outcomes of the interned schoolchildren because they, as a cohort, later became highly educated. Such reasoning, however, does not rule out that internment decreased educational attainment; the interned schoolchildren may have become even more educated in the absence of incarceration.

The first published economics paper analyzing Japanese American internment as a natural experiment is by Aimee Chin (2005). Chin (2005) examines how internment affected the long-run incomes of working-age internees. Internment possibly affected their long-run earn-

ing potential by interrupting their work experience and forcing internees to find new careers for which they were less suited. She finds that internment decreased income 25 years later by between nine and thirteen percent. As a part of her doctoral dissertation, Molly Cooper (2003) wrote a similar paper comparing internees who were adults to internees who were adolescents and to children, and finds that adults were affected the most by internment. In Saavedra (2012), I link WRA authority records to the Social Security Death Index and find that internees who were incarcerated within the first four years of life died approximately two years earlier. Internees with low socioeconomic status fathers drive almost the entire effect, decreasing their life spans by three years.

Both Aimee Chin and Molly Cooper use public-use micro data and a similar identification strategy as this study. Both Chin (2005) and Cooper (2003) compare internees who were adults to internees who were children, and find that the adult internees were more negatively affected. This does not mean that schoolchildren were unaffected, however. Many things affected adults that only indirectly affected children, such as loss of property. Consequently, internees who were adults during incarceration do not appear in any of my samples. Instead, I compare school age internees with internees who were just below school age. This allows me to more directly test whether school quality within the camps negatively affected children.

2.3 DATA

Public-use Census data does not contain internment status. To identifying internees, I follow the same strategy used by Aimee Chin (2005) and exploit the fact that the 97 percent of Japanese Americans on the West Coast were incarcerated, whereas only 1 percent of Japanese Americans outside of the West Coast were incarcerated. Because children are likely to reside in their birth states, race and birthplace almost determine internment status for individuals who were children in 1942. Following Chin (2005), I label an observation as being an internee if they are Japanese and were born in Arizona, California, Oregon, or Washington (the West Coast); I label Japanese Americans born outside of these states as being part of the uninterned Japanese American population.

Data on West Coast born and non-West Coast born Japanese Americans is not sufficient to examine the long-run effects of internment on adult outcomes. I also need data from non-Japanese Asians (both born on and outside of the West Coast) to analyze what differences we should expect between the West Coast born Japanese Americans and the non-West Coast born Japanese Americans in the absence of internment. Unfortunately, most Asians on the West Coast of the United States were Japanese, and there is not sufficient data on West Coast born non-Japanese Asians in any single Census year to complete a thorough analysis. Consequently, I pool data from the five percent samples of the 1980, 1990, and 2000 Censuses and focus on variables that are likely to be stable across time such as educational attainment. For example, an individual surveyed at age 40, 50, or 60 is likely to have the same educational attainment, and possibly the same occupation, at all three ages. An individual's educational attainment would have changed during these ages only if they enrolled in school after the age of 40. For this reason, I drop the small percentage of observations who were enrolled in school at the time of the Census.

I further restrict the sample to individuals who were born between 1930 and 1941. After 1941, relocation centers opened outside of the West Coast (e.g., Arkansas had two relocation centers), and Japanese Americans who lived in these states in 1941 were not subject to relocation. Consequently, I have no way to determine if an individual born in Arkansas in 1943 was born in an internment camp or in the Japanese Arkansas population. Lastly, I also drop individuals who were born outside of the United States, because the educational outcomes of first generation immigrants likely systematically differed from US-born Japanese Americans.

We must also determine which birth cohorts entered the internment camps at school age. Figure 7 displays school enrollment by age using data from the one percent sample of the 1940 Census downloaded from the Integrated Public Use Microdata Series (Ruggles et al., 2010). Six year olds were the youngest cohort in which over half of the population attended school. School enrollment was uncommon for younger children and nearly universal for older children. These statistics suggest that the internees who were born in or before 1936 were of school age when entering the internment camps.

Table 11 presents summary statistics for this sample. The majority of the sample is

Japanese (51.9 percent); 15.8 percent of the sample is Chinese; the remainder are non-Japanese and non-Chinese Asians. The sample is on average highly educated; 90 percent of observations have a high school diploma, 25.5 percent have at least a college diploma, and 10.1 percent have some post-collegiate education. West Coast born Asians compose 27.7 percent of sample. Internees (West Coast born Japanese Americans) compose 17.2 percent of sample (2,968 observations), and 11.0 percent of the sample (1,900 observations) were incarcerated during school age.

2.4 THE ECONOMETRIC MODEL

The ideal experiment to estimate the long-run effects of attending a WRA school would be if the WRA randomly assigned some internees to attend WRA schools and sent the remaining internees to nearby schools that were similar to the ones internees would have attended outside of the camps. Such schools did not exist near the remote WRA relocation centers, and the WRA never conducted such an experiment.

Without such an experiment, we could compare the proportion of school age internees who later obtained a college diploma with the proportion of non-school age internees who later obtained a college diploma. This estimate may be biased if the college graduation rate trends over time. Alternatively, we could compare the proportion of school age internees who later obtained a college diploma with the proportion of non-West Coast Japanese Americans from the same birth cohort who later obtained a college diploma. This estimate may be bias if there is a difference in college graduation rates between the West Coast and Hawaii (where most non-West Coast Japanese resided). To overcome these econometric challenges, I follow a non-linear difference-in-differences strategy. A non-linear difference-in-differences methodology accounts for cohort-invariant differences between the treatment (West Coast born Japanese Americans) and control (Japanese Americans born outside of the West Coast) groups and a secular cohort trend. Both Chin (2005) and Saavedra (2012) use non-West Coast born Japanese as a control group and employ a linear difference-in-differences strategy across birth cohorts (instead of across time) to examine the effects of Japanese American

internment on later-in-life outcomes. Esther Duflo (2001) also uses a linear difference-in-differences strategy across cohorts to examine the impact of a school construction program in Indonesia.

Restricting the control group to non-West Coast born Japanese Americans for the moment, let b index birthplace and let $c \in \{1930, 1931, \ldots, 1941\}$ index birth cohort. Let W_b indicate whether an observation born in birthplace b was born on the West Coast so that

$$S_c = \begin{cases} 1 & \text{if } c \leq 1936; \\ 0 & \text{otherwise.} \end{cases} \tag{2.1}$$

$$W_b = \begin{cases} 1 & \text{if } b \in \{\text{Arizona, California, Oregon, or Washington}\}; \\ 0 & \text{otherwise.} \end{cases} \tag{2.2}$$

Years of education is not available for individuals with fewer than nine years of schooling in Census; additionally, years of education is top coded. For these reasons, I focus a binary variable indicating collegiate attainment. Following Puhani (2008), suppose Y^0 and Y^1 are potential binary outcomes without and with treatment, respectively. An observation is treated if that observation attended school within a WRA relocation or assembly center. Because only West Coast Japanese Americans were incarcerated, it follows that an observation is treated if $W_b \times S_c = 1$. Furthermore, letting Y be the observed outcome, it follows that

$$Y = (W_b \times S_c)Y^1 + (1 - W_b \times S_c)Y^0. \tag{2.3}$$

The binary outcome $Y = 1$ if the individual has attained a specified level of education, and $Y = 0$ otherwise. We could use a linear probability difference-in-differences model. However, a linear probability model would not respect the binary nature of the outcome variable. For this reason, I estimate a non-linear difference-in-differences probit model. Letting the parameter γ be the incremental contribution of treatment to the linear index, we get that

$$Pr(Y^0 = 1|b, c, X) = E(Y^0|b, c, X) = \Phi(\alpha_b + \beta_c + X\theta) \tag{2.4}$$

and

$$Pr(Y^1 = 1|b, c, X) = E(Y^1|b, c, X) = \Phi(\alpha_b + \beta_c + \gamma + X\theta) \tag{2.5}$$

where $E[\cdot|b,c,X]$ is the conditional expectations function, and $\Phi(\cdot)$ is the CDF of the normal distribution. The parameters α_b and β_c are dummies for birthplace b and birth cohort c. The vector X is the set of controls. We do not observe both Y^0 and Y^1, of course. However, by combining equations (3), (4) and (5), we get the following estimable equation:

$$Pr(Y=1|b,c,X) = E(Y|b,c,X) = \Phi(\alpha_b + \beta_c + \gamma W_b S_c + X\theta) \tag{2.6}$$

Puhani (2008) shows that the treatment effect is the marginal effect of $W_b \times S_c$, which is

$$\begin{aligned} \tau &= E(Y^1|b,c,X) - E(Y^0|b,c,x) \\ &= \Phi(\alpha_b + \beta_c + \gamma + X\theta) - \Phi(\alpha_b + \beta_c + X\theta) \end{aligned} \tag{2.7}$$

The identifying assumption of difference-in-differences estimation in this setting is that in the absence of internment, there would have been no shocks specific to any birthplace-cohort combination. We can relax this assumption by adding non-Japanese Asians (both on the West Coast and outside of the West Coast) as another control group and obtain triple difference estimates. The identifying assumption of difference-in-difference-in-differences estimation is that in the absence of internment, there would have been no shock specific to any single race-birthplace-cohort combination. Letting J be a dummy variable indicating whether an observation is Japanese, we can estimate the equation

$$Pr(Y=1|b,c,X) = \Phi(\alpha_b + \beta_c + \delta_1 W_b S_c + \delta_2 W_b J + \delta_3 J S_c + \gamma W_b S_c J + X\theta) \tag{2.8}$$

The treatment effect in this case is, again, the incremental contribution of γ to the probability that $Y = 1$.

In additional to the probit difference-in-difference model, I estimate an ordered probit difference-in-differences model in which I exploit the fact that educational attainment is a ranked outcome. The four educational outcomes are less than high school, a high school diploma, a college degree, and post collegiate education. The marginal effects in this case are the effect of attending a WRA school on the probability of each of the four outcomes occurring. The sum of the marginal effects must sum to zero, of course.

2.5 RESULTS

2.5.1 Graphical Evidence

Figure 8 displays the proportion of individuals with post-collegiate and collegiate education by birthplace, birth year, and race. West Coast born Japanese Americans likely spent several years of their childhoods within WRA relocation centers. Japanese Americans born outside of the West Coast were likely part of the un-interned Japanese American population. Non-Japanese Asians were not incarcerated regardless of birthplace. Figure 8 displays three important facts about the data. First, non-Japanese Asians born in the West Coast become more educated than non-Japanese Asians born outside of the West Coast. Second, West Coast born Japanese Americans from the 1937-1941 birth cohort (who were incarcerated, but not during school age) became more educated than Japanese Americans born outside of the West Coast from the same birth cohort. It appears that West Coast born Japanese Americans were between four and nine percentage points more likely to attend some form of post-college education for this birth cohort. Third, this advantage disappears for the 1930 to 1936 birth cohort–the birth cohort that would have been of school age in 1942. For this birth cohort, Japanese Americans born on the West Coast became no more educated than Japanese American born outside of the West Coast.

I argue that in the absence of internment, West Coast Japanese Americans would have become more educated than Japanese Americans born outside of the West Coast, but the poor school quality within the internment camps eliminated that advantage for school age internees. Most of the internees who were not of school age would have left the camps in time to attend regular schools. This hypothesis hinges on the assumption that in the absence of internment, West Coast Japanese Americans would have become more educated than non-West Coast Japanese Americans. Because this assumption is by definition counterfactual, I cannot directly test it. However, the data from non-Japanese Asians in Figure 8 provides evidence for the assumptions validity.

2.5.2 The Effect of School Quality on Collegiate Attainment

Table 12 demonstrates how a linear difference-in-differences model would apply in this setting. Table 12 presents the percent of Japanese Americans with some post-college education by birthplace and by school-age status in 1942. Table 12 also presents the differences (holding either birth cohort or birthplace fixed) and the difference in these differences. For non-West Coast Japanese Americans, 12.1 percent of observations from the 1937-1941 birth cohorts attained some post-collegiate education, whereas 9.1 percent of observations from the 1930-1936 birth cohorts did, which gives us a difference of 3.0 percentage points. Because neither of these groups attended WRA schools, this difference reflects the secular cohort trend. For West Coast born Japanese Americans, 18.8 percent of observations from the 1937-1941 birth cohorts attained some form of post-collegiate education, whereas only 9.9 percent of observations from the 1930-1936 birth cohorts did, which gives a difference of 8.9 percentage points. Subtracting this from the secular cohort trend of 3.0 percentage points gives us the treatment effect of -5.9 percentage points. This estimate would coincide with a baseline linear probability difference-in-differences model.

The lower panel of Table 12 repeats this exercise for non-Japanese Asians. The estimated secular cohort trends are similar (2.4 and 2.5 percentage points), and the difference-in-differences estimate is statistically equal to zero. Because non-Japanese Asians were not incarcerated and neither birth cohort would have attended a WRA school, this result suggests that the difference-in-differences parallel trends assumption is satisfied.

Table 13 presents the main non-linear difference-in-differences regression results. Each column displays the average marginal effects from separate probit regressions. The probit difference-in-differences (DD) regressions use only non-West Coast Japanese Americans as a control group, whereas the probit difference-in-difference-in-differences (DDD) regressions also use non-Japanese Asians. Each regression controls for gender and contains a set of dummy variables for birth year, birthplace, and Census year. Standard errors are bootstrapped. Column (1) suggests that attending school within a WRA camp decreased the probability of attaining a college degree by 4.7 percentage points, which is significant at the five percent significant level. The triples differences estimates are smaller, suggesting that

treatment decreased the probability of attending college by 1.9 percentage points, but this estimate is imprecise. The treatment effect on the probability of attaining post-collegiate education is more precisely estimated, suggesting that treatment decreased the probability of attaining post-collegiate education by between 4.2 (DD) and 5.0 (DDD) percentage points, respectively. The estimates are statistically significant at the one and five percent significance levels, respectively.

Results from the order probit model appear in Table 14. The first column presents the average marginal effects from a non-linear DD regression using non-West Coast Japanese Americans as a control group and the second column present the DDD results using non-Japanese Asians as well. The results suggest that attending a WRA school increased the probability of not attaining a high school diploma by between 0.99 and 2.5 percentage points, and increased the probability of graduating with only a high school diploma by between 2 and 2.5 percentage points. Furthermore, treatment decreased the probability of attaining a college degree (but not a post-collegiate education) by between 1.5 and 2 percentage points, and increased the probability of attaining post-collegiate education between 2 and 2.5 percentage points. These coefficients are all significant at the five percent level.

Tables 13 and 14 pools data from the five percent samples of the 1980, 1990, and 2000 Census. Pooling data leaves open the possibility of double counting an individual that appears in more than one Census year. To address how this might affect the results, Table 15 presents non-linear difference-in-differences estimates for the effect on post-collegiate education using only one Census year at a time. These estimates yield coefficients between -0.037 and -0.052, but are less precisely estimated because the sample size is considerable larger when I pool the data. The fact that the magnitude of the coefficients are similar, and statistically significant for at least some regressions in spite of the smaller sample size suggests that double counting is not meaningfully affecting the results.

Table 16 presents the results of a placebo test. Table 16 presents the same regressions as Table 13, but instead of using data from Japanese Americans, Table 16 treats all Hispanic Americans as Japanese Americans. If the coefficient on the interaction term is statistically significant, then we should doubt that the previous estimates are true causal effects from poor school quality within the internment camps. Because there are many more Hispanics

in the United States than Japanese Americans, we should be able to pick up even small differences from zero in the interaction term. The results yield coefficients between -0.002 and 0.002. The estimates are precise yet statistically insignificant. The largest coefficient in this test is 25 times smaller than largest coefficient in Table 13. These results suggest that my previous estimates are due to the conditions within the internment camps, and not a result of a trend on the West Coast.

2.5.3 The Effect of School Quality on Occupational Income Score

Primary school quality could affect adult outcomes beyond educational attainment. For example, primary school quality could influence occupational choice directly or indirectly by affecting adult education attainment. Table 17 addresses the effect on occupational income score. Occupational income score is a measure of occupational earning power, and equals the median income of each occupation in 1950 in hundreds of dollars. For example, the median housekeepers earned $700 in 1950, and thus housekeepers have an occupational income score of seven. The median architects earned $5400 in 1950, and consequently architects have an occupational income score of 54.

I focus on occupation instead of wages or earnings, because occupation is likely to be stable across Census years, which allows me to pool the data. I drop individuals with occupational income score of 0 to omit individuals outside of the labor force. Table 17 presents the results from a linear difference-in-difference and a linear triple differences model in which occupational income score is the dependent variable. I run the regressions both with and without education dummies to assess whether school quality affects occupational choice directly or indirectly through affecting educational attainment. The coefficients are all negative (as expected), but only the first regression is statistically significant at the five percent level. Although the coefficients decrease in magnitude with the education dummies, they remain negative. I would need a larger sample to distinguish whether this is from sampling error of whether school quality indirectly affects occupational choice through channels other than education.

2.5.4 The Returns to Education

To analyze how school quality affects the returns to schooling, I use data on only 1980 census to ensure that all observations are still of working age. Restricting the control group to Japanese Americans outside of the West Coast, I model the effect of school quality on the returns to schooling using the following two-equation system:

$$y_i = \alpha_b + \beta_c + \gamma_s + \delta_i E_i + \epsilon_i \tag{2.9}$$

$$\delta_i = \delta_0 + \delta_1 W_b + \delta_2 S_c + \delta_3 S_c W_b \tag{2.10}$$

where y_i is the log of annual wage income, the parameters α_b and β_c are birth place and birth cohort fixed effects, respectively, and E_i is years of education. The returns to schooling for individual i is δ_i. Equation (10) relates cohorts to their returns of schooling. The dummy variable W_b denotes whether an observation born in birthplace b was born on the West Coast; S_c denotes whether or birth cohort c was of school age in 1942. Equation (10) allows the returns to schooling to vary across birthplace or birth cohort. The parameter δ_0 measures the returns to schooling for non-West Coast Japanese Americans from the 1937-1941 birth cohorts. The parameter δ_1 captures changes in the returns to schooling due to differences in on the West Coast, and the parameter δ_2 captures changes in the returns to schooling due to cohort trends. Under the assumption that there were not cohort-birthplace specific shocks other than treatment, δ_3 captures how attending a WRA school decreased the returns to schooling. Card and Krueger (1992) model the relationship between school quality and the returns to schooling using a similar two-equation system.

Plugging equation (10) into equation (9), we get that

$$y_i = \alpha_b + \beta_c + \gamma_s + \delta_0 E_i + \delta_1 W_b E_i + \delta_2 S_c E_i + \delta_3 S_c W_b E_i + \epsilon_i. \tag{2.11}$$

Because years is schooling is not available for those with fewer than nine years of schooling, those individuals are dropped from the sample. The results from this regression appear in Table 18. Column (1) estimates equation (11) with data from both males or females, and column (2) restricts to sample to males only. The results suggest that the returns to a year of school for Japanese Americans outside of the West Coast from the 1937-1941 was between

6 and 8 percent. These estimates are statistically significant at the 1 percent level. The returns to schooling are approximately 2.7 to 3.0 percent higher on the West Coast, which is significant at the ten percent level. The results suggest that attending schooling within the War Relocation Authority camps decreased the returns to school by between 1.1 and 1.4 percentage points. These estimates are statistically significant at the five and one percent significance level, respectively.

2.6 DISCUSSION

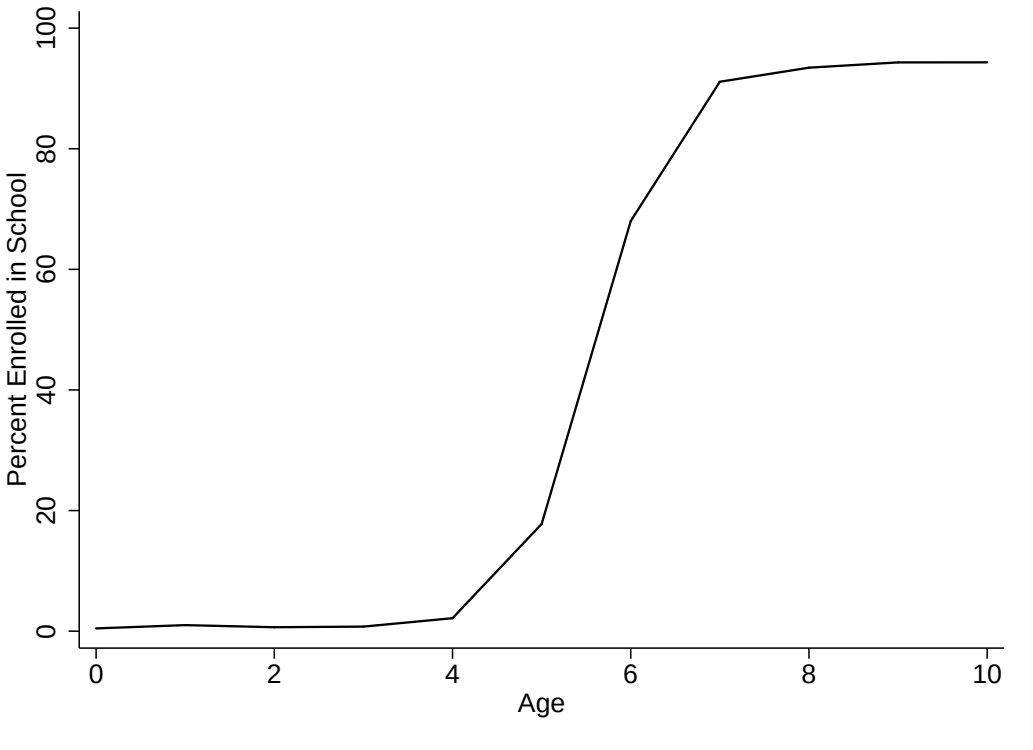
Using un-interned Japanese Americans and non-Japanese Asians as controls groups, this paper provides evidence that attending school within an internment camp decreased adult educational attainment and the returns to schooling. Several non-linear difference-in-differences and triple differences models all support this conclusion. Furthermore, I also find evidence supporting the identifying assumptions hold using data from Hispanics and non-Japanese Asians. Internees who attended War Relocation Authority schools were between 4.2 and 4.5 percentage points less likely to attain some post-collegiate education, and between 1.9 and 4.7 percentage points less likely to receive a college degree. Additionally, attending a WRA school decreased the returns to schooling by between 1.1 and 1.3 percentage points.

There are caveats to this study, however. For example, there is no way to know for sure that the estimated effects is caused by poor school quality, or whether internees benefited from school less than they would have otherwise because of the stress levels within the camps. Unfortunately, these two explanations may be impossibly to tease apart. Nonetheless, the results do suggest that schooling environment, whether it be poor environment at school or poor environment at home during schooling age, seems to have large effects on labor market outcomes.

Furthermore, the change in school quality induced by internment was much larger than changes most education policies propose, however. Nevertheless, schools in wealthy districts have more inputs than minority schools in inner cities. This paper suggests that equalizing school inputs could decrease disparities in educational attainment. School age internees only

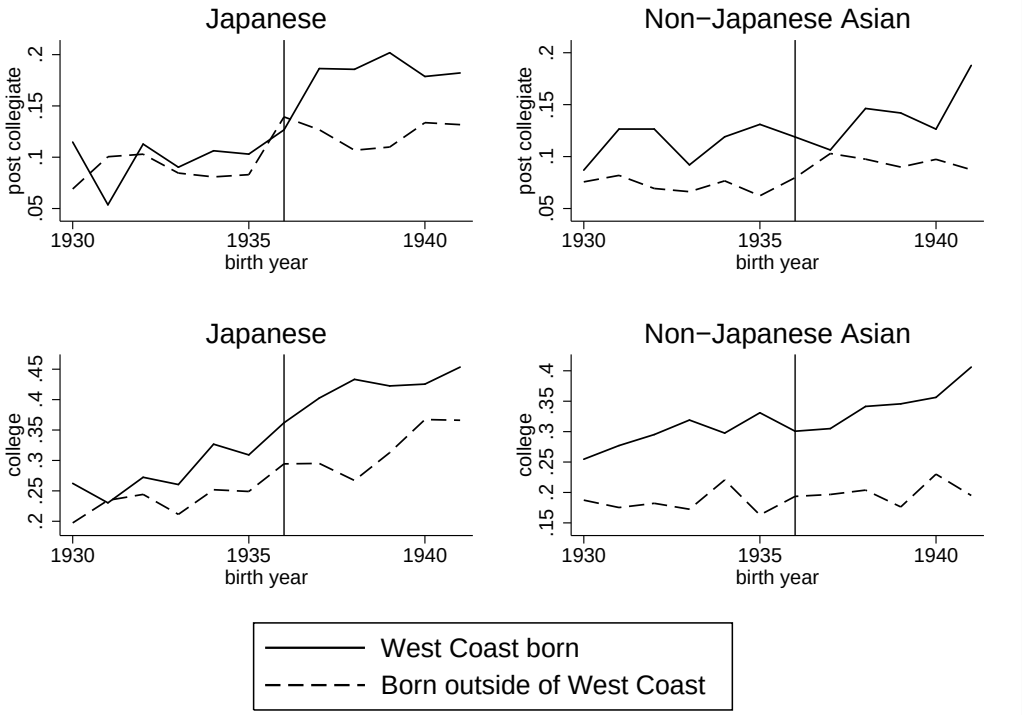
attended WRA schools for 3.5 years on average. If poor performing schools improved for the entire duration of a child's education, the benefits could be even larger.

Figure 7: School enrollment by age in 1940



Notes: Data are from the one percent sample of the 1940 US Census downloaded from IPUMS (Ruggles et al, 2010). Children who were six years old were the youngest cohort in which school enrollment exceeded 50 percent.

Figure 8: Education by race, birth cohort, and birth region



Notes: Data are pooled from the five percent samples of 1980, 1990, and 2000 Censuses. West Coast born Japanese Americans include those born in California, Oregon, and Washington. I restrict to sample to Japanese Americans born in the United States who were not enrolled in school. The West Coast 1930-1936 birth cohorts would have been of school age when entering the relocation centers.

Table 11: Summary Statistics

Variable	Mean	Standard deviation	Minimum	Maximum
Male	0.484	0.500	0	1
Japanese	0.519	0.500	0	1
Chinese	0.158	0.365	0	1
West coast born	0.277	0.448	0	1
School age in 1942	0.597	0.490	0	1
At least high school	0.897	0.304	0	1
At least college	0.254	0.436	0	1
Post-collegiate education	0.101	0.302	0	1
Age	53.99	9.10	38	70
Census year	1989.62	8.08	1980	2000
Birth year	1935.37	3.54	1930	1941

Notes: Data are pooled from the five percent samples of the 1980, 1990, and 2000 censuses. I restrict the data to Asians born in the United States between 1930 and 1941. I further restrict the data to individual who were not enrolled in school.

Table 12: Differences in the population proportion with post-collegiate education by race, birth cohort, and birthplace

Japanese			
Birth Cohort	Born Outside of West Coast	West Coast Born	Differences
1937-1941	0.121	0.188	-0.067
	(0.007)	(0.012)	(0.014)
1930-1936	0.091	0.099	-0.008
	(0.005)	(0.007)	(0.008)
Difference	0.030	0.089	-0.059
	(0.008)	(0.014)	(0.015)

Non-Japanese Asians			
Birth Cohort	Born Outside of West Coast	West Coast Born	Differences
1937-1941	0.094	0.139	-0.045
	(0.005)	(0.012)	(0.014)
1930-1936	0.069	0.113	-0.044
	(0.004)	(0.010)	(0.011)
Difference	0.024	0.025	-0.001
	(0.007)	(0.016)	(0.015)

Notes: Data are pooled from the five percent samples of the 1980, 1990, and 2000 census. I restrict the sample to individuals who were born in the United States and were not enrolled in school. Standard errors are in parentheses.

Table 13: Average marginal effect of attending a WRA school on educational attainment

Variables	College graduate		Post-collegiate education	
	(1)	(2)	(3)	(4)
Attended WRA school	-0.047	-0.019	-0.042	-0.050
	(0.020)	(0.029)	(0.014)	(0.020)
Male	0.092	0.085	0.047	0.051
	(0.008)	(0.007)	(0.007)	(0.005)
Method	Probit DD	Probit DDD	Probit DD	Probit DDD
Percent correctly classified	71.21	74.48	88.82	89.89
Pseudo R^2	0.0332	0.0631	0.0463	0.0625
Observations	8,956	17,244	8,956	17,244

Notes: Data are pooled from the five percent samples of 1980, 1990, and 2000 censuses. I restricted the sample to individuals who were born in the United States and were not enrolled in school. Each column presents the average marginal effects from separate probit regressions. Columns (1) and (3) present the average marginal effects of probit estimates from equation (6) and include only data from Japanese Americans. Columns (2) and (4) present the average marginal effects of probit estimates from equation (8) and include data from Japanese Americans and non-Japanese Asians. All regressions include dummy variable for every birthplace, birth cohort, age, and Census year. Every regressions controls for gender, and regressions in columns (2) and (4) control for race as well. Bootstrapped standard errors are in parentheses.

Table 14: Average marginal effects of treatment in an ordered probit model on educational outcomes

	(1)	(2)
Less than high school	0.0099	0.0252
	(0.0050)	(0.0134)
High school diploma	0.0257	0.0206
	(0.0130)	(0.0110)
College diploma	-0.0158	-0.0203
	(0.0080)	(0.0108)
Post-collegiate education	-0.0198	-0.0255
	(0.0100)	(0.0136)
Pseudo R^2	0.0245	0.0525
N	8956	17244

Notes: Data are pooled from the 5 percent samples of the 1980, 1990, and 2000 Censuses. Standard errors are bootstrapped. Each column is from a separate ordered probit regress with birthplace dummies, birth cohort dummies, gender, and Census dummies. Column (1) only uses non-West Coast born Japanese Americans as a control group, whereas Column (2) also uses non-Japanese Asians. Column (2) includes race dummies. Each cell is the marginal effect on the probability of obtaining a level of education.

Table 15: Average marginal effect of attending a WRA school on educational attainment restricting the data to a single Census year

Dependent Variable: Post-Collegiate Education			
	(1)	(2)	(3)
Attended WRA School	-0.037	-0.039	-0.052
	(0.028)	(0.023)	(0.021)
Male	0.043	0.047	0.050
	(0.013)	(0.010)	(0.011)
Year	1980	1990	2000
Percent Correctly Classified	83.81	90.98	91.49
Pseudo R^2	0.0267	0.0319	0.0422
Observations	2,946	3,115	2,855

Notes: The data from columns (1), (2), and (3) are from the five percent samples of the 1980, 1990, and 2000 Censuses, respectively. I restricted the sample to individuals who were born in the United States and were not enrolled in school. Each column presents the average marginal effects from separate probit regressions estimates of equation (6) and includes only data from Japanese Americans. All regressions include a dummy variable for every birthplace, birth cohort, and gender. Bootstrapped standard errors are in parentheses.

Table 16: Placebo tests treating Hispanics as Japanese

Variables	College graduate		Post-collegiate education	
	(1)	(2)	(3)	(4)
Placebo treatment effect	-0.002	0.002	-0.004	-0.001
	(0.003)	(0.009)	(0.002)	(0.007)
Male	0.043	0.046	0.023	0.025
	(0.002)	(0.002)	(0.001)	(0.001)
Method	Probit DD	Probit DDD	Probit DD	Probit DDD
Percent correctly classified	93.05	91.87	96.74	96.30
Pseudo R^2	0.0349	0.0684	0.0333	0.0540
Observations	97,699	105,987	97,699	105,987

Notes: Data are pooled from the five percent samples of 1980, 1990, and 2000 censuses. I restricted the sample to individuals who were born in the United States and were not enrolled in school. Each column presents the average marginal effects from separate probit regressions. Columns (1) and (3) present average marginal effects of probit estimates from equation (6) and include only data from Hispanic Americans. Columns (2) and (4) present the average marginal effects of probit estimates from equation (8) and include data from Hispanic Americans and non-Japanese Asians. All regressions include a dummy variable for every birthplace, birth cohort, age, and census year. Every regressions controls for gender and regressions in columns (2) and (4) control for race as well. Bootstrapped standard errors are in parentheses.

Table 17: The effect from attending a WRA school on occupational income score

Variables	Difference-in-differences		Triple differences	
	(1)	(2)	(3)	(4)
Attended WRA school	-1.224	-0.769	-1.36	-0.844
	(0.496)	(0.467)	(0.816)	(0.724)
Male	6.826	6.318	6.221	5.672
	(0.222)	(0.203)	(0.168)	(0.145)
Education dummies	No	Yes	No	Yes
R^2	0.1237	0.2245	0.1236	0.2202
Observations	7,515	7,515	14,313	14,313

Notes: Data are pooled from the five percent samples of the 1980, 1990, and 2000 Censuses and were downloaded from IPUMS (Ruggles et al., 2010). Each column presents results from a separate OLS regression. Every regression includes a set of dummy variables for age, birth year, birthplace, and Census year. The triple difference regressions include controls for race as well. Bootstrapped standard errors are in parentheses.

Table 18: The effect of school quality on the returns to schooling

	(1)	(2)
Years of education	0.079	0.063
	(0.013)	(0.014)
Years of education × School age in 1942	0.027	0.030
	(0.016)	(0.016)
West Coast × Years of education	-0.018	0.015
	(0.018)	(0.020)
School age in 1942 × West Coast × Years of education	−0.011	−0.014
	(0.005)	(0.005)
N	2577	1361
R squared	0.273	0.139
Males only	no	yes

Notes: Data are from the 5 percent sample of the 1980 census. The sample is restricted to Japanese American born in the US between 1930 and 1941. The sample excludes the bottom 2 percent of the education distribution, because years of education is group together for individuals with less than nine years of education. The dependent variable is the log of annual wage income. Each regress include birth state and birth cohort fixed effects. Column (1) includes a dummy for gender.

3.0 EARLY-LIFE DISEASE EXPOSURE AND OCCUPATIONAL STATUS: THE IMPACT OF YELLOW FEVER DURING THE 19TH CENTURY

Using city-of-birth data from the 100-percent sample of the 1880 Census merged to city-level fatality counts, I estimate the effect of early-life yellow fever exposure on adult occupational status. I find that *in utero*, neonatal, or postnatal yellow fever exposure decreased adult occupational status for white males with foreign-born mothers, whereas white males with US-born mothers were relatively unaffected. Furthermore, I find no evidence that epidemics 2 to 4 years after birth affect adult occupational status.

3.1 INTRODUCTION

During the nineteenth century, city dwellers carried a higher mortality risk than those in rural areas. Increased urbanization and transportation facilitated the spread of disease. Scientists had not yet discovered the vectors of many diseases, which prevented city official from investing in necessary sanitation. Diseases such as yellow fever, cholera, tuberculosis, dysentery, and typhoid fever increased the mortality rates in cities, a phenomenon described as the "urban mortality penalty." Once public health policies contained these diseases, the urban mortality penalty narrowed, eventually disappearing around 1940 (Haines 2001).

Health economists have linked early-life disease exposure to worse labor-market outcomes (Almond 2006; Barreca 2010; Almond and Currie 2011). 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 on mainly the effects of influenza, malaria, or famine-induced

malnutrition.

This study considers how early-life environment affects adult occupational outcomes in the context of the urban mortality transition by focusing on an epidemic disease that plagued cities: yellow fever. After the discovery of the yellow fever disease vector, the mosquito, public officials took measures to eradicate the disease. Yellow fever has not reached epidemic levels in the United States since 1905. In this paper, I ask whether yellow fever epidemics during early life decreased occupational status during adulthood, and if so, which demographic groups were most affected by these epidemics.

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 in 1851-1853 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.

I identify white males in the 1880 Census who were born in one of five US cities: New Orleans, LA; Mobile, AL; Charleston, SC; Norfolk, VA; and Washington, DC. I then merge this data with city/year level fatality counts. Using an ordered probit model, I find that whites who were born to immigrant mothers during yellow fever epidemics entered lower status occupations than whites with immigrant mothers born during non-epidemic years. For example, the results suggest that whites who were born to immigrant mothers during the 1853 yellow fever epidemic in New Orleans were 8.28 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 whites with US-born mothers. White immigrants were so much more susceptible to yellow fever that it earned the name "the strangers' disease" (Pritchett and Tulani 1995). Thus, this finding provides evidence that early-life disease exposure, as opposed to the wealthy fleeing cities or a stoppage of economic activity, drives the results. Additionally, I find that local yellow fever fatality rates not only during an individual's birth year, but also during the year following birth predicts

lower occupational status, whereas epidemics two to four years after an individual's birth year do not. Because children were less likely to contract yellow fever, these results suggest that the disease may have harmed children through their infected mothers during gestation and lactation. Lastly, I use linear models using 1900 occupational income and unemployment data. I find that early-life yellow fever exposure induce the children of immigrant mothers to enter lower-paying occupations, but they were no more likely to enter occupations with high unemployment rates.

Previous work on the effects of early-life disease exposure has examined influenza and malaria. In a seminal paper, Douglas 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 epidemic and finds evidence that *in utero* influenza exposure reduced educational attainment and wages. Alan 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 vector for malaria. He finds that *in utero* and post-natal malaria exposure worsened labor market outcomes.

The paper proceeds as follows. Section 2 covers the historical background of yellow fever epidemics. Section 3 presents the historical mortality data and the 100-percent sample of the 1880 Census. Section 4 discusses the econometric model, and Section 5 presents the results. Section 6 concludes.

3.2 HISTORICAL BACKGROUND

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 un-infected primates. Human-to-human contact cannot spread yellow fever (except possibly during gestation or lactation). Because mosquitoes are the yellow fever vector and are active mostly in summer, all yellow fever epidemics oc-

curred during the summer months and ended by the first frost of the year. Symptoms of mild infections 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.

Charles 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. 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.

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 from Latin America 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. During the mid-nineteenth century, New Orleans was the worst affected by yellow fever. In 1853, nearly 8,000 New Orleanians died of the disease.

Table 19 displays the number of outbreaks by city between 1668-1873, the number of outbreaks between 1835-1873, and the number of post-1835 outbreaks that killed at least one hundred inhabitants. It is clear from Table 19 that yellow fever plagued New Orleans the worst during the mid nineteenth century. New Orleans had twice as many outbreaks than 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 relatively immune. For example, during the 1854 epidemic in Charleston, SC, 96.1 percent of fatalities were white and 72.9 percent 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. Marys, GA, yellow fever took the lives of 42 of the town's 350 whites, while only taking three of the towns 150 blacks (Patterson 1992). Immigrants were more susceptible because natives would likely have gotten the disease during childhood, when cases tend to be mild, and acquired life-long immunity (Pritchett and Tulani 1995). Epidemic yellow fever during the nineteenth century had a fatality rate between 15 and 50 percent (Patterson 1992) implying that if 8 percent of a city died of yellow fever, then at least another 8 percent were infected and survived.

Because city officials did not know what caused yellow fever, cities tried various measures to stop the disease. American cities 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 percent of a city, Patricia Beeson and Werner 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.

There is some historical evidence to suggest that yellow fever infections of pregnant women negatively affected fetuses. In an 1894 JAMA article, Dr. Joseph Jones argued that yellow fever could be transmitted from the mother to fetus. 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 many similar cases with smallpox, and argued that a mother could transmit yellow fever to the fetus in a similar way. The case study discussed by Dr. Jones provides historical evidence that a mother contracting yellow fever during gestation could harm a child.

Recent case studies have suggested that the virus might spread to children during lactation as well. In 2009, a Brazilian woman received the yellow fever vaccine postpartum. Fifteen days after giving birth to a healthy infant, the woman received a yellow fever vaccination because yellow fever was spreading to a non-endemic region of Brazil. Eight days later, the infant refused to nurse and had a fever. The infant was admitted to the hospital, and an investigation determined that the infant received the yellow fever vaccine virus from breastfeeding (CDC 2010). Kuhn et al (2011) discuss a similar case study from a Canadian woman who received the vaccination to travel to Venezuela. More research is needed in this area, and where historical records are lacking, researchers could turn to the modern yellow fever epidemics in Latin America and Africa.

3.3 DATA

3.3.1 Fatality Data

Yellow fever fatality count data are from J.M Toner (1878). Toner pooled several sources from his medical library to document every yellow fever epidemic in the United States for which data was available. Toner's data appears to be complete after 1820, 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.

I convert fatality counts to fatality rates under the assumption that cities grow linearly between Census years. Figure 9 displays time-series yellow fever fatality rate data for New Orleans, LA, Charleston, SC, Mobile, AL, and Norfolk, VA. The data from Figure 9 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. Furthermore, an epidemic in one port city did not necessarily spread to others. For example, the worst yellow fever epidemic in New Orleans, LA, was in 1853, whereas Norfolk, VA, was not struck by yellow fever until 1855.

3.3.2 1880 Census

The micro occupational data are from the 100-percent sample of the 1880 Census available in the Integrated Public Use Microdata Series (Ruggles et al 2010). I restrict attention to white males born between 1835 and 1864, because labor force participation is nearly universal for this group in 1880. Normally, only state of birth is available in the IPUMS. However, in the 1880 Census the alphabetic birthplace string is available. For the main analysis, I searched Census records in which the enumerator included the individual's city of birth, allowing for misspellings and variations in punctuation. I include individuals born in one of five US cities: New Orleans, LA; Charleston, SC; Mobile, AL; Norfolk, VA; and Washington, D.C. I will refer this sample as Sample 1.

Yellow fever visited New Orleans, Charleston, Mobile, and Norfolk. I include Washington, DC, in the sample for various reasons. Including a city free of yellow fever allows me to estimate birth year fixed effects during years in which the four other cities experienced the disease. To make the birth year fixed effects as representative as possible of what would have happened in other cities in the absence of epidemics, we would like to include a city as similar as possible to those struck by yellow fever. Cities struck by yellow fever had warm summers, were on coasts or rivers, and were below an elevation of 500 feet (Toner 1873). Cities as far north as Baltimore and Philadelphia had smaller outbreaks of yellow fever during the time period, and Washington is the southern most major city that was not struck by yellow fever. Furthermore, Washington is on the Potomac River and has an elevation of only 23 feet. Another reason to include Washington, DC, in the sample is that Washington is the only city in which birth city corresponds with "state of birth." This feature of DC dramatically increases the sample size and allows my to precisely estimate the birth year fixed effects.

These sample restrictions result in 15,289 observations for Sample 1. Sample 1 includes 13,304 individuals born in Washington D.C., which comprise 87 percent of sample. The sample includes 1,631 individuals born in New Orleans, LA; 196 born in Charleston, SC; 139 born in Mobile, AL; and 19 born in Norfolk, VA. The top panel of Table 20 presents summary statistics for Sample 1. The average birth year is 1853.8. Individuals with a foreign-born mother and foreign-born fathers comprise 32 percent and 35 percent of the sample,

respectively. The occupational categories include occupational nonresponse (12 percent), farm workers (5 percent), unskilled laborers (13 percent), skilled laborers (39 percent), and professionals (31 percent). Because income is not available in the 1880 Census, in the analysis I use an ordered probit model treating occupational nonresponse as the lowest category, and professional as the highest. Although this approach is not perfect, it should capture socioeconomic status on average. Additionally, I analyze linear models in which I merge this data with occupational income and occupational unemployment data from 1900 compiled in Appendix A from Preston and Haines (1991).

Because only Census records with city of birth are included in the sample, this sample is not necessarily representative of individuals 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 opposite direction for those born during epidemic years and for those born during non-epidemic years. This kind of bias seems unlikely. To address this concern further, I construct an alternative sample without this problem. Sample 2 includes the universe of white males living in New Orleans, LA, Mobile, AL, Charleston, SC, or Washington, DC, who were also born in the same state. This sample is not perfect either. Sample 2 does not include those born in cities affected by yellow fever who then left the city. Sample 2 also includes those from the countryside of the same state that migrated to the city. Since those from the countryside would not have been exposed to yellow fever, this problem should attenuate the results. However, if the results from using Sample 2 align closely with those from using Sample 1, we can be more confident in the results.

Summary statistics for Sample 2 are in the bottom panel of Table 20. Those in Sample 2 are more likely to have foreign-born parents than those in Sample 1. This feature of Sample 2 is because immigrants are more likely to remain in the city. Sample 2 also contains fewer farm workers, because those who reside in the city are unlikely to work in farming. There is little overlap in the two samples. For example, Sample 2 is twice the size of Sample 1.

3.4 ECONOMETRIC MODEL

Because neither income nor educational attainment are available in the 100-percent sample of the 1880 Census, I focus on the effect of early-life disease exposure 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, farm workers, unskilled laborers, skilled laborers, and professionals. 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

$$o^*_{ibc} = \alpha_b + \beta_c + \gamma Y_{bc} + X_i'\theta + \epsilon_i \tag{3.1}$$

where α_b is a set of dummy variables for each birth year, β_c is a set of dummy variables for each birth city, and Y_{bc} is the yellow fever fatality rate in individual i's birth year b and birth city c. The vector X_i is a set of control variables containing dummy variables for each birthplace of individual i's mother. The term ϵ_i is distributed according to the standard normal. 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

$$\begin{aligned} Pr\left[o_{ibc} = j\right] &= \Phi\left(\mu_j - \alpha_b - \beta_c - \gamma Y_{bc} - X_i'\theta\right) \\ &\quad -\Phi\left(\mu_{j-1} - \alpha_b - \beta_c - \gamma Y_{bc} - X_i'\theta\right) \end{aligned} \tag{3.2}$$

where Φ is the CDF of the standard normal distribution.

One interpretation of this model is to view o^*_{ibc} as unobservable ability. Higher ability individuals enter high-income occupations. Early-life disease exposure reduces ability, and consequently moves the marginal individual into lower earning occupational categories.

I also estimate two variations of equation (1). The first variation allows the effect of yellow fever for individuals with foreign-born mothers, who were more susceptible to yellow fever, to be different from those with US-born mothers. The latent index variable becomes

$$\begin{aligned} o^*_{ibc} &= \alpha_b + \beta_c + \gamma_0 Y_{bc} \times \mathbf{1}\left[\text{Foreign-born mother}\right] \\ &\quad +\gamma_1 Y_{bc} \times \mathbf{1}\left[\text{US-born mother}\right] + X_i'\theta + \epsilon_i \end{aligned} \tag{3.3}$$

where **1** [Foreign-born mother] is an indicator variable that is equal to 1 if the individual's mother was born outside of the United States. Because all observations were born in one of five US cities, there are no immigrants in the sample. However, some were born into immigrant families. The parameters γ_0 and γ_1 represent the effects of early-life yellow fever for individuals born into immigrant families and for individuals born into native families, respectively, on the latent occupation index.

The second variation modifies equation (1) to include yellow fever fatality rates during an individuals year of birth, as well as the year before birth and the four years after birth. As in equation (2), 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 also report the marginal effect on the probability of entering specific occupational categories $\frac{\partial Pr[o_{ibc}=j]}{\partial Y_{bc}}$.

In section 5.2, I will also analyze linear models in which the dependent variable in the average income or average months unemployed for each occupation in 1900. The linear models use the same set of regressors but with non-categorical dependent variables.

3.5 RESULTS

3.5.1 Ordered Occupational Category Results

Estimates from equations (1) and (3) appear in the top panel of Table 21 for Sample 1 (the main sample). The first column displays the estimated coefficients from equation (1) and the second column displays the marginal effect on the probability of entering a professional occupation. The coefficients are negative and significant at five percent level, suggesting yellow fever during an individual's birth year decreased occupational status. The associated 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 1.4 percentage points. This implies that those born during the 1853 yellow fever epidemic

were 8.28 percentage points less likely to become professionals than they would have in the absence of the epidemic.

The third and fourth column display the estimates and marginal effects from equation (2), which allows for the effect of early-life yellow fever exposure to be different for those born to foreign-born mothers and for those born to US-born mothers. The results suggest that the children of US-born mothers were relatively unaffected. The estimated coefficient for those with US-born mothers is close to zero and statistically insignificant. However, the effect of early-life yellow fever exposure is negative and significant at the one percent level for those born to foreign-born mothers. The marginal effect predicts that the children of immigrant mothers who were born during epidemics that killed one percent of a city were 2.2 percentage points less likely to become professionals. Because immigrants were far more susceptible to the disease than natives, this provides evidence that the mechanism is disease exposure and not a temporary stoppage of economic activity.

Figure 10 displays the marginal effects from this specification. Children born during yellow fever epidemics to foreign-born mothers are less likely to be professional, and more likely to be unskilled laborers, farm workers, or blank occupations. The 95 percent confidence interval does not contain zero for any of these results. However, children born during yellow fever epidemics to US-born mothers are unaffected. The point estimate of the marginal effects are close to zero for all five occupational categories, and all 95 percent confidence intervals contain zero.

The results from the previous regressions are from individuals who reported a city of birth in the 100 percent sample of the 1880 Census (Sample 1). It is possible, even if unlikely, that this could bias the results. To see if this result is driven by the sample selection, I repeat the analysis for Sample 2 in the bottom panel of Table 21: all individuals who live in New Orleans, LA, Mobile, AL, Charleston, SC, or Washington, DC, in 1880 and were born in the same state. This sample is not perfect either, because it will contain individuals who were born in the countryside and then moved to the city. However, since these individuals were not exposed to yellow fever, if anything, this should only attenuate the results.

The results are strikingly similar. For example, the estimated marginal effect for the whole population is -1.38 in the first sample (significant at the five percent level), and -1.25

(significant at the one percent level) in the second sample. Similarly, the estimated marginal effect of yellow fever for those born to foreign-born mothers is -2.20 in the first sample, and -1.76 percent in the second sample, both of which are significant at the one percent level. The estimated marginal effects from those born to US-born mothers is statistically insignificant in both samples. The results are remarkably similar given that there is little overlap between these two samples (the second sample is twice the size of the first).

So far, I have only considered the effect of yellow fever epidemics during an individual's year of birth. Table 22 includes the yellow fever fatality rate in an individual's year of birth, as well as the year before birth, and the four years following birth. As in equation (3), I interact these variables with a dummy variable indicating if an individual was born to a foreign-born mother. The results suggest that yellow fever during an individual's year of birth and the following year predict lower occupational status for individuals born to a foreign-born mother. These results are significant at the ten and one percent levels, respectively. Yellow fever epidemics during other years are not significantly different from zero. Epidemics during any age do not significantly affect individuals born to US-born mothers.

Figure 11 visually displays the marginal effects from this specification. As suggested by the results in Table 22, the 95 percent confidence intervals for all occupations and for all ages contains zero for those born to US-born mothers. However, yellow fever epidemics during an individual's birth year or during the year after birth predict a lower probability of entering a professional occupation, and higher probability of becoming unskilled laborers, farm workers, or report occupational nonresponse for those with foreign-born mothers. Epidemics during the year before birth, or two to four years after birth do not significantly predict occupational status.

3.5.2 1900 Occupational Income Results

In the previous subsection, I assumed occupations are ordered. Although this should measure socioeconomic status on average, it has some problems. The highest paid skilled laborers probably earn more than the lowest paid professionals. Unfortunately, occupation is the only meaningful labor-market outcome in the 100-percent sample of the 1880 Census. Oc-

cupational income scores do not exist for the time period. However, Appendix A of Preston and Haines (1991) present average income and average months unemployed by occupation in 1900. In this section, I merge this data to the 1880 Census microdata, and use a linear model to analyze the effects of early-life yellow fever exposure on the log of average occupational earnings and on occupational job security (proxied through average months unemployed).

The results are in Table 23. The top panel uses data from Sample 1 (individuals who reported being born in one of the New Orleans, Mobile, Charleston, Norfolk, and DC), and the bottom panel uses data from Sample 2 (individuals living in New Orleans, Mobile, Charleston, or DC, and were born in the same state). As in the previous subsection the results from the two samples are remarkably similar.

The results suggests that being born during a yellow fever epidemic that killed one percent of the city decreased occupational earnings by between 0.7 percent (Sample 2) and 1.4 percent (Sample 1). These estimates are significant at the five percent and ten percent levels, respectively. Epidemics affected those born to immigrant mothers and did not affect those born to native mothers. A yellow fever epidemic that killed one percent of the city decreased occupational earnings by between 1 percent (Sample 2) and 2.2 percent (Sample 1). These estimates are significant at one and five percent levels, respectively. There is no evidence that early-life yellow fever exposure increased the likelihood of individuals entering occupations with a high risk of unemployment.

Figures 12 and 13 present these results visually. These figures present the residuals of a regression of the log of average occupational earnings on a set of birth year fixed effects, birthplace fixed effects, and mother's birthplace fixed effects. I then plot these residuals against the yellow fever fatality rate during an individual's birth year for those with immigrant mothers and for those with native mothers. Each point is a birth year/birth city combination. The size of each circle is proportional to the number of observations in that birth year/birth city cell. The residuals are downward sloping for those with immigrant mothers, but the lines are flatter for those with US-born mothers, suggesting that yellow fever epidemics do not explain residual income for this group.

3.6 CONCLUSION AND DISCUSSION

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 benefit of eradicating the disease may be higher than previously thought. Furthermore, if the effects from other urban diseases such as cholera, tuberculosis, dysentery, and typhoid fever had similar effects, then the benefits from the urban mortality transitions would be even larger.

There are several caveats to this study. First, city-of-birth data is only available for a small subset of the 100-percent 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 year fever exposure, it should not affect the results. Furthermore, using individuals currently living in these cities and who were born in the same state in 1880 yields remarkably similar results. Linear models using 1900 occupational data suggest that early-life yellow fever exposure induced the children of immigrant mothers to enter lower paying occupations, however, they were no more likely to enter occupations with high unemployment rates.

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. Yellow fever epidemics struck during the summer months, implying that many individuals who were *in utero* during the epidemic would have been born during the same year. However, the results are at least suggestive that yellow fever may have been transmitted to children during gestation and lactation. Only year of birth, and the year following birth predict lower occupational status for those born to immigrant mothers. This timing is consistent with the *in utero*, neonatal, and postnatal stages of life. Second, although yellow fever infected many adults (including mothers), children were less likely to become infected. Cases in which children became infected were typically mild.

Table 19: 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

Source: J.M. Toner (1873)

Table 20: Summary statistics

Sample 1

Variable	obs	mean	s.d.	min	max
Foreign-born mother	15289	0.319	0.466	0	1
Foreign-born father	15289	0.353	0.478	0	1
Birth year	15289	1853.82	7.85	1835	1864
Yellow fever fatality rate during birth year	15289	0.083	0.522	0	12.49
Urban (during adulthood)	15289	0.869	0.337	0	1
Non response	15289	0.124	0.329	0	1
Farm worker	15289	0.047	0.212	0	1
Unskilled laborer	15289	0.129	0.335	0	1
Skilled laborer	15289	0.388	0.487	0	1
Professional	15289	0.312	0.464	0	1

Sample 2

Variable	obs	mean	s.d.	min	max
Foreign-born mother	34603	0.481	0.500	0	1
Foreign-born father	34603	0.522	0.500	0	1
Birth year	34603	1854.49	7.482	1835	1864
Yellow fever fatality rate during birth year	34603	0.444	1.107	0	6.03
Urban (during adulthood)	34603	1	0	1	1
Non response	34603	0.109	0.312	0	1
Farm worker	34603	0.014	0.118	0	1
Unskilled laborer	34603	0.183	0.387	0	1
Skilled laborer	34603	0.315	0.465	0	1
Professional	34603	0.378	0.485	0	1

Notes: Sample 1 includes white males in the 100-percent sample of the 1880 Census who reported being born in either New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, or Washington, DC, allowing for misspellings and variations in punctuation. Sample 2 includes white males in the 100-percent sample of the 1880 Census that lived in New Orleans, LA, Mobile, AL, Charleston, SC, or Washington, DC, and were born in the same state.

Table 21: The effect of yellow fever during an individual's birth year

Panel A: Sample 1

Variable	coefficient	marginal effect	coefficient	marginal effect
Y_b	-0.0408^{**}	-0.0138^{**}		
	(0.0206)	(0.0070)		
$Y_b \times \mathbf{1}$ [US-born mother]			-0.0045	-0.0015
			(0.0345)	(0.0116)
$Y_b \times \mathbf{1}$ [Foreign-born mother]			-0.0651^{***}	-0.0220^{***}
			(0.0233)	(0.0079)
N	15289		15289	
Pseudo R^2	0.0369		0.0369	

Panel B: Sample 2

Variable	coefficient	marginal effect	coefficient	marginal effect
Y_b	-0.0345^{***}	-0.0125^{***}		
	(0.0087)	(0.0032)		
$Y_b \times \mathbf{1}$ [US-born mother]			-0.0119	-0.0043
			(0.0116)	(0.0042)
$Y_b \times \mathbf{1}$ [Foreign-born mother]			-0.0486^{***}	-0.0176^{***}
			(0.0094)	(0.0034)
N	34603		34603	
Pseudo R^2	0.0318		0.0319	

Notes: Sample 1 includes white males in the 100-percent sample of the 1880 Census who reported being born in either New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, or Washington, DC, allowing for misspellings and variations in punctuation. Sample 2 includes white males in the 100-percent sample of the 1880 Census that lived in New Orleans, LA, Mobile, AL, Charleston, SC, or Washington, DC, and were born in the same state. Columns (1) and (3) are coefficients from ordered probit regressions and Columns (2) and (4) are the associated marginal effects on the probability of entering a professional occupation. Each regression contains a set of dummy for each birth year, birth city, and birth state/country of the mother. Robust standard errors are in parenthesis.
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Table 22: The effect of yellow fever during different ages

Variable	coefficient	s.e.	z	p-value
$Y_{b-1} \times \mathbf{1}$ [Foreign-born mother]	-0.0054	0.0226	-0.24	0.811
$Y_{b-1} \times \mathbf{1}$ [US-born mother]	-0.0497	0.0318	-1.56	0.118
$Y_b \times \mathbf{1}$ [Foreign-born mother]	-0.0466	0.0240	-1.95	0.052
$Y_b \times \mathbf{1}$ [US-born mother]	0.0054	0.0352	0.15	0.877
$Y_{b+1} \times \mathbf{1}$ [Foreign-born mother]	-0.0757	0.0270	-2.80	0.005
$Y_{b+1} \times \mathbf{1}$ [US-born mother]	-0.0375	0.0347	-1.08	0.280
$Y_{b+2} \times \mathbf{1}$ [Foreign-born mother]	-0.0376	0.0235	-1.60	0.110
$Y_{b+2} \times \mathbf{1}$ [US-born mother]	0.0081	0.0401	0.20	0.840
$Y_{b+3} \times \mathbf{1}$ [Foreign-born mother]	0.0213	0.0268	0.80	0.426
$Y_{b+3} \times \mathbf{1}$ [US-born mother]	0.0013	0.0272	0.05	0.961
$Y_{b+4} \times \mathbf{1}$ [Foreign-born mother]	-0.0276	0.0351	-0.78	0.433
$Y_{b+4} \times \mathbf{1}$ [US-born mother]	0.0285	0.0346	0.82	0.411
N	15289			
Pseudo R^2	0.0373			

Notes: Sample includes white males in the 1880 Census who reported being born in either New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, or Washington, DC. Each regression contains a set of dummy for each birth year, birth city, and birth state/country of the mother. Standard errors are robust to heteroskedasticity.

Table 23: The effect of yellow fever during an individual's birth year

Panel A: Sample 1

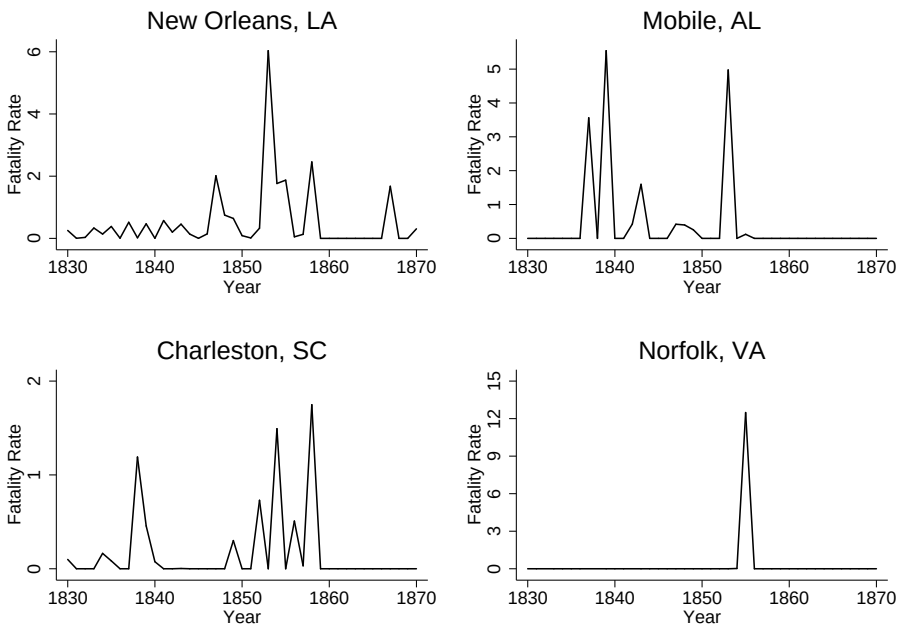
Variable	log(income)		Month unemployed	
	(1)	(2)	(3)	(4)
Y_b	-0.0139^{*}		0.0137	
	(.0076)		(0.0192)	
$Y_b \times \mathbf{1}$ [US-born mother]		-0.0012		-0.0032
		(0.0102)		(0.0303)
$Y_b \times \mathbf{1}$ [Foreign-born mother]		$-.0222^{**}$		0.0256
		(0.0095)		(0.0218)
N	10,934	10,934	14,371	14,371
R^2	0.0562	0.0564	0.0458	0.0458

Panel B: Sample 2

Variable	log(income)		Month unemployed	
	(1)	(2)	(3)	(4)
Y_b	-0.0073^{**}		-0.0024	
	(0.0030)		(0.0095)	
$Y_b \times \mathbf{1}$ [US-born mother]		-0.0020		-0.0222
		(0.0038)		(0.0118)
$Y_b \times \mathbf{1}$ [Foreign-born mother]		-0.0107^{***}		0.0111
		(0.0034)		(0.0103)
N	26,321	26,321	32,592	32,592
R^2	0.0813	0.0814	0.0398	0.0401

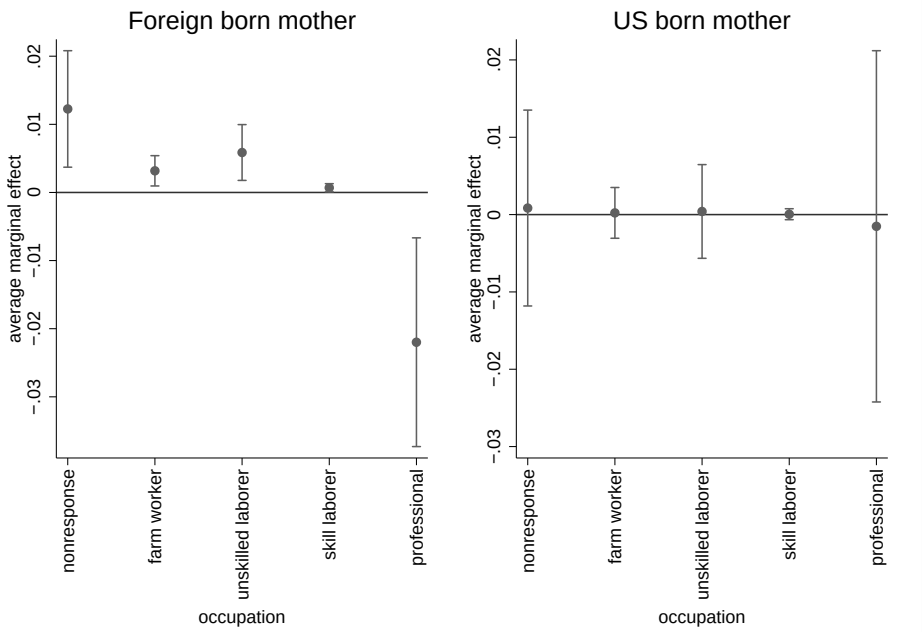
Notes: Sample 1 includes white males in the 100-percent sample of the 1880 Census who reported being born in either New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, or Washington, DC, allowing for misspellings and variations in punctuation. Sample 2 includes white males in the 100-percent sample of the 1880 Census that lived in New Orleans, LA, Mobile, AL, Charleston, SC, or Washington, DC, and were born in the same state. The dependent variable in the log of average occupational income and the average months unemployed for each occupation in 1900 as reported in Appendix A of Preston and Haines (1991). Each regression include birth year and birth place fixed effects, as well as dummies for the mother's birth place. Robust standard errors are in parenthesis.
$*$ 10 percent significance; $**$ 5 percent significance; $***$ 1 percent significance

Figure 9: Yellow fever fatality rates



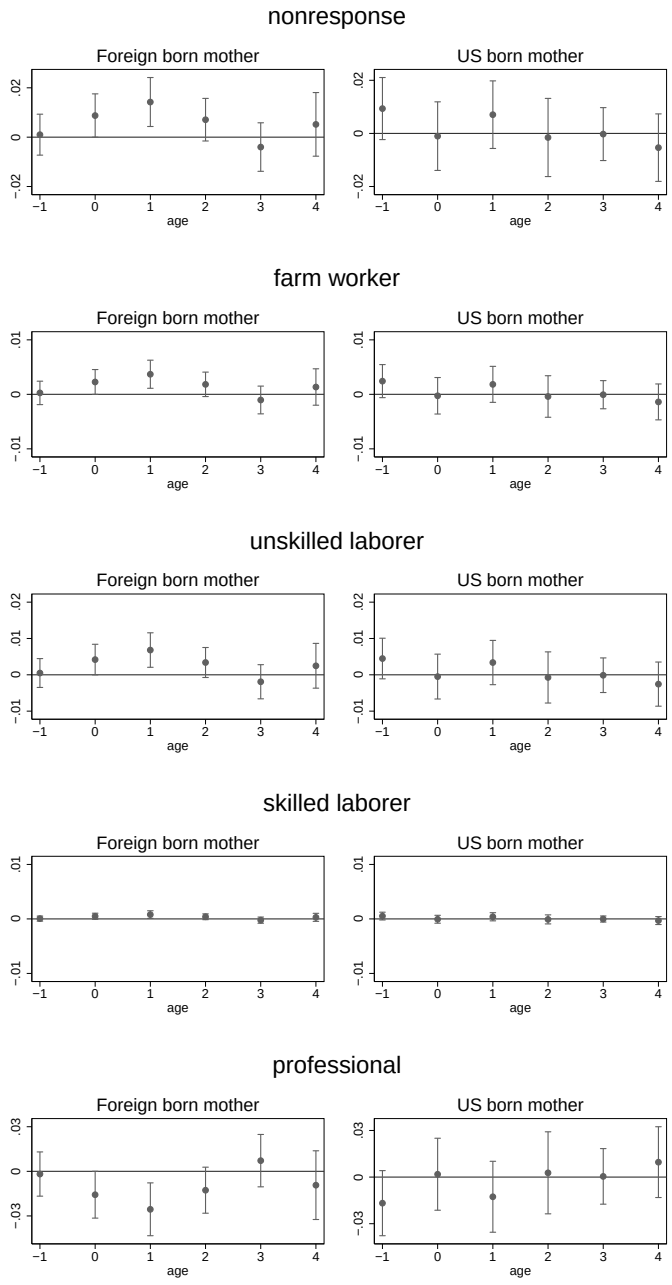
Notes: Data are from Toner (1873). I convert fatality counts into fatality rates by assuming city population grows linearly between Census years.

Figure 10: Marginal effects of yellow fever during an individual's birth year



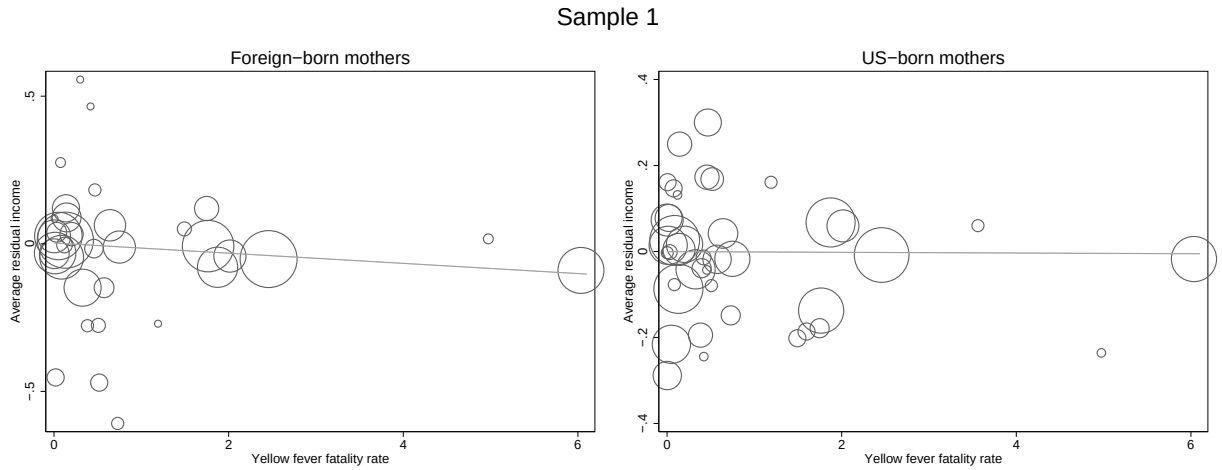
Notes: Sample includes white males in the 1880 Census who reported being born in either New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, or Washington, DC. Marginal effects are from the ordered probit estimates in Table 21.

Figure 11: Marginal effects of yellow fever during various ages of childhood



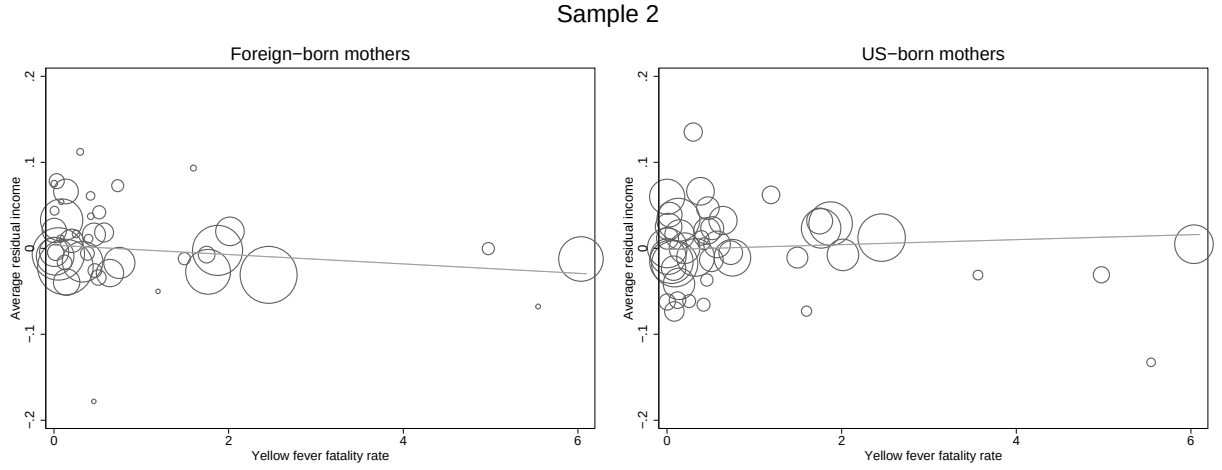
Notes: Sample includes white males in the 1880 Census who reported being born in either New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, or Washington, DC. Marginal effects are from the ordered probit estimates in Table 23.

Figure 12: Average residual income



Notes: Sample 1 includes white males in the 100-percent sample of the 1880 Census who reported being born in either New Orleans, LA, Mobile, AL, Charleston, SC, Norfolk, VA, or Washington, DC, allowing for misspellings and variations in punctuation. The dependent variable is the residual log of occupational income of a regression of average occupational income in 1900 on a set of birth year, birth place, and mother's birth place fixed effects. Each circle is a birthplace/birth year cell and is proportional to sample size.

Figure 13: Average residual income



Notes: Sample 2 includes white males in the 100-percent sample of the 1880 Census that lived in New Orleans, LA, Mobile, AL, Charleston, SC, or Washington, DC, and were born in the same state. The dependent variable is the residual log of occupational income of a regression of average occupational income in 1900 on a set of birth year, birth place, and mother's birth place fixed effects. Each circle is a birthplace/birth year cell and is proportional to sample size.

4.0 MITIGATING THE NEGATIVE EFFECTS OF LOW BIRTH WEIGHT: EVIDENCE FROM QUASI-RANDOMLY ASSIGNED ADOPTEES

Infants who are underweight at birth earn less, score lower on tests, and become less educated as adults. Previous studies have found mixed evidence that socioeconomic status mitigates this effect. In this paper, we reconcile these findings using a unique dataset in which adoptees were quasi-randomly assigned to families. We find that the average income within a zip code mitigates the effects of low birth weight, as in Currie and Morreti (2007), whereas other family characteristics do not, as in Currie and Hyson (1999). These results cannot be explained by differences in genetics, prenatal healthcare or neonatal healthcare. This chapter is co-authored with Brian Beach.

4.1 INTRODUCTION

Infants who are underweight at birth earn less, score lower on tests, and become less educated as adults.[1]. Researchers have found mixed evidence that socioeconomic status (SES) mitigates the negative effects of low birth weight (LBW). Currie and Hyson (1999) find little evidence that SES, as measured by fathers occupational status, mitigates the negative effects of LBW.[2] On the other hand, Currie and Moretti (2007) find that LBW women are more likely to have LBW children, and these effects are larger for individuals born in low-income zip codes.

[1] See Case, Fertig and Paxson (2005), Black et al (2007), and Currie and Almond (2011)

[2] Black et al (2005) also find no evidence that the effect of LBW is mitigated by mothers education, family income, or birth order, but they cannot rule out small effects because of their sample size.

Why these papers find conflicting results remains an open question. Almond and Currie (2011) argue that the differences between Currie and Hyson (1999) and Currie and Moretti (2007) are due to improvements in technology. They note that Currie and Hyson analyze a 1958 birth cohort and that it is possible that there were few effective interventions for LBW infants in 1958. Improvements in neonatal technology that might only be accessible to high SES families may explain the results in Currie and Moretti (2007), which uses data from a later period. Alternatively, these papers might differ because they look at different populations or because they use different measures of socioeconomic status. For example, socioeconomic status is multidimensional and some measures may mitigate the negative effects of LBW whereas other measures might not.

In this paper, we ask whether childhood environment mitigates the effects of LBW using five measures of SES: mothers education, fathers education, family income, family size, and mean neighborhood income. We address this question using data from Sacerdote (2007) in which an adoption agency quasi-randomly assigned Korean orphans to American adoptive families. Because we have random assignment, childhood environment is not confounded by genetics, prenatal health, or neonatal healthcare. Consistent with the findings of Currie and Hyson (1999), we find no evidence that parents education, family income, or family size mitigate the negative effects of LBW. However, mean neighborhood income does mitigate the effects of LBW, which is consistent with Currie and Moretti (2007). Our results indicate that channels other than neonatal healthcare can mitigate the negative effects of LBW. However, this does not imply that neonatal healthcare does not mitigate the effects of LBW. In fact, Prashant et al (2013) find evidence that LBW infants with access to neonatal healthcare have lower mortality rates and score higher on tests.

These results raise the question of how socioeconomic status might mitigate the negative effects of low birth weight through channels other than neonatal healthcare. Healthcare after the neonatal period might treat symptoms that manifest during childhood. Human capital investments might compensate for lower ability induced by poor prenatal health. Alternatively, access to networks might provide opportunities that cushion the effects of LBW. Socioeconomic status could be correlated with any of these channels. The correlation between SES and healthcare quality, however, might differ by country. For example, SES

should be a weaker predictor of healthcare quality in countries with universal healthcare.

Birth weight is determined by gestational age, genetics, and fetal nutritional intake. Infants born prematurely and the children of mothers who were born LBW are more likely to be LBW. Furthermore, infants exposed to air pollution or famines while in utero have lower birth weights, higher infant mortality rates, and higher adult mortality (Currie, Neidell, and Schmieder (2009); Almond et al, (2010); Barker (1995)). Maternal behaviors such as smoking or drinking during pregnancy also negatively influence birth weight (Markowitz et al (2013); Evans and Ringel (1999)). Public policies targeting pregnant women have been successful at increasing birth weights (Almond et al (2011); Currie and Gruber (1996)).

Because many of the determinants of prenatal health are unobservable, researchers often use LBW as a proxy for poor prenatal health. In most data sets, the unobserved determinants of LBW (genetics, prenatal environment, and maternal behaviors) are correlated with childhood SES (both observed and unobserved).[3] In our data, family environment is quasi-randomly assigned; therefore, the unobserved determinants of birth weight are orthogonal to childhood SES.

In addition to short-run health costs, LBW also has long-run economic costs. Individuals who are LBW earn less income, become less educated, and are less likely to be married when they reach adulthood (Case, Fertig, and Paxson (2005); Currie and Hyson (1999)). To identify the causal effect of fetal nutritional intake while holding genetics and family environment constant, many studies have used twin designs (Almond et al (2005); Oreopoulos et al (2008); Black et al (2007); Royer (2009)). Birth weight has the potential to affect long-run economic outcomes through several channels, such as cognitive development and adult health. Consistent with this literature, we find that low birth weight infants earn less and become less educated.

[3]Although children from low SES families are more likely to be LBW, Costa (1998) shows that this difference has narrowed since 1900.

4.2 DATA

Holt International Childrens Services is an organization that places Korean orphans into adoptive families. As of 2005, the program had placed over 100,000 Korean children with American families. A feature of the Holt institution is that children were quasi-randomly assigned to families. Holt assigned children to families using their application date and a first come, first served rule. Because potential applicants did not know whether the next available child is healthy or not, adoptive families could not have strategically timed their applications to increase the likelihood of adopting a healthier child. To be eligible to adopt, adoptive parents had to be married for at least three years, have no more than four children, have a household income greater than 125% of the Federal Poverty Line, and be between the ages of 25 and 45.

Sacerdote (2007) surveyed adoptive families and obtained information regarding their family characteristics at the time of adoption and the adult outcomes for each of their children.[4] Sacerdote linked this data to Holt records and showed that family characteristics do not predict an adoptees initial health, age at time of adoption, or gender. Because family environment was quasi-randomly assigned to adoptees, this data allowed Sacerdote to separately identify the effects of family environment (nature) and genetics (nurture). Sacerdote finds height, weight, and BMI to be determined by genetics; smoking and drinking behavior appear to be influenced by family environment; education and income appear to be influenced by both nature and nurture.

Holt records report the weight that first appears in the adoptees' medical records, but this initial weight is not always an adoptees birth weight. To ensure that we accurately capture birth weight we restrict the sample to adoptees with an initial weight less than 4.5 kilograms, the top of the U.S. birth weight distribution. Figure 14 shows that with this restriction the distribution of Holts initial weights resembles the U.S. birth weight distribution (obtained from the 1988 Integrated Health Interview Series). In the first panel of Figure 1 we plot the truncated adoptee distribution and the distribution of birth weights for the U.S. population.

[4]The response rate of this survey was 34%; however, after re-surveying a subset of the non-respondents Sacerdote finds that non-response was not correlated with the adoptees adult outcomes.

In the second panel we plot the truncated adoptee distribution and the distribution of birth weights for the subset of the U.S. population whose mothers receive Aid to Families with Dependent Children (AFDC). It should be noted that although adoptees are lower birth weight than the U.S. population as a whole, they are similar to children whose mothers were on AFDC. Although we use adoptees with an initial weight less than 4.5 kilograms for our analysis, our regression results are not sensitive to this cutoff. This is illustrated in Section IV, where we relax this restriction.

Table 24 produces the summary statistics restricting Sacerdotes dataset to adoptees that weighed less than 4.5 kg at their initial medical history. The data contains 535 adoptees, of which nearly 450 were at least 25 years old at the time of the survey. We restrict attention to adoptees over the age of 25 when examining adult income, educational attainment, and college attendance. The sample is only 25 percent male, suggesting that females were more likely to be given up for adoption (see Edlund and Lee (2013) for a discussion on son preference in Korea). Most adoptees arrived to the United States around age one. The adoptive mothers had an average of 15.06 years of schooling while the adoptive fathers had an average of 15.89. Adoptees over the age of 25 have an average of 15.03 years of schooling.

Table 25 presents the correlations between each of our five measures of SES. Although income, neighborhood income, and parents education are positively correlated, with the exception of mothers and fathers education, the correlation between any of the other measures never exceeds 0.20. This highlights the multidimensionality of SES and illustrates why papers that only use one measure of SES might find conflicting results.

4.3 RESULTS

Figure 15 visually displays our basic results. Figure 2 presents local linear smooth regressions of the adoptees years of schooling for four family characteristics: mothers education, log of family income, the number of children in the family (including the adoptee), and the log of the average income within the neighborhood. Several features of Figure 2 are notable. First, LBW adoptees become less educated than normal birth weight adoptees for every level

of mothers education, family income, family size, and neighborhood income. Second, the effect of LBW (measured by the gap between the two nonparametric regression lines) remains approximately constant across levels of mothers education, family income, and family size. In other words, these three measures of socioeconomic status are not mitigating the negative effects of LBW. The birth weight gap does narrow for adoptees from higher income neighborhoods. Figure 2 indicates that a LBW adoptee assigned to a low-income neighborhood would attain one-half fewer years of education, on average, than a normal birth weight adoptee assigned to the same neighborhood. However, that gap almost disappears for adoptees from high-income neighborhoods. Neighborhood income appears to mitigate the effects of low birth weight.

These nonparametric regression lines do not control for other variables. We analyze the effect of LBW on adult outcomes and the extent to which quasi-randomly assigned childhood characteristics mitigate the negative effects of birth weight by estimating variations of the following equation:

$$y_i = \alpha + \beta LBW_i + Z'\Gamma + (LBW_i \times Z)'\Delta + X_i'\Lambda + \epsilon \tag{4.1}$$

and where y_i is either years of schooling, log of income, college attendance, or BMI. The variable LBW_i is an indicator equal to one if the adoptees birth weight is less than 2.5kg. This is the LBW cutoff used by Currie and Hyson (1999) and Currie and Moretti (2007). It is also the cutoff used by the CDC, WHO, and many other academic papers. The vector Z_i contains measures of socioeconomic status. The interaction term measures the extent that variables in Z_i mitigate the effects of LBW. This paper is interested in understanding the coefficients within the Δ vector. Each regression also includes indicators for gender and age.

Table 26 presents our results. For each outcome variable we present three specifications. First, we include the LBW dummy variable with no interactions. Second, we interact LBW with log of neighborhood income. Third, we interact LBW with log of neighborhood income, mothers education, fathers education, family income, and the number of children within the family. This is the specification presented in equation (1). We find that LBW adoptees earn approximately 20% less and attain one-half fewer years of education. Because the interaction with neighborhood income is positive and significant at the 5% level for all regressions, we

conclude that neighborhood income mitigates the effects of LBW. None of the other SES measures are significant at the 10% level. We repeat this analysis in Table 27 using BMI as the dependent variable. For BMI, we find no evidence of a LBW effect or mitigation at conventional levels of significance for any measure of SES.

At first these results might appear to conflict with Sacerdote (2007) because he does not find that neighborhood income affects economic outcomes. This difference occurs because neighborhood income only affects LBW adoptees (see Figure 2). The majority of the sample is normal birth weight, which explains why Sacerdote finds no effect from neighborhood income.

4.4 ROBUSTNESS

This section examines the robustness of our results. Specifically, we show that our results are not sensitive to the truncation of the sample or the inclusion of specific interaction terms.

As discussed in Section II, we do not observe birth weights for adoptees entering Holt with an incomplete medical history. Low birth weight adoptees that cross the LBW threshold before their medical history begins will be misclassified as normal birth weight. Normal birth weight children, however, should not be misclassified as LBW because weight tends to increase monotonically during the first years of life. Classifying LBW adoptees as normal birth weight attenuates our results and biases estimates towards zero. In Tables 3 and 4 we address this bias by restricting our analysis to adoptees with an initial weight less than 4.5kg. This restriction allows us to recover a birth weight distribution resembling that of the United States. To illustrate that our results are not sensitive to this cutoff, we relax this restriction in Table 28.

In the first four columns of Table 28 we estimate the effect of LBW and its interaction with each measure of SES for the non-truncated sample. In the last four columns we restrict analysis to adoptees that entered Holt before age one we assume that adoptees arriving by age one are more likely to arrive with a complete medical history. In both cases, we find results similar to those in Tables 3 and 4. For economic outcomes, the effect of LBW is

always negative and is significant for four of the six specifications. Moreover, the interaction of LBW and log of neighborhood income is positive and significant for each specification. The interaction of LBW and the other four measures of SES appears 32 times in Table 28, but only two of those coefficients are significant at the 10% level.

Because we use five measures of SES, it is possible to model the effect of LBW and its interaction with SES in many different ways. We can estimate the effect of LBW by itself; we can include an interaction with one measure of SES; we can include all five measures of SES; or we can include any subset of those interactions. There are 31 unique ways to organize our five interactions, 32 if we include the specification with no interactions. In Table 6, we ask whether our results are consistent across these specifications. For each outcome variable, we run each of the 32 specifications. Each interaction appears sixteen times, and Table 29 reports the number of times the interaction produces a p-value less than 0.05 or less than 0.01. Regardless of whether the outcome variable is log of income, educational attainment, or college attendance, the interaction of LBW and log of neighborhood income is significant at the 5% level for every specification that it appears in. For these outcome variables, none of the other interactions are ever significant at the 5% level. When the outcome of interest is BMI, the interaction of LBW and log of neighborhood income is never significant. With regards to the effect on BMI, the only interaction that is significant at the 5% level is the interaction of LBW with mothers education. However, that interaction is only significant in one of the sixteen specifications. These results are consistent with the results presented in Tables 3 and 4 neighborhood income mitigates the effects of low birth weight for economic outcomes but it does not affect BMI.

4.5 EXTERNAL VALIDITY

The previous sections present evidence that LBW decreases incomes and educational attainment for Korean adoptees. Furthermore, for these adoptees, neighborhood quality mitigates the negative effects of LBW, whereas family characteristics do not. A policy implication of these results is that orphanages placing LBW adoptees should give greater weight to an adop-

tive familys neighborhood. For example, suppose an orphanage is placing two adoptees, one that is normal birth weight and one that is low birth weight, into two potential adoptive families. Further, suppose that these families are identical except that one lives in a high-income neighborhood and one lives in a low-income neighborhood. If the orphanages objective is to maximize the sum of the adoptees adult economic outcomes, then the orphanage should assign the LBW adoptee to the high-income neighborhood.

Although the unique nature of the Holt Institution ensures that genetics and in utero environment are orthogonal to SES, it also raises the concern of whether these results are externally valid to the general non-adopted population. The set of families eligible to adopt from Holt are not representative of families with young children in the United States. Adoptive families must have had an income of at least 125% of the Federal Poverty Line, must be married, and must be between the ages of 25-45. Any policy attempting to mitigate the negative effects of LBW for non-adoptees would likely affect poorer and younger families as well as single mothers. In this section we address this concern by testing whether our results are sensitive to weighting our observations so that the family income, neighborhood income, and mothers education distributions resemble that of the U.S. population. To construct sample weights, we use data from the 1970 Census Form 1 and Form 2 samples. This 2% sample of the U.S. population includes family income, neighborhood income, and educational attainment. We restrict the sample to households with a unique head of household and households with either zero or one spouse. We also restrict the sample to households with at least one child. For constructing the weights, we create bins of income, neighborhood income, and education.[5] Let $N_j^U S$ be the number of households in the Census sample in SES bin j, and $N^U S$ be the total number of households in the sample. Similarly, let N_j^A be the number of adoptees who were adopted into a household in SES bin j, and N^A be the total number of adoptees. We rerun the analysis from Table 3 weighting each adoptee by $\frac{\left(\frac{N_j^U S}{N^U S}\right)}{\left(\frac{N_j^A}{N^A}\right)}$. These regressions will give higher weights to adoptees from families that are more representative

[5]For constructing weights, we group family income and neighborhood income into the following categories–less than 20,000; 20,000 to 30,000; 30,000 to 50,000, and greater than 50,000. For mothers education we use the following categories–less than 12 years of schooling (did not complete high school), 12 to 15 years of schooling (high school and some college), 16 years of schooling (college degree), and greater than 16 years of schooling (graduate education).

of the U.S. population so that the weighted density of the adoptee sample will match that of the U.S.

The results from these weighted regressions are in Table 7. The first three columns weight the observations by neighborhood income, the next three columns weight by mothers education, and the last three weight the observations by family income. The results are of the same sign and of similar magnitude as in Table 3, however, the standard errors are larger. Although not all estimates are significant for all specifications, LBW children have lower income and are less likely to go to college. Furthermore, neighborhood income mitigates the effects of LBW, which is significant for six out of the nine specifications. Of the 36 coefficients in which the other four interaction terms appear, only one is significant at the 10% level. Admittedly, the above analysis does not fully address the question of external validity. Applying these results to the general population should be viewed cautiously.

4.6 MODEL

4.6.1 Generalized Model

This section provides an example demonstrating that parents investing in children by living in better neighborhoods and sending their children to better quality schools will lead to neighborhood income mitigating the negative effects of low birth weight. Furthermore, parent's income only mitigates the negative effects of low birth weight through its effect on neighborhoods.

Parents have income Y_p. They must decide how much of good x to consume, and how much to invest in living in a higher quality neighborhood, I_s. Schools are funded locally, and parents purchase better schools by paying higher rents. The price of good x is normalized to 1, and the parent's budget constraint is:

$$Y_p = x + pI_s \tag{4.2}$$

where p is the price of a one unit increase in school quality in terms of good x.

Children are endowed with health θ_H and parents with background θ_B. Higher earning parents have children with higher θ_B. Notationally, $Y_p = g(\theta_B)$, where g is an increasing function. Children's outcomes depend on their endowments and investments in the form of school quality:

$$Y_c = f(\theta_H, \theta_B, I_s). \tag{4.3}$$

The function f is the technology of skill formation, and satisfies:

$$\frac{\partial f}{\partial \theta_H} > 0, \frac{\partial f}{\partial \theta_B} > 0, \frac{\partial f}{\partial I_s} > 0, \tag{4.4}$$

and

$$\frac{\partial^2 f}{\partial \theta_H^2} \leq 0, \frac{\partial^2 f}{\partial \theta_B^2} \leq 0, \frac{\partial^2 f}{\partial I_s^2} \leq 0. \tag{4.5}$$

Parents pick I_s to maximize utility $U(x, Y_c)$ subject to their budget constraint and the technology of skill formation. The first order condition is

$$\frac{\partial U}{\partial x} p = \frac{\partial U}{\partial Y_c} \frac{\partial f}{\partial I_s}. \tag{4.6}$$

For schooling investments to mitigate the negative effects of low birth weight and for family income to only mitigate low birth weight through its effects of schooling, we need:

$$\frac{\partial^2 f}{\partial \theta_H \partial I_s} < 0, \frac{\partial^2 f}{\partial \theta_H \partial \theta_B} = 0. \tag{4.7}$$

This condition implies that schooling investment and health endowments are substitutes.

4.6.2 Example and Simulation

For purposes of example, suppose that:

$$Y_c = \gamma\theta_B + \sqrt{\theta_H + I_s} \tag{4.8}$$

$$U(x, Y_c) = \min\{\delta x, (1-\delta)Y_c\} \tag{4.9}$$

The parameter δ measures how much utility parents attach to the consumption good x relative to their child's achievement Y_c. Further suppose that $\gamma = 0.1$, and $\theta_B = Y_p$. Also θ_B, θ_H, and δ are all independently and uniformly distributed over the unit interval. I now solve the model and generate simulated data. I then regress:

$$Y_c = \beta_0 + \beta_1\theta_B + \beta_2 I_s + \beta_3 LBW_i + \beta_4 LBW_i \times \theta_B + \beta_5 LBW_i \times I_s + \epsilon \tag{4.10}$$

where LBW_i is a dummy variable equal to one if individual i is in the bottom half of the health distribution. The estimates for this regression are in Table 31. Notice that the main effect of LBW is negative, the interaction on LBW and family income is statistically zero, while the interaction of LBW and neighborhood income is statistically significant and positive.

4.7 CONCLUSION

Does socioeconomic status mitigate the negative effects of low birth weight? The answer appears to depend on how socioeconomic status is measured. Currie and Hyson (1999) measure SES with fathers education and find little evidence that SES mitigates the effects of LBW. Currie and Moretti (2007), on the other hand, measure SES as the poverty rate within a zip code and find that SES does mitigate the effects of LBW. Using five measures of SES, we find that zip code income mitigates the LBW effect whereas other family characteristics do not. This result cannot be explained by genetics, prenatal healthcare, or neonatal healthcare

being correlated with family environment because adoptees were quasi-randomly assigned to families.

This analysis raises the question of why neighborhood income matters more than other family characteristics. One possibility is schooling. Schools in high-income neighborhoods might offer better remedial programs to help struggling students. Another possibility is that neighborhood characteristics are a better proxy for individual wealth than individual income or education. It could also be that high-income neighborhoods have access to better healthcare during the adoptees childhood. These explanations are neither mutually exclusive nor jointly exhaustive, but two aspects of our dataset prevent us from addressing these mechanisms. First, detailed neighborhood-level education, wealth, and healthcare data are not available for the 1964 to 1985 time period (the years in which these orphans were adopted). Second, although children were randomly assigned to families, families were not randomly assigned to neighborhoods. Therefore, families sorting into neighborhoods along unobservable dimensions could drive this result. However, this sorting must be independent of birth weight, genetics, and prenatal environment. Why neighborhood income mitigates the negative effects of LBW remains an open question for future research.

Figure 14: Birth Weight Densities

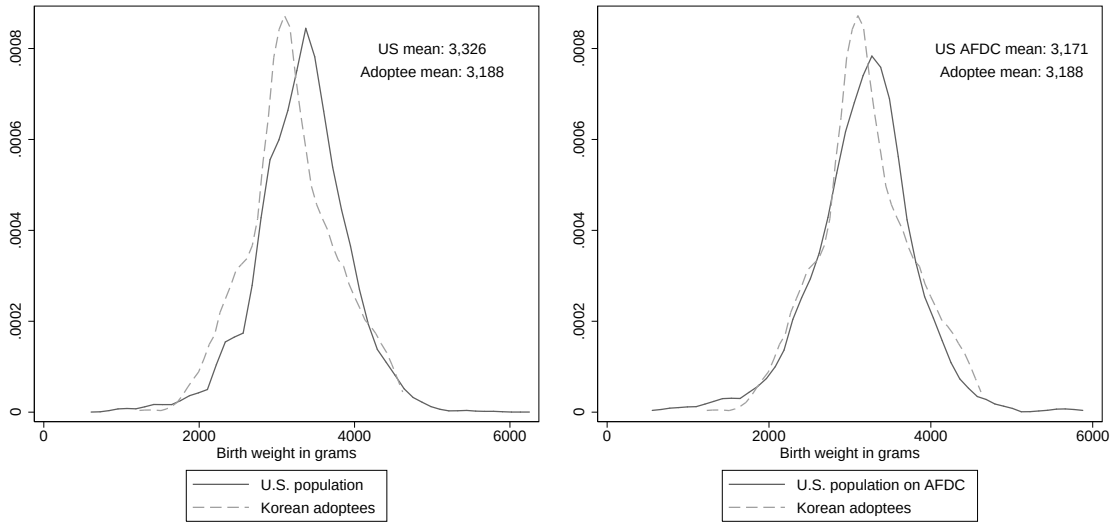


Figure 15: Years of Schooling by Birth Weight and Socioeconomic Status

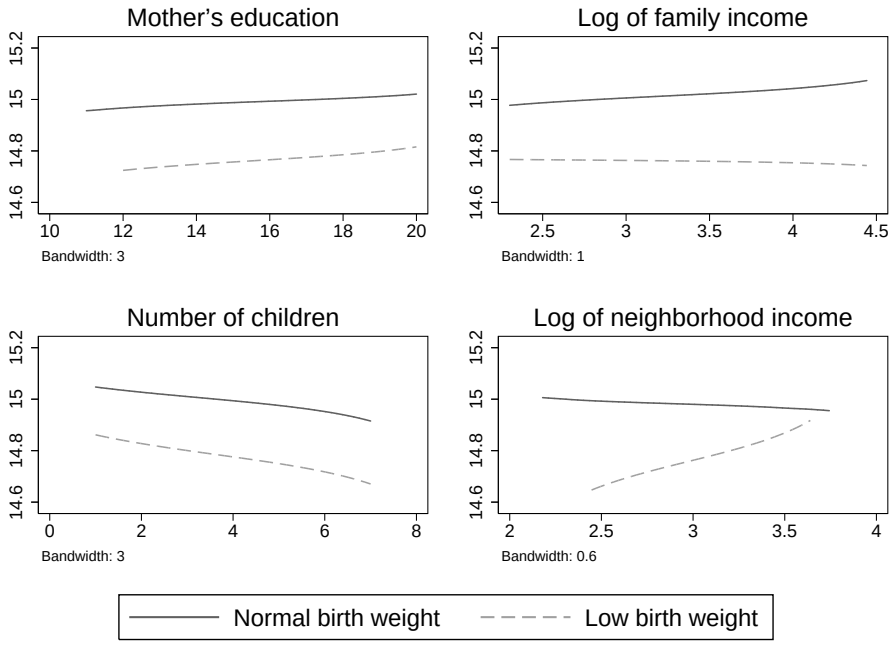


Table 24: Summary Statistics

Characteristics of the adoptee

	Mean	SD	Min	Max	Observations
Male	0.234	0.424	0	1	535
Age	28.867	3.834	19	40	535
U.S. arrival age	1.108	0.545	1	5	529
Married	0.426	0.495	0	1	530
BMI	23.040	3.828	16.499	38.008	522
Income*	50.252	36.640	10	200	437
Attended college*	0.565	0.496	0	1	448
Education*	15.033	2.110	9	21	448

Characteristics of the adopting family at the time of adoption

	Mean	SD	Min	Max	Observations
Mother's education	15.060	2.450	9	20	535
Father's education	15.888	2.864	9	20	535
Log of family income	3.165	0.672	2.303	5.298	535
Log of neighborhood income	2.953	0.256	2.179	3.743	535
Number of children	3.2	1.398	1	7	535

Notes: Sample restricted to adoptees with an initial weight of less than 4.5kg.
* Restricted to those at least 25 years old.

Table 25: Correlations between measures of socioeconomic status

	log neighborhood income	log family income	Mother's education	Father's education	Number of children
log neighborhood income	1	0.119	0.148	0.119	-0.013
log family income		1	0.157	0.043	-0.049
Mother's education			1	0.562	0.057
Father's education				1	0.045
Number of children					1

Note: Correlations reported for adoptees with an initial weight less than 4.5kg.

Table 26: The Effect of LBW on Adult Socioeconomic Status

	Log of income			Educational attainment			College attendance		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Low birth weight	-0.196**	-2.791***	-2.810**	-0.422	-7.444**	-5.681*	-0.082	-1.836***	-1.562*
	(0.0988)	(1.027)	(1.272)	(0.262)	(3.098)	(3.293)	(0.0622)	(0.701)	(0.844)
Log of neighborhood income * LBW		0.885**	0.863**		2.395**	2.503**		0.598**	0.581**
		(0.343)	(0.341)		(1.066)	(1.131)		(0.239)	(0.249)
Mother's education * LBW			-0.003			0.041			0.008
			(0.039)			(0.129)			(0.029)
Father's education * LBW			0.017			-0.038			0.002
			(0.035)			(0.126)			(0.028)
Log of family income * LBW			-0.036			-0.625			-0.079
			(0.140)			(0.440)			(0.104)
Number of children * LBW			-0.010			-0.056			-0.042
			(0.078)			(0.160)			(0.039)
Marginal effect of LBW	-0.196**	-0.176*	-0.175*	-0.422	-0.371	-0.408	-0.082	-0.069	-0.072
	(0.0988)	(0.095)	(0.096)	(0.262)	(0.261)	(0.270)	(0.0622)	(0.061)	(0.064)
Observations	437	437	437	448	448	448	448	448	448
R-squared	0.146	0.157	0.158	0.089	0.099	0.104	0.080	0.091	0.094

Notes: Sample restricted to adoptees over the age of 25. Robust standard errors clustered at the family level. Each regression controls for log of familys zip code income, mothers education, fathers education, and gender. Each regression also includes a set of dummy variables for parents income category, the childs age, and the number of children within the family.
* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

Table 27: The Effect of LBW on BMI

	(1)	(2)	(3)
Low birth weight	0.514	-1.967	5.254
	(0.534)	(5.395)	(7.177)
Log of neighborhood income * LBW		0.843	1.709
		(1.826)	(1.820)
Mother's education * LBW			-0.264
			(0.193)
Father's education * LBW			-0.143
			(0.198)
Log of family income * LBW			-0.491
			(0.859)
Number of children * LBW			-0.602
			(0.389)
Marginal effect of LBW	0.514	0.526	0.627
	(0.534)	(0.534)	(0.516)
Observations	522	522	522
R-squared	0.099	0.100	0.115

Notes: Robust standard errors clustered at the family level. Each regression controls for log of familys zip code income, mothers education, fathers education, and gender. Each regression also includes a set of dummy variables for parents income category, the childs age, and the number of children within the family.
* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

Table 28: The Effect of LBW on Adult Outcomes

	Non-truncated sample				Adoptees entering Holt before age one			
	Log income	Education	College	BMI	Log income	Education	College	BMI
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Low birth weight	-2.366**	-5.455*	-1.446*	5.971	-2.035*	-3.038	-0.910	6.809
	(1.157)	(3.012)	(0.760)	(6.644)	(1.223)	(3.174)	(0.813)	(7.137)
Log of neighborhood income * LBW	0.918***	2.505**	0.574**	1.098	0.857***	2.101*	0.492**	0.755
	(0.305)	(1.042)	(0.225)	(1.624)	(0.327)	(1.154)	(0.249)	(1.794)
Mother's education * LBW	-0.014	-0.026	-0.003	-0.338*	-0.022	-0.012	-0.001	-0.180
	(0.033)	(0.124)	(0.027)	(0.183)	(0.037)	(0.129)	(0.029)	(0.189)
Father's education * LBW	-0.027	-0.018	0.001	-0.059	-0.019	-0.063	-0.003	-0.132
	(0.031)	(0.125)	(0.028)	(0.190)	(0.0347)	(0.126)	(0.029)	(0.197)
Log of family income * LBW	0.054	-0.482	-0.048	-0.457	0.011	-0.788*	-0.133	-0.637
	(0.126)	(0.428)	(0.100)	(0.809)	(0.148)	(0.458)	(0.105)	(0.918)
Number of children * LBW	-0.019	-0.069	-0.050	-0.344	-0.028	-0.019	-0.051	-0.574
	(0.066)	(0.149)	(0.034)	(0.379)	(0.072)	(0.148)	(0.036)	(0.406)
Marginal effect of LBW	-0.172**	-0.446*	-0.085	0.644	-0.183*	-0.531**	-0.094	0.409
	(0.086)	(0.260)	(0.060)	(0.488)	(0.095)	(0.263)	(0.062)	(0.511)
Observations	856	877	877	995	572	585	585	673
R-squared	0.061	0.030	0.022	0.058	0.046	0.031	0.033	0.082

Notes: For log of income, educational attainment, and college attendance outcomes the sample is restricted to adoptees over the age of 25. The non-truncated sample includes all adoptees regardless of initial weight and treats those with an initial weight greater than 2.5kg as normal birth weight. In columns 5-8, uses the full sample but restricts analysis to adoptees that arrived at Holt by age one. Robust standard errors clustered at the family level. Each regression controls for log of familys zip code income, mothers education, fathers education, and gender. Each regression also includes a set of dummy variables for parents income category, the childs age, and the number of children within the family.

* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

Table 29: Proportion of specifications with significant coefficients

	Log of income		Educational attainment		College attendance		BMI	
	$p < 0.05$	$p < 0.01$	$p < 0.05$	$p < 0.01$	$p < 0.05$	$p < 0.01$	$p < 0.05$	$p < 0.01$
Low birth weight	17/32	2/32	9/32	0/32	12/32	1/32	3/32	0/32
Log of neighborhood income * LBW	16/16	5/16	16/16	0/16	16/16	2/16	0/16	0/16
Mother's education * LBW	0/16	0/16	0/16	0/16	0/16	0/16	1/16	0/16
Father's education * LBW	0/16	0/16	0/16	0/16	0/16	0/16	0/16	0/16
Log of family income * LBW	0/16	0/16	0/16	0/16	0/16	0/16	0/16	0/16
Number of children * LBW	0/16	0/16	0/16	0/16	0/16	0/16	0/16	0/16

Note: This tables summarizes the results of repeating the analyses in Tables 3 and 4 for every possible subset of the interactions between LBW and the five measures of SES.

Table 30: Weighted estimates of the effect of low birth weight on adult outcomes

	Weighted by neighborhood income			Weighted by mother's education			Weighted by family income		
	Log income	Education	College	Log income	Education	College	Log income	Education	College
Low birth weight	-2.847**	-5.372	-1.335	-0.587	-8.329**	-1.758*	-1.092	-3.844	-1.136
	(1.241)	(3.564)	(0.922)	(1.720)	(3.856)	(1.058)	(1.772)	(3.834)	(0.987)
Log of neighborhood income * LBW	0.917***	2.678**	0.580**	0.685	3.353**	0.604	1.082***	2.226*	0.369
	(0.326)	(1.223)	(0.269)	(0.540)	(1.591)	(0.376)	(0.375)	(1.217)	(0.308)
Mother's education * LBW	-0.002	0.048	0.010	-0.083	0.108	-0.018	-0.091	0.008	0.000
	-0.038	(0.129)	(0.030)	(0.075)	(0.212)	(0.048)	-0.069	(0.209)	(0.050)
Father's education * LBW	0.009	-0.072	-0.008	0.035	-0.128	0.015	-0.012	-0.058	0.013
	(0.035)	(0.120)	(0.029)	(0.0567)	(0.144)	(0.0349)	(0.058)	(0.158)	(0.041)
Log of family income * LBW	-0.022	-0.766*	-0.115	-0.232	-0.505	0.002	-0.239	-0.851	-0.073
	(0.137)	(0.457)	(0.110)	(0.225)	(0.650)	(0.153)	(0.231)	(0.620)	(0.151)
Number of children * LBW	-0.016	-0.030	-0.036	-0.071	0.003	-0.039	-0.019	0.090	0.005
	(0.074)	(0.166)	(0.040)	(0.116)	(0.218)	(0.054)	(0.112)	(0.222)	(0.056)
Observations	437	448	448	437	448	448	437	448	448
R-squared	0.148	0.108	0.096	0.466	0.359	0.366	0.239	0.216	0.193

Notes: SES is weighted to resemble the 1970 US distribution. We calculate these weights using data from the 1970 Neighborhood Census (Form 1 and Form 2). For each column we restrict analysis to adoptees over the age of 25. Robust standard errors clustered at the family level. Each regression controls for log of familys zip code income, mothers education, fathers education, and gender. Each regression also includes a set of dummy variables for parents income category, the childs age, and the number of children within the family. * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

Table 31: Simulation Estimates

Variable	coefficient	S.E.	t stat	p-value
neighborhood income	70.440729	0.002759	159.75	0
parents' income	0.106974	0.004345	24.62	0
low birth weight (LBW)	-0.37757	0.004983	-75.77	0
neighborhood income × LBW	0.156925	0.005383	29.15	0
parents' income × LBW	0.011106	0.009309	1.19	0.233
Constant	0.86417	0.002354	367.12	0
N	9952			
R-squared	0.8500			

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