THREE ESSAYS IN HEALTH ECONOMICS

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THREE ESSAYS IN HEALTH ECONOMICS

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This dissertation investigates the effects of government programs and policy on health. The first essay estimates the health impacts of the Empowerment Zone (EZ) program—a federal program that gave sizeable grants and tax breaks to certain high-poverty census tracts in selected cities. Using differences-in-differences and synthetic control methods I find that the EZ program decreased fertility rates by 10 percent, decreased the prevalence of low birth weight by 8 percent, and increased overall birth weight by 0.8 percent. This increase in infant health was not driven by changes in the composition of births. I compare the Chicago, New York City, and Philadelphia EZs to a control group of areas which applied for, but were not granted the EZ program in the first round. Estimates using an alternate control group support these findings. Recent research on the later-life impacts of low birth weight suggest that the health impacts of this program may have substantial long-term benefits. The second essay examines the effects of public insurance expansions among children in the 1980s and 1990s on their future educational attainment. We find that expanding health insurance coverage for low-income children increases the rate of high school and college completion. These estimates are robust to only using federal Medicaid expansions, and mostly are due to expansions that occur when the children are not newborns. Our results indicate that the long-run benefits of public health insurance are substantial. In the third essay, we estimate the impact of stress during early adulthood on later-life health. We use the risk of induction into the armed forces from age 18 ½ to 26 during

the Vietnam War as a proxy for stress, and obesity and self-reported health as measures of later-life health. We exploit variation in risk of induction based on an individual's birth month and year and therefore the age at which he became eligible for military induction. We find that induction risk is associated with worse health outcomes. Importantly, these effects are present for men but not women, which is consistent with them being the result of stress about military induction rather than unobserved trends.

BIOGRAPHICAL SKETCH

Daniel Grossman graduated from Northwestern University in 2005 with a BA in economics. After graduation, he joined the United States Peace Corps and served as a small enterprise development volunteer for two years. While in Senegal he met his wife Nichole. Upon returning to the US, Daniel worked as an associate in research for Frank Sloan, a health economist at Duke University, for three years. Working for Dr. Sloan introduced a strong interest in health policy and health economics and led Dan to return to graduate school to pursue a doctorate in Policy Analysis and Management at Cornell University. He completed his doctorate in August 2015 and has accepted a position as assistant professor of economics at West Virginia University this Fall.

This document is dedicated to my family. To my parents for encouraging me to pursue my interests and instilling the importance of education in me at an early age.

To Sai Sai, for providing me company while I worked and for demanding walks to interrupt my work. For keeping me in shape and allowing me to clear my head and organize my thoughts, without which I may never have finished my degree.

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CHAPTER 1

THE EFFECT OF URBAN EMPOWERMENT ZONES ON FERTILITY AND HEALTH: A CASE STUDY OF CHICAGO, NEW YORK CITY, AND PHILADELPHIA

1.1. Introduction

Poor neonatal health imposes large costs on individuals and society. Despite the fact that low birth weight rates are only 8 percent nationally, nearly half of all neonatal hospital costs are incurred by low birth weight babies (Almond et al. 2010, Russell et al. 2007). Additionally, rates of low birth weight differ greatly by race, with non-Hispanic blacks nearly twice as likely to have a low birth weight baby compared to non-Hispanic whites (Reichman 2005). The costs of low birth weight are not limited to hospital costs: low birth weight is predictive of worse later-life education, labor market, and health outcomes as well (Almond and Currie 2011). Therefore, policies that improve low birth weight rates, especially those that affect mothers who are most likely to have low birth weight babies, are important.

In this paper, I explore whether the empowerment zone (EZ) program—a federal stimulus package aimed at improving infrastructure and labor market conditions in low-income, high-poverty urban areas—had unintended effects on fertility and health. The EZ program offered generous tax credits to businesses both operating in these areas and hiring EZ residents, and granted $100 million for infrastructure projects. I study EZs because previous research has shown this program increased employment and earnings among zone residents (Busso, Gregory, and Kline

2013, Ham et al. 2011), but no prior study has investigated the health effects of this program.

In addition to providing the first analysis of the EZ program on health, which is an important extension of the literature investigating the overall effects of the EZ program, this paper contributes to the growing literature on the effects of the social safety net on birth outcomes. Previous work has demonstrated that income-targeted programs, including the earned income tax credit (Hoynes, Miller, and Simon 2015) and food stamps (Almond, Hoynes, and Schanzenbach 2011), improve low birth weight rates. Where my work differs and provides an additional contribution is that the EZ is a place-based policy, meaning that the program benefits have to be spent within the program's geographic limits or claimed by individuals living within the zone.

This study also relates to the strand of research investigating the relationship between macroeconomic conditions and health. While there is evidence of decreases in fertility and improvements in birth outcomes during times of high unemployment (Dehejia and Lleras-Muney 2004), the multi-pronged nature of the EZ program makes it particularly interesting to study. It incorporated block grants to be spent on infrastructural improvements and business tax credits to improve the labor markets in relatively small, urban areas. The EZ program may affect health not only through the direct effects of higher employment and wages, but also the indirect effects of urban renewal, increased infrastructure investment, an improved business environment, and potentially safer neighborhoods. While it may be more difficult to disentangle the mechanisms at work, the potential effects may be larger than that of an income

transfer program or a macroeconomic shock because of these additional pathways. This makes the overall program effects of the EZ program especially pertinent.

I use vital statistics natality data from 1990-2002 which I link for 9 cities and states to create a unique data set.[1] The data set consists of restricted-access, administrative records with geocoded census tract of residence for mothers. This level of geographic detail is necessary to study the EZ program, as each EZ is composed of mostly contiguous census tracts. In my main specification, I use differences-in-differences models, comparing areas that received round I EZs to areas that applied for but did not receive EZs or received EZs at a later point in time. These control areas all qualified for EZ status under the program guidelines and are a natural comparison for the EZs. Indeed, these are the areas used as control zones in much of the previous EZ literature (Busso, Gregory, and Kline 2013, Hanson 2009, Hanson and Rohlin 2011, 2013, Reynolds and Rohlin 2013).

To account for pre-treatment trends, I also estimate synthetic control models which create a weighted control group matched on pre-treatment trends. This creates a treatment and control group that follow similar trajectories in the pre-period, thus creating a counterfactual comparison by which to compare any changes that occur in the treatment area following the implementation of the EZ program. To address concerns about whether control areas from different cities differ on trends of unobservable characteristics, I perform differences-in-differences and synthetic control models using an alternate control group composed of areas in the same city as the EZs.

[1] I have data from Colorado, Florida, Kentucky, Pennsylvania, Oregon, Ohio, Texas, the City of Chicago, and New York City.

I find a large and statistically significant effect of EZs on fertility rates, birth weight, and low birth weight. Fertility rates decrease by 10 births per 1,000 females of age 15 to 44 in EZs compared to control areas, a 10 percent decrease. Birth weight increases by 26 grams on average in EZs, while low birth weight rates decrease by 0.8 percentage points in EZs compared to control zones, an 8 percent decline. Fertility effects by age suggest that the decreases in fertility do not reflect women delaying pregnancy to a later point in time, but an overall decrease in fertility. Furthermore, I present evidence that the infant health increases were not driven by changes in the composition of births, but rather reflect increased health among mothers. These results are robust to using an alternate group of control zones from the same city as the EZ. I find evidence of heterogeneous treatment effects across EZs as well, with fertility effects strongest in Chicago and Philadelphia. Birth weight results are strongest in New York but are present in Chicago as well, albeit with a smaller magnitude.

These results suggest that policy makers and researchers should consider the health effects of any program that affects income and infrastructure when developing, implementing and evaluating these programs. Ignoring the health effects of these programs underestimates the overall program effect, especially given the importance of birth outcomes on later life labor and health outcomes.

In the next section, I describe the institutional details of the EZ program. Section 1.3 discusses the theoretical framework, potential mechanisms through which the EZ program may affect health, and provides a literature review. Section 1.4 discusses the data, while section 1.5 provides information on methods and

identification strategy. In Section 1.6, I present the main findings, heterogeneous treatment effects, and robustness checks. Section 1.7 concludes.

1.2. Background: Empowerment Zones

In 1993, Congress announced a competitive bidding process for municipalities to apply to become EZs. The application zones were composed of multiple, generally contiguous census tracts chosen by the city. To qualify, all tracts in the zone were required to have poverty rates above 20 percent, with 90 percent of the tracts having poverty rates above 25 percent and 50 percent of the tracts having rates above 35 percent (Government Accountability Office 2010). Additionally, only tracts with unemployment rates above 6.3 percent were eligible. The actual average unemployment rates in the initial EZs were over 24 percent.[2] Eligible applicant zones must have had a total population below 200,000 and below the greater of 50,000 and ten percent of the most populous city in the area.[3] Both New York and Chicago had to exclude tracts from their applications because of this population restriction; each had over 199,000 individuals living in their EZ tracts. Urban areas of the program were to be administered by the U.S. Department of Housing and Urban Development.

The EZ program provided large employment tax credits for employers operating in these areas and exemptions from capital gains taxes. Firms operating in EZs were eligible to receive up to a 20 percent federal tax credit on the first $15,000 of an employee's wages, but only for employees who both lived and worked in the zones. Therefore, the firm could claim up to $3,000 in tax credits per eligible

[2] All poverty and unemployment restrictions were based on the 1990 Census.
[3] This was to ensure that the treatment would be large enough to make a difference in the selected area while preventing the money from being spent across an entire city.

employee annually. Additionally, because these credits are federal tax credits, the local governments in these areas would still reap the tax benefits of attracting new businesses or preventing businesses from closing (Hanson and Rohlin 2011).

EZs also were eligible to receive tax exempt bonds and up to $100 million in Social Service Block Grant funds for business assistance, infrastructure investment, physical development, training programs, youth services, promotion of home ownership, and emergency housing assistance.[4] EZ areas allocated these funds based on the strategic plans the communities originally submitted during the application process. However, each EZ area had latitude over how exactly these funds were spent, which depended in large part on strategic goals and local governance in general. Private funds provided upwards of $7 of additional investment for every $1 of Social Service Block Grant funds spent in EZs.

In December 1994, Congress authorized the funding of six initial urban EZs: portions of Atlanta, Baltimore, Chicago, Detroit, New York City, and Philadelphia/Camden and 60 "Enterprise Communities." Enterprise communities are areas that applied for EZ status but were denied. They were eligible for between $2.5 and $3 million in Social Service Block Grant funds and tax-exempt bond financing, but they were not eligible for business tax credits. If anything, this slight treatment will bias my results towards zero where the enterprise communities are used as a control group.

I present maps of the Chicago, New York, and Philadelphia EZs in Figures 1-3. These maps show the full city, census tracts that received the EZ program in black,

[4] The New York EZ also received guarantees of $100 million from both the state and the city government for a total of $300 million as part of its initial EZ agreement with the department of Housing and Urban Development.

and tracts that did not receive the program, but had poverty rates above 20 percent in gray. These areas were not include in the city EZ application, but had poverty rates that made them eligible for the program.

By the year 2000, over $400 million dollars in block grant funds had been spent in EZs and over $200 million in tax credits had been claimed. Tax credit claims grew each year between 1994 and 2000 with over $55 million claimed in 2000 alone (Busso, Gregory, and Kline 2013).

1.3. Mechanisms and Literature Review

1.3.1 Theoretical Discussion

The Becker Model of Fertility (1960, 1965) provides the theoretical basis for this study. In the Becker model, children are normal goods and individuals maximize their utility by choosing the quantity and quality of children to have.[5] Parents raise children by investing both own time and in goods for the purpose of child development. The amount parents invest in each good depends on the value of parents' time and cost of market goods. As in a standard utility maximizing equation, parents will invest in each good such that the marginal utility of an additional hour is the same across products, providing child health and other products in the most efficient way.

An increase in wages and income will have an ambiguous effect on fertility because of counteracting substitution and income effects. Wages and expected income determine the price of a parent's time, so an increase in wages will raise the opportunity cost of children. This increase in opportunity cost will decrease the

[5] Recent literature suggests it is valid to assume that children are normal goods (see e.g. Black et al. 2013, Lovenheim and Mumford 2013, Lindo 2010)

demand for children. However, children are normal goods so any increase in wages and income will increase family's purchasing power, thereby increasing the demand for children. Following similar arguments, the predictions are ambiguous for health behaviors that produce in utero health for pregnant women.[6]

EZs may affect infant health through the following channels: fertility, income, local macroeconomic conditions, and infrastructure and neighborhood effects. Infant health is a particularly important outcome of interest given the long-term effects of poor in utero health and early infant conditions (e.g., Figlio et al. 2013; Almond and Mazumder 2011; Almond, Edlund and Palme 2009; Almond, 2006; Black, Devereux, and Salvanes 2007; Oreopoulos et al. 2008; Royer 2009). To the extent that the EZ affects health, the long-term benefits of the program may be quite substantial. Below, I briefly review the EZ literature, and discuss each potential mechanism in turn.

1.3.2. Previous EZ Literature

Previous studies examining the EZ program have estimated the effect of the program on wages (Busso, Gregory, and Kline 2013, Oakley and Tsao 2006, Reynolds and Rohlin 2013), employment (Busso, Gregory, and Kline 2013), property values (Hanson 2009), owner occupied housing (Krupka and Noonan 2009), firm relocation (Hanson and Rohlin 2011), and geographic spillover effects of the program (Hanson and Rohlin 2013). While some find EZs had little impact (Oakley and Tsao 2006), newer work, using restricted employment and administrative data at both the household and establishment level, report large wage and employment effects (Busso,

[6] Mothers invest in health behaviors, like receiving prenatal care, such that the marginal utility of an additional unit of, for example, prenatal care is equal to the marginal utility of an additional unit of a market good. An increase in wages will lead to mothers substituting away from time-intensive health behaviors, while it also will lead to individuals investing more in these health behaviors through the income effect.

Gregory, and Kline 2013). This paper extends that literature by estimating impacts of the EZ program on fertility and health.

1.3.3. Mechanisms for Possible Effects

How do these increases in wages and employment in EZs affect fertility? An increase in wages will lead to an increase in demand for a normal good. A burgeoning literature confirms that children are normal goods as fertility rates rise due to exogenous shocks to income and wealth (Black et al. 2013, Lovenheim and Mumford 2013, Lindo 2010), while fertility rates decrease during periods of high unemployment (e.g., Becker 1960, Dehejia and Lleras-Muney 2004). Perry (2004) decomposes fertility responses by income, finding that higher earning women increase fertility during periods of high employment while less-educated, lower earning women are less likely to reproduce in response to better labor market conditions. This implies that for lower earning women, the substitution effect on fertility may be stronger than the income effect. Because of the high poverty and unemployment rates in EZs, it is likely that, despite the improving labor market conditions, women in these areas will reproduce less.

Any effects of income on fertility rates also can affect infant health. While higher unemployment rates lead to decreases in fertility, they also lead to selection into fertility which may affect infant health through compositional changes (Dehejia and Lleras-Muney 2004). Thus, accounting for whether EZs change the demographic composition of who has children, as well as overall fertility rates is vital. This is especially important when studying place-based policies as the policy itself can cause distributional changes in composition in these areas.

Increases in income affect infant health by raising the demand for health inputs in the birth weight production function leading to health improvements (Corman and Grossman 1985, Currie 2009, Mocan, Raschke, and Unel 2013). Finding plausibly exogenous income shocks that do not affect the outcome of interest can be difficult (Almond and Currie 2011). A recent literature uses changes in the social safety net to study the effect of income on health. Hoynes, Miller, and Simon (2015) find between a 7 and 11 percent reduction in low birth weight probability associated with a $1,000 increase in the earned-income tax credit among single women with a high school diploma or less. Investigating the effect of the rollout of food stamps on infant health, Almond, Hoynes, and Schanzenbach (2011) find similarly sized decreases in the probability of low birth weight.[7] Income targeting programs have their own endogeneity issues in that income itself may be endogenous. Thus, studying the effect of place-based programs on health provides an additional data point by which to evaluate the effect of income on health.

Income shocks also can affect child health through expenditures on market goods. Investigating the effect of a partner's job loss on birth outcomes, Lindo (2011) finds large decreases in birth weight, especially among the lower half of the birth weight distribution, with suggestive evidence that individuals may reduce food expenditures after a job separation.

However, the macroeconomy and health literature suggests a potential negative effect of improved labor markets on birth outcomes. During periods of high

[7] Other work in this literature has investigated the effects of Women Infants and Children (Hoynes, Page, and Stevens 2011) on birth outcomes. Kenkel, Schmeiser, and Urban (2012) and Cowan and Tefft (2012) also investigate the effect of the earned-income tax credit on smoking.

unemployment, pregnant individuals reduce drinking and smoking, are more likely to exercise, have better sleep habits, and receive improved prenatal care compared to periods of lower unemployment (Aparicio and Gonzalez 2014, Dehejia and Lleras-Muney 2004).

A related literature investigates the health effects of exogenous income shocks among American Indians following the distribution of casino profits. Akee et al. (2013) provide suggestive evidence that income transfers from casino profits improve child health by reducing obesity, while Wolfe et al. (2012) find improvements in health and health behaviors following these income shocks. These results imply that income shocks affect health throughout the age distribution, not just infant health.

1.3.4. Neighborhood Conditions on Health

Because of the geographic limits on where EZ funds can be spent, and the large amount of money spent in EZs, the neighborhood effects of the program on health are potentially large. Neighborhood improvements include better local amenities, which hedonic price models predict will raise real estate prices (Roback 1982). Despite large infrastructural improvements and evidence of increased business establishments in EZs (Hanson and Rohlin 2011), Busso, Gregory, and Kline (2013) report little evidence of increases in property values in EZs for owner occupied housing among those who have lived in their houses for less than five years.[8]

While there is a long history of research suggesting neighborhood environments affect children and families, identifying these effects is challenging (see

[8] Individuals who have lived in a house for less than five years likely are more knowledgeable about the actual value of their home, although others report fairly large increases in property values following the EZ program (Hanson 2009, Krupka and Noonan 2009).

e.g. Ellen and Turner 1997). Moving to Opportunity, which provided housing vouchers for low-income families living in high poverty areas to move to areas with lower poverty rates, is one experiment which plausibly identifies neighborhood effects. Improving the neighborhood in which one lives can have substantial effects on an individual's well-being and general health (Ludwig et al. 2012, 2013).

Estimates from sociology find that more disadvantaged neighborhoods are associated with lower physical activity (Wen, Browning, and Cagney 2007), while higher crime rates are associated with poorer health (see e.g. Browning, Cagney, and Iveniuk 2012). These studies provide evidence that improvements in infrastructure and neighborhoods affect health outcomes.

1.4. Data

I use restricted-access, state-specific, geocoded vital statistics natality data because they contain census tract of residence of mothers, a necessary variable for investigating the EZ program. The EZ program application and qualification process was based on 1990 census tract level characteristics, and the program was implemented at the census tract level. To my knowledge, these natality data sets are the only ones available at such a fine geographic level that also contain a sufficient sample size to analyze EZ program effects. Further, these data contain the universe of births in these areas.

These geocoded data are only available on a state by state basis. To compile the data set I use in this project, I applied separately to nearly 30 states in which a potential EZ or control zone existed and for which these data were available. Through this effort, I created a unique dataset consisting of geocoded data from 9 cities and

states. States that provided me with these data include Colorado, Florida, Kentucky, Ohio, Oregon, Pennsylvania, and Texas. I also have natality data specifically for the City of Chicago and New York City, both of which are treatment zones. Pennsylvania contains both treatment and control zones.[9] Because of data availability I limit my analysis to Chicago, New York, and Philadelphia.[10] The years for which I have these data are 1990-2002. The EZ program began in 1995, so I have pre-EZ data for 1990-1994 and post-EZ data for 1995-2002 from which to analyze the program effects.[11]

Vital Statistics Natality data from all states listed above contain birth weight and parental demographic characteristics such as race, ethnicity, and educational attainment. These data also contain age, prenatal care measures, including whether a woman received any prenatal care and the month in which care began, whether a woman drinks or smokes during pregnancy, gestational period, a plural birth indicator, and geocoded addresses to identify the census tract of the mother's residence.[12] Not all variables are available from all states. Table 1 provides a chart describing the data available from each state. I limit my sample to singular births because of higher incidence of low birth weights and additional risks associated with plural births.[13]

I also use Census tract level summary files of neighborhood characteristics from the 1990 and 2000 decennial Censuses and the American Community Survey 5-

[9] I applied for data from all EZs, but only Chicago, New York, and Philadelphia zones provided me with these data.
[10] Philadelphia and Camden were technically one EZ, but they were managed by separate entities with input from separate state and city governments and received a predetermined amount of the social service block grants. Philadelphia received $79 of the $100 million. I do not have New Jersey data so I treat the Philadelphia EZ as if it were the whole EZ based on its receipt of the majority of funds and its separate management.
[11] Because I use date of conception rather than date of birth, I technically have data from 1989-2001.
[12] In some cases, states provided me with raw maternal address data that I geocoded to find the exact latitude and longitude of residence using ArcGIS software by Esri. I then used these coordinates to find the census tract of residence.
[13] Over 97 percent of births in the US over the study window were singular births (http://www.cdc.gov/nchs/data/databriefs/db80.pdf accessed June 12, 2014)

year file (2005-2009) to create tract-level demographic characteristics. I use these data sets to calculate the fertility rate, which is the number of births occurring in a given zone *s* in year *t* divided by the total population of females, aged 15 to 44, in area *s* in year *t*:

$$FertilityRate_{st} = 1000 * \frac{Births_{st}}{Female\ Population\ 15-44\ _{st}} \quad (1)$$

Because the decision to conceive occurs approximately nine months before a women gives birth, I use the year of conception, calculated from the estimated gestational age, rather than the year of birth for all fertility rate calculations. I similarly use year of conception for all birth outcomes, although I perform additional analyses using year of birth.[14] I calculate this female population between ages 15 and 44 for each year from 1990 to 2002 by linearly interpolating Census data from 1990, 2000, and 2005-2009. Annual census tract-level covariates described below also are calculated in this fashion.

1.5. Empirical Methods

The primary research question of this paper is: does the EZ program affect fertility rates, birth weight, or the probability of having a low birth weight baby? To answer these questions, I estimate differences-in-differences models as my basic specification. The differences-in-differences model compares outcomes in treated areas to control areas using time periods before and after the treatment. I obtain an estimate of the average treatment effect of the EZ program on health by calculating the

[14] States collect these data at the time of birth, so I assume that mothers were living in the same residence at the time of conception.

difference between the average change in outcomes between the post and pre period in the treatment and control groups.

The differences-in-differences model is:

$$Y_{ist} = \alpha_1 + \alpha_2(EZ * Post)_{ist} + \beta X_{ist}{}' + \delta_c + \gamma_t + \varepsilon_{ist}\,, \qquad (2)$$

where α_2, the coefficient on the interaction term, is the main parameter of interest. Y_{ist} is a birth outcome for child *i* in zone *s* in year *t*. Birth outcomes include birth weight, very low birth weight (<1500 grams), and low birth weight (<2500 grams). Other outcomes include fertility rates per 1,000 women aged 15 to 44, gestational age of at least 37 weeks or full-term births, whether a mother received any prenatal care, the month mothers received prenatal care for the first time,[15] and smoking status of mothers. *EZ* is an indicator variable that takes a value of 1 if a mother resided within a census tract that received round I EZ status (i.e., is in the treatment group) when she gave birth and *Post* is an indicator variable that is 1 if a baby was born after the implementation of the EZ program in 1995. The main effect of the variable *EZ* is subsumed by the census tract fixed effects (δ_c). Year fixed effects (γ_t) absorb the main effect of the variable *Post*. *X* is a vector of individual demographic characteristics including gender of the baby, maternal race and ethnicity, age, and education level of the mother, and a vector of census tract level characteristics of the tract in which a mother resides when she gave birth including population, gender, race and ethnicity, median household income, vacancy rates, and poverty rates. I cluster

[15] Mothers who did not receive prenatal care receive the value 10 for month prenatal care first received (Mocan, Raschke, and Unel 2013).

standard errors in all regressions at the EZ or control zone level, as this is the level of treatment, to allow for serial correlation of error terms.

I discuss the differences-in-differences model for fertility rates separately because fertility is a population-level measure and thus requires aggregating data up to the EZ or control zone level.[16] I estimate the same general differences-in-differences equation as in equation (2) with the following qualifications: the variable on the left hand side of the equation, fertility rate, and all individual- and census tract-level characteristics are aggregated to the EZ or control zone-level, and I use EZ- or control zone-level fixed effects. I also calculate age-specific models of fertility rates for ages 15 to 19, 20 to 24, 25 to 29, 30 to 34, 35 to 39, and 40 to 44.

In the differences-in-differences specification, I use a control group composed of areas that applied for but did not receive Round I EZ funding, or areas that received later round EZ funding. The strengths of using this group of control areas include that these areas are similar to EZs in that all qualified for EZ status under the poverty and population restrictions described in Section 1.2. They also were chosen by their respective cities as application zones, such that if there exists selection into being included as an application tract, as long as the selection is consistent across cities this should not be a concern. These strengths have led others to rely on a similar set of controls (Busso, Gregory, and Kline 2013, Hanson 2009, Hanson and Rohlin 2011, 2013, Reynolds and Rohlin 2013). Ham et al. (2011) raise the concern that using controls from other cities and states may fail the conditional independence assumption: even after controlling for observables such as demographic characteristics

[16] I also calculate fertility rates by census tract. These results are similar to those aggregated to the zone level.

of the zones and of the mothers giving birth in these zones, unobservable characteristics in these areas may still differentially affect outcomes. Yet using same city control areas may mask geographic spillover effects, either positive or negative, of the EZ program. In the case of positive spillovers, this would bias my estimates towards zero. I perform robustness checks using same city control areas in which I exclude tracts adjacent to EZs.

While parts of Atlanta, Baltimore, Chicago, Detroit, New York City, and Philadelphia/Camden originally received Round I EZ status, because of data availability I limit my analysis to Chicago, New York, and Philadelphia. I use these 3 EZs and 16 control zones.[17] The data I collected contain 24 control zones,[18] however I limit my sample to zones in cities with a population of at least 100,000 and application zones of at least 10 census tracts similar to Busso, Gregory, and Kline (2013). This creates a comparison group that is more similar to my treatment zones, which are all in major population centers. Appendix Table 1 lists all EZs and control areas used in this paper.

The differences-in-differences model has two main assumptions: (1) the common trends assumption and (2) no unobserved shocks occur contemporaneously with EZ implementation. The common trends assumption states that absent the EZ program these areas would have continued to follow similar trends in the post period. If areas are granted an EZ in response to worsening outcomes in these areas, my estimates will be biased towards zero. They will be biased towards finding an effect if

[17] The 16 control zones I use are Austin, Texas; Cincinnati, Ohio; Corpus Christi, Texas; Dallas, Texas; Denver, Colorado; El Paso, Texas; Houston, Texas; Jacksonville, Florida; Louisville, Kentucky; Miami, Florida; Pittsburgh, Pennsylvania; Portland, Oregon; San Antonio, Texas; Summit, Ohio; Tampa, Florida.
[18] Omitted potential control zones include: Bellmead/Waco, Texas; Chester, Pennsylvania; Cleveland, Ohio; Dauphin, Pennsylvania; Fort Lauderdale, Florida; Fort Worth, Texas; Greeley/Evans, Colorado; Port Arthur, Texas.

EZs were granted in response to improving conditions in these areas. Figure 4 provides annual unadjusted-means of fertility rates by EZ status, with fitted lines demonstrating the pre-treatment trends. Figures 5 and 6 provide graphs with monthly unadjusted-means for birth weight, low and very low birth weight, and full-term births. From these figures, EZs and control areas appear to be trending in similar ways in the period prior to the policy which suggests common trends may be a reasonable assumption.[19] To the extent that slight differences exist in pre-trends by EZ status in Figures 5-6, I use synthetic control methods which I describe below to create a better pre-period match on trends.

The second assumption is that no unobserved shocks occur at the same time as the EZ program that differentially affects trends in the outcome variable in treated or control zones.[20] I estimate models using an alternate same city control group to account for potential unobserved shocks. Considering that these areas are similarly economically-disadvantaged areas and from the same cities as the EZs, for this assumption to fail in this context cities would have had to have implemented additional programs only in the EZ section of the city at the same time as the EZ program.

Ideally, EZs would have been either randomly assigned or assigned based on a numerical scoring method of the relative strengths of each municipal applicants' proposal. However, while the selection process was based on the strengths of each

[19] I present similar figures for each EZ separately in Appendix Figures A1-A3.

[20] One of the main contemporaneous changes occurring during this time period is the expansion of state Medicaid systems via federal waivers for expanding family planning coverage (see Kearney and Levine 2009). However, treatment zones in New York and Illinois did not apply for these waivers until 2002, after my study period, while Pennsylvania never applied for this waiver. Therefore this program is unlikely to affect fertility analyses in this paper.

city's proposal, there was no formal grading process and final decision for EZs were made by the Secretary of the Department of Housing and Urban Development, Henry Cisneros. This raises the issue of policy endogeneity which I attempt to address below.

The differences-in-differences model has a number of potential weaknesses in this context. First of all, because I only have 3 treatment zones and 16 control zones the model has the problems associated with a small number of clusters (Bertrand, Duflo, and Mullainathan 2004). Standard errors in this circumstance may be underestimated, affecting statistical significance interpretations. Uncertainty about whether the control group accurately captures the counterfactual trend of treated areas, had the treatment not occurred, is another concern (Abadie, Diamond, and Hainmueller 2010).

To account for these concerns with the differences-in-differences method, I employ synthetic control models (Abadie, Diamond, and Hainmueller 2010). This method is similar to a matching estimator (Rosenbaum and Rubin 1983), except inference is very different using synthetic control methods which I discuss more below. I create a weighted control area matched on pre-EZ trends, including the outcome of interest, such that the vector of weights (W) minimizes:

$$\|X_1 - X_0 W\|_v = \sqrt{(X_1 - X_0 W)' V (X_1 - X_0 W)} \quad (3)$$

where X_1 is a vector of pre-intervention characteristics of the treatment zones and X_0 denotes a similar vector for control zones. The vector of characteristics for the treatment zone is unweighted. The pool of control areas consists of all application and future zones, but if a control area is trending differently from the treatment, it can

receive zero weight. This method creates a weighted comparison group that minimizes the root mean squared error of the outcome variables in the pre-treatment period, which is the standard deviation in the difference between the actual outcome value of the treatment group and the predicted outcome value of the synthetic control group (Abadie, Diamond, and Hainmueller 2010).

I estimate synthetic control models separately by outcome so that a potentially different synthetic control group is used for fertility than is used for birth weight, or any other birth outcome. I focus on fertility rates for the remainder of this section for the sake of brevity. The basic specification adjusts for the pre-period outcome of interest in each pre-EZ year (1989, 1990, 1991, 1992, 1993, and 1994),[21] and the average of the following variables over the same pre-period: race of the mother (non-Hispanic black, non-Hispanic white, Hispanic, and other race), gender of the baby, census tract level demographic characteristics for poverty level, race, gender, and educational attainment. I include each year separately for the outcomes because it creates the best pre-treatment match both on trend and level, which is important in the inference tests that use minimized root mean squared prediction errors described below. In my main specification, I match on just trend to remove issues of matching on levels that affect my ability to create a valid synthetic control group, especially in instances in which an area has an outcome at either extreme compared to other areas. The high fertility rates in Chicago are an example of this problem.[22] I accomplish this

[21] I also have estimated models in which I control for the outcome averaged over 1989-1994. The results are similar to the main results in Table 3.
[22] While I focus on results matched on trend, I present those matched on trend and level in Appendix Table 3.

by subtracting the mean of the outcome of interest over all pre-period years from the mean of the outcome in each year *t*:

$$\tilde{Y}_t = Y_t - \frac{\sum_{t=1989}^{1994} Y_t}{6} \ , \forall \ t$$

Starting in 1995, I compare the difference in fertility rates following the EZ program between the EZs and the weighted control group to determine the average treatment effect of the program. This is calculated as the difference in the change in fertility rates by treatment status in the post- and pre-treatment period.

The main strengths of this method are it creates a matched control group that follows similar pre-trends in terms of the outcome of interest, and it allows for rigorous inference testing. Because the control areas follow similar pre-trends to the treatment areas, they are plausibly a better counterfactual representation of what one would expect to have happened to outcomes in Round I EZs absent the treatment. A key assumption of the synthetic control method is that the better pre-period match implies a better post-period counterfactual, which is untestable. Differences-in-differences and synthetic control models are identified under different assumptions. To the extent that the results from these two methods are the same lends credibility to estimates from both.

The inference testing consists of systematically assigning treatment to each control zone, creating a synthetic control group using the actual EZ as a control area as well as the full pool of control zones, minus the zone assigned to treatment. As in the case of the EZ, I calculate the average treatment effect of assigning treatment to each control zone. This creates a distribution of average treatment effects by which to

evaluate the average treatment effect of the actual EZ program. So if there are 30 average treatment effects and the EZ effect is larger than 27 of the control area average treatment effects, the estimate is statistically significant at the 10% level.

I also calculate the root mean squared prediction error in the post-period for each of these estimates. This is the out of sample analog to the pre-root mean squared error described above. I then divide the post root mean squared error by the pre root mean squared error and create a distribution of these ratios (Abadie, Diamond, and Hainmueller 2010). Thus, if areas had a poor pre-trend match, this method implicitly controls for this difference. I then use the distribution of these ratios as a confidence interval to compare the ratio obtained using the actual EZ.

For this method to fail, one would have to assume that EZs were selected endogenously because they were improving before the EZ program began and additionally that the rate of improvement changed differentially following the EZ program for reasons entirely separate from the EZ program. This is essentially a second derivative difference in the post period. That is, even after controlling for trends using synthetic control models, the EZs improved even more than places that were trending similarly in the pre-period following the EZ program implementation.

1.5.1 Summary Statistics

In Table 2, I find that areas that received EZ status differed from control zones on a number of demographic characteristics and birth outcomes. However, it is important to note that trends, and not levels, are what matter in the differences-in-differences model.

The unadjusted differences-in-differences in Column 7 show fertility rates in EZs decrease by nearly 20 births per 1,000 women compared to control zones. Moreover, birth weight in EZs increase by 33 grams while low birth weight rates decrease by 1.3 percentage points and full-term births increase by 1.6 percentage points relative to control zones. Trends in racial composition of mothers are similar across EZs and control areas. Thus, there appears to be a change in fertility and birth outcomes with little evidence of a change in the composition of mothers. The remainder of the paper examines whether these patterns remain when employing more advanced empirical methods.

1.6. Results

1.6.1. Differences-in-Differences and Synthetic Control Estimates

1.6.1.1. Baseline Results

In Table 3, I present fertility and birth outcome results from estimating equations (2) and (3). In Panel A, I use areas that applied for but did not receive the EZ program or areas that received the EZ program at a later point in time as control zones. This is my preferred control group. Panel B, alternatively, uses a control group of individuals who lived in census tracts within the same city as EZs with poverty rates above 20 percent that did not apply for the EZ program. These were similarly economically-disadvantaged areas that, based on their poverty rates, qualified for the EZ program. For each control group I estimate both differences-in-differences and synthetic control models. Each cell in the table presents results from a separate regression where the dependent variable is denoted in each column.

In Panel A using differences-in-differences models, fertility rates in EZs decrease by 10 births per 1,000 women aged 15 to 44 compared to control zones after the designation of round I EZs. This is a 11 percent decrease in fertility rates relative to a pre-EZ program mean fertility rate of approximately 89 births per 1,000 women between 15 and 44 years of age. Birth weight increases by 26 grams in EZs compared to control zones. While these relative increases in birth weight are statistically significant at conventional levels, the magnitude of these effects is rather small. Compared to the mean birth weight of approximately 3200 grams, a 26 gram increase is equivalent to a 0.8 percent increase in birth weight. However, when I examine this effect closer using clinical birth weight thresholds, a picture of distributional effects emerges.

EZs decrease low birth weight rates by 0.8 percentage points. The pre-treatment mean for low birth weight is approximately 10 percent, so this is an 8 percent decrease. The EZ program appears to have little effect on rates of very low birth weights in zones compared to control zones. Previous work studying the earned income tax credit (Hoynes, Miller, and Simon 2015) and the initial roll-out of food stamps (Almond, Hoynes, and Schanzenbach 2011) suggests these income-targeting programs decrease low birth weight rate by between 5 and 11 percent, which is similar to the effect size I find.

Additionally, the EZ program increases the probability a mother will carry a baby to full-term, or for 37 weeks or more. The effect size implies an increase in full-term births of 0.9 percentage points, or a 1 percent increase relative to control zones.

1.6.1.2. Synthetic Control and Inference Results

Because the differences-in-differences model does not match on trends in the pre-period, I use synthetic control methods as a robustness test. This method creates a control group with a similar pre-trend in the outcome variable compared to the EZ group. Row (2) of Table 3 summarizes the results for fertility rates and birth outcomes using the synthetic control method and inference testing as described in section 1.5. The first row of parenthetical numbers under row (2) presents implied p-values of the EZ average treatment effect evaluated against the distribution of placebo average treatment effects, while the row immediately beneath that contains implied p-values of the ratio of post to pre-root mean squared prediction error for the EZ compared to the distribution from inference testing.

The results for fertility rates are robust to synthetic control methods. Results for additional birth outcomes provide estimates of similar magnitudes to the differences-in-differences estimates. Birth weight in EZs increases by nearly 18 grams relative to synthetic control models, although this estimate is not statistically significant at conventional levels. The effect of EZs on low birth weight implies a 1.3 percentage point reduction in low birth weight rates compared to the synthetic control and a 1.8 percentage point increase in full-term births. These estimate are statistically significant using the root mean squared error distribution.

Figure 7 presents fertility rate results from Table 3, Panel B graphically.[23] Panel B of Figure 7 shows the difference between treatment and synthetic control groups for models in which each control is designated as the treatment. Fertility rates

[23] Appendix Figures 1-4 provide similar figures of synthetic control models for birth weight, low birth weight, very low birth weight, and full-term.

decrease following the EZ program and continue to decrease in the years after, with an implied reduction in fertility rates of 15 births per 1,000 females compared to synthetic controls. Importantly, the reduction in fertility rates in EZs, the solid line in Figure 7, Panel B, is larger than the placebo effects of assigning treatment to any of the control zones, depicted in the dotted lines. Panels C and D present the cumulative distribution of average treatment effects and ratio of post- to pre-root mean squared prediction errors, respectively, from assigning treatment systematically to each control. The vertical line shows the estimate for the actual EZs. In each case, the vertical line is at the extreme, lending credence to the statistical significance of these estimates.

1.6.1.3. Same City Control Areas

As a robustness check, I evaluate the EZs compared to a control group consisting of births occurring to mothers who lived in Chicago, New York, and Philadelphia using a differences-in-differences model in Panel B of Table 3. Using same city control groups ostensibly reduces the concern that unobservable differences between control groups and treatment areas may affect outcomes. Figures 1-3 present areas in each city with poverty rates above 20 percent as well as areas that receive the EZ program. I exclude births to mothers living in census tracts adjacent to tracts that received the program to ensure that any spillover effects of the program do not confound my estimates.

These estimates are consistent with the main differences-in-differences results. Fertility rates decrease by 11 births per 1,000 females between the ages of 15 and 44. Birth weight and low birth weight results are similar to the main results in magnitude,

although neither estimate is statistically significant. The effect size for full-term is less than half the size of that in Panel A and lacks statistical significance.

Results using synthetic control methods with same city control zones, which control for pre-trends and potential unobserved city-wide shocks, are presented in row (4) of Table 3. The fertility results are robust to this specification. Birth outcomes are of similar magnitude to those in row (3) and are consistent with the results in Panel A. These estimates provide supporting evidence that the effects I find in Panel A reflect the true program effects on health.

1.6.2. Mechanisms

The differences-in-differences results in Table 3 present the average treatment effect of the EZ program on birth weight. Despite using clinically relevant thresholds for low and very low birth weight, these results do not completely illuminate the birth weight distribution effects of the EZ. Using an unconditional quantile regression model (Firpo, Fortin, and Lemieux 2009), I estimate the effect of the EZ program on every fifth percentile of the birth weight distribution. I include the same variables as in the differences-in-differences specification described earlier. Figure 8 shows that the improvements in birth weight from the EZ program are concentrated among the lowest quantiles of the birth weight distribution.

In Table 4, I explore potential mechanisms through which EZs may improve birth outcomes using a sample of Chicago, New York, and Philadelphia EZs and my preferred control sample. These estimates provide suggestive evidence of improved prenatal care in EZs: the probability of receiving no prenatal care decreases in EZs compared to control zones by 1.8 percentage points and time to receipt of a first

prenatal care visit decreases by 0.16 months, or nearly a week. However, neither of these results is statistically significant.

Smoking in EZs increases relative to control areas by 2 percentage points, a 15 percent increase. If anything, this increase in smoking should make birth outcomes worse as smoking while pregnant is associated with lower birth weight (Currie, Neidell, and Schmeider 2009).

An additional mechanism through which the EZ program may affect health is through compositional change in these zones. However, there is little evidence of differential compositional change following the EZ program compared to control areas. In Table 5, I present the proportion of individuals still living in the same house, the same county, and the same state as they were 5 years prior to the 1990 and 2000 decennial censuses by EZ status. The last column of this table provides unadjusted differences-in-differences calculations for each row. The results show no differential change in the probability of remaining in the same area following the EZ program.

In Table 2, Panel B, I find little evidence of compositional change specifically among mothers corresponding to EZ adoption. The unadjusted differences-in-differences in Column 7 show that trends in racial composition of mothers are similar across EZs and control groups. Educational attainment of expecting mothers does not change in statistically significant ways, although there is some evidence of EZ mothers becoming slightly more educated than non-EZ mothers. Mothers in EZs are three-tenths of a year older than mothers in control zones at the time of birth following the EZ program, a statistically significant but modest difference.

To test for demographic shifts using regression tools, I use data from before the EZ program and regress each outcome variable on maternal age, race and ethnicity, educational attainment, and child's gender. These regressions include all births conceived before 1995. Using these estimates, I predict the value of each dependent variable over the entire sample, not just the pre-period sample used in the original regression. Next, using a differences-in-differences model, I regress the predicted outcome on an indicator variable for whether the birth occurred in an EZ, the interaction of the variable *EZ* and *Post*, an indicator for whether the baby was conceived in 1995 or later, and year fixed effects which subsume the main effects of the variable *Post*.

Because fertility is a population-level measure, I create predicted fertility rates by categories of age and race based on fertility rates in 1990. I assign predicted fertility as the demographically weighted predicted fertility in zone *s*:

$$Predicted\ Fertility_s = \sum_{r=1}^{4}(\sum_{k=1}^{4} fertility\ rate_{rk} * P_{rks})$$

where $fertility\ rate_{rk}$ is the 1990 national fertility rate of race *r*, non-Hispanic white, non-Hispanic black, Hispanic, or other, and age *k*, a vector of age bins 15-24, 25-29, 30-34, and 35-44. P_{rks} is the proportion of females between the ages of 15 and 44 of a particular race *r*, in age bin *k*, living in zone *s*.

The results for predicted fertility, presented in Table 6, imply that based on the age and race of mothers in EZs, I would expect little change in fertility rates following the EZ program implementation in EZs compared to control zones. For birth

outcomes, the model predicts that birth weight in these areas should decrease slightly, low birth weight should increase slightly, and full-term births should be unaffected relative to control areas. The actual results from the main differences-in-differences specification are oppositely signed and statistically significant. These results imply demographic compositional changes cannot explain the decrease in fertility rates and improvements in birth outcomes following the EZ program.[24]

1.6.3. Heterogeneous Treatment Effects

1.6.3.1. Fertility Analysis by Age

I show in section 1.6.1 that fertility rates decrease in EZs. However, whether this is simply a timing effect in that women delay fertility to a later age, or a true decrease in fertility requires an age-specific analysis. In this section I estimate the same models as in Table 3 separately by age groups 15 to 19, 20 to 24, 25 to 29, 30 to 34, 35 to 39, and 40 to 44. In Table 7, I find the largest decreases in fertility rates in EZs among those aged 20 to 24 and 25 to 29. Fertility decreases by 18 to 19 births per 1,000 women among those aged 20 to 24 and by 19 to 20 births among those aged 25 to 29 compared to control zones. EZs decrease fertility rates among those aged 30 to 34 by approximately 7 births per 1,000. These results are robust to alternate control groups and synthetic control methods. Finally, there is some evidence that EZs decrease fertility rates among those aged 15 to 19, but these results are not robust across specifications. Importantly, these results suggest that the fertility decrease in EZs is not just a timing effect, but a decrease in total fertility.

[24] Appendix Table 2 contains results of a similar analysis using a same city control group.

1.6.3.2. Subgroup Analysis by Education

To examine heterogeneous treatment effects, in Table 8 I separately estimate equation (2) by educational attainment and race. These estimates compare EZs to my preferred control group of application areas and future EZs for all panels of Table 8. I cannot estimate fertility rates by educational attainment because of imprecise estimates of the denominator of women aged 15 to 44 by educational attainment.[25]

For the sample of mothers with a high school diploma or less, which I display in Panel A of Table 8, I find nearly identical point estimates and statistical significance compared to the main results in Table 3. That this estimate is robust to limiting the sample to less educated women is further evidence that compositional changes are not driving my results. However, Panel B presents results using only mothers with at least some college education. These estimates demonstrate that birth weight increases by 43 grams among those living in EZs compared to those living in control zones, with little effect on low birth weight. These estimates of low birth weight are similar to those in Hoynes, Miller, and Simon (2012), who find decreases of between 7 and 11 percent among less-educated women but little effect on low birth weight among those with more than a high school degree.

1.6.3.3. Subgroup Analysis by Race

Because of differences in birth weights and the incidence of low birth weight babies by race,[26] I estimate models separately by mother's race in Panels C through E of Table 8. For blacks, estimates are of both a similar magnitude and statistical

[25] Estimates of the population of women aged 15 to 44 come from decennial Census summary files. These summary files also collect data on the educational attainment of individuals aged 25 or older but do not collect educational attainment by gender and age groups. Any attempt at classifying educational attainment for those under age 25 would be inherently biased.

[26] Black babies have rates of low birth weight nearly twice that of white babies (See e.g. Reichman 2005).

significance compared to those of the main differences-in-differences estimates. The point estimate on low birth weight is slightly larger than the main results, a 1.3 percentage point decrease, but the rate of low birth weight among blacks is 14 percent in the pre-period implying a similar 9 percent reduction in low birth weight due to EZs. For Hispanics, the estimates imply little effect of EZs on fertility rates, but similar improvements in both birth weight and low birth weight rates in EZs compared to control areas. Conversely to other races and ethnicities, whites, who compose just 4 percent of the EZ sample, do not appear to be affected by the EZ program. Despite similar reductions in fertility rates, the point estimate on birth weight is negative but not statistically significant.

1.6.3.4. Heterogeneous Treatment Effects by Zone

The overall average treatment effects of the EZ program masks considerable heterogeneous treatment effects of this program in each EZ. In this section, I present results from estimates that limit the analysis sample to comparisons of each EZ individually to a full set of zones that applied for but did not receive or received the EZ program at a later point in time, estimating both differences-in-differences and synthetic control models. I also estimate these models using same city control areas. However, this sample differs from the same city control sample used in section 1.6.1 in that I only include control areas from the specific EZ city. For example, the analysis of the Chicago EZ uses a control group composed only of Chicago control areas.

Importantly, for each EZ individually, the results are robust to both differences-in-differences and synthetic control methods *and* to alternate, same city

control groups. However the results vary substantially by zone, which I discuss in more detail below.

1.6.3.4.1. Chicago

Estimates using only the Chicago EZ, shown in Table 9, provide substantively similar results to the main estimates using all EZs. Fertility rates in the Chicago EZ decrease substantially compared to control zones using both methods for each control group. These point estimates are statistically significant in all cases. For birth outcomes, the Chicago EZ increases birth weight relative to control areas by between 8 and 27 grams. These results are statistically significant in all cases. Low birth weight rates decrease by between 0.6 and 1.5 percentage points in the Chicago EZ compared to control areas with all estimates, except the synthetic control result in row (2), exhibiting statistical significance. In Panel A of Figure 9, I find that distributional effects of the EZ program using unconditional quantile regressions are substantively similar to the results using all EZs shown in Figure 8.[27]

1.6.3.4.2. New York City

The New York EZ exhibits the smallest decreases in fertility rates of the three EZs, but the largest improvements in birth outcomes as shown in Table 10. The EZ decreases fertility rates by between 4 and 6 births per 1,000. These results are statistically significant in 2 of the 4 specifications. Birth weight in the New York EZ increases by between 43 and 45 grams compared to the main control group using both differences-in-differences and synthetic control methods, but increases between 13 and 27 grams compared to a same city control group. These effects are statistically

[27] This is the same specification as in Figure 8, but performed separately by EZ.

significant in all cases except the same city synthetic control method in row (4). The EZ program decreases low birth weight rates by between 0.5 and 1.8 percentage points compared to control areas. The results are consistent and fairly robust across methods, with the only exception an attenuated point estimate for the same city synthetic control method. Babies born in the New York EZ were 1.6 percentage points more likely to be of full-term. Figure 9, Panel B shows similar distributional effects for New York compared to the pooled results shown in Figure 8.

1.6.3.4.3. Philadelphia

Fertility rates for Philadelphia are of a similar magnitude to those of the main results, as shown in Table 11. However, birth outcomes worsen in the Philadelphia EZ, the only EZ that exhibits this effect. Despite persistent evidence of increases in birth weight in the Philadelphia EZ, low and very low birth weight increase substantially compared to both control groups. The magnitude of these effects is between a 0.5 and 1.2 percentage point increase in very low birth weight and is robust to all control groups and model specifications. Distributional effects for Philadelphia, in Figure 9, Panel C, similarly exhibit opposite results compared to each of the other EZs and the overall EZ effects. They imply worse outcomes for those in the lowest quantiles of the birth weight distribution.

1.6.3.4.4. Potential Explanations for Heterogeneity by Empowerment Zones

The nature of the EZ program provided each zone with a large degree of autonomy over whether to focus resources on economic opportunities or social and community development, and how and when to spend the program resources. The disbursement of funds differed greatly by city both in terms of time and goals.

Nearly two-thirds of the New York EZ's projects focused on economic activity, while this number was closer to one-third for both Chicago and Philadelphia. Meanwhile, nearly two-thirds of projects in Chicago and Philadelphia focused on community development (Government Accountability Office 2006). Projects intended to improve public housing were a substantial part of the Chicago and Philadelphia EZs, but were not a major part of the New York EZ. On the other hand, workforce development was a major aim of the EZ program in New York and Chicago, but less so in Philadelphia (Hebert et al. 2001).

The Philadelphia EZ had significant delays in commencing infrastructure projects. The reasons for these delays include high staff turnover, community activist dissatisfaction, and mayoral interference (Gittell et al. 2001, Hebert et al. 2001). While similar complaints have been made against other EZs, these issues were particularly pronounced in the Philadelphia EZ. Yet, despite these delays, each of these 3 EZs had spent a similar percent of the total amount of block grants received by 2000. Chicago had spent approximately $62 million, while New York had spent approximately $48 million,[28] and Philadelphia spent $50 million.[29] However, it is unclear how much of these funds had been spent on completed projects that could provide tangible benefits to city residents. Despite these various differences in program implementation, no clear explanation for these differences in outcomes emerges.

[28] New York spent an additional $48 million in matching funds from both the city and state by this time for a total of $144 million in EZ funds spent on infrastructure projects.
[29] These numbers come from the US Housing and Urban Development Annual Performance Measurement System (available at http://www5.hud.gov/urban/perms/perms.asp).

1.6.4. Additional Robustness Checks

I perform the synthetic control method matching on both trend and level in Appendix Table 3. The results are consistent with my preferred specification matching on just level. I also perform the main analysis using a control pool of all 24 control zones in Appendix Table 4, using a differences-in-differences model in Panel A and a synthetic control model matched on trend in Panel B and trend and level in Panel C. These results are fairly robust across specifications. Next, I perform all analyses using the year of birth, rather than the year of conception to classify pregnancies relative to the EZ program. I do this because vital statistics data contain residence at the time of birth, not time of conception. These results are both qualitatively and quantitatively similar to those using time of conception and are presented as Appendix Table 5.

1.7. Discussion

This study provides the first estimates of the effect of the empowerment zone program on fertility and birth outcomes. The EZ program is a federal program that potentially increases household income and provides large scale changes in neighborhood infrastructure. It is one of the largest place-based program ever implemented in the US and health effects are an important metric by which to explore the overall effects of this program. Overall, my estimates suggest the EZ program decreases fertility and improves birth outcomes among zone residents compared to control areas.

While I find substantial gains in birth weight and reductions in low birth weight that are similar to those found for the earned income tax credit (Hoynes, Miller, and Simon 2015) and the initial roll-out of food stamps (Almond, Hoynes, and

Schanzenbach 2011), important distinctions between the works remain. First, Hoynes, Miller, and Simon (2012) find very little effect of an increase in income on overall birth weight and the effects on low birth weight are specific to a less-educated, single mother sample. My results for low birth weight are similar in magnitude to theirs, but I also find substantial increases in birth weight. Also, fertility rates decrease by nearly 10 births per 1,000 women in EZs while the earned-income tax credit had little effect on fertility rates (Baughman and Dickert-Conlin 2009).

Interestingly, the per capita costs of the EZ program are much lower than those of the earned income tax credit and the Supplemental Nutrition Assistance Program on an annual basis.[30] By 2000, approximately $600 million in federal funds had been spent on the EZ program, in areas with a total population of approximately 700,000 individuals or $850 per capita over 6 years. For comparison, in 2011 28 million families claimed the earned income tax credit at a total program cost of $60 billion while 45 million individuals were enrolled in SNAP at a cost of $72 billion. These annual per capita costs are between $1000 and $2000.

The income effects of the EZ program (Busso, Gregory, and Kline 2013, Ham et al. 2011) likely led to an increase in demand for higher quality children, decreasing fertility rates and leading parents to invest more in the birth weight production function. Increases in income may lead to substitution away from having children, especially among low earning women, thus improvements in labor market conditions in these cities may explain this decline in fertility (Heckman and Walker 1990, Perry 2004). My sample consists nearly entirely of less-educated women living in high

[30] The Supplemental Nutrition Assistance Program is the current name of the food stamp program.

poverty, high unemployment areas so any improvements in the labor market likely led to the substitution effect swamping the income effect of higher wages on fertility. Moreover, fewer babies born in these areas would reduce demand for prenatal services, potentially affecting overall health by increasing availability of health services.

Parents may invest more in the birth weight production function through more or higher quality prenatal care, or through health behaviors that my model cannot capture such as eating healthier or exercising more while pregnant (Aparicio and Gonzalez 2013). While I find a non-statistically significant increase in receipt of prenatal care, these estimates are suggestive of a potential pathway through which the EZ program affects health. Increases in full-term births may partially explain improvements in birth weight and low birth weight as gestational age is associated with intra-uterine growth and increased birth weight (see e.g. Olsen et al. 2010).

This paper focuses on the short-term health effects of the EZ program. However, the fetal origins literature provides a fairly strong prior for predicting long-term health and labor market effects of the EZ program. This program also reduced poverty, improved labor markets, and improved infrastructure in these neighborhoods. The potential long-term benefits of reducing low birth weight together with the neighborhood-level gains in labor market outcomes could have staggering welfare effects in these areas and may justify the cost of the EZ program.

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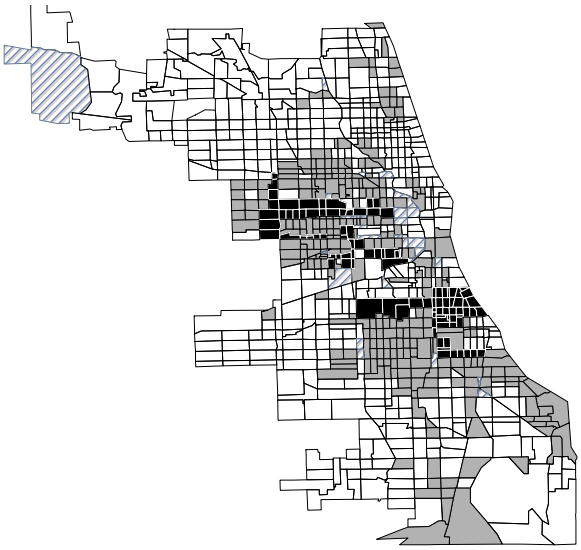
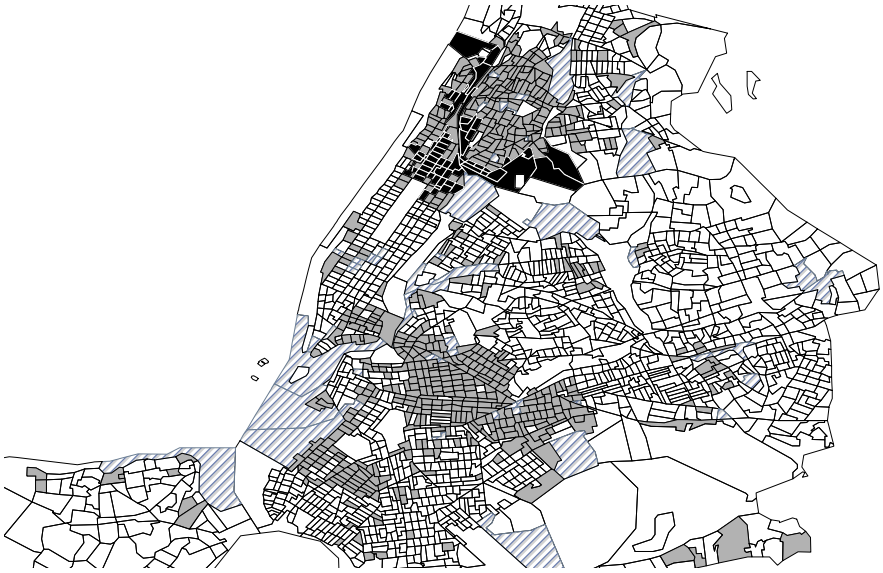
Figure 1.1: Map of Chicago, the Chicago Empowerment Zone, and the Census Tracts that Qualified for Empowerment Zone Status



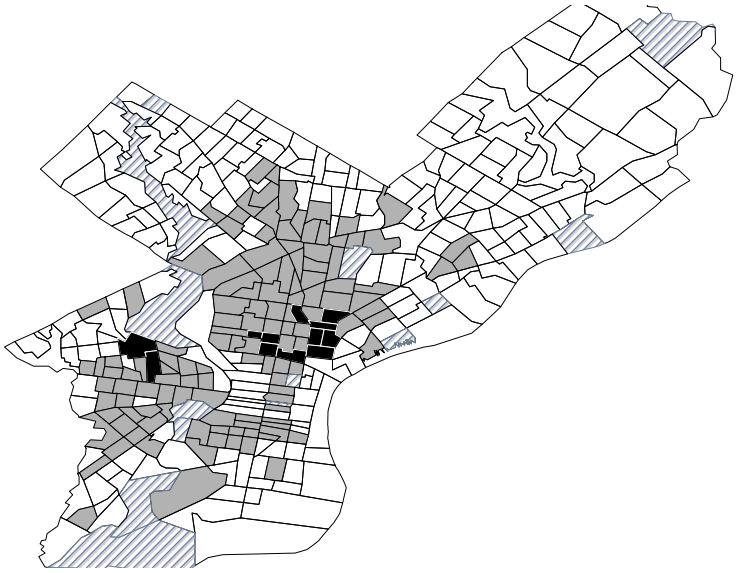
Source: Poverty rates are from the 1990 Decennial Census. Empowerment Zone is shown in black. Areas that qualified for EZ status, with poverty rates above 20% and at least 500 inhabitants, are denoted in gray. Striped areas did not qualify because of populations below 500 individuals. White areas did not have poverty rates above 20%.

Figure 1.2: Map of New York City, the New York Empowerment Zone, and the Census Tracts that Qualified for Empowerment Zone Status



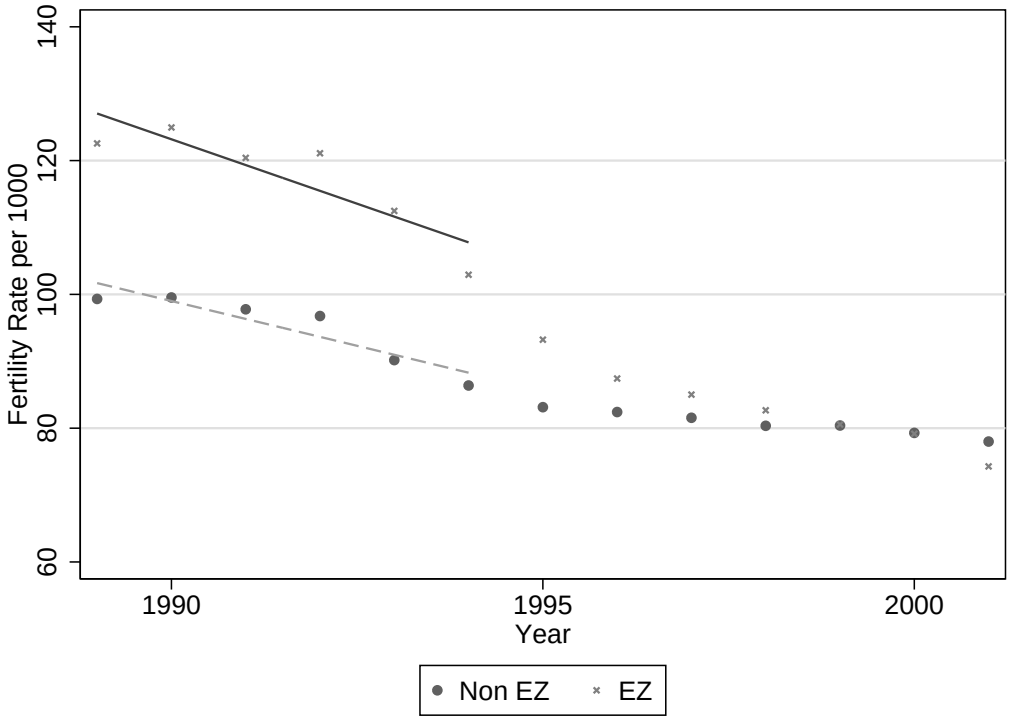
Source: Poverty rates are from the 1990 Decennial Census. Empowerment Zone is shown in black. Areas that qualified for EZ status, with poverty rates above 20% and at least 500 inhabitants, are denoted in gray. Striped areas did not qualify because of populations below 500 individuals. White areas did not have poverty rates above 20%.

Figure 1.3: Map of Philadelphia, the Philadelphia Empowerment Zone, and the Census Tracts that Qualified for Empowerment Zone Status



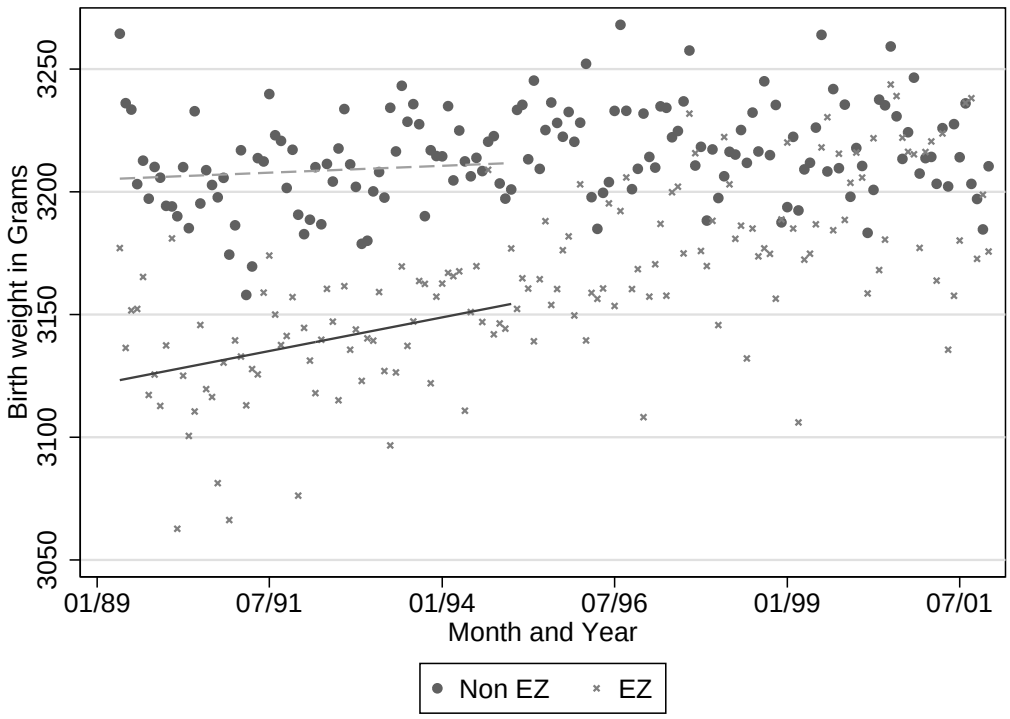
Source: Poverty rates are from the 1990 Decennial Census. Empowerment Zone is shown in black. Areas that qualified for EZ status, with poverty rates above 20% and at least 500 inhabitants, are denoted in gray. Striped areas did not qualify because of populations below 500 individuals. White areas did not have poverty rates above 20%.

Figure 1.4: Average Fertility Annually by Empowerment Zone Status



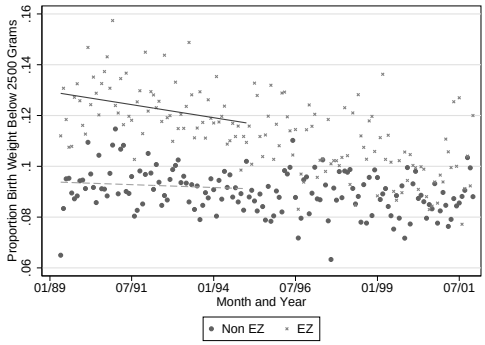
Source: Fertility rates collapsed by year, with linear trend lines for the pre-empowerment zone periods. Chicago, New York, and Philadelphia EZs compared to the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

Figure 1.5: Average Birth Weight by Empowerment Zone Status

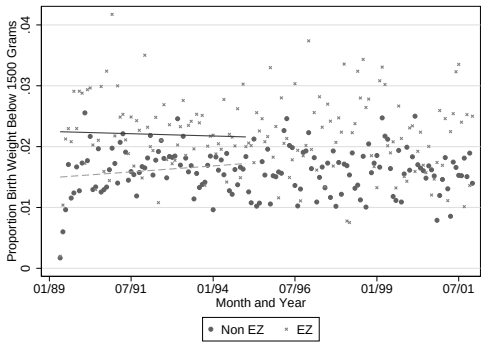


Source: Birth weights collapsed by month, with linear trend lines for the pre-empowerment zone periods. Chicago, New York, and Philadelphia EZs compared to the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

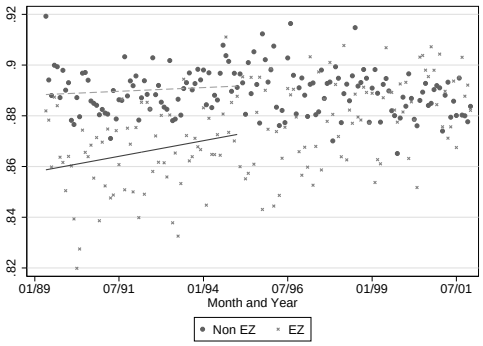
Figure 1.6: Birth Outcomes Separately by Empowerment Zone Status



(a) Low Birth Weight



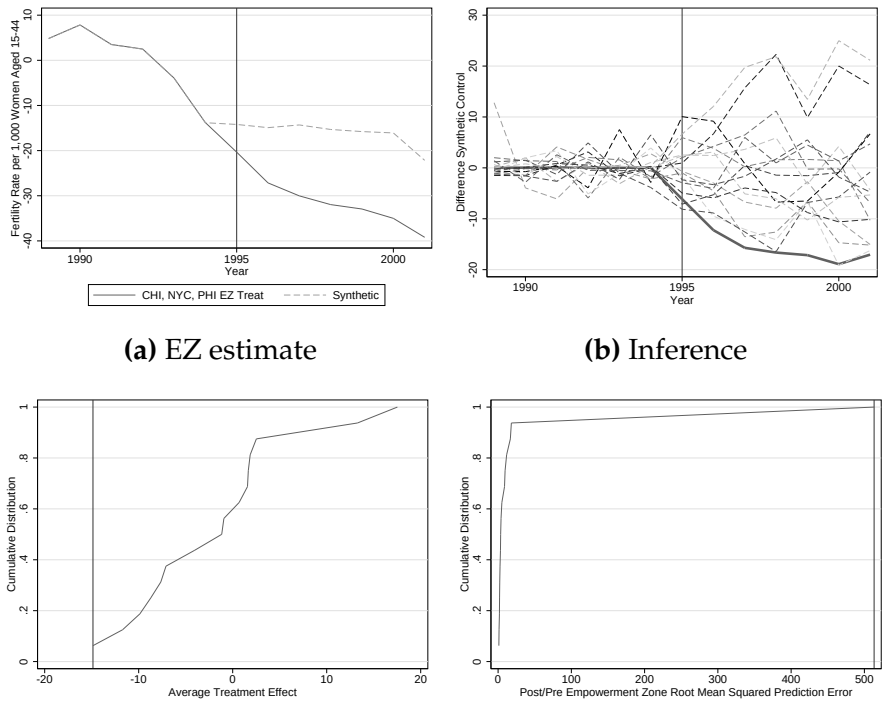
(b) Very Low Birth Weight



(c) Full-term

Source: Low, very low birth weight rates, and full-term birth rates collapsed by month, with linear trend lines for the pre-empowerment zone periods. Chicago, New York, and Philadelphia EZs compared to the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

Figure 1.7: The Effect of Empowerment Zone Status on Fertility Using Synthetic Control Methods

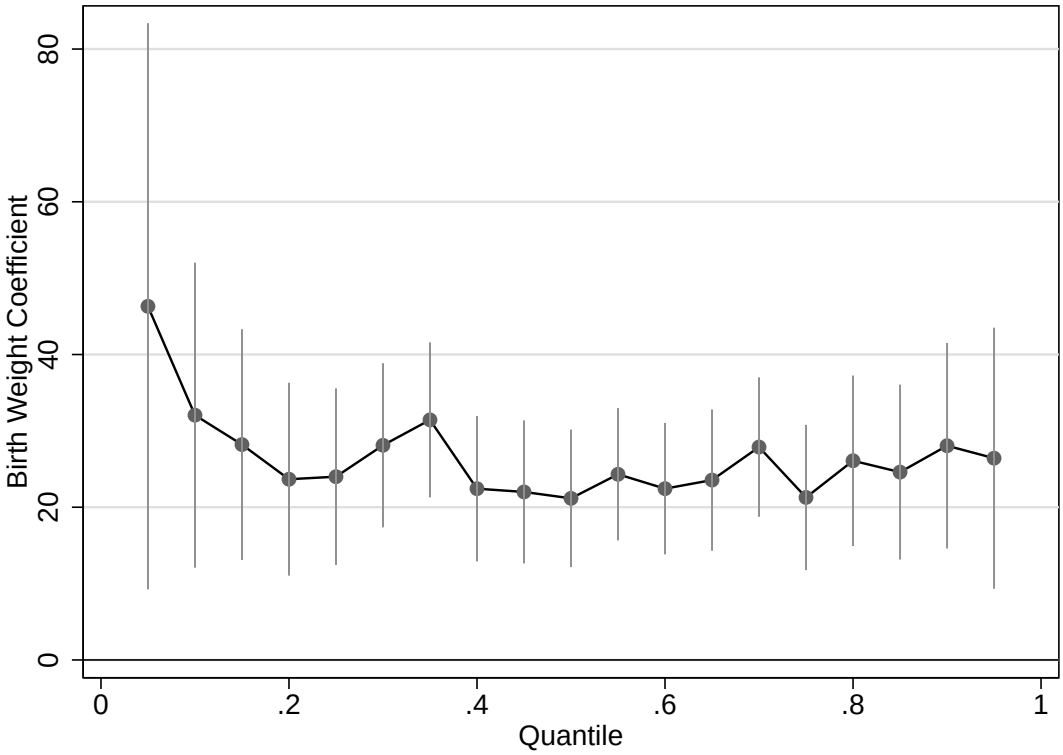


(a) EZ estimate **(b)** Inference

(c) Average Treatment Effect **(d)** Root Mean Sqared Error

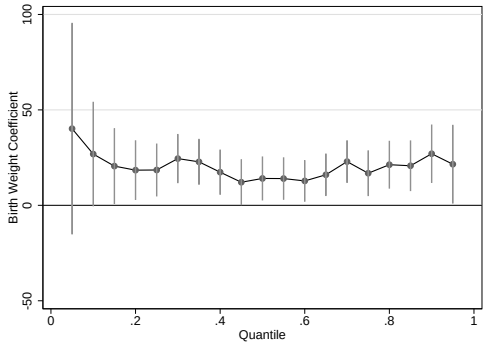
Source: Author's estimation of equation (3) in the text, using the full sample of EZs and the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. Panel (a) provides estimates of the synthetic control method using EZ as the treatment. Panel (b) provides estimates of the effect of EZ status, systematically assigning EZ status to the actual EZ (the solid line) and all control zones (the dotted lines). Panel (c) provides the cumulative distribution of average treatment effects using estimates from the full inference sample, with a vertical line showing the average treatment effect for the actual EZ. Panel (d) provides the cumulative distribution of the ratio of post- to pre-root mean squared prediction error for the full inference sample, with a vertical line showing the value for the actual EZ.

Figure 1.8: The Effect of Empowerment Zone Status on Birth Weight by Quantile

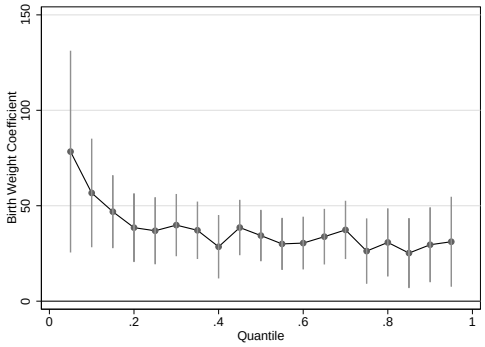


Source: This figure presents results of separate unconditional quantile regressions (Firpo et al. 2009) for every fifth percentile of the birth weight distribution. It uses the full sample of EZs and the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

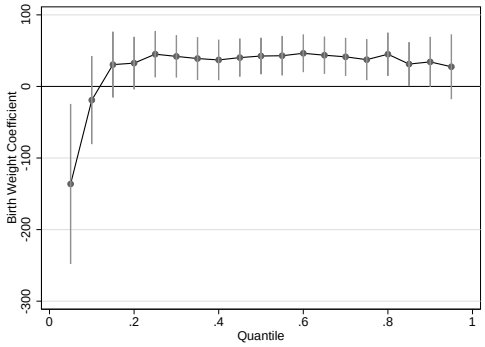
Figure 1.9: The Effect of Empowerment Zone Status on Birth Weight by Quantile



(a) Chicago



(b) New York



(c) Philadelphia

Source: These figures present results of separate unconditional quantile regressions (Firpo et al. 2009) for every fifth percentile of the birth weight distribution. The sample of EZs used in each panel is listed below the figures. Each panel uses the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

Table 1.1: The Effect of Empowerment Zone Status on Birth Weight

Variable	Illinois	New York	Pennsylvania	Colorado	Florida	Kentucky	Ohio	Oregon	Texas
Empowerment Zone	X	X	X						
Control Zone			X	X	X	X	X	X	X
Birth Weight	X	X	X	X	X	X	X	X	X
Mother Race	X	X	X	X	X	X	X	X	X
Mother Ethnicity	X	X	X	X	X	X	X	X	X
Mother Age	X	X	X	X	X	X	X	X	X
Marital Status	X	X	X	X	X	X	X	X	
Educational Attainment	X	X	X	X	X	X	X	X	X
APGAR Score	X	X	X	X	X	X		X	
Prenatal Care	X	X	X	X	X	X	X	X	X
Gestational Period	X	X	X	X	X	X	X	X	X
Plural Birth Indicator	X	X	X	X	X	X	X	X	X
Drinking	X	X	X	X	X	X	X	X	X
Smoking	X	X	X	X	X	X	X	X	X

Source: Vital statistics natality data from each state. I only mark a state as providing a variable if the variable was provided for all years 1990-2002.

Table 1.2: Unadjusted Differences-in-Differences Comparing Demographic Characteristics of Mothers by EZ status and Time

	EZ			Control			
	Pre	Post	Diff	Pre	Post	Diff	D-in-D
Panel A. Dependent Variables							
Fertility Rates (per 1,000 Women Aged 15-44)	117.4	83.19	-34.213***	94.99	80.74	-14.256***	-19.957***
	(18.55)	(11.14)	(5.996)	(16.70)	(20.51)	(2.538)	(5.608)
Fertility Rates (per 1,000 Women Aged 15-19)	147.1	109.4	-37.717***	132.3	110.9	-21.426***	-16.291*
	(34.69)	(27.24)	(5.720)	(26.63)	(24.99)	(5.238)	(7.079)
Fertility Rates (per 1,000 Women Aged 20-24)	204.6	152.2	-52.372***	167.1	146.9	-20.235***	-32.136**
	(48.75)	(26.47)	(12.917)	(43.82)	(49.22)	(5.510)	(12.100)
Fertility Rates (per 1,000 Women Aged 25-29)	145.4	106.4	-39.084***	108.8	103.0	-5.857	-33.227***
	(13.28)	(10.07)	(6.000)	(27.77)	(29.80)	(4.309)	(6.600)
Fertility Rates (per 1,000 Women Aged 30-34)	91.83	71.49	-20.339***	68.41	63.67	-4.746	-15.593***
	(14.18)	(13.61)	(1.919)	(17.48)	(20.99)	(2.650)	(3.092)
Fertility Rates (per 1,000 Women Aged 35-39)	44.21	36.67	-7.543**	32.05	28.73	-3.320***	-4.222
	(6.245)	(8.400)	(2.391)	(10.26)	(10.49)	(0.960)	(2.213)
Fertility Rates (per 1,000 Women Aged 40-44)	10.13	8.935	-1.200	7.744	7.131	-0.613	-0.587
	(2.072)	(2.106)	(0.785)	(4.308)	(3.082)	(0.553)	(0.857)
Birth Weight (BWT)	3138.9	3182.1	43.200*	3209.4	3219.2	9.791	33.408
	(627.7)	(625.5)	(17.681)	(593.6)	(592.3)	(11.480)	(18.732)
Low BWT (< 2500 g)	0.123	0.106	-0.016*	0.0919	0.0882	-0.004	-0.013
	(0.328)	(0.308)	(0.007)	(0.289)	(0.284)	(0.003)	(0.007)
Very Low BWT (< 1500 g)	0.0218	0.0215	-0.000	0.0159	0.0160	0.000	-0.000
	(0.146)	(0.145)	(0.002)	(0.125)	(0.126)	(0.001)	(0.001)
Full-term (37+ Weeks Gestational Age)	0.867	0.881	0.014	0.891	0.889	-0.002	0.016*
	(0.339)	(0.324)	(0.008)	(0.311)	(0.314)	(0.004)	(0.008)
Observations	69043	59267	128310	118099	121698	239797	368094

Table 1.2: (Continued)

	EZ			Control			
	Pre	Post	Diff	Pre	Post	Diff	D-in-D
Panel B. Individual-Level Characteristics							
Child Male Gender	0.509	0.509	(0.000)	0.459	0.511	0.052***	-0.052***
	(0.500)	(0.500)	(0.004)	(0.498)	(0.500)	(0.012)	(0.012)
Mother White	0.0367	0.0337	-0.003	0.115	0.106	-0.008*	0.005
	(0.188)	(0.181)	(0.007)	(0.319)	(0.308)	(0.004)	(0.007)
Mother Black	0.592	0.546	-0.046***	0.381	0.332	-0.049**	0.003
	(0.491)	(0.498)	(0.001)	(0.486)	(0.471)	(0.016)	(0.016)
Mother Other Race	0.00696	0.00923	0.002	0.0149	0.0145	-0.000	0.003
	(0.0831)	(0.0956)	(0.003)	(0.121)	(0.119)	(0.001)	(0.003)
Mother Hispanic	0.365	0.410	0.045***	0.489	0.547	0.058***	-0.013
	(0.481)	(0.492)	(0.008)	(0.500)	(0.498)	(0.018)	(0.019)
Mother Years of Schooling	10.99	11.13	0.142***	10.50	10.71	0.205**	-0.063
	(2.521)	(2.721)	(0.021)	(2.784)	(2.729)	(0.079)	(0.081)
Mom HS Grad	0.489	0.525	0.036***	0.458	0.475	0.016	0.019
	(0.500)	(0.499)	(0.011)	(0.498)	(0.499)	(0.009)	(0.013)
Mom Some College	0.160	0.205	0.045**	0.137	0.161	0.023***	0.021
	(0.367)	(0.404)	(0.015)	(0.344)	(0.367)	(0.005)	(0.013)
Mom College	0.0381	0.0560	0.018	0.0327	0.0457	0.013***	0.005
	(0.191)	(0.230)	(0.009)	(0.178)	(0.209)	(0.003)	(0.008)
Mom Grad	0.0129	0.0205	0.008	0.0113	0.0170	0.006***	0.002
	(0.113)	(0.142)	(0.006)	(0.106)	(0.129)	(0.002)	(0.005)
Mother Age	24.72	25.25	0.531***	23.99	24.22	0.232*	0.299*
	(6.129)	(6.364)	(0.109)	(5.957)	(5.954)	(0.101)	(0.136)
Panel C. Census Tract-Level Characteristics							
Observations	69043	59267	128310	118099	121698	239797	368094

Table 1.2: (Continued)

	EZ			Control			
	Pre	Post	Diff	Pre	Post	Diff	D-in-D
Females Between Age 15-44	600.7	625.7	24.964	681.4	712.3	30.944	-5.979
	(550.5)	(564.4)	(29.311)	(447.6)	(405.5)	(23.219)	(33.755)
Population Area	2600.8	2610.5	9.734	3132.5	3152.0	19.534	-9.800
	(2249.0)	(2310.0)	(118.554)	(1895.7)	(1697.0)	(92.146)	(135.331)
Percent Male Area	0.464	0.468	0.004	0.489	0.491	0.002	0.002
	(0.0829)	(0.0645)	(0.003)	(0.0665)	(0.0655)	(0.003)	(0.004)
Percent White Area	0.119	0.128	0.009	0.384	0.393	0.009	0.000
	(0.177)	(0.168)	(0.013)	(0.282)	(0.265)	(0.011)	(0.015)
Percent Black Area	0.701	0.676	-0.026	0.468	0.443	-0.025*	-0.001
	(0.362)	(0.362)	(0.023)	(0.361)	(0.347)	(0.010)	(0.022)
Percent Other Race Area	0.177	0.196	0.019	0.145	0.163	0.018***	0.001
	(0.237)	(0.233)	(0.011)	(0.173)	(0.163)	(0.004)	(0.010)
Percent Hispanic Area	0.257	0.276	0.019*	0.289	0.313	0.023***	-0.005
	(0.329)	(0.334)	(0.008)	(0.355)	(0.358)	(0.005)	(0.008)
Percent No HS Grad	0.536	0.461	-0.075***	0.495	0.441	-0.054***	-0.021*
	(0.137)	(0.127)	(0.007)	(0.150)	(0.156)	(0.007)	(0.009)
Percent HS Grad	0.464	0.549	0.085***	0.505	0.570	0.065***	0.019*
	(0.137)	(0.134)	(0.005)	(0.150)	(0.162)	(0.007)	(0.008)
Percent Some College	0.160	0.202	0.042***	0.171	0.190	0.020***	0.023***
	(0.0679)	(0.0864)	(0.002)	(0.0726)	(0.0673)	(0.004)	(0.005)
Percent College Grad	0.0646	0.0909	0.026***	0.0828	0.107	0.024***	0.002
	(0.0603)	(0.0738)	(0.002)	(0.0820)	(0.0985)	(0.005)	(0.005)
Median HH Income	19712.5	23577.4	3864.952**	20822.1	24209.3	3387.238***	477.714
	(9061.9)	(10556.6)	(1326.526)	(8201.6)	(9401.2)	(442.206)	(1198.105)
Observations	69043	59267	128310	118099	121698	239797	368094

Table 1.2: (Continued)

	EZ			Control			
	Pre	Post	Diff	Pre	Post	Diff	D-in-D
Percent Vacant Housing	0.154	0.162	0.008	0.166	0.143	-0.023*	0.031*
	(0.107)	(0.111)	(0.009)	(0.0851)	(0.0707)	(0.011)	(0.013)
Percent Below Poverty	0.476	0.423	-0.053***	0.436	0.388	-0.047***	-0.006
	(0.163)	(0.145)	(0.014)	(0.135)	(0.126)	(0.006)	(0.014)
Percent Below Poverty Age 18-44	0.432	0.380	-0.052***	0.414	0.393	-0.021**	-0.031***
	(0.153)	(0.119)	(0.004)	(0.142)	(0.138)	(0.007)	(0.008)
Percent Below Poverty Age 18-24	0.460	0.425	-0.035***	0.467	0.453	-0.014	-0.022
	(0.184)	(0.160)	(0.009)	(0.170)	(0.167)	(0.009)	(0.012)
Percent Below Poverty Age 25-34	0.422	0.374	-0.048***	0.407	0.382	-0.025**	-0.023**
	(0.182)	(0.157)	(0.003)	(0.156)	(0.156)	(0.008)	(0.008)
Percent Below Poverty Age 35-44	0.392	0.339	-0.054***	0.366	0.346	-0.019**	-0.035***
	(0.158)	(0.134)	(0.001)	(0.168)	(0.155)	(0.007)	(0.007)
Percent Below Poverty Age 18-64	0.426	0.384	-0.042*	0.390	0.350	-0.040***	-0.002
	(0.162)	(0.137)	(0.017)	(0.135)	(0.124)	(0.006)	(0.015)
Percent Below Poverty Under Age 17	0.599	0.572	-0.028*	0.571	0.564	-0.007	-0.021
	(0.180)	(0.176)	(0.014)	(0.167)	(0.157)	(0.011)	(0.016)
Females Between Age 15-44	600.7	625.7	24.964	681.4	712.3	30.944	-5.979
	(550.5)	(564.4)	(29.311)	(447.6)	(405.5)	(23.219)	(33.755)
Females Between Age 15-24	206.1	211.3	5.241	251.7	263.1	11.447	-6.206
	(187.3)	(192.8)	(10.523)	(219.7)	(209.4)	(10.772)	(13.888)
Percent Females Between Age 15-44	0.228	0.235	0.007	0.223	0.226	0.003	0.004
	(0.0616)	(0.0457)	(0.005)	(0.0494)	(0.0421)	(0.002)	(0.005)
Observations	69043	59267	128310	118099	121698	239797	368094

Source: Data are from vital statistics data from the following states: Colorado, Florida,

Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. The limited sample of controls (Busso et al. 2013) is used, excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. The sample consists of 3 empowerment zones and 16 control zones. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 0.1% level, ** indicates significance at the 1% level, * indicates significance at the 5% level.

Table 1.3: The Effect of Empowerment Zone Status on Birth Weight

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Cross City Control Zones					
(1) Differences-in-	-9.6***	25.5***	-0.008***	0.000	0.009**
Differences	(2.914)	(8.290)	(0.003)	(0.001)	(0.004)
Obs	234	360526	360526	360526	354477
Dep Var Mean	89.3	3197.9	0.098	0.017	0.883
(2) Synthetic Controls	-14.86*+	17.90	-0.013+	0.001	0.018+
	(0.063)	(0.250)	(0.188)	(0.563)	(0.875)
	(0.000)	(0.125)	(0.000)	(0.500)	(0.063)
Panel B. Same City Control Zones					
(3) Differences-in-	-11.4***	12.8	-0.006	0.001	0.004
Differences	(3.425)	(11.329)	(0.004)	(0.001)	(0.004)
Obs	1285	938829	938829	938829	923646
Dep Var Mean	79.5	3197.5	0.101	0.018	0.891
(4) Synthetic Controls	-10.2+	18.7	-0.007	0.0006	0.006
	(0.179)	(0.262)	(0.238)	(0.571)	(0.667)
	(0.036)	(0.488)	(0.452)	(0.452)	(0.440)

Source: Author's estimation of equation (2) and (3) in the text using all 3 empowerment zones and control zones listed in the panel. Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. In Panel A, the analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Panel B uses a control sample of babies born to mothers living in census tracts in the same city as EZs with poverty rates above 20%. Each cell in the table comes from a separate regression. The estimates include controls for mother's race and ethnicity, child's gender, and year fixed effects, controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates, and tract level fixed effects. Fertility rate regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level. The numbers in parentheses for synthetic controls represent the implied p value of the average treatment effect (ATE), and the implied p value of the ratio of the post to pre root mean squared prediction error (MSPE). * indicates significance at the 10% level using ATE, + indicates significance at the 10% level using MSPE.

Table 1.4: The Effect of Empowerment Zone Status on Maternal Outcomes: Differences-in-Differences Results

	No Prenatal	Month Prenatal	Smoke
Panel A. Cross City Control Zones			
All EZs	-0.018	-0.157	0.020**
	(0.016)	(0.176)	(0.009)
Obs	349759	344051	354643
Dep Var Mean	0.061	3.231	0.148
Panel B. Same City Control Zones			
All EZs	0.001	0.049	-0.005
	(0.013)	(0.159)	(0.011)
Obs	885761	842673	934350
Dep Var Mean	0.054	3.875	0.105

Source: Author's estimation of equation (2) in the text using all 3 empowerment zones and control zones listed in the panel. In Panel A, the analysis sample consists of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Panel B uses a control sample of babies born to mothers living in census tracts in the same city as EZs with poverty rates above 20%. Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Each cell in the table comes from a separate regression. No Prenatal denotes the mother did not receive prenatal care during pregnancy, Month Prenatal refers to the month a mother first received prenatal care, contingent on ever receiving such care, Apgar score is a composite measure used to assess infant health at 5 minutes of age and a score of 7 or above is considered normal, Smoking refers to ever smoking during pregnancy, and Full-term refers to gestational age of 37 weeks or more. The estimates include controls for mother's race and ethnicity, child's gender, and year fixed effects. Additional specifications labeled TractDemographics also include controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates. Specification TractFE additionally includes tract level fixed effects. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level.

Table 1.5: The Proportion of Individuals Living in the Same House, County, and State as 5 Years Ago as of the 1990 and 2000 Decennial Census

	Empowerment Zones		Control Areas		D-in-D
	1990	2000	1990	2000	
Living in the Same House as 5 Years Ago	0.600	0.594	0.489	0.466	0.017
	(0.137)	(0.145)	(0.137)	(0.135)	(0.019)
Living in the Same County as 5 years Ago	0.891	0.887	0.859	0.835	0.020
	(0.139)	(0.088)	(0.132)	(0.142)	(0.018)
Living in the Same State as 5 years Ago	0.935	0.928	0.921	0.899	0.014
	(0.078)	(0.060)	(0.070)	(0.094)	(0.011)
N	168	168	303	303	940

Source: Summary statistics from the 1990 and 2000 Decennial Censuses. D-in-D presents results from an unadjusted differences-in-differences model.

Table 1.6: The Effect of Empowerment Zone Status on Predicted Birth Outcomes Using 1990-1994 Data to Predict Outcomes

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Chicago, New York, and Philadelphia Zones Compared to Control Zones					
Predicted	0.209	-4.259	0.001	0.0001	-0.0001
	(1.904)	(4.037)	(0.001)	(0.0002)	(0.001)
Panel B. Only Chicago Zones Compared to Control Zones					
Predicted	3.610***	-8.571**	0.002**	0.0003	-0.001
	(1.105)	(3.684)	(0.001)	(0.0002)	(0.001)
Panel C. Only New York Zones Compared to Control Zones					
Predicted	-2.490**	-6.780*	0.002*	0.0003	-0.001
	(1.105)	(3.769)	(0.001)	(0.0002)	(0.0008)
Panel D. Only Philadelphia Zones Compared to Control Zones					
Predicted	-0.492	-11.00***	0.003***	0.0004*	-0.002*
	(1.105)	(3.729)	(0.001)	(0.0002)	(0.001)

Source: Author's estimation using predicted values of fertility rates calculated using demographically weighted 1990 national age-race fertility rates. Predicted values of birth outcomes calculated by regressing birth outcome on maternal demographic characteristics including race and ethnicity, educational attainment, and mothers age at the time of birth for 1989-1994. Predicted estimates come from regressing predicted outcomes on an indicator variable for whether the birth occurred in an EZ, the interaction of the variable EZ and post, an indicator for whether the baby was conceived in 1995 or later, and year fixed effects which subsume the variable post. I use all EZs and the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level.

Table 1.7: The Effect of Empowerment Zone Status on Fertility Rates by Age

	15-19	20-24	25-29	30-34	35-39	40-44
Panel A. Cross City Control Zones						
(1) All EZs	-1.608	-17.800*	-19.225***	-6.484**	-2.939	-0.176
	(4.626)	(9.416)	(4.092)	(3.033)	(1.920)	(0.907)
Obs	234	234	234	234	234	234
Dep Var Mean	134.765	173.374	114.935	72.317	34.074	8.143
% Change	-1.2	-10.3	-16.7	-9.0	-8.6	-2.2
(2) Synthetic Controls	-17.427^{+}	-29.722^{+}	-19.205*$^{+}$	-10.841	-1.053	-0.716
	(0.313)	(0.125)	(0.063)	(0.250)	(0.500)	(0.313)
	(0.000)	(0.000)	(0.000)	(0.188)	(0.188)	(0.563)
Panel B. Same City Control Zones						
(3) All EZs	-16.685*	-19.141*	-19.561***	-7.410**	0.256	-0.903
	(8.833)	(10.500)	(5.037)	(3.081)	(3.612)	(0.584)
Obs	1283	1285	1285	1285	1285	1284
Dep Var Mean	88.130	134.485	104.016	74.395	36.311	8.213
% Change	-18.9	-14.2	-18.8	-10.0	0.7	-11.0
(4) Synthetic Controls	-25.742*	-22.305	-12.028	-17.110*	-0.929	-1.560
	(0.072)	(0.107)	(0.202)	(0.095)	(0.476)	(0.262)
	(0.157)	(0.131)	(0.119)	(0.321)	(0.381)	(0.286)

Source: Authors estimation of equation (2) in the text using all 3 empowerment zones and control zones listed in the panel. Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The analysis sample is composed of fertility rates calculated as the number of babies born to mothers living in empowerment zones or control areas between the stated ages divided by the total population of women in these areas between those ages. In Panel A, the analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Panel B uses a control sample of babies born to mothers living in census tracts in the same city as EZs with poverty rates above 20%. Each cell in the table comes from a separate regression. All regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. The estimates include controls for mother's age, race and ethnicity, child's gender, high school graduate, some college, and college graduate, and year fixed effects, controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates, and program-level fixed effects. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level.

Table 1.8: The Effect of Empowerment Zone Status on Birth Weight: Differences-in-Differences Heterogeneous Treatment Effects

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. High School Diploma or Less					
All EZs		23.374***	-0.009**	0.000	0.009*
		(9.042)	(0.004)	(0.001)	(0.005)
Obs		302760	302760	302760	297367
Dep Var Mean		3187.956	0.100	0.017	0.881
Panel B. More Than a High School Diploma					
All EZs		43.092***	-0.003	0.002	0.011***
		(7.753)	(0.003)	(0.002)	(0.003)
Obs		57766	57766	57766	57110
Dep Var Mean		3249.889	0.085	0.017	0.895
Panel C. Black Only					
All EZs	-11.131**	35.319***	-0.013**	-0.000	0.018*
	(4.510)	(13.105)	(0.007)	(0.002)	(0.010)
Obs	234	155288	155288	155288	152832
Dep Var Mean	101.177	3073.125	0.140	0.026	0.846
Panel D. Hispanic Only					
All EZs	-2.107	26.405***	-0.005**	0.000	-0.001
	(16.098)	(9.160)	(0.002)	(0.001)	(0.002)
Obs	232	170883	170883	170883	167519
Dep Var Mean	115.359	3291.944	0.064	0.010	0.916
Panel E. White Only					
All EZs	-7.460	-17.742	-0.004	0.004	0.008
	(12.274)	(18.751)	(0.007)	(0.003)	(0.008)
Obs	234	30457	30457	30457	30288
Dep Var Mean	68.495	3302.113	0.076	0.012	0.906

Source: Author's estimation of equation (2) in the text using all 3 empowerment zones and control zones listed in the panel . Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Each cell in the table comes from a separate regression. The estimates include controls for mother's race and ethnicity, child's gender, and year fixed effects, controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates, and tract level fixed effects. Fertility rate regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. Standard errors clustered

at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level.

Table 1.9: The Effect of Empowerment Zone Status on Birth Weight: Chicago Results

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Only Chicago Zone Compared to Cross City Control Areas					
(1) Differences-in-Differences	-13.2***	18.1***	-0.007***	0.001	0.005
	(2.145)	(6.368)	(0.002)	(0.001)	(0.004)
Obs	208	298963	298963	298963	293550
Dep Var Mean	88.9	3202.1	0.095	0.017	0.886
(2) Synthetic Controls	-21.7*+	8.7+	-0.0120	0.001	0.012+
	(0.063)	(0.375)	(0.250)	(0.625)	(0.750)
	(0.063)	(0.000)	(0.125)	(0.563)	(0.063)
Panel B. Only Chicago Zone Compared to Same City Control Areas					
(3) Differences-in-Differences	-12.4***	16.1***	-0.006***	-0.001	0.004*
	(3.245)	(5.676)	(0.002)	(0.001)	(0.002)
Obs	377	203937	203937	203937	200794
Dep Var Mean	79.5	3174.7	0.107	0.020	0.882
(4) Synthetic Controls	-16.5+	26.8	-0.015+	-0.001	0.004+
	(0.200)	(0.250)	(0.150)	(0.526)	(0.650)
	(0.000)	(0.100)	(0.000)	(0.211)	(0.000)

Source: Author's estimation of equations (2) and (3) in the text using the Chicago empowerment zone and control zones listed in the panel. Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, Ohio, Oregon, Pennsylvania, and Texas. In Panel A, the analysis sample is composed of babies born to mothers living in the Chicago EZ or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Panel B uses a control sample of babies born to mothers living in census tracts in Chicago with poverty rates above 20%. Each cell in the table comes from a separate regression. The estimates include controls for mother's race and ethnicity, child's gender, and year fixed effects, controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates, and tract level fixed effects. Fertility rate regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level. The numbers in parentheses for synthetic controls represent the implied p value of the average treatment effect (ATE). The numbers in parentheses the next row represent the implied p value of the ratio of the post to pre root mean squared prediction error (MSPE). * indicates significance at the 10% level using ATE, + indicates significance at the 10% level using MSPE.

Table 1.10: The Effect of Empowerment Zone Status on Birth Weight: New York Results

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Only New York Zone Compared to Cross City Control Areas					
(1) Differences-in-	-4.1	42.6***	-0.015***	-0.002	0.016***
Differences	(3.208)	(6.589)	(0.003)	(0.001)	(0.003)
Obs	208	287105	287105	287105	282458
Dep Var Mean	87.1	3212.4	0.092	0.016	0.888
(2) Synthetic Controls	-4.6^{+}	44.9*	-0.018	-0.004	0.030
	(0.375)	(0.000)	(0.125)	(0.125)	(0.875)
	(0.063)	(0.313)	(0.438)	(0.375)	(0.188)
Panel B. Only New York Zone Compared to Same City Control Areas					
(3) Differences-in-	-6.4**	27.5***	-0.012***	0.000	0.011***
Differences	(2.539)	(3.851)	(0.002)	(0.001)	(0.001)
Obs	546	540209	540209	540209	530265
Dep Var Mean	69.8	3238.6	0.088	0.017	0.900
(4) Synthetic Controls	-4.6	13.3	-0.005	0.000^{+}	0.012
	(0.244)	(0.317)	(0.268)	(0.439)	(0.854)
	(0.439)	(0.415)	(0.195)	(0.049)	(0.512)

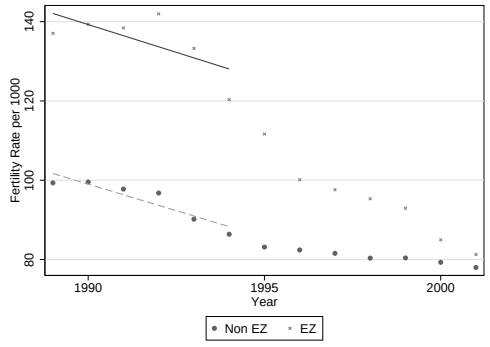
Source: Author's estimation of equations (2) and (3) in the text using the New York empowerment zone and control zones listed in the panel. Data are from vital statistics data from the following states: Colorado, Florida, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. In Panel A, the analysis sample is composed of babies born to mothers living in the New York EZ or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Panel B uses a control sample of babies born to mothers living in census tracts in Chicago with poverty rates above 20%. Each cell in the table comes from a separate regression. The estimates include controls for mother's race and ethnicity, child's gender, and year fixed effects, controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates, and tract level fixed effects. Fertility rate regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level. The numbers in parentheses for synthetic controls represent the implied p value of the average treatment effect (ATE). The numbers in parentheses the next row represent the implied p value of the ratio of the post to pre root mean squared prediction error (MSPE). * indicates significance at the 10% level using ATE, + indicates significance at the 10% level using MSPE.

Table 1.11: The Effect of Empowerment Zone Status on Birth Weight: Philadelphia Results

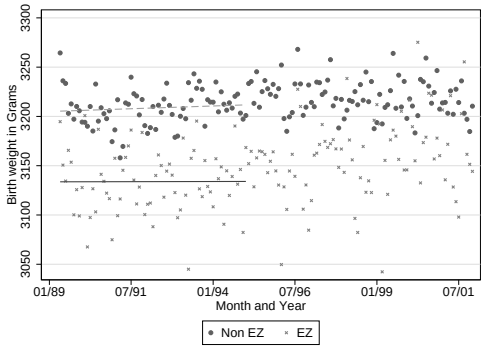
	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Only Philadelphia Zone Compared to Cross City Control Areas					
(1) Differences-in-Differences	-11.5***	16.1**	0.008***	0.009***	-0.003
	(3.238)	(7.830)	(0.003)	(0.001)	(0.004)
Obs	208	245536	245536	245536	241431
Dep Var Mean	88.1	3212.3	0.091	0.016	0.891
(2) Synthetic Controls	-21.5*+	28.6*	-0.002	0.007	-0.003
	(0.063)	(0.063)	(0.375)	(0.938)	(0.563)
	(0.063)	(0.188)	(0.588)	(0.688)	(0.188)
Panel B. Only Philadelphia Zone Compared to Same City Control Areas					
(3) Differences-in-Differences	-14.5***	0.6	0.009**	0.012***	-0.003
	(3.057)	(8.353)	(0.004)	(0.002)	(0.005)
Obs	362	105712	105712	105712	104642
Dep Var Mean	78.9	3130.5	0.121	0.023	0.865
(4) Synthetic Controls	-14.4	6.4+	0.010	0.005	0.002
	(0.125)	(0.360)	(0.800)	(0.840)	(0.583)
	(0.375)	(0.000)	(0.520)	(0.800)	(0.500)

Source: Author's estimation of equations (2) and (3) in the text using the Philadelphia empowerment zone and control zones listed in the panel. Data are from vital statistics data from the following states: Colorado, Florida, Kentucky, Ohio, Oregon, Pennsylvania, and Texas. In Panel A, the analysis sample is composed of babies born to mothers living in the Philadelphia EZ or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Panel B uses a control sample of babies born to mothers living in census tracts in Philadelphia with poverty rates above 20%. Each cell in the table comes from a separate regression. The estimates include controls for mother's race and ethnicity, child's gender, and year fixed effects, controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates, and tract level fixed effects. Fertility rate regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level. The numbers in parentheses for synthetic controls represent the implied p value of the average treatment effect (ATE). The numbers in parentheses the next row represent the implied p value of the ratio of the post to pre root mean squared prediction error (MSPE). * indicates significance at the 10% level using ATE, + indicates significance at the 10% level using MSPE.

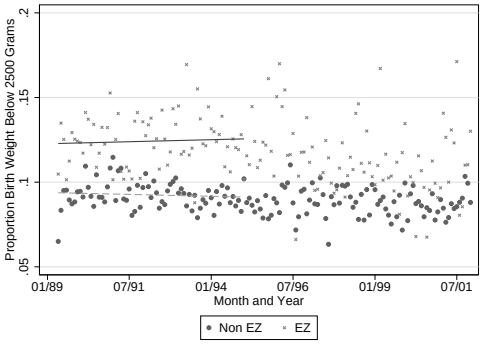
Figure 1.A1: Fertility and Birth Outcomes Separately by Empowerment Zone Status: Chicago EZ Only



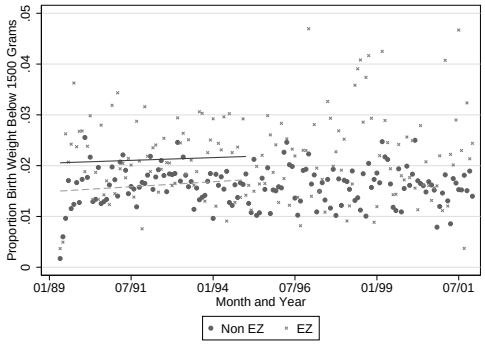
(a) Fertility



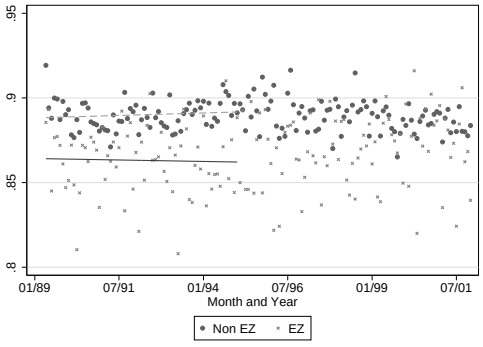
(b) Birth Weight



(c) Low Birth Weight



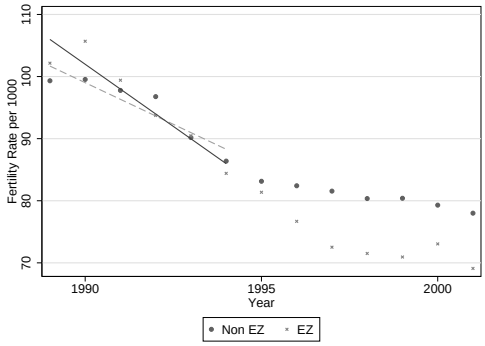
(d) Very Low Birth Weight



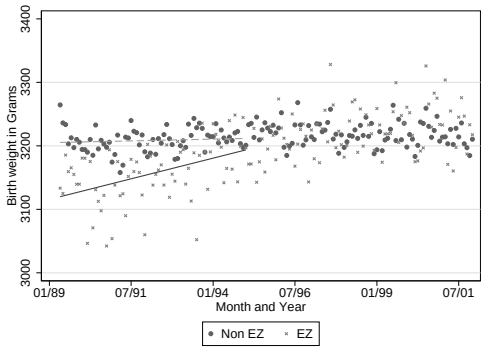
(e) Full-term

Source: Fertility and birth outcomes collapsed by year or month, with linear trend lines for both the pre- and post- empowerment zone periods. Chicago EZ compared to the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

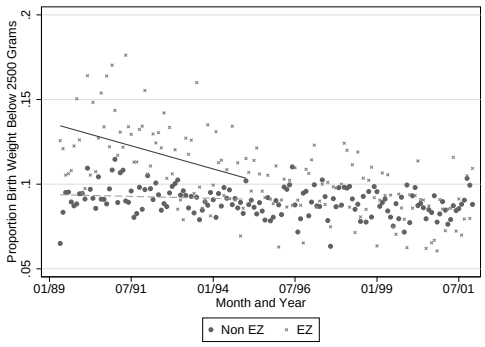
Figure 1.A2: Fertility and Birth Outcomes Separately by Empowerment Zone Status: New York EZ Only



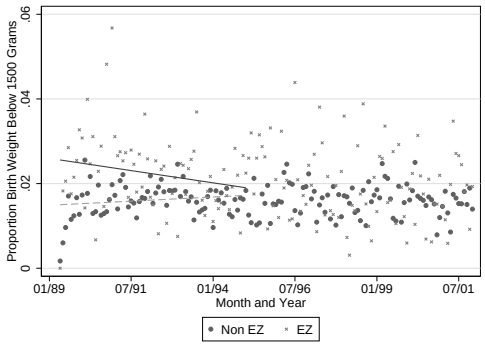
(a) Fertility



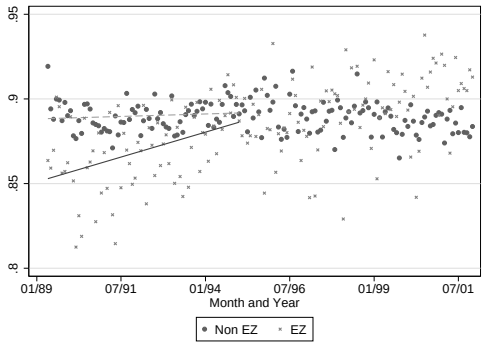
(b) Birth Weight



(c) Low Birth Weight



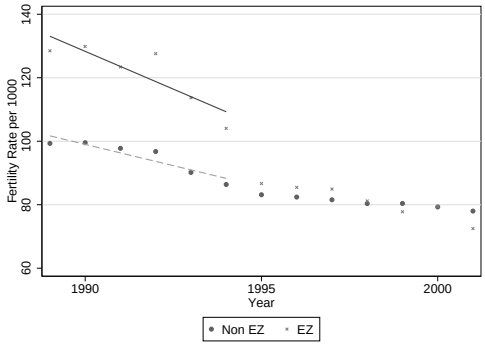
(d) Very Low Birth Weight



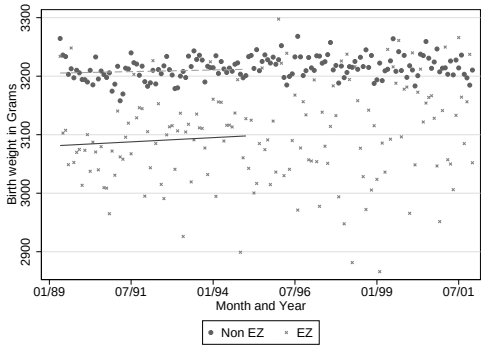
(e) Full-term

Source: Fertility and birth outcomes collapsed by year or month, with linear trend lines for both the pre- and post- empowerment zone periods. New York EZ compared to the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

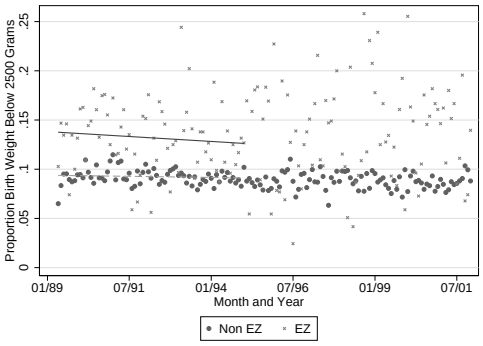
Figure 1.A3: Fertility and Birth Outcomes Separately by Empowerment Zone Status: Philadelphia EZ Only



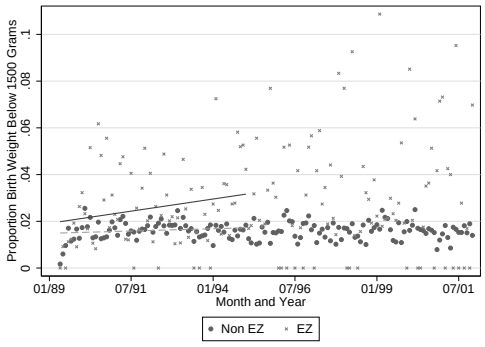
(a) Fertility



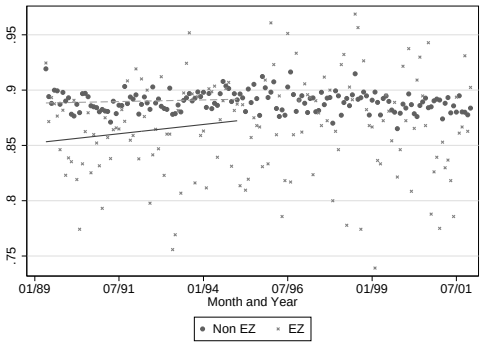
(b) Birth Weight



(c) Low Birth Weight



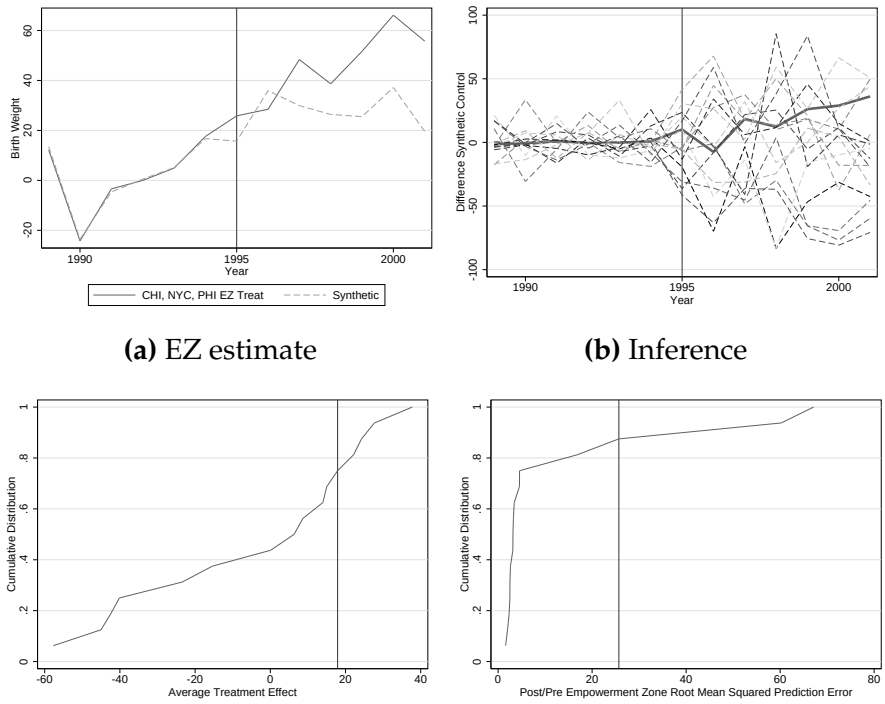
(d) Very Low Birth Weight



(e) Full-term

Source: Fertility and birth outcomes collapsed by year or month, with linear trend lines for both the pre- and post- empowerment zone periods. Philadelphia EZ compared to the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone.

Figure 1.A4: The Effect of Empowerment Zone Status on Birth Weight Using Synthetic Control Methods

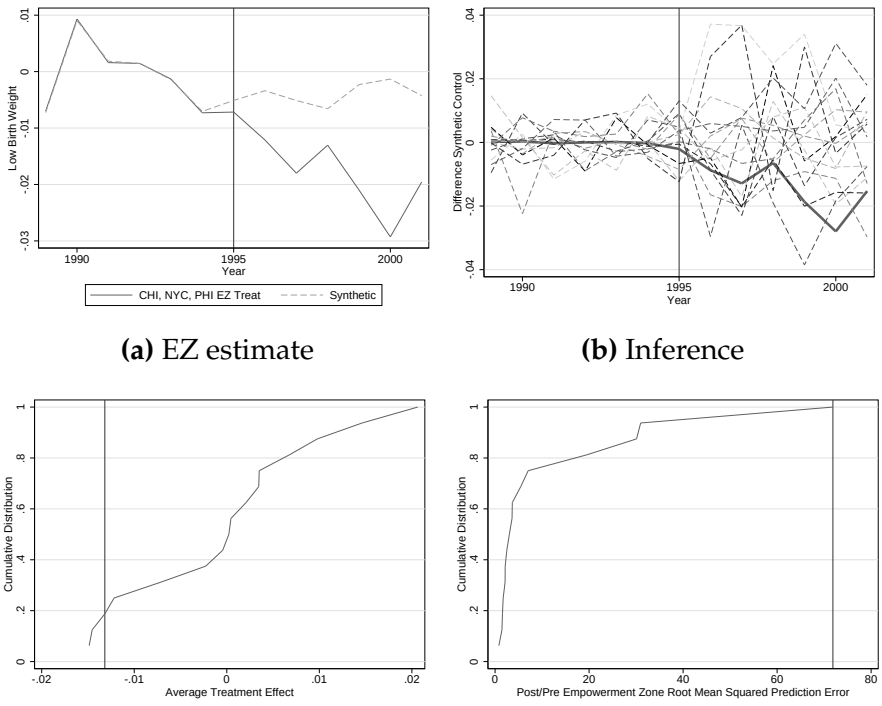


(a) EZ estimate **(b)** Inference

(c) Average Treatment Effect **(d)** Root Mean Sqared Error

Source: Author's estimation of equation (3) in the text, using the full sample of EZs and the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. Panel (a) provides estimates of the synthetic control method using EZ as the treatment. Panel (b) provides estimates of the effect of EZ status, systematically assigning EZ status to the actual EZ (the solid line) and all control zones (the dotted lines). Panel (c) provides the cumulative distribution of average treatment effects using estimates from the full inference sample, with a vertical line showing the average treatment effect for the actual EZ. Panel (d) provides the cumulative distribution of the ratio of post- to pre-root mean squared prediction error for the full inference sample, with a vertical line showing the value for the actual EZ.

Figure 1.A5: The Effect of Empowerment Zone Status on Low Birth Weight Using Synthetic Control Methods

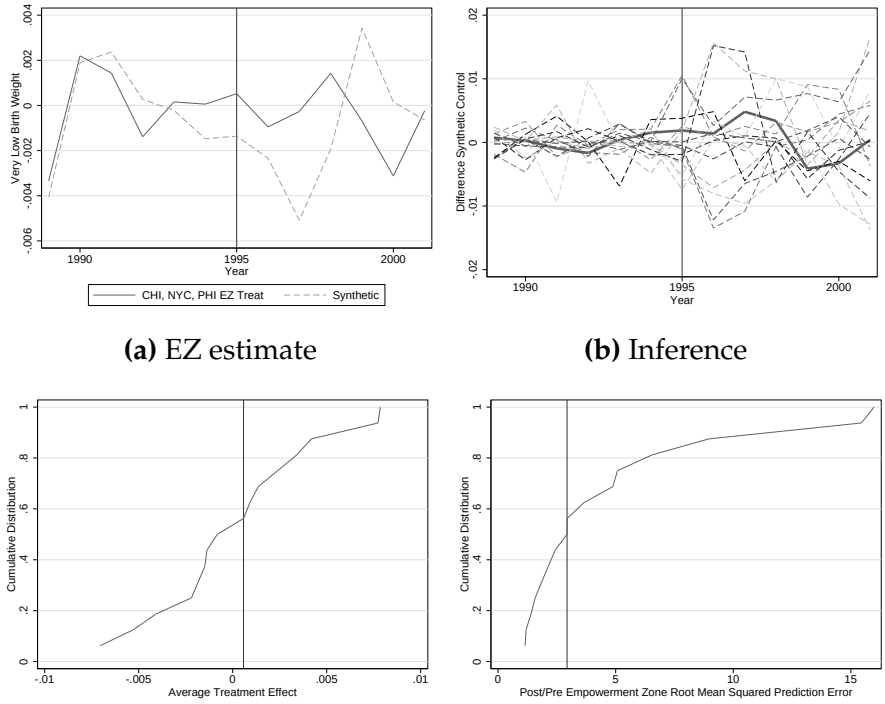


(a) EZ estimate **(b)** Inference

(c) Average Treatment Effect **(d)** Root Mean Sqared Error

Source: Author's estimation of equation (3) in the text, using the full sample of EZs and the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. Panel (a) provides estimates of the synthetic control method using EZ as the treatment. Panel (b) provides estimates of the effect of EZ status, systematically assigning EZ status to the actual EZ (the solid line) and all control zones (the dotted lines). Panel (c) provides the cumulative distribution of average treatment effects using estimates from the full inference sample, with a vertical line showing the average treatment effect for the actual EZ. Panel (d) provides the cumulative distribution of the ratio of post- to pre-root mean squared prediction error for the full inference sample, with a vertical line showing the value for the actual EZ.

Figure 1.A6: The Effect of Empowerment Zone Status on Very Low Birth Weight Using Synthetic Control Methods

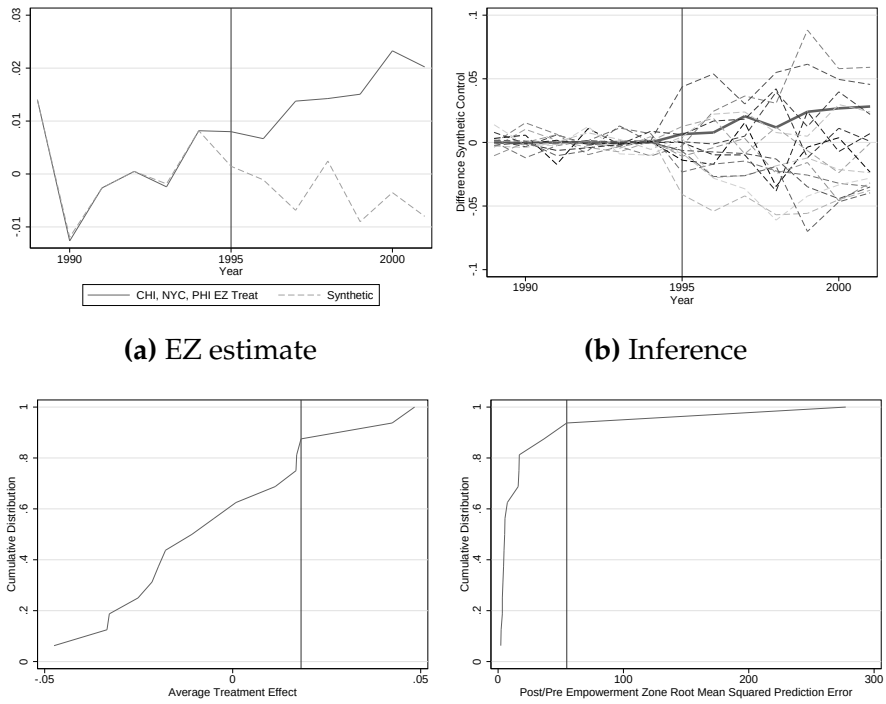


(a) EZ estimate **(b)** Inference

(c) Average Treatment Effect **(d)** Root Mean Sqared Error

Source: Author's estimation of equation (3) in the text, using the full sample of EZs and the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. Panel (a) provides estimates of the synthetic control method using EZ as the treatment. Panel (b) provides estimates of the effect of EZ status, systematically assigning EZ status to the actual EZ (the solid line) and all control zones (the dotted lines). Panel (c) provides the cumulative distribution of average treatment effects using estimates from the full inference sample, with a vertical line showing the average treatment effect for the actual EZ. Panel (d) provides the cumulative distribution of the ratio of post- to pre-root mean squared prediction error for the full inference sample, with a vertical line showing the value for the actual EZ.

Figure 1.A7: The Effect of Empowerment Zone Status on Gestational Age Using Synthetic Control Methods



(a) EZ estimate **(b)** Inference

(c) Average Treatment Effect **(d)** Root Mean Sqared Error

Source: Author's estimation of equation (3) in the text, using the full sample of EZs and the limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. Panel (a) provides estimates of the synthetic control method using EZ as the treatment. Panel (b) provides estimates of the effect of EZ status, systematically assigning EZ status to the actual EZ (the solid line) and all control zones (the dotted lines). Panel (c) provides the cumulative distribution of average treatment effects using estimates from the full inference sample, with a vertical line showing the average treatment effect for the actual EZ. Panel (d) provides the cumulative distribution of the ratio of post- to pre-root mean squared prediction error for the full inference sample, with a vertical line showing the value for the actual EZ.

Table 1.A1: Empowerment Zones and Control Areas Used in this Study

Program Name	Treatment	Limited Sample
Austin, TX		X
Bellmead/Waco, TX		
Chester, PA		
Chicago, IL	X	
Cincinnati, OH		X
Cleveland, OH		
Columbus, OH		X
Corpus Christi, TX		X
Dallas, TX		X
Dauphin, PA		
Denver, CO		X
El Paso, TX		X
Fort Lauderdale, FL		
Fort Worth, TX		
Greeley/Evans, CO		
Houston, TX		X
Jacksonville, FL		X
Louisville, KY		X
Miami, FL		X
New York City, NY	X	
Philadelphia, PA	X	
Pittsburgh, PA		X
Port Arthur, TX		
Portland, OR		X
San Antonio, TX		X
Summit, OH		X
Tampa, FL		X

Source: Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The limited sample of controls excludes controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone (Busso et al. 2013).

Table 1.A2: The Effect of Empowerment Zone Status on Predicted Birth Outcomes Using 1990-1994 Data to Predict Outcomes Using Same City Control Areas

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Chicago, New York, and Philadelphia Zone Compared to Control Zones					
Predicted	1.253	2.022	0.000	0.0001	-0.0002
	(1.380)	(2.690)	(0.001)	(0.0001)	(0.0008)
Panel B. Only Chicago Zone Compared to Control Zones					
Predicted	3.415***	0.360	-0.0003	-0.00003	0.0002
	(0.715)	(4.956)	(0.002)	(0.0003)	(0.001)
Panel C. Only New York Zone Compared to Control Zones					
Predicted	0.747	1.330	0.0005	0.0002	-0.0008
	(0.713)	(1.856)	(0.0007)	(0.0002)	(0.0007)
Panel D. Only Philadelphia Zone Compared to Control Zones					
Predicted	-0.950	12.07***	-0.003**	-0.0005**	0.003***
	(0.895)	(4.497)	(0.001)	(0.0002)	(0.001)

Source: Author's estimation using predicted values of fertility rates calculated using demographically weighted 1990 national age-race fertility rates. Predicted values of birth outcomes calculated by regressing birth outcome on maternal demographic characteristics including race and ethnicity, educational attainment, and mothers age at the time of birth for 1989-1994. Predicted estimates come from regressing predicted outcomes on an indicator variable for whether the birth occurred in an EZ, the interaction of the variable EZ and post, an indicator for whether the baby was conceived in 1995 or later, and year fixed effects which subsume the variable post. I use all EZs and a control group composed of babies born to mothers living in census tracts in the same city as EZs with poverty rates above 20%. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level.

Table 1.A3: The Effect of Empowerment Zone Status on Birth Weight Matched on Trend and Level: Synthetic Control Inference Results

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
All EZs	-13.050	23.700^{+}	-0.011	-0.001	0.003^{+}
	(0.125)	(0.250)	(0.188)	(0.500	(0.625)
	(0.750)	(0.063)	(0.125)	(0.625)	(0.063)

Source: Author's estimation of equation (3) in the text using the empowerment zones listed in the column and limited sample of controls (Busso et al. 2013), excluding controls from cities with populations less than 100,000 or with less than 10 census tracts in the original application zone. Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. The sample consists of 3 empowerment zones and 16 control zones. Each cell represents the average treatment effect from a separate synthetic control estimate. The numbers in parentheses in row 2 represent the implied p value of the average treatment effect (ATE). The numbers in parentheses in row 3 represents the implied p value of the ratio of the post to pre root mean squared prediction error (MSPE). * indicates significance at the 10% level using ATE, + indicates significance at the 10% level using MSPE. The estimates include controls for child's gender, mother's race and ethnicity, tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates. All outcome, individual and tract level demographic characteristics are aggregated up to the program level.

Table 1.A4: The Effect of Empowerment Zone Status on Birth Weight, Using Year of Birth

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Differences-in-Differences using Cross City Controls					
Differences-in-Differences	-9.570***	27.058***	-0.009***	0.000	0.009**
	(2.832)	(8.611)	(0.003)	(0.001)	(0.004)
Obs	216	366501	366501	366501	360419
Dep Var Mean	88.100	3197.203	0.098	0.017	0.885
Panel B. Synthetic Controls Matched on Trend					
Synthetic Controls	-12.429	51.809^{+}	-0.018^{*+}	0.001	0.001^{+}
	(0.188)	(0.125)	(0.063)	(0.688)	(0.625)
	(0.188)	(0.000)	(0.000)	(0.188)	(0.000)
Panel C. Synthetic Controls Matched on Level and Trend					
Synthetic Controls		12.524	-0.013^{+}	-0.001^{+}	0.003
		(0.375)	(0.250)	(0.500)	(0.563)
		(0.438)	(0.000)	(0.063)	(0.125)

Source: Author's estimation of equation (2) and (3) in the text using the empowerment zones listed in the panel and controls. Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. Panel B estimates equation (3) matched on the trend of the dependent variable. Panel C estimates equation (3) matched on the trend and level of the dependent variable. Each cell in the table comes from a separate regression. The estimates include controls for mother's race and ethnicity, child's gender, and year fixed effects, controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates, and tract level fixed effects. Fertility rate regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level. The numbers in parentheses for synthetic controls represent the implied p value of the average treatment effect (ATE), and the implied p value of the ratio of the post to pre root mean squared prediction error (MSPE). * indicates significance at the 10% level using ATE, + indicates significance at the 10% level using MSPE.

Table 1.A5: The Effect of Empowerment Zone Status on Birth Weight, Using the Full Set of Controls

	Fertility per 1,000	Birth Weight in Grams	Low Birth Weight	Very Low Birth Weight	Full-term 37+ Weeks
Panel A. Using Year of Conception					
(1) Differences-in-Differences	-12.559***	23.834***	-0.008***	0.001	0.010**
	(3.589)	(7.424)	(0.003)	(0.001)	(0.004)
Obs	351	420560	420560	420560	414307
Dep Var Mean	95.434	3185.318	0.102	0.018	0.885
(2) Synthetic Controls	-13.387*+	28.222*	-0.012	0.000	0.011+
	(0.042)	(0.042)	(0.167)	(0.625)	(0.792)
	(0.000)	(0.125)	(0.125)	(0.333)	(0.083)
Panel B. Using Year of Birth					
(3) Differences-in-Differences	-11.574***	24.014***	-0.009***	0.001	0.010**
	(3.840)	(7.988)	(0.003)	(0.001)	(0.004)
Obs	324	427587	427587	427587	421300
Dep Var Mean	85.235	3195.600	0.098	0.018	0.886
(4) Synthetic Controls	-26.152*+	33.925+	-0.013+	0.001	0.016+
	(0.042)	(0.125)	(0.167)	(0.583)	(0.750)
	(0.042)	(0.000)	(0.042)	(0.375)	(0.000)

Source: Author's estimation of equation (2) in the text using the empowerment zones listed in the panel and the full sample of controls. Data are from vital statistics data from the following states: Colorado, Florida, Illinois, Kentucky, New York, Ohio, Oregon, Pennsylvania, and Texas. The analysis sample is composed of babies born to mothers living in empowerment zones or control areas, which are areas that applied for, but did receive, empowerment zone status or areas that received empowerment zone status in a later round. The sample consists of 3 empowerment zones and 24 control zones. Each cell in the table comes from a separate regression. The estimates include controls for mother's age, race and ethnicity, child's gender, high school graduate, some college, and college graduate, and year fixed effects. Additional specifications labeled Tract Demographics also include controls for tract level demographic characteristics including race and ethnicity, educational attainment, median household income, vacancy rates, and poverty rates. Specification Tract FE additionally includes tract level fixed effects. Fertility rate regressions are performed at the aggregated EZ program level. All individual and tract level demographic characteristics are aggregated up to the program level, while all FE are program level FE rather than tract level FE for these regressions. Standard errors clustered at the program level, either the empowerment zone or the control zone, are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, * indicates significance at the 10% level.

CHAPTER 2

THE EFFECT OF CHILD HEALTH INSURANCE ACCESS ON SCHOOLING: EVIDENCE FROM PUBLIC INSURANCE EXPANSIONS

2.1. Introduction

Whether and how to provide access to affordable healthcare for low-income Americans has become a central policy issue in the US. The importance of this issue is underscored by the intense debate surrounding the passage and implementation of the 2010 Affordable Care Act (ACA), one of the largest expansions of public health insurance in US history. Most individuals from low-income households obtain medical insurance through Medicaid. Since its inception in 1965, Medicaid has gone through repeated expansions that have greatly increased the scope of the program as well as the public sector's role in health insurance provision. As a result, over 50 percent of children in the United States currently are eligible for publicly-provided health insurance through this program,[31] and health insurance coverage is high amongst this population (DeNavas-Walt et al., 2013).

The expansions that generated this high level of coverage were expensive. In 2012, total state and federal spending on Medicaid was $415.2 billion (Henry J. Kaiser Family Foundation, 2014), which makes it the largest government program that targets low-income Americans.[32] The substantial public funds devoted to providing health insurance to low-income children, as well as recent debates over the value of such

[31]. Throughout this paper, we refer to "public health insurance" and Medicaid synonymously. Publicly-provided health insurance also includes State Children's Health Insurance Plans (SCHIP). Medicare, however, is not included in our definition of public health insurance for purposes of this paper.

[32]. As a point of reference, total expenditures on food stamps (SNAP) in 2012 were $78.4 billion, and spending on Temporary Aid for Needy Families (TANF) was $31.4 billion. Total Medicare expenditures were $536 billion, which highlights that the Medicare and Medicaid/SCHIP programs are of roughly similar size.

insurance that surrounded the passage of the ACA, highlight the importance of understanding what benefits, if any, accrue to individuals due to health insurance access when they are young.

The effect of Medicaid expansions on access to healthcare and on subsequent child health has been studied extensively, (Currie and Gruber, 1996a, 1996b; Moss and Carver, 1998; Baldwin et al., 1998; Cutler and Gruber, 1996, LoSasso and Buchmueller, 2004; Gruber and Simon, 2008), typically showing that Medicaid expansions increase access to healthcare, decrease infant mortality, and improve childhood health. Furthermore, these expansions and Medicaid access more generally have been linked to a lower likelihood of bankruptcy and to less medical debt (Gross and Notowidigdo, 2011; Finkelstein et al., 2012). Notably, this literature has focused almost exclusively on the short- or medium-run effects of Medicaid on health and financial outcomes. Such effects are of considerable policy importance, but without an understanding of how Medicaid eligibility when young impacts long-run outcomes, it is difficult to fully assess the impact of this large government program. Estimating the long-run effects of Medicaid has received very little attention in the literature, and it is the focus of this paper.

We provide the first evidence on how expanding health insurance for children throughout their youth influences their eventual educational attainment. Our analysis focuses on education for several reasons. First, there is a strong argument from human capital theory that the improvements in child health and increased financial stability associated with Medicaid could have large effects on educational attainment. Second, cohorts affected by the Medicaid increases we study have been exposed to persistently

high returns to human capital investment (Autor 2014; Autor, Katz, and Kearney 2008). Thus, examining the effects of Medicaid expansions on long-run educational attainment is of considerable policy interest.

Similar to prior work on Medicaid, we exploit the expansions of Medicaid and the State Children's Health Insurance Program (SCHIP) that took place in the 1980s and 1990s to examine how the educational attainment of these children was affected by access to these programs. We use data on 22-29 year olds born between 1980 and 1990 from the 2005-2012 American Community Survey (ACS) that allow us to match each respondent to his or her state of birth. We then use data from the March Current Population Survey (CPS) to calculate Medicaid eligibility by age, state, year, and race that we link to our ACS sample. With these data, we follow the method of simulated instrumental variables pioneered by Currie and Gruber (1996a, 1996b) and Cutler and Gruber (1996) to account for the fact that the demographic composition of a state may be endogenous to Medicaid eligibility rules. By using a fixed sample to calculate eligibility, the model is identified using eligibility rule changes only.

We make several contributions to the literature. First, we estimate the effect of health insurance access among both young and school-age children on their long-run educational attainment. Second, we focus on Medicaid eligibility throughout one's childhood rather than just at birth. Virtually all of the prior work on Medicaid expansions focuses on point-in-time eligibility, particularly eligibility at birth (Levine and Schanzenbach, 2009; Currie and Gruber, 1996b).[33] From a policy perspective,

[33]. Currie, Decker and Lin (2008) present suggestive evidence that exposure to Medicaid expansions when young lead to better health in adolescence, which suggests there could be an effect on educational attainment as well. In a related study, Brown, Kowalski, and Lurie (2015) use IRS tax data to show that the eligibility expansions in the

focusing on eligibility at older ages is important because of the large amount spent on providing health insurance to non-newborn children. We present direct evidence that focusing just on point-in-time eligibility at birth provides an incomplete characterization of the effect of Medicaid on educational attainment. Our results show that it is repeated exposure throughout one's childhood that impacts these long-run outcomes, which has not been demonstrated previously.

Third, we are able to examine heterogeneous effects by the age at which a child is exposed to Medicaid expansions. There is a sizable body of research demonstrating a link between fetal health as well as the provision of fetal healthcare services on future educational outcomes (Figlio et al., 2014; Levine and Schanzenbach, 2009; Currie and Gruber, 1996b), but the effect of children's access to health insurance on their educational attainment has not been studied. Socioeconomic disparities in educational outcomes begin at young ages and largely persist throughout the lifecycle (Carneiro and Heckman, 2002; Todd and Wolpin, 2007). Our study provides insight into the ages at which Medicaid expansions have the largest long-run impacts on children in order to help close these educational gaps.

Fourth, we develop a new robustness test that allows us to assess the extent to which state-level Medicaid eligibility expansions are endogenously related to underlying trends in outcomes. Specifically, we isolate the variation in state Medicaid eligibility that comes from changes in federal rules. These changes impact states differentially based on their pre-existing welfare eligibility rules. Importantly, these expansions are unlikely to be related to outcome trends in any one state, which makes

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1980s led to higher earnings by the time individuals reached the age of 31. Their work does not examine educational attainment, but their results and ours strongly complement one another.

these estimates robust to state-specific educational attainment trends. This is a particularly important strategy in this paper because of our focus on average eligibility during one's youth. There are no sharp breaks across cohorts in childhood eligibility, but rather continuous increases the size of which are based on one's state and year of birth. This makes our estimates potentially more sensitive to state-specific trends than the prior literature that focuses on point-in-time eligibility. Our development and use of the federal eligibility instrument, in addition to our use in some specifications of state-specific time trends, provides evidence that our estimates are not being influenced by secular trends. That our estimates are similar when only using federal eligibility suggests as well that state Medicaid expansions are not endogenous, which helps validate the large body of work that uses them.

Finally, we contribute to the literature by showing that our results are insensitive to using current state versus state of birth. Because there are few datasets that include state of birth, most long-run analyses are forced to use current state as a proxy for childhood exposure. This is problematic if there is endogenous mobility related to Medicaid eligibility. Our estimates are inconsistent with such mobility, and thus our findings expand the possibilities for examining long-run Medicaid effects using other datasets that only contain current state of residence.

We find consistent evidence that Medicaid exposure when young increases later educational attainment. Our baseline estimates suggest a 10 percentage point increase in average Medicaid eligibility between the ages of 0-17 decreases the high school dropout rate by 0.4 of a percentage point, increases the likelihood of college enrollment by 0.3 of a percentage point, and increases the four-year college attainment

rate (BA receipt) by 0.7 of a percentage point. These estimates translate into declines in high school non-completion of about 4 percent, increases in college enrollment of 0.5 percent, and increases in BA attainment of about 2.5 percent relative to the sample means. In separate estimates by race, we find that the high school completion effects are larger among nonwhites, while the college enrollment and completion rate impacts are largest among white children. However, both groups experience substantial increases in educational attainment due to Medicaid expansions that occurred during their youth.

Our results on heterogeneity by age at the time of expansion, while imprecise, suggest that Medicaid expansions among children aged (4-8) are the most important. We also find evidence that expansions among teens aged 14-17 increase educational attainment, though interestingly, there is little effect of expansions for children at birth or in their first few years of life. These findings highlight the importance of examining childhood eligibility rather than point-in-time eligibility and suggest that there are sizable returns to covering older children in Medicaid.

Overall, our results point to large effects of Medicaid expansions for children on their eventual educational attainment. These effects are particularly important because lower-income families are most affected by Medicaid and SCHIP expansions, and it is children from these families that have exhibited the most sluggish growth in educational attainment over the past 30 years (Bailey and Dynarski, 2011). Our estimates suggest that the long-run returns to providing health insurance access to children are larger than just the short-run gains in health status.

The rest of this paper is organized as follows: Section 2.2. describes the public health expansions we use in our analysis, and Section 2.3. reviews the literature on the effects of health insurance on health and family finances as well as the literature examining the links between health, family resources, and educational outcomes. Section 2.4. provides a description of the data. We outline our empirical strategy and detail our results in Sections 2.5. and 2.6., respectively, before concluding in Section 2.7.

2.2. Medicaid and Public Health Care Expansions for Children

The Medicaid program was introduced in 1965 and phased in mostly over the late 1960s as a health insurance component for state-based cash welfare programs that targeted low-income, single-parent families. Beginning in the mid-1980s, the Medicaid program was slowly separated from cash welfare, first by extending benefits to low-income children in two-parent families and then by raising the income eligibility thresholds for two groups: children and pregnant women (Gruber, 2003; Gruber and Simon, 2008).[34] Since the 1980s, Medicaid has been expanded to many low-income families who did not previously qualify due to their income levels, family composition and/or labor force participation. As a result of these expansions, by the mid-1990s, most children in America below the poverty line, and all young children below 133 percent of the poverty line, were eligible for Medicaid. In certain states, their parents were as well.

Importantly, for most of these expansions, states could choose to implement the expansion based on their own eligibility preferences. By the early 1990's, states

[34]. For more details on Medicaid expansions, see Currie and Gruber (1996a), Gruber (2003), and Gruber and Simon (2008).

were required to cover all children below 100 percent of the poverty line, and children under age six below 133 percent of the poverty line. Many states opted to provide more generous coverage, however, for which the federal government would provide matching funds up to a certain threshold. In 1997, Congress passed the State Children's Health Insurance Plan (SCHIP), which was one of the largest expansions of public health insurance to date. SCHIP provided matching funds to states to expand coverage to children from households with incomes below 200 percent of the poverty line. Prior to SCHIP, states were permitted to cover children up to 200 percent of the poverty line, but, without federal matching funds, very few states did so.

In this paper, we exploit these expansions in Medicaid generosity in the 1980s and 1990s that were phased in at different times, and with different generosity levels across states, to identify the effect of Medicaid eligibility on long-run educational attainment. Thus, our identification strategy uses both state-level variation, which assumes the timing of state eligibility changes is exogenous with respect to underlying trends in educational attainment of residents, and federal variation to explicitly test the robustness of our estimates to the assumption that the state Medicaid variation is exogenous.

2.3. Previous Literature

The effect of Medicaid eligibility on education flows through two main potential channels: better health due to Medicaid enrollment as well as higher household resources stemming from the insurance protection provided by Medicaid. There is a large literature that shows Medicaid expansions both increase medical care usage and improve health among children and adults (Currie and Gruber, 1996a, 1996b; Currie,

2000; Kaestner et al., 2000; Kaestner et al., 2001; Almeida, Dubay, and Ko, 2001; Banthin and Selden, 2003; Dafny and Gruber, 2005; Buchmueller et al. 2005).[35] To the extent that health enters into the education production function, the health effects of Medicaid expansions could lead to higher educational attainment among affected children.

How are such changes in child health from Medicaid expansions predicted to affect educational attainment? Surprisingly little work has been done on this question. While much existing research has documented that better fetal health translates into better educational and adult outcomes (Miller and Wherry, 2014; Figlio et al., 2014; Almond and Mazumder, 2011; Almond, Edlund, and Palme, 2009; Almond, 2006; Black, Devereaux, and Salvanes, 2007; Oreopoulos et al., 2008; Royer, 2009), very little research estimates how childhood health after birth impacts such outcomes. Currie et al. (2010) find that children with health problems in early childhood have poorer long-run health, a higher likelihood of being on social assistance, and lower educational outcomes. Case, Fertig, and Paxson (2005) and Case, Lubotsky, and Paxson (2002) both show that worse health in childhood is negatively associated with long-run outcomes, such as health, educational attainment, and labor market outcomes.[36]

Cox and Reback (2013) as well as Lovenheim, Reback, and Wedenoja (2014) examine the effect of access to health care *services* on educational attainment using the rollout of school-based health centers in the US. The former study finds that center

[35]. Levy and Meltzer (2008) provide a recent review of this literature.
[36]. See Almond and Currie (2011) for a comprehensive overview of the fetal origins hypothesis and Eide and Showalter (2011) for evidence on the effect of health on human capital outcomes throughout the life cycle.

openings lead to higher attendance rates, while the latter shows they cause lower teen birth rates but do not affect high school dropout rates. The students treated by these centers are typically in high school, so the differences between these estimates and the large effects of health found by researchers examining younger children may potentially be due to heterogeneity in the effects of health at different times during childhood.

Another main channel through which Medicaid can influence educational attainment is through its effect on family resources. Recent work has suggested that public health insurance successfully shelters low-income families from financial risk associated with negative health shocks (Gross and Notowidigdo 2011; Dave et al. 2013; Finkelstein et al. 2012). Thus, Medicaid expansions better the financial position of households, which much prior work demonstrates can positively affect educational investments (Dahl and Lochner 2012; Lovenheim 2011; Michelmore 2013).

While we provide the first analysis in the literature of the long-run effects of Medicaid on educational attainment, there are two papers in the literature that are closely related to ours. The first paper is Levine and Schanzenbach (2009), which analyzes the effect of Medicaid and SCHIP expansions *at birth* on future educational achievement as measured by state-level National Assessment of Educational Progress (NAEP) scores. This paper is typical of the literature in its focus on point-in-time eligibility (at birth) rather than eligibility over a period of one's childhood. They examine differences in Medicaid expansions by state and the differences between age cohorts in a triple difference framework. Their results suggest that a 50 percentage

point increase in Medicaid eligibility corresponds to a 0.09 standard deviation increase in reading test scores, but there are no effects on math scores.

Our analysis is distinguished from theirs along several dimensions. First, we focus on the effects of expanding health insurance throughout one's youth. This question is particularly important given the amount of money spent in the US on providing health care to non-newborn children through Medicaid.[37] Indeed, our results indicate that expanding eligibility to non-newborns is an important driver of the long-run effects of Medicaid; estimates using point-in-time eligibility at birth show little effect of Medicaid on educational attainment. Second, we examine effects on long-run educational attainment rather than on test scores at younger ages. A growing body of evidence suggests that the effects of given educational interventions on test scores are poor predictors of their effects on the longer-run outcomes that are of greater interest, such as educational attainment and earnings (Ludwig and Miller, 2007; Chetty et al., 2011; Deming et al., 2013).[38]

The second related work is a currently unpublished working paper by Brown, Kowalski, and Lurie (2015). They use IRS tax data to examine the effect of Medicaid expansions throughout a child's early life on earnings. They find results that are highly complementary to our own: Medicaid eligibility increases from 0-18 are associated with higher earnings, lower EITC receipt, and higher labor force participation. That

[37]. If health insurance among school-age children did not positively affect these children, ostensibly the government could only offer Medicaid to pregnant women and households with very young children.

[38]. Much of this evidence suggests that it is particularly problematic to use effects on contemporaneous test scores to predict long-run outcomes. Levine and Schanzenbach (2009) examine effects on the NAEP scores of 4th and 8th graders, which themselves are longer-run test score outcomes. Furthermore, instructors are unlikely to manipulate NAEP scores endogenously with respect to Medicaid eligibility rates, which would not necessarily be the case for contemporaneous test scores used to evaluate a given educational intervention. Nevertheless, it is not at all clear that effects on NAEP scores would translate into higher educational attainment, which underscores the importance of our analysis.

they obtain these estimates on a different dataset using somewhat different cohorts is notable. Together, our results point to large effects of Medicaid expansions on the long-run outcomes of affected children.

2.4. Data

We use three sources of data in our analysis of the effects of insurance expansions on educational attainment. Below, we describe these sources of data, as well as the construction of the variables that we use in our investigation.

2.4.1. Medicaid Eligibility Data

Our Medicaid eligibility data are constructed for the years during which the 1980-1990 birth cohorts are between the ages of 0-17 using the March Current Population Survey (CPS). We construct two eligibility measures using state and year information on eligibility rules similar to those used in Gross and Notowidigdo (2011) and Gruber and Simon (2008).[39] Eligibility calculations are based on the household's income, the age and number of children in the household, and the gender and unemployment status of the head of household.

The first Medicaid eligibility measure we construct is the proportion of households of a given race (white, nonwhite) with children of age *i* in state *s* and year *t* who are eligible for Medicaid, where $i \in (0,1,\ldots,17)$. Thus, for example, we calculate the proportion of households with five-year-olds in New York who are eligible for Medicaid in each year between 1985 (the 1980 birth cohort) and 1995 (the 1990 birth cohort). We calculate eligibility separately by child's race due to the strong correlation between race and Medicaid eligibility: a given change in eligibility rules is likely to

[39]. We are extremely grateful to Tal Gross and Kosali Simon for providing us with the computer code that forms the basis for our eligibility calculations.

impact nonwhites differently than whites even though the Medicaid rules themselves are race-neutral.

These calculations allow us to measure the proportion of children of each age and race group that are Medicaid-eligible in each state and in each year between 1980 and 2007. As described below, our outcome data span the years 2005-2012 and include the 1980-1990 birth cohorts. These cohorts are between the ages of 22 and 29 in 2005-2012, which is why our CPS sample ends in 2007 (when the 1990 birth cohort is 17).[40] We use three-year moving averages of calculated eligibility instead of yearly eligibility because the small sample sizes in the CPS within each age-race-state cell lead to measurement error in eligibility. While this measurement error is not problematic for our instrumental variables strategy, using one-year eligibility likely would attenuate the OLS estimates considerably. This makes comparisons between our OLS and IV estimates less informative.[41] Furthermore, the use of three-year moving averages is standard in the recent Medicaid literature that employs simulated instrument methods (Gruber and Simon 2008; Gross and Notowidigdo 2011; DeLeire, Lopoo, and Simon 2011), which facilitates comparisons between our estimates and those in prior work. Aside from making the estimates more precise, our use of these moving averages has little effect on the results. We refer to this measure of Medicaid eligibility as "actual eligibility."

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[40]. We have conducted extensive sensitivity analyses using different birth cohort ranges and ACS age ranges. Our results are not very sensitive to the age range or birth cohorts used. These sensitivity analyses are available from the authors upon request.

[41]. This method necessitates the use of CPS data through 2009 (which contains 2008 income information), to enable the construction of our 3-year moving average. In Appendix Table A-9, we show our estimates are robust to using 1-year averages, although as expected the OLS estimates are attenuated. And, in Appendix Table A-7, we show they are robust to dropping all states that include cell sizes for 0-17 eligibility that come from under 100 observations (3.3 percent of the sample). Online appendix tables are available at http://jhr.uwpress.org/.

Actual eligibility varies within states over time due to changes in eligibility rules, changes in demographic composition, and changes in the economic circumstances of households. In order to isolate the variation in Medicaid eligibility due only to eligibility rule changes, we follow the method first used in Currie and Gruber (1996a, 1996b) and Cutler and Gruber (1996) and calculate "simulated fixed eligibility," which is the proportion of the population in each state, age, race, year cell that would be eligible for Medicaid, calculated using a fixed national sample that does not vary across states or over time. We use the 1986 CPS and calculate the share of this fixed population with a child of age *i* in year *t* and race *r* that would be eligible for Medicaid in each state using that state's Medicaid eligibility rules in that year, adjusting family income for inflation using the Consumer Price Index for All Urban Consumers. Critically, this sample does not vary by demographic characteristics across states or over time and thus is unaffected by state-specific trends in population or economic conditions that relate to both eligibility and coverage, (such as a state-level recession). Finally, we collapse these estimates into unique state-year-age-race cells that yield the proportion of the fixed sample eligible for Medicaid in each cell.

Our baseline estimates include Medicaid eligibility variation coming from federal Medicaid expansions, state decisions about whether they will provide more generous benefits than required by federal law, as well as the timing of state expansions. Among these sources of variation, the one that is most worrisome is the timing of state expansions: state expansion decisions may be endogenous with respect to underlying trends in educational attainment. Thus, we also construct measures of Medicaid eligibility that only are a function of federal rules. Federal Medicaid rules

have different impacts on states due to pre-existing state-level AFDC policies. Hence, we fix AFDC rules in each state as of 1980 and then calculate the 3-year moving average of actual eligibility, as well as yearly fixed simulated eligibility for each age, race, and state that would occur *only* due to changes in federal regulations governing Medicaid eligibility thresholds. Put differently, our federal eligibility measures yield state-year-age-race eligibility that would occur if no states provided more generous Medicaid access than required under federal law. The reason this is not simply a cohort-based analysis, then, is that the effect of federal rules varies by state according to (fixed) welfare policies. By design, this source of Medicaid eligibility variation is unlikely to be correlated with any decisions states can make regarding Medicaid eligibility.

Trends in our Medicaid eligibility measures, both overall and by race, are shown in Figure 1. For each birth cohort, we show the average eligibility between the ages of 0-17 to which the cohort was exposed. The panels of the figure show, for the 1980-1990 birth cohorts, actual eligibility that is a function of both state and federal rules as well as eligibility that uses only federal rules. As demonstrated in Figure 1, there was a dramatic rise in Medicaid eligibility that took place across the birth cohorts we study. Overall, average eligibility rates over the course of childhood increased 172 percent between the 1980 and 1990 birth cohorts. Much of this was the non-linear increase in eligibility that came from the 1990 federal Medicaid expansion that extended eligibility to all children born after September 30, 1983 in families up to 100 percent of the poverty line. In Panels B and C of Figure 1, we show that the proportional increases experienced between whites and nonwhites were similar, but

the higher baseline eligibility rates among nonwhites in 1980 led to much higher eligibility among the 1990 cohort than among the 1980 cohort. In our data, over 50 percent of nonwhites born in 1990 were eligible for Medicaid over the course of their childhood, while less than 30 percent of whites were eligible among this birth cohort.

Figure 1 also shows that the trends in overall eligibility track the trends in federal eligibility closely, especially after the 1984 birth cohort, which highlights the importance of federal Medicaid policies for identification. The simulated eligibility trends are very close to the actual trends as well. Thus, most of the aggregate pattern in Medicaid eligibility is due to policy changes rather than demographic shifts in the US population.

2.4.2. Educational Attainment

The main outcome data we use come from the 2005-2012 American Community Survey (ACS). The ACS was designed to replace the Census, and thus the variables and design across the two surveys are almost identical. The sample for our analysis consists of birth cohorts from 1980-1990 who are between 22 and 29 in 2005-2012. Thus, for each individual in our sample, we observe eligibility in his or her birth state at each age between 0 and 17. Table 1 shows the birth cohorts included in our analysis sample at each age and year. The top row shows the ACS (calendar) year, and the column shows the age of the respondent. For example, in the 2008 ACS, observations of 25 year olds come from the 1983 cohort. This table illustrates that we do not observe each birth cohort in each ACS survey due to our constructed age cutoffs. For example, 29 year olds are observed in 2009-2012 and come from the 1980-1983 birth cohorts only, whereas 25 year olds come from the 1980-1987 birth

cohorts and are included in each of the ACS years in this analysis. Our use of 1980 as the earliest birth cohort is driven by our lack of information about state-specific Medicaid eligibility pre-1980, which makes it infeasible to use earlier birth cohorts.[42] Furthermore, we examine individuals only up to age 29, since by age 29 most education has been completed (Bound, Lovenheim, and Turner, 2010). Including older individuals would reduce the number of calendar years in which we can identify eligibility for such respondents.

We calculate, for each respondent, indicators for whether the person did not complete high school, whether she attended any college, and whether she obtained a Bachelor's Degree (BA).[43] We collapse the data to birth cohort, state of birth, survey year, race (white/nonwhite) means for all variables, using the individual census weights. We then link each birth cohort, state-of-birth, race, survey year cell to the Medicaid eligibility means discussed in Section 4.1.[44] In particular, we calculate average eligibility for each birth cohort (*c)* in each survey year (*t*), state of birth (*s*) and race (*r*) over their childhood ages ($i \in [0,17]$):

(1) $$eligibility_{scrt} = \frac{1}{18}\sum_{i=0}^{17} \overline{elig}_{scirt},$$

where $\overline{elig}_{scirt}$ is the average Medicaid eligibility in birth state *s*, cohort *c*, survey year *t*, and of race *r* when the birth cohort was age *i*.

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[42]. We also note that Medicaid eligibility was very low pre-1980 and there were few expansions. Thus, our focus on birth cohorts between 1980 and 1990 captures most of the policy-driven variation in Medicaid exposure that has occurred since the program's inception.

[43]. Our measure of high school completion includes GEDs, which is potentially problematic if Medicaid eligibility shifts students from obtaining a traditional high school diploma to a GED given the low returns to GED receipt found in the literature (Heckman and LaFontaine 2006). In 2008 and after, however, the ACS asks directly about GED completion. We show in Table 4 that our main high school completion results are not being driven by GEDs.

[44]. Public insurance expansions can potentially alter the character of medical care for both individuals who experience a change in insurance coverage and also those who do not (Finkelstein, 2007). Because we adopt an aggregated cohort-based empirical approach, we allow for the presence of these "spillovers" within cohorts.

We construct an identical measure using fixed simulated eligibility:

(2) $$fs\_eligibility_{scrt} = \frac{1}{18}\sum_{i=0}^{17}\overline{fs\_elig}_{scirt},$$

where $\overline{fs\_elig}_{scirt}$ is simulated Medicaid eligibility that is calculated using a constant sample from the 1986 CPS, as described above.

Descriptive tabulations of the analysis data for the full sample and by race group are shown in Table 2. In the full sample, the average respondent is 25, and about 68 percent of the respondents are white. The gender and age composition of the sample varies little across race groups. Furthermore, the educational attainment of nonwhites is much lower than that of whites, while average Medicaid eligibility is much higher for nonwhites. Both of these patterns reflect the strong correlation between socioeconomic status and race, which highlights the potential importance of any effect of Medicaid eligibility on educational attainment to help address gaps in educational outcomes between whites and nonwhites.

2.5. Empirical Methodology

In order to motivate our empirical models, it is helpful first to consider the ideal experiment one would use to identify the effect of Medicaid on long-run outcomes. Similar to the lottery for access to Oregon's Medicaid program (Finkelstein et al. 2012), the most credible way to estimate the program effects of interest would be to randomly assign families with children of different ages eligibility for Medicaid. Such eligibility would last throughout the remainder of the child's schooling years, unless the household finances made them ineligible. With a long enough panel, we then could simply compare educational attainment among children who were

randomly assigned Medicaid eligibility relative to those who were not. One also could calculate the effect of Medicaid coverage, using randomized eligibility as an instrument (Finkelstein et al. 2012).

While such an experiment would identify the effect of Medicaid eligibility over one's childhood, in practice such an analysis is not currently feasible. The Oregon experiment did not target children,[45] and there is no other randomized Medicaid experiment of which we are aware. However, we can exploit the changes in both state and federal Medicaid eligibility rules that occurred over the 1980s and 1990s to approximate this experimental ideal. Because these policy changes never make Medicaid eligibility less generous, once a child's family is eligible for Medicaid in a state, he or she remains eligible for the duration of childhood unless the family's income or assets rise sufficiently. As we argue below, the variation in eligibility on which we focus is unrelated to demographic differences across individuals or to secular trends in educational attainment. Thus, these eligibility expansions mirror the assignment mechanism one would use in the ideal experiment.

We exploit the state and federal Medicaid eligibility expansions that occurred since 1980 using a difference-in-difference model that estimates how within-state changes in Medicaid eligibility across cohorts over their childhood impacted their educational attainment. Specifically, we estimate models of the following form:

(3) $$Y_{scart} = \beta_0 + \beta_1 eligibility_{scrt} + \beta_2 X_{sart} + \gamma_{rs} + \delta_{rt} + \theta_{ra} + \varepsilon_{scart},$$

[45]. Adult access to Medicaid through the Oregon lottery might indirectly influence children's outcomes through family financial stability or better parental health. However, the experiment occurred too recently to test its effects on children's long-run outcomes.

where Y_{scart} is the educational outcome (high school non-completion rate, college attendance rate or college graduation rate) [46] in state-of-birth *s*, birth cohort c, age *a*, of race *r*, in survey year *t*. The variable $eligibility_{scrt}$ comes from equation (1) above and denotes the mean fraction of individuals of a given race and in a given birth cohort and birth state who were eligible for Medicaid.

In the baseline specification, the vector X_{sart} consists only of an indicator for whether the observation is for the nonwhite sample or not. As we discuss below, we then include in X_{sart} some measures of potential confounding policies. In the baseline specification, the model includes as well a set of race-by-age fixed effects (θ_{ra}), race-by-state-of-birth fixed effects (γ_{rs}), and race-by-calendar year fixed effects (δ_{rt}).[47] The race-by-age fixed effects in particular are important because they account for the fact that older individuals have more time to complete their education and that this age pattern might be different across whites and nonwhites. The race-by-state fixed effects control for fixed differences across states that are correlated with both Medicaid eligibility and educational attainment, such as the higher education structure and the industrial mix in the state, which we allow to vary by race as well. The race-by-year fixed effects account for any economy-wide shocks that could be correlated with prior Medicaid expansions and that might be different across racial groups.

The coefficient of interest in equation (3) is β_1. It thus is important to clarify the sources of variation identifying this parameter, conditional on other controls in the model. As discussed above, we are exploiting variation from Medicaid eligibility

[46]. The "some college" outcome contains both college dropouts and those who receive an Associates Degree (AA). In Online Appendix Table A-8, we show estimates that use "Associates Degree" rather than "Some College." The estimates are very similar in showing little effect of Medicaid eligibility on whether an individual obtains an AA.
[47]. Henceforth, we will refer to "state fixed effects" and "state-of-birth fixed effects" synonymously.

expansions over the course of one's childhood. With the inclusion of state fixed effects, we are focusing on within-state changes in eligibility across cohorts and relating these to within-state changes in educational attainment. That is, within each state, we are using the fact that Medicaid eligibility for older cohorts is lower than that for younger cohorts, and thus we are essentially comparing across cohorts within states to identify β_1. When we pool all states, we are averaging these within-state effects together. Furthermore, the time-varying nature of the Medicaid expansions *across* states allows us to partial out age effects from calendar year effects.[48] As a result, our identifying variation comes from cross-cohort changes in childhood Medicaid eligibility within each state as well as cross-state variation in the timing of eligibility expansions.

Equation (3) incorporates a potentially restrictive set of assumptions about the cross-state variation we use, namely that the state and age fixed effects are constant across calendar years. We can relax this assumption by include race-state-year and race-age-year fixed effects in the model:[49]

(4) $$Y_{scart} = \beta_0 + \beta_1 eligibility_{scrt} + \beta_2 X_{sart} + \gamma_{rst} + \theta_{rat} + \varepsilon_{scart}$$

In equation (4), including the age-by-year-by-race effects allows for any national birth cohort-specific shocks that could impact educational attainment or for more recent cohorts to obtain their degrees later. State-by-year-by-race fixed effects account for

[48]. If we estimated this model using one state, we could not estimate both age and year fixed effects. The reason is that within a state, birth cohort fully describes the treatment intensity, and birth cohort and age-year interactions are perfectly collinear with each other.

[49]. Note that we do not control for race-by-state-by-age fixed effects. Thus, some of the identifying variation could be coming from fixed differences across ages within a state. However, this would require the existence of shocks to specific ages (but not birth cohorts) in a state that happen to be correlated with Medicaid eligibility differences. We have estimated models using these fixed effects, and the results are qualitatively similar (if somewhat less precise). We do not include them in the analysis because there is little economic justification for these controls. Furthermore, note that the estimates that use only federal variation would be unaffected by any such shocks.

any state macroeconomic changes that could influence contemporaneous educational attainment. While equation (4) is more flexible, it also is much more demanding of the data, which leaves us with less statistical power. As a result, these estimates tend to be imprecise.

Both equations (3) and (4) are identified off of the fact that states expanded their Medicaid eligibility rules differentially across cohorts and the fact that the timing and size of these changes varied across states. These models therefore are difference-in-difference specifications, where the treatment dose varies across different cohorts depending on the state and year of birth, as well as depending on one's race. As discussed in Section 4, this variation comes from two sources: the first is rule changes that expand Medicaid eligibility to different age groups within each state, and the second is demographic shifts that expand the proportion of individuals who meet pre-existing eligibility criteria.

For our analysis, the second source of variation is potentially problematic even conditional on the fixed effects. If there are demographic changes that affect the proportion of people eligible for Medicaid, these changes are likely to be correlated with educational attainment. Our limited set of demographic controls cannot fully account for such changes, although demographic changes that expand Medicaid eligibility most likely generate a negative bias in estimating the effect of Medicaid on educational attainment. We therefore use an instrumental variables strategy that is robust to demographic shifts. This IV strategy amounts to using *fs_eligibility* from equation (2) as an instrument for *eligibility*. Because *fs_eligibility* is based on

eligibility rules in each year using a fixed sample of individuals from the 1986 CPS, it is only affected by eligibility rule changes over time within states.

Similar to any difference-in-difference analysis, there are two main assumptions we invoke. The first is that Medicaid expansions are not correlated with underlying trends in educational attainment across cohorts at the state level. A particular concern for our identification strategy would be if Medicaid expansions are occurring in states that are becoming more affluent. Then, even simulated fixed eligibility changes would be positively correlated with underlying trends in educational attainment. We do not believe such a situation is likely, however, since states probably would be more compelled to expand Medicaid eligibility due to increased, not decreased, demand for public insurance. This is a common identification assumption that has been invoked repeatedly in the Medicaid literature (Currie and Gruber, 1996a, 1996b; Cutler and Gruber, 1996; Gross and Notowidigdo, 2011; Gruber and Simon, 2008). The second assumption underlying our identification strategy is that there are no other state-level policies that are correlated with Medicaid expansions that themselves might affect educational attainment.

We provide an extensive set of robustness checks to provide additional confidence that our results are not being driven by endogenous state Medicaid eligibility expansions or by other policies. First, in some specifications we control in X_{sart} for average state EITC amounts between the ages of 0-17 for each cohort. Prior work linking EITC policies to educational outcomes suggests EITC generosity could

be a confounding factor if it is correlated with Medicaid generosity.[50] We also control for average school spending per pupil in the years in which each cohort was 5-17, separately by urban, rural and suburban districts. Although there is a tenuous link between school expenditures and education outcomes (see Hanushek, 2003 for an overview of this literature), recent work has linked school spending increases from school finance reforms to better long-run outcomes (Jackson, Johnson, and Persico, 2014). We view these factors as the ones that are most likely to produce confounding effects, but our estimates that control for these policies provide evidence that this is not the case.

We provide more direct evidence that endogenous state Medicaid expansions are not biasing our estimates by using only federal Medicaid eligibility rules as discussed in Section 4.1. The race-by-state-of-birth fixed effects control for the fixed differences in AFDC rules across states, and the identifying variation in the federal model comes solely through the fact that federal rule changes have differential impacts on states due to pre-existing AFDC policies. Thus, there is no scope in these models for endogenous state decisions regarding Medicaid, and to the extent we obtain similar results using this variation, it will provide confidence in the validity of the results that use state Medicaid variation. This is the first paper to provide estimates using only federal eligibility variation, so these results are of interest in their own right insofar as they help validate the widely-employed assumption that state Medicaid expansions are exogenous.

[50]. See Michelmore (2013) for an overview of state-level EITC laws. We thank Kathy Michelmore for providing us with these data.

We also conduct robustness tests that include race and state of birth specific linear trends across birth cohorts. These models are identified off of the non-linear increases in Medicaid eligibility that followed from state and federal law changes, and they help guard against any upward bias from correlated secular trends in educational attainment and Medicaid eligibility. We further provide a robustness check in which we randomly assign observed eligibility levels across age-state-year cells. Overall, our estimates are robust to using variation in Medicaid eligibility from different sources and to the addition of controls for other policies affecting low-income populations. These findings support the validity of our identification strategy.

Because errors are unlikely to be independent within states of birth over time, we cluster all standard errors at the state-of-birth level. All estimates also are weighted using sample weights provided in the ACS.

2.6. Results

2.6.1. Main Results

Table 3 presents the main results from our estimation of equations (3) and (4). Each cell in the table comes from a separate regression, with Panel A showing results that use all Medicaid eligibility and Panel B showing results using only federal eligibility. The first column in the table presents the first stage, which shows how a change in fixed simulated eligibility translates into actual eligibility. The table also shows the effect of actual Medicaid eligibility ("OLS") and fixed simulated eligibility ("RF," for reduced form) on high school non-completion, college enrollment and four-year college completion, as well as the associated IV estimates.

Across outcomes and the specifications shown in different rows, we find consistent evidence that Medicaid eligibility when young increases educational attainment. Focusing on the baseline IV results in row (1), a 10 percentage point increase in Medicaid eligibility reduces high school non-completion by 0.39 of a percentage point, increases college enrollment by 0.35 of a percentage point, and increases BA attainment by 0.66 of a percentage point. The high school and college completion estimates are statistically significantly different from zero at the 5 percent level. Relative to the mean attainment rates shown in Table 2, these estimates translate into a 4.1 percent decline in high school dropouts, a 0.5 percent increase in college enrollment, and a 2.5 percent increase in BA receipt. As shown in Figure 1, there was a 24 percentage point increase in average eligibility during childhood between the 1980 and 1990 birth cohorts. Our estimates suggest this change would have reduced high school non-completion by 10.0 percent, increased college enrollment by 1.3 percent, and increased college completion by 6.0 percent.

To put these effects in perspective, it is helpful to compare them to educational attainment trends over this period. Murnane (2013) shows that high school graduation rates increased by about 6 percentage points between the 1980 and 1990 birth cohorts. Since our estimates show that a 24 percentage point increase in Medicaid would increase high school completion by 0.94 percentage points, this implies that 15.6 percent of this increase can be attributed to Medicaid expansions. Our tabulations from the Current Population Survey indicate that college completion rates among 23-year olds between the 1980 and 1990 birth cohorts increased by 4.8 percentage points. A 24 percentage point Medicaid eligibility increase would increase BA attainment by 1.6

percentage points using the baseline results, which implies that Medicaid expansions can explain 33.3 percent of the overall BA attainment increases over this period.

The results in Table 3 represent the effect of exposure to Medicaid eligibility throughout one's childhood on educational attainment (the intent-to-treat). From a policy perspective, this is a parameter of interest because the government cannot compel the take-up of Medicaid. It also is the parameter on which much of the Medicaid literature focuses.[51] However, it is of interest as well to understand how enrollment in Medicaid affects educational attainment (the treatment effect on the treated). This is a difficult calculation because we lack the ability to track how average eligibility in one's childhood relates to Medicaid take-up during childhood. The existing estimates on take-up in the literature are not the appropriate "first stages" in our context, as they provide the contemporaneous effects on enrollment, where we would need an estimate of the effect on take-up over one's entire childhood to scale our results.

In order to estimate the treatment on the treated effect, we use the marginal take-up rate of 0.156 calculated by Gruber and Simon (2008) for the period 1996-2002 and assume this rate represents a yearly "risk" of taking up Medicaid. That is, we assume that in each year of childhood, 15.6 percent of the eligible population that has not yet taken up Medicaid does so. For example, at age 0, 15.6 percent of eligibles will have taken up Medicaid and 84.4 percent will have not, and at age 1, [15.6 + (15.6*84.4)] =28.8 percent will have taken it up and 71.2 percent will have not (and so

[51]. The other relevant papers that use simulated instruments to examine effects on child or family outcomes, namely Levine and Schanzenbach (2009), Currie and Gruber (1996b), and Gross and Notowidigdo (2011), only report these intent to treat estimates.

forth). This method implies that for a child eligible at birth, he or she has a 95 percent chance of being on Medicaid at some point before the age of 18. Because children are made eligible at different ages, we calculate the associated likelihood of taking up Medicaid conditional on first being eligible at each age between 0 and 17. We then average over these take-up estimates by age and find that expanding eligibility increases the likelihood a child takes up Medicaid at some point during childhood by 71.4 percent. This average take-up estimate matches the average Medicaid take-up rate of 73 percent quite closely (Currie, 2004). Thus, treating marginal take-up rates as a constant risk of Medicaid enrollment reconciles the evidence on low marginal but high average take-up rates, which provides some validation for the method we use to calculate treatment on the treated effects.

We calculate the treatment effect on the treated by dividing our IV parameter estimates for eligibility by the 71.4 percent take-up rate. These calculations allow us to interpret our results from the standpoint of an individual becoming eligible for Medicaid (eligibility changing from zero to one) rather than from the standpoint of a policymaker who can expand eligibility by a given percentage among the state population. Treatment on the treated estimates show that enrolling in Medicaid decreases the likelihood of dropping out of high school by 5.5 percentage points and leads to a 9.2 percentage point increase in the likelihood of completing a BA. To put the magnitude of these results in perspective, they are similar to the estimated effects on educational attainment of attending a higher-quality high school (Deming et al. 2014) and of Head Start (Garces, Thomas, and Currie 2002).

Rows (2)-(4) of Table 3 show our conclusions are largely robust to adding additional controls for EITC and school spending (row 2). In rows 3 and 4, the addition of race-state-year and race-age-year fixed effects reduces precision considerably. For the high school non-completion outcome, the magnitudes of the point estimates decline, while for the college graduation outcome, the point estimates increase. However, in both cases they are qualitatively similar to the baseline estimates and the confidence intervals include the baseline estimates. Overall, adding controls for other potentially confounding policies as well as a large array of fixed effects do not change the conclusion that Medicaid eligibility expansions had sizable positive effects on long-run educational attainment.

Table 3 also demonstrates that the OLS and IV results are quite different from each other. The OLS estimates in Panel A show Medicaid eligibility increases are associated with smaller high school dropout declines (in absolute value) and with smaller college completion increases. These results are suggestive that the bias from failing to account for the correlation between demographics and Medicaid eligibility would cause one to find a smaller effect of Medicaid on educational attainment.

As discussed in Section 5, an important identification concern with the estimates that use state-level policy variation is that this variation is potentially correlated with secular trends in educational outcomes. This is especially relevant in this study relative to the rest of the Medicaid literature, since we are using average Medicaid eligibility over one's childhood. As a result, there are no sharp breaks in average eligibility that we can exploit. In Panel B of Table 3, we thus show estimates using only federal Medicaid eligibility that are unlikely to be correlated with the trends

associated with any one state. Focusing on the baseline estimates in row (5), we find that increases in Medicaid eligibility reduce high school dropout and increase college enrollment and completion. Comparing the estimates in row (5) to the baseline results in row (1), the point estimates for the reduced form are smaller in absolute value when only the federal variation is used. As the IV estimates show, this difference mostly reflects the smaller first stage. In Panel A, the first-stage estimates are around 0.9, suggesting that a 10 percentage point change in fixed simulated eligibility is associated with a 9 percentage point change in actual eligibility.[52] As expected, the link between federal Medicaid rules and actual eligibility is much weaker because we are ignoring state responses to the federal regulation changes. However, the first stage for the federal variation still is sizable in magnitude and is statistically significant from zero at the 1 percent level.

Comparing the IV estimates from similar models across panels shows that using the federal-only variation produces results that are quantitatively and qualitatively similar to the estimates that use state variation as well. For high school non-completion in the baseline specification (row 1), the estimates indicate a 10 percentage point eligibility increase during childhood reduces dropout by 0.39 of a percentage point using all Medicaid variation, and it reduces dropout by 0.55 of a percentage point using only federal variation (row 5). For college enrollment, the estimates in row (5) are smaller than those in row (1), and they are inconsistent with all but a small increase in college attendance. Finally, for college completion, the IV

[52]. Our first-stage estimates are similar to what has been found in prior work. Cutler and Gruber (1996) report a first-stage of 0.84 for children and 0.95 for women, while Gross and Notowidigdo (2011) have an implied first-stage estimate of 0.61.

coefficients across panels of Table 3 show very similar effects of Medicaid eligibility expansions. Comparisons of rows (2) and (6) show that our estimates using federal variation are comparable when including the EITC and school spending controls as well.[53] That these two models yield similar estimates of the effect of changes in Medicaid eligibility among children on long-run educational attainment supports our use of all Medicaid variation, as it suggests state Medicaid eligibility variation is not endogenous with respect to long-run educational outcomes.

A final potential concern with the results in Table 3 is that the high school completion variable groups GED and high school diploma recipients together. Starting in 2008, the ACS began asking separately about high school diploma and GED receipt, and in Table 4 we present estimates using 2008-2012 data where we separate high school diploma non-receipt from diploma and GED non-receipt. As the table demonstrates, the effects are extremely similar across the two measures of high school completion, suggesting that our baseline estimates do not obscure potential shifts between traditional diplomas and GEDs. In addition, the some college and college plus estimates are similar in the 2008-2012 sample, if somewhat larger among all outcomes. These results suggest our estimates are not driven by the particular sample period we chose.

2.6.2. Educational Attainment Results by Age at Expansion

While these results indicate a beneficial overall effect of Medicaid expansions on educational attainment, from a policy perspective, it is important to discern whether

[53]. We do not present federal variation results that include race-state-year and race-age-year fixed effects. Due to the limited amount of variation in federal Medicaid eligibility, including these fixed effects yields large standard errors that make the resulting estimates uninformative. Furthermore, the goal of using the federal variation is to find a source of variation that is unlikely to be related to state trends. As a result, there is little theoretical justification for including the race-state-year and race-age-year fixed effects in these models.

it matters if one measures eligibility at a point-in-time (typically birth) relative to over one's childhood, as well as whether there are effects of health insurance access at different ages. In Table 5, we present IV estimates that control for eligibility at birth (similar to what was done in Levine and Schanzenbach (2009) and Currie and Gruber (1996b)). Using both equations (3) and (4), we find very little evidence that Medicaid eligibility at birth is associated with long-run educational attainment. With the exception of our college completion measure (and only when we include the full range of fixed effects), the rest of the estimates are small in magnitude and either are "wrong-signed" or are not statistically significant. This finding is suggestive that the test score gains found by Levine and Schanzenbach (2009) do not translate into higher educational attainment. However, when we add in eligibility at ages 1-17, we find that Medicaid eligibility does lead to more education among affected cohorts. It is the eligibility at older ages that is responsible for this relationship; eligibility at birth continues to be uncorrelated with long-run educational outcomes.[54] The age 0 and age 1-17 estimates are statistically different from each other at the 10 percent level for no high school and at the 5 percent level for BA plus in the first column. But, when we add in the fixed effects in the second column, the loss of precision makes these estimates not statistically different from each other (although they still remain qualitatively different from each other).

Table 6 expands upon the finding that the age at which one experiences Medicaid eligibility might matter for long-run outcomes. In this table, we estimate the

[54]. The one exception is again for the BA Plus outcome when estimating equation (4). Here, we see a positive effect of eligibility at birth on college completion. But, the effect of eligibility at ages 1-17 still is larger (although also less precisely estimated).

effects of eligibility using ages 0-3, ages 4-8, ages 9-13, and ages 14-17. These categories are selected to correspond to those Medicaid eligibility age categories delineated in Currie et al. (2008) as well as to correspond roughly to different schooling levels (pre-school, elementary school, etc.). Panel A shows results from the baseline specification (equation 3), while in Panel B we include our full set of fixed effects (equation 4).

While the results are somewhat imprecise, they show evidence that eligibility at ages 0-3 has little impact on educational attainment. For high school completion, it is eligibility at ages 4-8 that is the most important.[55] For college completion, the estimates are less consistent across panels. Focusing on Panel B, eligibility at all ages except 9-13 are positively related to BA attainment. But, in Panel A, only eligibility during teenage years impacts college completion. We also find evidence of a college enrollment effect due to eligibility expansions among teenagers. At least some of this effect may be due to reproductive services that can be purchased with Medicaid (Lovenheim, Reback, and Wedenoja 2014; Kearney and Levine 2009). Taken together, the results from Tables 5 and 6 demonstrate that estimates of Medicaid eligibility at birth provides an incomplete characterization of how Medicaid affects educational attainment and that eligibility among older, school-age children is particularly important for driving attainment outcomes.

[55]. This is not to say that this insurance has no effect as they age. Indeed, one reason why Medicaid expansions among younger children might be more effective is because they are likely to be eligible for a longer proportion of their childhood. Of course, this does not explain why expansions among very young children do not affect educational attainment.

2.6.3. Educational Attainment Results by Race

Thus far, we have estimated models that pool effects across racial groups. But, given persistent racial disparities in educational attainment, heterogeneous effects by race are of considerable interest. In Online Appendix Tables A-1 and A-2,[56] we estimate our models separately for whites and nonwhites, respectively. For whites, the effects on high school non-completion are negative, but they are smaller in absolute value than in the pooled model and they are not statistically significant at conventional levels. There is a larger effect of Medicaid on college completion for whites, although these estimates also are not statistically significantly different from zero at conventional levels. The effect is on the order of 1.0 to 1.3 percentage points for each 10 percentage point increase in Medicaid eligibility. There is a sizable, positive effect on college completion for whites using the federal variation as well. While these point estimates are large – suggesting a 2.5 percentage point increase from a 10 percentage point Medicaid eligibility increase – they are consistent with observed increases in white college completion across these cohorts.[57]

Among nonwhites, the effects on high school non-completion are larger, particularly in the baseline model. High school non-completion is reduced by 0.46 percentage points for each 10 percentage point increase in Medicaid eligibility.[58] There also is evidence of a positive college completion effect on the order of 0.4 of a

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[56]. All online appendices can be found at http://jhr.uwpress.org/.

[57]. CPS tabulations indicate that college completion rates among white 23 year olds increased by 6.4 percentage points between the 1980 and 1990 birth cohorts. White Medicaid eligibility expanded by 19 percentage points across cohorts, which would increase BA attainment rates by 4.75 (=0.25*0.19*100) percentage points. This is 74 percent of the total BA attainment increase over this period.

[58]. It is notable that these estimates become much smaller in absolute value when we include the full set of fixed effects. However, they also become much less precise such that the baseline estimates are still within the 95 percent confidence intervals. Furthermore, the estimates using federal variation show a large effect of eligibility increases on high school completion.

percentage point for each 10 percentage point increase in eligibility. However, as we show in Online Appendix Table A-6, the BA estimates for non-whites are not robust to the inclusion of state-specific birth cohort trends. Overall, these results are consistent with a larger effect of Medicaid eligibility for whites on higher education completion and a larger effect for nonwhites on high school completion.[59] Other than for the college completion estimates using federal eligibility variation, the estimates by race are not statistically different from each other, however.

2.6.4. Robustness Checks

In this section, we present several additional robustness checks that yield additional insight into the validity of our central identifying assumption, namely that there are not differential underlying trends in educational attainment correlated with public health insurance eligibility expansions. First, in Table 7, we present results from the models presented in Table 3 that also include state-specific linear birth cohort trends, separately by race. If there are differential trends in educational attainment correlated with Medicaid expansions, these results should yield substantively different results from our baseline model. For the high school graduation rate estimates, the results are extremely similar to baseline. However, adding state-specific linear time trends reduces the college completion estimates that include state-level eligibility. As shown in Tables A-5 (whites) and A-6 (non-whites), this average result is mostly due to the fact that there is a large effect of Medicaid eligibility on whites when including state-specific linear time trends but no effect on non-whites. These results also

[59]. In Online Appendix Tables A-3 and A-4 we also show estimates by gender. Although the estimates are somewhat noisy, they suggest a high school completion effect exists for both males and females, while the college enrollment and completion results are isolated to males.

highlight that the federal variation estimates are robust to including state-specific linear time trends. Thus, for whites, there continues to be a large effect of eligibility expansions on college completion, while for non-whites the effects of eligibility expansions are localized to high school completion.

Second, in Table 8, we show the mean and the 2.5^{th} and 97.5^{th} percentiles from 500 simulations that randomly assign Medicaid eligibility and fixed simulated eligibility across age-state-year cells. That is, we take combinations of actual and fixed simulated eligibility, and as a pair randomly assign them to different age-state-year cells, separately by race. This assignment is done with replacement. Both for the baseline model and for the model including race-state-year and race-age-year fixed effects, the average estimates are very close to zero. Furthermore, the non-parametric confidence intervals suggest these null estimates are precisely estimated. This robustness check suggests the results presented in Table 3 are due to the specific way the Medicaid eligibility expansions were rolled out over time within states. When we randomly assign eligibility levels, they are no longer meaningfully related to educational attainment.

In tables A-7 through A-11 of the online appendix, we also present results that explore the sensitivity of our results to several modeling assumptions we have made throughout the analysis. In Table A-7, we estimate our models excluding the small states that generate fewer than 100 observations for an underlying age-race-cohort-year eligibility calculation. The results are virtually identical to those in Table 3. Table A-8 replaces the some college outcome with whether an individual earns an Associates (AA) Degree. We fail to find an effect of Medicaid expansions on AA

attainment, which supports our finding that the main impacts of Medicaid on educational attainment come through high school and BA completion. In Table A-9, we use 1-year instead of 3-year Medicaid eligibility. Again, our results are very similar to baseline.

In Table A-10, we assign Medicaid eligibility based on an individual's state of residence rather than state of birth. The results are very similar to the baseline analysis. This is especially notable since there are very few sources of data that include in them an individual's state of birth. Thus, any long-run analysis of Medicaid eligibility requires researchers to use an individual's current state of residence as a proxy for childhood exposure, which is problematic if there is endogenous mobility related to Medicaid eligibility. Our estimates are inconsistent with such mobility, and thus our findings expand the possibilities for examining long-run Medicaid effects using other datasets that only contain current state of residence. Finally, in order to assess whether the results are sensitive to cohort exposure to local labor market conditions, in Table A-11, we control for average unemployment rates in each state-of-birth and for each birth cohort. The estimates change little from those in Table 3. Overall, these results show our conclusions are robust to different ways of constructing our analysis sample and to different modeling assumptions.[60]

2.7. Conclusions

In this paper, we provide the first evidence on the effects of public health insurance expansions on long-run educational attainment in the US. Overall, our

[60]. In an earlier version of our paper, we present evidence using outcomes from the Youth Risk Behavior Surveillance System (YRBSS) that better health is one of the mechanisms driving our results by showing that Medicaid eligibility when young translates into better teen health. While our estimates from this analysis were typically not statistically significant at conventional levels, they provide support for the idea that better health is an important mechanism that drives at least part of the increased educational attainment we document.

results suggest large effects of childhood Medicaid expansions on eventual educational outcomes. Our baseline estimates indicate that a 10 percentage point increase in Medicaid eligibility between the ages of 0 and 17 decreases the likelihood of not completing high school by approximately 4 percent and increases the 4-year college completion rate by 2.5 percent. The effects on high school completion are largest among nonwhites, while the effects on college completion are largest for whites. We also present evidence that public health insurance expansions when children are of school age are closely linked with long-run educational attainment; eligibility expansions beyond birth lead to higher educational attainment. To the best of our knowledge, these are the first estimates to demonstrate the importance of health insurance eligibility among older children, particularly as it relates to educational outcomes.

Although the public health insurance expansions that we study occurred in the past several decades, our results have several implications that are important for current public policy. First, they suggest that the long-run benefits of providing health insurance to low-income children may be much larger than the short-run gains. Evidence pointing to the large and growing returns to educational attainment (Autor, Katz, and Kearney, 2008) as well as the importance of education in increasing intergenerational economic mobility (Black and Devereaux, 2011; Chetty et al., 2014) suggests that the returns on the public investments in health insurance in the 1980s and 1990s will be realized for some time.

Second, our results relate to current policy discussions over the future of the SCHIP program, which have accompanied the larger debate over the ACA. More

specifically, the ACA prohibits states from imposing eligibility and enrollment standards for Medicaid and SCHIP that were more restrictive than those in place in March 2010 (when the ACA was passed) until 2019. However, there have been attempts in Congress to repeal these provisions, which would essentially allow states to cut SCHIP benefits and eligibility. In addition, SCHIP funding is up for re-authorization in 2015, and its passage is far from assured. A back-of-the-envelope calculation indicates that eliminating the SCHIP program would reduce eligibility for public health insurance by 15.4 percentage points. Our baseline estimates suggest such a decline would increase the high school dropout rate by six-tenths of a percentage point and would decrease the college enrollment rate by five-tenths of a percentage point and the college completion rate by one percentage point. The results from this study highlight the need to account for the long-run effects of public health insurance provision when considering changes to the publicly provided health care system that is targeted at low-income children.

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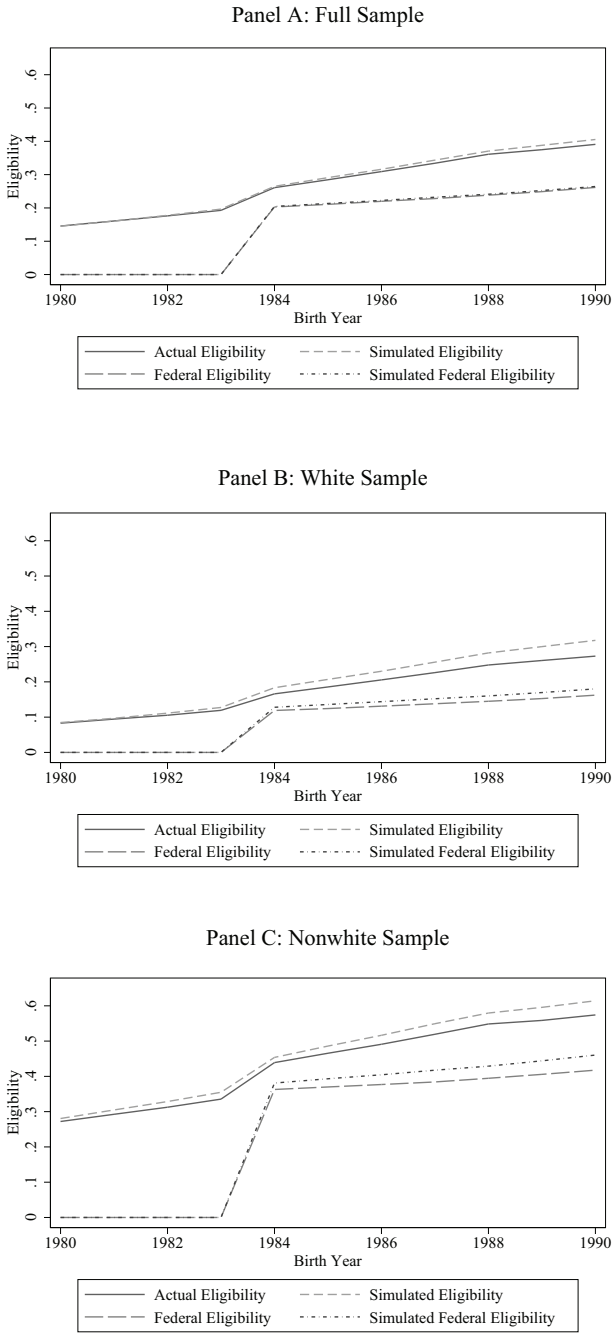
Figure 2.1: **Medicaid Eligibility by Birth Cohort and Race**



The figure shows average eligibility of 0-17 year olds by birth cohort calculated using 1980-2007 CPS data combined with state by year Medicaid eligibility rules. Eligibility is calculated separately for whites and non-whites. Simulated fixed eligibility is calculated by applying state-by-year rules to 1986 CPS data. Federal eligibility uses only federal Medicaid rules, applied to each state using fixed 1980 AFDC rules.

Table 2.1: **Birth Cohorts by Age in Each ACS Year**

Age	2005	2006	2007	2008	2009	2010	2011	2012
22	1983	1984	1985	1986	1987	1988	1989	1990
23	1982	1983	1984	1985	1986	1987	1988	1989
24	1981	1982	1983	1984	1985	1986	1987	1988
25	1980	1981	1982	1983	1984	1985	1986	1987
26		1980	1981	1982	1983	1984	1985	1986
27			1980	1981	1982	1983	1984	1985
28				1980	1981	1982	1983	1984
29					1980	1981	1982	1983

Table 2.2: **Summary Statistics for Analysis Samples**

Variable Name	All	White	Nonwhite
No High School	0.094	0.071	0.143
	(0.048)	(0.029)	(0.045)
No High School or GED	0.126	0.102	0.176
	(0.054)	(0.038)	(0.050)
At Least Some College	0.656	0.694	0.572
	(0.086)	(0.062)	(0.071)
College Graduate	0.265	0.309	0.172
	(0.108)	(0.096)	(0.065)
Age	25.001	25.031	24.936
	(2.156)	(2.155)	(2.157)
Male	0.504	0.508	0.497
	(0.039)	(0.032)	(0.049)
White	0.683	1.000	0.000
	(0.466)	(0.000)	(0.000)
Black	0.143	0.000	0.451
	(0.266)	(0.000)	(0.290)
Hispanic	0.123	0.000	0.386
	(0.230)	(0.000)	(0.255)
Other Race	0.052	0.000	0.163
	(0.108)	(0.000)	(0.135)
Age 0-17 3-year Average	0.236	0.156	0.410
Medicaid Eligibility	(0.152)	(0.077)	(0.127)
Age 0-17 Average Fixed	0.254	0.171	0.431
Simulated Medicaid Eligibility	(0.154)	(0.084)	(0.118)
Age 0-17 3-year Federal Average	0.113	0.068	0.208
Simulated Medicaid Eligibility	(0.140)	(0.070)	(0.194)
Age 0-17 Average Federal Fixed	0.122	0.074	0.224
Simulated Medicaid Eligibility	(0.147)	(0.073)	(0.204)
Observations	5494	2754	2740

Source: Author's tabulations from the 2005-2012 ACS. The samples consist of 1980-1990 birth cohorts aged 22-29, for whom we observe Medicaid eligibility in every year in their birth state from age 0 through 17. All tabulations were done using ACS sample weights. Standard deviations are shown in parentheses. Average eligibility is calculated using 3-year moving averages. The GED tabulations only include ACS years 2008-2012. Federal Medicaid eligibility is calculated using federal rules only, interacted with 1980 state AFDC rules as described in the text.

Table 2.3: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment**

Specification	1st Stage	No HS			Some College			College Plus		
		OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.927***	-0.030**	-0.036**	-0.039**	0.023	0.032	0.035	0.019	0.061**	0.066**
	(0.111)	(0.014)	(0.014)	(0.015)	(0.018)	(0.022)	(0.025)	(0.017)	(0.029)	(0.033)
(2) EITC & School Spending	0.966***	-0.023	-0.036**	-0.038**	0.024	0.033	0.034	0.016	0.067**	0.069**
	(0.076)	(0.015)	(0.015)	(0.015)	(0.019)	(0.021)	(0.022)	(0.020)	(0.030)	(0.032)
(3) EITC, School Spending, R-S-Y & R-A-Y FE	0.943***	-0.000	-0.019	-0.021	0.045	0.076*	0.081*	0.042	0.096	0.102
	(0.111)	(0.020)	(0.024)	(0.024)	(0.031)	(0.042)	(0.042)	(0.028)	(0.064)	(0.064)
(4) Baseline + R-S-Y & R-A-Y FE	0.890***	-0.001	-0.022	-0.025	0.010	0.087	0.099	0.036	0.080	0.091
	(0.170)	(0.020)	(0.025)	(0.027)	(0.033)	(0.059)	(0.071)	(0.026)	(0.066)	(0.070)
Panel B: Federal Eligibility										
(5) Baseline	0.212***	-0.030**	-0.012***	-0.055***	0.023	0.002	0.011	0.019	0.017***	0.078***
	(0.030)	(0.014)	(0.004)	(0.021)	(0.018)	(0.007)	(0.032)	(0.017)	(0.006)	(0.028)
(6) EITC & School Spending	0.210***	-0.023	-0.011***	-0.054**	0.024	0.002	0.011	0.016	0.016***	0.077***
	(0.032)	(0.015)	(0.004)	(0.021)	(0.019)	(0.007)	(0.033)	(0.020)	(0.006)	(0.027)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.4: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment, Separating GED and HS Diplomas, 2008-2012**

		No HS Diploma		No GED or HS		Some College		College Plus	
Specification	1^{st} Stage	OLS	IV	OLS	IV	OLS	IV	OLS	IV
Panel A: All Eligibility									
(1) Baseline	0.927***	-0.020	-0.047**	-0.023	-0.043**	0.009	0.047	0.025	0.085**
	(0.115)	(0.018)	(0.019)	(0.020)	(0.020)	(0.020)	(0.037)	(0.019)	(0.042)
(2) EITC & School Spending	0.959***	-0.013	-0.046***	-0.015	-0.042**	0.021	0.039	0.021	0.087**
	(0.078)	(0.019)	(0.018)	(0.020)	(0.020)	(0.022)	(0.027)	(0.022)	(0.041)
(3) EITC, School Spending R-S-Y & R-A-Y FE	0.944***	0.010	-0.023	0.003	-0.016	0.032	0.079**	0.025	0.099
	(0.110)	(0.021)	(0.024)	(0.023)	(0.028)	(0.028)	(0.039)	(0.031)	(0.071)
(4) Baseline + R-S-Y & R-A-Y FE	0.889***	0.009	-0.027	0.003	-0.022	-0.003	0.100	0.020	0.090
	(0.173)	(0.020)	(0.027)	(0.023)	(0.029)	(0.030)	(0.072)	(0.027)	(0.075)
Panel B: Federal Eligibility									
(5) Baseline	0.210***	-0.020	-0.075***	-0.023	-0.075***	0.009	-0.004	0.025	0.086***
	(0.030)	(0.018)	(0.025)	(0.020)	(0.026)	(0.020)	(0.038)	(0.019)	(0.030)
(6) EITC & School Spending	0.206***	-0.013	-0.073***	-0.015	-0.073***	0.021	0.000	0.021	0.087***
	(0.031)	(0.019)	(0.026)	(0.020)	(0.026)	(0.022)	(0.041)	(0.022)	(0.031)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old respondents from the 2008-2012 ACS. Each cell in the table comes from a separate regression (N=3957). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.5: **IV Estimates of the Effect of Average Medicaid Eligibility At Birth on Educational Attainment by Age at Eligibility**

Medicaid Age	Age 0		Age 0, 1-17	
Eligibility	Baseline	FE	Baseline	FE
No High School:				
Age 0 Eligibility	-0.006	-0.011	-0.004	-0.008
	(0.011)	(0.013)	(0.011)	(0.013)
Age 1-17 Eligibility			-0.038***	-0.022
			(0.014)	(0.029)
Any College:				
Age 0 Eligibility	-0.016	-0.001	-0.017	-0.011
	(0.012)	(0.014)	(0.012)	(0.017)
Age 1-17 Eligibility			0.032	0.100
			(0.023)	(0.070)
BA Plus:				
Age 0 Eligibility	-0.024*	0.046**	-0.026*	0.039**
	(0.013)	(0.019)	(0.013)	(0.019)
Age 1-17 Eligibility			0.062**	0.070
			(0.031)	(0.068)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old respondents from the 2005-2012 ACS. All estimates include an indicator for the cell being nonwhite or not, race-by-age-fixed effects, race-by-calendar year fixed effects and race-by-state of birth fixed effects. "FE" estimates are from equation (4) and include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects. Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.6: **IV Estimates of the Effect of Average Medicaid Eligibility During School Years on Educational Attainment, by Age at Eligibility**

Age Range	No HS Diploma	Any College	College Plus
	Panel A: Baseline Model		
0-3	-0.011	0.019	-0.003
	(0.014)	(0.015)	(0.017)
4-8	-0.030***	0.013	0.037***
	(0.010)	(0.014)	(0.014)
9-13	0.007	-0.025*	-0.026
	(0.010)	(0.015)	(0.017)
14-17	-0.012	0.061***	0.069***
	(0.008)	(0.011)	(0.016)
	Panel B: Baseline + R-S-Y and R-A-Y FE		
0-3	0.003	-0.010	0.053
	(0.020)	(0.030)	(0.036)
4-8	-0.030	0.032	0.051
	(0.020)	(0.026)	(0.057)
9-13	0.003	0.012	-0.002
	(0.012)	(0.029)	(0.022)
14-17	-0.013	0.072***	0.025
	(0.015)	(0.028)	(0.025)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). All estimates include an indicator for the cell being nonwhite or not, race-by-age-fixed effects, race-by-calendar year fixed effects and race-by-state of birth fixed effects. Estimates in Panel B come from equation (4) and also include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects. Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.7: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment, Including State of Birth Linear Time Trends**

		No HS			Some College			College Plus		
Specification	1^{st} Stage	OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	1.009***	-0.033**	-0.043***	-0.042***	0.011	-0.019	-0.019	0.028	0.026	0.026
	(0.059)	(0.016)	(0.015)	(0.015)	(0.022)	(0.029)	(0.028)	(0.022)	(0.025)	(0.024)
(2) EITC & School Spending	1.013***	-0.032**	-0.041***	-0.040***	0.007	-0.016	-0.016	0.022	0.035	0.034*
	(0.059)	(0.016)	(0.015)	(0.014)	(0.020)	(0.027)	(0.026)	(0.018)	(0.022)	(0.020)
(3) EITC, School Spending, R-S-Y & R-A-Y FE	1.019***	-0.009	-0.031	-0.029	0.046	0.000	-0.002	0.014	0.005	0.006
	(0.148)	(0.033)	(0.048)	(0.043)	(0.040)	(0.062)	(0.054)	(0.026)	(0.049)	(0.043)
(4) Baseline + R-S-Y & R-A-Y FE	1.033***	-0.003	-0.023	-0.021	0.044	-0.000	-0.003	0.017	0.013	0.014
	(0.147)	(0.032)	(0.047)	(0.041)	(0.039)	(0.060)	(0.052)	(0.027)	(0.046)	(0.040)
Panel B: Federal Eligibility										
(5) Baseline	0.220***	-0.033**	-0.013***	-0.059***	0.011	0.004	0.019	0.028	0.018***	0.082***
	(0.032)	(0.016)	(0.004)	(0.021)	(0.022)	(0.007)	(0.030)	(0.022)	(0.006)	(0.026)
(6) EITC & School Spending	0.223***	-0.032**	-0.012***	-0.054**	0.007	-0.000	0.000	0.022	0.011*	0.049**
	(0.032)	(0.016)	(0.004)	(0.021)	(0.020)	(0.007)	(0.030)	(0.018)	(0.006)	(0.025)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Estimates also include race by state of birth linear time trends. Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.8: **Placebo Test with Randomly Assigned Medicaid Eligibility**

	No HS Graduation		Some College		BA	
	RF	IV	RF	IV	RF	IV
Baseline	0.0001	0.0001	-0.0004	-0.0005	$2.87e^{-5}$	$2.31e^{-5}$
	(-0.006, 0.007)	(-0.007, 0.007)	(-0.011, 0.010)	(-0.013, 0.012)	(-0.010, 0.011)	(-0.011, 0.012)
Baseline +	0.0002	0.0002	-0.0004	-0.0005	0.0001	0.0001
R-S-Y & R-A-Y FE	(-0.008, 0.007)	(-0.009, 0.008)	(-0.012, 0.011)	(-0.014, 0.013)	(-0.010, 0.011)	(-0.011, 0.012)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old respondents from the 2005-2012 ACS. We randomly assign age-state-year eligibility and fixed simulated eligibility, as a pair, across different age-state-year cells. This is done separately by race. We conduct 500 separate simulations for each outcome, both including and excluding state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects. All estimates include an indicator for the cell being non-white or not as well as race-by-age fixed effects, race-by-calendar year fixed effects and race-by-state of birth fixed effects. The table shows the mean estimate across all 500 runs, as well as the 2.5^{th} and 97.5^{th} percentiles in parentheses. The range in parentheses thus show the non-parametric 95% confidence interval. IV estimates are constructed by dividing the reduced form (RF) by the first stage, which also is estimated using this method. First stage estimates are available upon request from the authors.

Table 2.A-1: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment using the White Sample**

Specification	1st Stage	No HS			Some College			College Plus		
		OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.729***	-0.021	-0.022	-0.030	-0.005	0.026	0.036	0.018	0.070	0.096
	(0.132)	(0.018)	(0.023)	(0.032)	(0.032)	(0.029)	(0.043)	(0.042)	(0.049)	(0.075)
(2) EITC & School Spending	0.809***	-0.012	-0.032	-0.040	-0.004	0.026	0.032	0.018	0.080	0.099
	(0.085)	(0.019)	(0.023)	(0.030)	(0.033)	(0.029)	(0.036)	(0.046)	(0.052)	(0.069)
(3) EITC, School Spending, S-Y & A-Y FE	0.777***	0.018	-0.029	-0.038	0.029	0.078*	0.100*	0.100	0.103	0.133
	(0.124)	(0.022)	(0.034)	(0.042)	(0.050)	(0.044)	(0.052)	(0.064)	(0.093)	(0.115)
(4) Baseline + S-Y & A-Y FE	0.692***	0.016	-0.027	-0.039	-0.010	0.107*	0.155	0.087	0.084	0.121
	(0.192)	(0.019)	(0.033)	(0.047)	(0.041)	(0.058)	(0.103)	(0.059)	(0.101)	(0.137)
Panel B: Federal Eligibility										
(5) Baseline	0.233***	-0.021	-0.012	-0.051	-0.005	0.029*	0.126**	0.018	0.058***	0.249***
	(0.028)	(0.018)	(0.010)	(0.041)	(0.032)	(0.015)	(0.062)	(0.042)	(0.015)	(0.068)
(6) EITC & School Spending	0.236***	-0.012	-0.012	-0.052	-0.004	0.030*	0.127**	0.018	0.059***	0.252***
	(0.029)	(0.019)	(0.010)	(0.040)	(0.033)	(0.015)	(0.062)	(0.046)	(0.016)	(0.070)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old white respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=2754). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include age fixed effects, calendar year fixed effects and state of birth fixed effects. Rows 3 and 4 include state of birth by calendar year (S-Y) fixed effects and age by calendar year (A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-2: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment using the Nonwhite Sample**

		No HS			Some College			College Plus		
Specification	1st Stage	OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	1.148***	-0.035*	-0.053**	-0.046**	0.040	0.039	0.034	0.020	0.050**	0.044**
	(0.129)	(0.019)	(0.023)	(0.019)	(0.025)	(0.032)	(0.028)	(0.014)	(0.020)	(0.019)
(2) EITC & School Spending	1.109***	-0.035*	-0.048**	-0.043**	0.042	0.037	0.033	0.013	0.048**	0.044**
	(0.102)	(0.018)	(0.022)	(0.019)	(0.027)	(0.035)	(0.031)	(0.016)	(0.022)	(0.020)
(3) EITC, School Spending, S-Y & A-Y FE	1.153***	-0.013	-0.006	-0.006	0.060	0.073	0.066	0.002	0.087	0.078
	(0.177)	(0.031)	(0.049)	(0.038)	(0.046)	(0.091)	(0.071)	(0.032)	(0.055)	(0.048)
(4) Baseline + S-Y & A-Y FE	1.200***	-0.013	-0.014	-0.012	0.023	0.056	0.050	0.001	0.074	0.064
	(0.243)	(0.030)	(0.052)	(0.039)	(0.051)	(0.102)	(0.079)	(0.029)	(0.060)	(0.054)
Panel B: Federal Eligibility										
(5) Baseline	0.207***	-0.035*	-0.012**	-0.056**	0.040	-0.004	-0.019	0.020	0.007	0.032
	(0.032)	(0.019)	(0.006)	(0.028)	(0.025)	(0.008)	(0.036)	(0.014)	(0.007)	(0.031)
(6) EITC & School Spending	0.201***	-0.035*	-0.012**	-0.059**	0.042	-0.005	-0.023	0.013	0.006	0.029
	(0.034)	(0.018)	(0.006)	(0.029)	(0.027)	(0.008)	(0.038)	(0.016)	(0.007)	(0.033)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old nonwhite respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=2726). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include age fixed effects, calendar year fixed effects and state of birth fixed effects. Rows 3 and 4 include state of birth by calendar year (S-Y) fixed effects and age by calendar year (A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-3: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment using the Male Sample**

Specification	1st Stage	No HS			Some College			College Plus		
		OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.728***	-0.013	-0.023	-0.031	-0.020	0.079**	0.108*	0.060	0.129**	0.177*
	(0.132)	(0.029)	(0.031)	(0.042)	(0.051)	(0.039)	(0.061)	(0.049)	(0.061)	(0.095)
(2) EITC & School Spending	0.820***	0.002	-0.028	-0.033	-0.019	0.068*	0.088*	0.067	0.135**	0.171**
	(0.079)	(0.030)	(0.031)	(0.040)	(0.055)	(0.036)	(0.047)	(0.051)	(0.060)	(0.083)
(3) EITC, School Spending, S-Y & A-Y FE	0.792***	0.034	-0.017	-0.019	0.024	0.160***	0.212***	0.110	0.114	0.148
	(0.114)	(0.032)	(0.044)	(0.054)	(0.079)	(0.058)	(0.079)	(0.074)	(0.112)	(0.141)
(4) Baseline + S-Y & A-Y FE	0.689***	0.020	-0.016	-0.023	-0.013	0.195**	0.284*	0.096	0.100	0.144
	(0.192)	(0.030)	(0.044)	(0.059)	(0.070)	(0.081)	(0.156)	(0.070)	(0.116)	(0.157)
Panel B: Federal Eligibility										
(5) Baseline	0.233***	-0.013	-0.019	-0.083	-0.020	0.023	0.098	0.060	0.067***	0.288***
	(0.028)	(0.029)	(0.014)	(0.060)	(0.051)	(0.027)	(0.111)	(0.049)	(0.019)	(0.087)
(6) EITC & School Spending	0.236***	0.002	-0.020	-0.084	-0.019	0.024	0.103	0.067	0.068***	0.288***
	(0.030)	(0.030)	(0.014)	(0.059)	(0.055)	(0.028)	(0.112)	(0.051)	(0.019)	(0.088)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old male respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-4: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment using the Female Sample**

Specification	1st Stage	No HS			Some College			College Plus		
		OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.731***	-0.028*	-0.019	-0.026	0.004	-0.033	-0.045	-0.034	0.002	0.003
	(0.133)	(0.015)	(0.022)	(0.029)	(0.039)	(0.044)	(0.058)	(0.046)	(0.050)	(0.068)
(2) EITC & School Spending	0.824***	-0.029*	-0.023	-0.028	0.009	-0.019	-0.028	-0.041	0.020	0.024
	(0.079)	(0.016)	(0.023)	(0.029)	(0.037)	(0.044)	(0.056)	(0.051)	(0.055)	(0.071)
(3) EITC, School Spending, S-Y & A-Y FE	0.795***	0.002	-0.029	-0.041	0.024	-0.014	-0.023	0.073	0.077	0.099
	(0.114)	(0.027)	(0.033)	(0.040)	(0.047)	(0.076)	(0.097)	(0.061)	(0.095)	(0.120)
(4) Baseline + S-Y & A-Y FE	0.695***	0.014	-0.036	-0.052	-0.017	0.004	0.006	0.066	0.054	0.078
	(0.193)	(0.023)	(0.030)	(0.046)	(0.041)	(0.065)	(0.085)	(0.059)	(0.104)	(0.136)
Panel B: Federal Eligibility										
(5) Baseline	0.233***	-0.028*	-0.005	-0.020	0.004	0.037	0.157	-0.034	0.048**	0.208**
	(0.028)	(0.015)	(0.013)	(0.053)	(0.039)	(0.024)	(0.104)	(0.046)	(0.020)	(0.085)
(6) EITC & School Spending	0.237***	-0.029*	-0.005	-0.022	0.009	0.036	0.154	-0.041	0.048**	0.205**
	(0.029)	(0.016)	(0.013)	(0.053)	(0.037)	(0.024)	(0.102)	(0.051)	(0.020)	(0.084)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old female respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-5: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment, using the White Sample and Including State of Birth Linear Time Trends**

		No HS			Some College			College Plus		
Specification	1st Stage	OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.848***	-0.030	-0.051*	-0.060	0.033	-0.010	-0.012	0.093	0.046	0.055
	(0.062)	(0.032)	(0.031)	(0.037)	(0.051)	(0.048)	(0.055)	(0.061)	(0.047)	(0.054)
(2) EITC & School Spending	0.865***	-0.029	-0.050	-0.058	0.054	0.035	0.041	0.133***	0.129***	0.150***
	(0.059)	(0.031)	(0.032)	(0.037)	(0.042)	(0.044)	(0.050)	(0.045)	(0.039)	(0.047)
(3) EITC, School Spending, S-Y & A-Y FE	0.799***	-0.030	-0.138*	-0.172*	0.064	0.005	0.007	0.126	0.110	0.138
	(0.152)	(0.049)	(0.074)	(0.098)	(0.074)	(0.107)	(0.121)	(0.077)	(0.114)	(0.128)
(4) Baseline + S-Y & A-Y FE	0.828***	-0.010	-0.116	-0.140	0.060	0.002	0.002	0.127	0.117	0.141
	(0.157)	(0.047)	(0.076)	(0.095)	(0.069)	(0.102)	(0.111)	(0.083)	(0.120)	(0.129)
Panel B: Federal Eligibility										
(5) Baseline	0.249***	-0.030	-0.016	-0.063*	0.033	0.033**	0.134**	0.093	0.063***	0.251***
	(0.027)	(0.032)	(0.010)	(0.037)	(0.051)	(0.016)	(0.059)	(0.061)	(0.015)	(0.061)
(6) EITC & School Spending	0.257***	-0.029	-0.014	-0.053	0.054	0.029*	0.112*	0.133***	0.056***	0.217***
	(0.028)	(0.031)	(0.010)	(0.037)	(0.042)	(0.016)	(0.059)	(0.045)	(0.014)	(0.054)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old white respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=2754). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include age fixed effects, calendar year fixed effects and state of birth fixed effects. Rows 3 and 4 include state of birth by calendar year (S-Y) fixed effects and age by calendar year (A-Y) fixed effects as shown in equation (4). Estimates include state of birth specific linear time trends across birth cohorts. Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-6: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment, using the Nonwhite Sample and Including State of Birth Linear Time Trends**

		No HS			Some College			College Plus		
Specification	1^{st} Stage	OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	1.109***	-0.034*	-0.038	-0.034	0.002	-0.025	-0.023	0.002	0.014	0.013
	(0.071)	(0.019)	(0.024)	(0.020)	(0.024)	(0.032)	(0.028)	(0.021)	(0.026)	(0.023)
(2) EITC & School Spending	1.105***	-0.032	-0.033	-0.029	-0.003	-0.033	-0.030	-0.016	-0.007	-0.006
	(0.073)	(0.021)	(0.026)	(0.023)	(0.026)	(0.033)	(0.029)	(0.021)	(0.024)	(0.021)
(3) EITC, School Spending, S-Y & A-Y FE	1.159***	0.000	0.039	0.036	0.039	-0.006	-0.009	-0.038	-0.061	-0.051
	(0.164)	(0.034)	(0.056)	(0.046)	(0.051)	(0.079)	(0.061)	(0.034)	(0.050)	(0.040)
(4) Baseline + S-Y & A-Y FE	1.165***	-0.000	0.036	0.033	0.036	-0.001	-0.005	-0.034	-0.054	-0.044
	(0.157)	(0.034)	(0.058)	(0.047)	(0.053)	(0.078)	(0.059)	(0.034)	(0.048)	(0.038)
Panel B: Federal Eligibility										
(5) Baseline	0.213***	-0.034*	-0.012**	-0.058**	0.002	-0.003	-0.013	0.002	0.008	0.036
	(0.034)	(0.019)	(0.006)	(0.027)	(0.024)	(0.008)	(0.034)	(0.021)	(0.007)	(0.030)
(6) EITC & School Spending	0.213***	-0.032	-0.011*	-0.053*	-0.003	-0.005	-0.023	-0.016	0.002	0.011
	(0.034)	(0.021)	(0.006)	(0.030)	(0.026)	(0.008)	(0.034)	(0.021)	(0.006)	(0.029)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old nonwhite respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=2726). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include age fixed effects, calendar year fixed effects and state of birth fixed effects. Rows 3 and 4 include state of birth by calendar year (S-Y) fixed effects and age by calendar year (A-Y) fixed effects as shown in equation (4). Estimates include state of birth specific linear time trends across birth cohorts. Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-7: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment Removing the Small Samples from NH, VT, ME, WV**

		No HS			Some College			College Plus		
Specification	1st Stage	OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.939***	-0.034**	-0.041***	-0.044***	0.025	0.036	0.038	0.018	0.065**	0.069**
	(0.112)	(0.014)	(0.014)	(0.015)	(0.018)	(0.023)	(0.026)	(0.017)	(0.029)	(0.033)
(2) EITC & School Spending	0.978***	-0.027*	-0.041***	-0.042***	0.027	0.037*	0.038*	0.015	0.072**	0.073**
	(0.077)	(0.015)	(0.014)	(0.014)	(0.019)	(0.022)	(0.022)	(0.020)	(0.031)	(0.033)
(3) EITC, School Spending, S-Y & A-Y FE	0.966***	-0.003	-0.022	-0.022	0.043	0.079*	0.081*	0.037	0.103	0.107*
	(0.114)	(0.021)	(0.024)	(0.023)	(0.032)	(0.043)	(0.042)	(0.028)	(0.066)	(0.065)
(4) Baseline + S-Y & A-Y FE	0.913***	-0.004	-0.025	-0.027	0.007	0.090	0.099	0.033	0.088	0.097
	(0.174)	(0.020)	(0.026)	(0.027)	(0.034)	(0.061)	(0.071)	(0.026)	(0.067)	(0.069)
Panel B: Federal Eligibility										
(5) Baseline	0.211***	-0.034**	-0.012***	-0.056***	0.025	0.003	0.016	0.018	0.016***	0.077***
	(0.031)	(0.014)	(0.004)	(0.021)	(0.018)	(0.007)	(0.032)	(0.017)	(0.006)	(0.028)
(6) EITC & School Spending	0.208***	-0.027*	-0.012***	-0.056***	0.027	0.003	0.016	0.015	0.016***	0.076***
	(0.032)	(0.015)	(0.004)	(0.021)	(0.019)	(0.007)	(0.033)	(0.020)	(0.006)	(0.028)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old respondents from the 2005-2012 ACS. States with race-state-age-year cells of under 100 are dropped from the analysis. Each cell in the table comes from a separate regression (N=5076). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-8: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment With Associates Degrees**

Specification	1st Stage	No HS			Associates Degree			College Plus		
		OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.927***	-0.030**	-0.036**	-0.039**	-0.010	-0.011	-0.012	0.019	0.061**	0.066**
	(0.111)	(0.014)	(0.014)	(0.015)	(0.011)	(0.019)	(0.019)	(0.017)	(0.029)	(0.033)
(2) EITC & School Spending	0.966***	-0.023	-0.036**	-0.038**	-0.017	-0.020	-0.021	0.016	0.067**	0.069**
	(0.076)	(0.015)	(0.015)	(0.015)	(0.011)	(0.017)	(0.017)	(0.020)	(0.030)	(0.032)
(3) EITC, School Spending, S-Y & A-Y FE	0.943***	-0.000	-0.019	-0.021	-0.015	-0.004	-0.003	0.042	0.096	0.102
	(0.111)	(0.020)	(0.024)	(0.024)	(0.021)	(0.044)	(0.042)	(0.028)	(0.064)	(0.064)
(4) Baseline + S-Y & A-Y FE	0.890***	-0.001	-0.022	-0.025	-0.015	0.016	0.019	0.036	0.080	0.091
	(0.170)	(0.020)	(0.025)	(0.027)	(0.021)	(0.049)	(0.050)	(0.026)	(0.066)	(0.070)
Panel B: Federal Eligibility										
(5) Baseline	0.212***	-0.030**	-0.012***	-0.055***	-0.010	-0.007**	-0.033**	0.019	0.017***	0.078***
	(0.030)	(0.014)	(0.004)	(0.021)	(0.011)	(0.004)	(0.014)	(0.017)	(0.006)	(0.028)
(6) EITC & School Spending	0.210***	-0.023	-0.011***	-0.054**	-0.017	-0.007*	-0.032**	0.016	0.016***	0.077***
	(0.032)	(0.015)	(0.004)	(0.021)	(0.011)	(0.004)	(0.015)	(0.020)	(0.006)	(0.027)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old male respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-9: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment Using 1-Year Medicaid Eligibility**

		No HS			Some College			College Plus		
Specification	1^{st} Stage	OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.985***	-0.018*	-0.036**	-0.038***	0.005	0.032	0.033	0.013	0.061**	0.061**
	(0.125)	(0.011)	(0.014)	(0.014)	(0.015)	(0.022)	(0.024)	(0.014)	(0.029)	(0.031)
(2) EITC & School Spending	1.028***	-0.012	-0.036**	-0.037***	0.005	0.033	0.032	0.011	0.067**	0.065**
	(0.092)	(0.010)	(0.015)	(0.014)	(0.016)	(0.021)	(0.021)	(0.016)	(0.030)	(0.030)
(3) EITC, School Spending, S-Y & A-Y FE	0.971***	0.007	-0.019	-0.020	0.010	0.076*	0.078*	0.021	0.096	0.103
	(0.128)	(0.013)	(0.024)	(0.023)	(0.022)	(0.042)	(0.041)	(0.020)	(0.064)	(0.063)
(4) Baseline + S-Y & A-Y FE	0.915***	0.006	-0.022	-0.024	-0.007	0.087	0.095	0.019	0.080	0.092
	(0.185)	(0.013)	(0.025)	(0.027)	(0.023)	(0.059)	(0.070)	(0.018)	(0.066)	(0.068)
Panel B: Federal Eligibility										
(5) Baseline	0.241***	-0.018*	-0.012***	-0.049**	0.005	0.002	0.012	0.013	0.017***	0.068***
	(0.038)	(0.011)	(0.004)	(0.019)	(0.015)	(0.007)	(0.028)	(0.014)	(0.006)	(0.026)
(6) EITC & School Spending	0.239***	-0.012	-0.011***	-0.048**	0.005	0.002	0.011	0.011	0.016***	0.067***
	(0.040)	(0.010)	(0.004)	(0.019)	(0.016)	(0.007)	(0.029)	(0.016)	(0.006)	(0.025)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old male respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-10: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment Using Current State of Residence**

Specification	1st Stage	No HS			Some College			College Plus		
		OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.932***	-0.039***	-0.045***	-0.048***	0.039*	0.025	0.027	0.056***	0.084***	0.090***
	(0.107)	(0.011)	(0.014)	(0.015)	(0.023)	(0.027)	(0.028)	(0.021)	(0.028)	(0.033)
(2) EITC & School Spending	0.966***	-0.036***	-0.045***	-0.046***	0.050*	0.028	0.029	0.062***	0.083***	0.086***
	(0.076)	(0.012)	(0.014)	(0.014)	(0.026)	(0.028)	(0.028)	(0.021)	(0.026)	(0.029)
(3) EITC, School Spending, S-Y & A-Y FE	0.940***	-0.018	-0.021	-0.022	0.107**	0.081	0.084	0.136***	0.180***	0.192***
	(0.113)	(0.018)	(0.024)	(0.023)	(0.042)	(0.056)	(0.053)	(0.048)	(0.069)	(0.070)
(4) Baseline + S-Y & A-Y FE	0.893***	-0.015	-0.023	-0.026	0.054	0.084	0.092	0.121***	0.185**	0.207**
	(0.166)	(0.018)	(0.022)	(0.023)	(0.040)	(0.063)	(0.069)	(0.046)	(0.083)	(0.088)
Panel B: Federal Eligibility										
(5) Baseline	0.216***	-0.039***	-0.012***	-0.054***	0.039*	0.002	0.012	0.056***	0.016***	0.074***
	(0.030)	(0.011)	(0.004)	(0.021)	(0.023)	(0.007)	(0.034)	(0.021)	(0.005)	(0.026)
(6) EITC & School Spending	0.215***	-0.036***	-0.012***	-0.055**	0.050*	0.003	0.013	0.062***	0.016***	0.074***
	(0.031)	(0.012)	(0.004)	(0.021)	(0.026)	(0.007)	(0.035)	(0.021)	(0.005)	(0.027)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old male respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not as well as race-by age, race-by-calendar year and race-by-state of residence fixed effects. Rows 3 and 4 include race by state of residence by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-residence level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 2.A-11: **The Effect of Average Medicaid Eligibility During School Years on Educational Attainment Controlling for Unemployment During Childhood**

		No HS			Some College			College Plus		
Specification	1st Stage	OLS	RF	IV	OLS	RF	IV	OLS	RF	IV
Panel A: All Eligibility										
(1) Baseline	0.916***	-0.033**	-0.039***	-0.042***	0.021	0.031	0.033	0.010	0.053*	0.058*
	(0.112)	(0.015)	(0.015)	(0.016)	(0.019)	(0.023)	(0.026)	(0.018)	(0.029)	(0.033)
(2) EITC & School Spending	0.964***	-0.025*	-0.040**	-0.041***	0.022	0.029	0.030	0.009	0.057*	0.060*
	(0.078)	(0.015)	(0.016)	(0.016)	(0.019)	(0.021)	(0.021)	(0.021)	(0.029)	(0.031)
(3) EITC, School Spending, S-Y & A-Y FE	0.942***	-0.003	-0.022	-0.024	0.047	0.077*	0.083**	0.041	0.094	0.101
	(0.112)	(0.019)	(0.025)	(0.024)	(0.031)	(0.041)	(0.041)	(0.027)	(0.063)	(0.064)
(4) Baseline + S-Y & A-Y FE	0.886***	-0.007	-0.026	-0.030	0.015	0.091*	0.104	0.034	0.079	0.090
	(0.168)	(0.018)	(0.024)	(0.025)	(0.032)	(0.055)	(0.068)	(0.026)	(0.065)	(0.069)
Panel B: Federal Eligibility										
(5) Baseline	0.205***	-0.033**	-0.014***	-0.067***	0.021	0.001	0.004	0.010	0.011*	0.053*
	(0.034)	(0.015)	(0.004)	(0.021)	(0.019)	(0.007)	(0.032)	(0.018)	(0.006)	(0.029)
(6) EITC & School Spending	0.211***	-0.025*	-0.014***	-0.067***	0.022	-0.001	-0.004	0.009	0.009	0.043
	(0.034)	(0.015)	(0.004)	(0.021)	(0.019)	(0.007)	(0.033)	(0.021)	(0.006)	(0.028)

Source: Authors' estimation of equations (3) and (4) in the text using 22-29 year old male respondents from the 2005-2012 ACS. Each cell in the table comes from a separate regression (N=5480). The "OLS" columns refer to models that use a three-year moving average of actual eligibility as the independent variable, and the "RF" columns refer to models that use fixed simulated eligibility as the independent variable. The "IV" columns refer to models that instrument for actual eligibility using fixed simulated eligibility. All estimates include an indicator for the cell being nonwhite or not, average state unemployment rates when respondents were 0-17 in their state of birth, race-by age, race-by-calendar year and race-by-state of birth fixed effects. Rows 3 and 4 include race by state of birth by calendar year (R-S-Y) fixed effects and race by age by calendar year (R-A-Y) fixed effects as shown in equation (4). Standard errors clustered at the state-of-birth level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

CHAPTER 3

THE EFFECT OF STRESS ON LATER-LIFE HEALTH: EVIDENCE FROM THE VIETNAM DRAFT

3.1 Introduction

Bodily stress response serves an important biological function in humans: it increases blood flow, directs energy towards the brain and muscles, and is part of our adaptive process, allowing our bodily systems to stabilize and maintain life (McEwen 2005, Schneiderman et al. 2005). However, long-term exposure to stress can have deleterious effects on the body including increased risk of cardiovascular events, higher susceptibility to infection, and increased risk of emotional problems (Schneiderman et al. 2005, Lupien et al. 2009). While much has been written about the long term effects of in utero exposure to stress and illness (see e.g. Currie and Almond 2011), the effect of stress during one's late teens and early adulthood has received relatively little attention despite the fact that this is an important developmental time for the brain (Kaestner and Yarnoff 2011, Dahl 2004). Moreover, these are ages at which individuals make important human capital, career and family formation decisions that have long-run consequences for their well-being.

We exploit variation in the risk of being inducted into the military. We focus on the risk between 1955 (after the Korean war) and 1973 (when the US switched to an all-volunteer force), and examine the effect of this risk on later life health outcomes. The personnel needs of the army varied greatly over this time period due to the Vietnam War and the continued Cold War threat creating varying levels of risk depending on when a person became eligible to be inducted into the military. We

argue this prolonged uncertainty about whether one would be conscripted into military service between the ages 18 ½ and 26 caused large amounts of stress on young men at this time. Highly publicized calls for increased troop levels in Vietnam and increasingly large numbers of U.S. servicemen being killed or wounded were well documented by media outlets, which made this risk particularly salient.

We find consistent evidence that early adulthood stress as measured by induction risk has negative effects on later life health. A 10 percentage point increase in induction risk, which is a 2 standard deviation increase, between the ages of 18 ½ and 26 is associated with a 0.2 unit (0.08%) increase in BMI, a 1.5 percentage point (1%) increase in the probability of being obese, a 1 percentage point (1%) increase in the probability of being in fair or poor health and a 2 percentage point (0.3%) decrease in the probability of being in very good or excellent health. These associations do not exist for women or men who served in the military, which is consistent with the effect for male non-Vietnam-veterans being due to draft risk as opposed to changes in unobserved variables around that time.

This paper contributes to the literature by examining a novel source of stress: risk of induction to the military, and examines its association with later-life health outcomes. Previous literature has used induction risk as an instrumental variable for education (de Walque 2007, Grimard and Parent 2007) to estimate the effect of education on smoking. This literature did not examine whether the risk could directly affect later-life health controlling for education.

Additionally, we focus on a period of the war before the implementation of the draft lottery, a period largely ignored in previous studies, but a particularly morbid and

lethal time for American soldiers.[61] By focusing on men who did not serve in the military we remove the potential for endogenous health selection caused by combat exposure and avoid potential selection into educational attainment through the GI Bill (Angrist and Chen 2011).

In the next section, we review the relevant literatures. Section 3.3 provides background on the Vietnam War and describes the evolving process of military induction during the Vietnam War. Section 3.4 describes the data and in Section 3.5 we describe our identification strategy. We present our results in Section 3.6 before concluding in Section 3.7.

3.2. Literature Review

The armed forces induction system may affect health through at least three routes: 1) increased stress caused by the risk of serving in the armed forces; 2) military service; 3) through draft-avoidance behaviors such as attending college or getting married to receive military exemptions. We discuss each of these possibilities in turn.

3.2.1. Clinical Effects of Stress on Bodily Function and Health

Risk of being inducted into the military during the Vietnam War caused great uncertainty about one's future and may have increased individuals' stress levels for long periods of time.[62] Below, we describe the pathways through which long-term risk of military service, a proxy for stress, may lead to worse long-term health. While bodily responses to acute stress events, or adaptive processes, which return the body to

[61] Over 80 percent of US casualties were suffered before the first draft lottery occurred December 1, 1969, although public outcry and a shift from explicitly pro-US government media coverage to a more nuanced approach covering public dissent and occasionally showing explicit, disturbing war images suggest a particular salience to risk of being inducted during the draft lottery years (The U.S. National Archives and Records Administration, 2013, Griffin, 2010, Hallin, 1984).

[62] For most of the war, individuals were eligible to be drafted from age 18 ½ to 26.

a state of normal function are an important facet of human adjustment to stress, chronic or repeated stress can lead to harmful health effects (Schneiderman et al. 2005, Lupien et al. 2009). Exposure to stress leads to biological responses from the nervous, cardiovascular, endocrine, and immune systems (Schneiderman et al. 2005). Individual response patterns vary based on early life experiences, the age at which stress occurs, coping mechanisms, the type and persistence of stressors, genetic endowments, and personal environment and constitution (Schneiderman et al. 2005, Lupien et al. 2009). The pathways through which stress may affect health are complex, including psychological and biochemical responses, as well as behavioral changes which may be initiated through these channels (Schneiderman et al. 2005).

Stressors lead to the release of hormones such as epinephrine and cortisol which increase sources of energy through higher blood sugar and the breaking down of fats into useable energy (Schneiderman et al. 2005, Sapolsky et al. 2000). The body diverts this energy to tissues that become more active during stress (skeletal muscles and brain) and away from less critical activities like eating, growth, and sexual activity (Schneiderman et al. 2005). This temporarily increases blood pressure through increased heart rate and stroke volume (the amount of blood pumped with each beat). Chronic mobilization of these processes is associated with high blood pressure, cardiac hypertrophy (the thickening of the heart muscle, which reduces the heart chamber size), damaged arteries and plaque formation, suppressed immunity including atrophied wound healing, slower surgery recovery, higher susceptibility to viruses including upper respiratory infections, worse antibody response to vaccines, and increased inflammation which can exacerbate many of the aforementioned conditions

(Schneiderman et al. 2005, Sapolsky et al. 2009). Importantly, stress can lead to psychological problems like anxiety and depression, which are associated with sleep problems, substance abuse, heavier cigarette consumption, and higher alcohol consumption (Schneiderman et al. 2005).

Stress may be especially harmful in high demand and low control situations, in which an individual is under a large amount of strain and has little overall control, such as military induction (Karasek and Theorell 1990). In such situations, individuals have limited control over whether they are inducted, what their military occupation would be, where they might be deployed, and what dangers they might face, and this lack of control may greatly increase stress.

3.2.2. The Effects of Military Service on Later-Life Outcomes

Researchers have studied the effect of military service and risk of military service on labor market outcomes (Angrist 1990), education outcomes (Card and Lemieux 2001), and violent crimes (Lindo and Stoecker 2013). See Dobkin and Shabani (2009) for a discussion of the health effects of military service, and Gimbel and Booth (1996) for a discussion of stress among veterans.

3.2.3 The Effects of Draft Avoidance Behaviors on Later-life Outcomes

For certain periods of the Vietnam War, otherwise draft-eligible men could receive deferments for being enrolled in college; for this reason, college attendance became a draft-avoidance behavior. This led to an increase in college attendance, and several papers have used induction risk as an instrumental variable for educational

attainment (see e.g. Card and Lemieux 2001)[63], in some cases to estimate the effect of college education on later-life smoking (de Walque 2007, Grimard and Parent 2007) or mortality (Buckles et al. 2013).

Grimard and Parent (2007) use variation in military induction risk among the 1945-1950 birth cohorts to instrument for educational attainment and find that additional education is associated with a lower probability of smoking, while de Walque (2007) uses a similar measure of induction risk based on birth cohort to instrument for educational attainment and finds that education decreases the probability of smoking and increases the probability of quitting smoking. Both of these studies use a similar measure of induction as our study, but we extend their measures by exploiting birth cohort variation by month of birth as well as year.

Additional variation in induction risk existed at the state level. Buckles et al. (2013) exploits the across-state variation to estimate the effect of college completion on mortality. They find rather sizable effects of education on mortality using both national and state level induction risk to instrument for both educational attainment and veteran status, noting this effect likely operates through decreased smoking, improved financial security, and better health resources. It seems unlikely that state-level variation in induction risk was well known at the time, raising the question of whether draft-eligible men would have responded to it (Buckles et al. 2013). Thus, the national risk of induction may be a better measure of stress and perceived incentives for draft avoidance behavior.

[63] However, whether the increase in educational attainment during the Vietnam War is due to draft avoidance or use of the GI Bill is a matter of debate (Angrist and Chen 2011).

Other draft avoidance behavior included getting married and having children to exploit marital and paternal draft deferments (Kutinova 2009). Kutinova (2009) finds evidence of increased first births occurring between 8 and 10 months following an executive order ending marital deferments for childless men, while Hanson (2011) finds young men married a younger ages during periods in which marital deferments existed.

A large and growing literature in economics estimates the effect of early life stressors on a wide variety of later-life outcomes, including educational attainment, earnings, and health (see Almond and Currie 2011). This literature generally focuses on stressors in utero or during infancy, but other work, examining the long term effects of leaving school during a recession on later life health, has found significant negative effects on health for men (Maclean 2013, Maclean et al. 2015). In contrast, women leaving school during periods of high unemployment have lower rates of depressive symptoms compared to women leaving school during periods of lower unemployment rates (Maclean 2013, Maclean et al. 2015). These studies provide evidence of long-term effects of state and national-level conditions on adult health.

The innovation of this paper is to examine the impact of induction risk on later-life health, controlling for education, income, and marriage – all of which may have been altered by the risk. Our research provides new information about the impact of induction risk during young adulthood on later-life health, as well as the validity of draft risk as an instrument for education; specifically, we find evidence that draft risk may have a direct effect on later-life health, even controlling for possible draft avoidance behaviors such as education and marriage.

3.3. The Vietnam Conflict and the Risk of Induction into the U.S. Military

Following the French military departure from Vietnam in 1954, the US began to directly aid the South Vietnamese government, and US military advisors began training the South Vietnamese military in 1955 ("Chronology of Events Relative to Vietnam, 1954-1965," 1965). This growing influence in Vietnam did not affect US military inductions until the Gulf of Tonkin Incident in 1964 in which a US ship engaged the North Vietnamese navy in the Gulf of Tonkin ("Chronology of Events Relative to Vietnam, 1954-1965,"1965). Induction rose rapidly, peaking in October 1966, and again in the first half of 1968 before decreasing rapidly; see Figure 1. Prior to U.S. involvement in Vietnam, induction level also were high in the second half of 1961 due to the Berlin Wall Crisis, and in the early 1950s due to the Korean conflict.

As the U.S. death toll rose in Vietnam, political unrest in the US grew with student protests and draft card burnings. During his 1968 Presidential campaign, Nixon campaigned on a platform that included abolishing the draft and implementing an All-Volunteer Force (Rostker 2006). It was not until 1973 that Nixon was able to create the all-volunteer force when Congress allowed the legislative mandate to induct citizens into the military to expire. Later in 1973 Congress passed the Case-Church amendment that officially ended direct US involvement in Vietnam.

3.3.1. Induction Process

At this time, individuals could enter military service through two routes: voluntary enlistment or conscription (induction). Voluntary enlistments alone were not enough to maintain an adequate fighting force during this era (Annual Report of the Director of the Selective Service System, 1955). Draft-eligible males would

sometimes volunteer in order to choose their branch of service; as a result, enlistments and inductions were correlated - when inductions dropped, enlistments also dropped, necessitating inductions to meet military manpower needs.

Induction and enlistment differed on the following parameters: enlistments required an additional 1-2 years of service beyond that required of inductees, but allowed individuals to choose their branch of service and provided the possibility of officer and other specialty training. Those enlisting voluntarily were given additional flexibility on start date (House Armed Services Committee, 1970 via Angrist, 1991). Voluntary inductees, alternatively, received similar treatment to non-voluntary inductees: shorter tours of duty but less choice and flexibility in assignment.[64]

The process of inductions proceeded as follows: The Director of the Selective Service System received a monthly call for inductions from the President and Secretary of Defense (Annual Report, 1955). The director then delivered these calls for induction to State-level directors of Selective Service, who sent these requisitions to the local boards in the state. To ensure an equitable system by state, requisitions were sent out proportionately depending on the number of eligible registrants by state, and states further received credit for the number of residents currently serving in the military (Annual Report, 1955).

Local boards conducted pre-induction examinations in order to have induction-eligible individuals ready when they received calls from the State Director. Approximately half of all examinees were cleared for service (Annual Report, 1966).

[64] These volunteers for induction were generally younger than regular inductees and may have been trying to join the military so that their service did not interrupt their lives at a later age; they received no preferential treatment besides expedited delivery into the Armed Forces (Annual Report, 1955, Semi-Annual Report 1968).

An additional 20 percent were rejected for service after being delivered for induction (Annual Report, 1966; Angrist, 1991). Those passing pre-induction medical exams were sent to Armed Forces induction stations.

A priority system dictated the order of induction; generally, delinquents received the highest draft priority, followed by volunteers for induction.[65] The priority of induction varied during the Vietnam War period, with various marital, paternal, and student deferments created, modified, and repealed. The lexis chart in Table 1 summarizes which ages of young adults were eligible for induction by year, and Figures 2 and 3 give a brief summary of changes in the induction system from 1948 through the end of the induction system and the beginning of an all-volunteer force in 1973. Our data do not allow us to determine an individual's eligibility for specific deferments, so they are not used in our calculation of induction risk.

Executive order 10659 provided a new order for inductions, one created to prevent older registrants (those 26 or older) and fathers from being high priority inductees (Annual Report 1955, pg. 27). Under this order, the next highest priority groups after delinquents and volunteers became those aged 19-25, without children, chosen by age, oldest first,[66] followed by those of similar ages with children. Then those aged 26 or older were chosen by age, with the youngest receiving calls for induction first. Some deferments, including student deferments, extended the age at

[65] Delinquency is defined as failure to comply with the Universal Military Training and Service Act. Examples are refusal to register, failure to supply board with information, failure to report for pre-induction examination, or failure to report for induction (Annual Report 1952). Any person of 18-26, under provision part 1630 of SS regulations, can offer themselves for induction at any point in time. Persons between age 17 and 18, with the approval of a guardian, also can volunteer for induction (Annual Report 1955).

[66] Previous research using induction risk as an instrumental variable for education looked only at risk during certain college ages (e.g. deWalque, 2007, used ages 19-22), but actual risk extended well past that. The priority of drafting the oldest first underscores the importance of including post-college years in induction risk.

which one could be inducted to age 35 (Annual Report 1953).[67] Finally, if local boards could not fill their quotas using the previous groups, those aged 18 ½, but not yet 19, could be called, oldest first.

The US selective service enacted the first draft lottery on December 1, 1969 (Selective Service System, 2012). Birth dates were randomly selected to determine draft priority for those born between January 1, 1944 and December 31, 1950.[68] Individuals were at risk of induction for the calendar year 1970 after which point they could not be called for induction. Subsequent draft lotteries included single birth cohorts; e.g. the 1970 draft applied only to the 1951 birth cohort. The final draft lottery from which inductees were called for service occurred on August 5, 1971 and affected the 1952 birth cohort. However, the Selective Service continued to conduct the draft lottery annually until 1975, with the understanding that should Congress reinstate the draft, inductions would be based on these draft lotteries. The last inductee was called in December 1972 and reported for duty in July 1973 (Selective Service System, 2012). Soon after in August 1973, Congress passed the Case-Church ending direct US involvement in the Vietnam War.

3.4. Data

The primary data used in this project are: 1) the National Health Interview Survey (NHIS) and 2) induction data taken from Reports of the Director of the Selective Service. In this section, we describe these data and document how we constructed the variables used in our analysis.

[67] Few, if any, individuals over the age of 26 were called for induction.
[68] The 1969 draft lottery was skewed towards higher priority calls for those with December birthdays apparently due to inadequately mixed draft balls (Fienberg 1971 via Lindo and Stoecker 2014). This issue was corrected in later draft lotteries.

3.4.1. National Health Interview Survey (NHIS)

The NHIS is a cross-sectional, nationally representative data set of the US non-institutional population. Each year, NHIS interviews approximately 100,000 individuals, asking questions about basic demographic and socioeconomic characteristics, military service, height and weight (from which we calculate body mass index or BMI),[69] and self-reported health. We use data from the 1982-1996 NHIS for our main analysis sample. Based on our sample of interest - men born from 1937 to 1956 - our main analysis sample is aged 25 to 59 during 1982-1996. The sample size varies between 119,000 and 140,000 depending on the regression model specification, due to missing values for certain variables.

Because height and weight measures come from self-reports, they likely suffer from reporting error (Bound et al. 2001; Cawley et al., 2015). We adjust these measures for reporting error using data from the National Health and Nutrition Examination Survey (NHANES) III (1988-1994), which include both self-reports and measurements of weight and height and are from a similar time period as our NHIS data.[70]

---

[69] $BMI = 703 * \left(\frac{weight\ in\ lbs}{height\ in\ inches^2}\right)$

[70] Using the NHANES data for the same age range as our NHIS data (age 25-59), we estimate the following regressions separately by gender:

$$MeasuredWeight_i = \beta_0 + \beta_1 SelfReport_i + \beta_2 SelfReport_i^2 + X_i + \varepsilon_i \qquad (1)$$

Where SelfReport refers to self-reported weight, and X includes age and race (non-Hispanic Black, non-Hispanic other race, and Hispanic). We then save the constant and coefficient estimates from these regressions and create an adjusted measure for weight in the NHIS sample by multiplying the NHIS self-reported weight values and demographic characteristics by their coefficients from NHANES regressions, and adding them together, along with the constant from the regression model. We perform a similar adjustment for self-reported height. Using these constructed values, we create an adjusted measure of BMI and obesity (BMI≥30) which we use throughout the rest of this manuscript. We also estimate all models using the non-adjusted BMI and obesity measures and find slightly larger and more statistically significant results using unadjusted variables, accentuating the importance of adjusting these variables.

From 1982-96, NHIS respondents were asked whether they had ever served in the US military and, if so, during which period did they serve (e.g. World War II, Korea, Vietnam). We do not use NHIS data from before 1982 because in those earlier years the NHIS data do not include month of birth, which we use to construct a more accurate measure of induction risk. We do not use NHIS data from after 1996 because in those later years the NHIS only asked whether respondents had been "honorably discharged" from the military, not whether they had served during the Vietnam era (or served at all but without an honorable discharge). We focus only on the years in which we have the more complete military variable (1982-96), however we extend our sample through 2012 as a sensitivity analysis.

Our sample is limited to individuals born in the calendar years 1937 to 1956. We do this to avoid any contamination of our main analysis sample with individuals serving in the Korean War, which ended in July 1953. Thus any individual born in 1937 would not have been eligible to serve in Korea. de Walque (2007) also imposes this sample restriction.[71] Because we are interested in educational attainment, we limit our sample to those over age 25 at interview as most individuals have completed their schooling by this age. We further restrict our sample to those with valid measurements of height and weight because we use these to create BMI.[72]

[71] We also perform analyses using 1930-1956 birth cohorts in sensitivity analyses reported in 6.3. This sample would have been completely ineligible to serve in WWII, which is an important restriction as we only want to consider the post WWII induction system, created in 1948.

[72] To be consistent across the many years of NHIS samples we use, we drop individuals below 59 inches and above 76 inches, and those weighing less than 100 pounds and more than 285 pounds, the most restrictive data publishing policies used by NHIS over this time.

3.4.2. Selective Service Reports

We use annual (1955-1966) and semi-annual (1967-1975) reports of the Director of the Selective Service for induction eligibility, the number of monthly inductions, as well as for additional institutional details and troop level data. These induction data are available by month, which we use to construct our measure of induction risk. We calculate induction risk as the number of inductions in a given 12 month period, using month and year of birth and monthly induction numbers from annual reports to construct a measure of induction risk by month of birth, divided by the number of individuals at risk or:

$$R18_{m,y} = \frac{Inductions\ (12\ Months)}{Cohort\ at\ Risk} \qquad (2)$$

Where R18 is the risk at age 18 for those born in month m, year y. We calculate the denominator, *Cohort at Risk*, using the induction reports for the official number of individuals who have registered for selective service and are between the ages of eligibility (18 ½ to 26) for the given period.[73] We calculate risk separately for age 18 ½, 19, …, 26, which we then sum to calculate average risk from 18 ½ - 26. We focus on 18 ½ to 26 rather than college years (e.g. 19-22) that was the focus of previous research (Card and Lemieux 2001, de Walque 2007, Grimard and Parent 2007, Buckles et al. 2013) because 18 ½ - 26 were the actual, legislated, ages at which an individual was at risk of induction.[74] We calculate the total risk as follows:

[73] We also perform this calculation using measures of the cohort of age 17 year olds as reported by the Department of Education (and creating measures of those between the ages of 18 ½ - 26, by assuming the cohort of 17 year olds in a given year will be the correct number of 18 year olds in the following year). Our results are robust to these multiple methods so we use the official number of individuals registered via the selective service throughout.
[74] For years in which the draft lottery system was in place, we only consider eligibility for those in the birth cohorts at risk.

$$
\begin{aligned}
InductionRisk18\ 1/2-26_{m,y} &= R18_{m,y} + \left(1-R18_{m,y}\right)R19_{m,y} \\
&+ \left(1-R18_{m,y}\right)\left(1-R19_{m,y}\right)R20_{m,y} \\
&+ \left(1-R18_{m,y}\right)\left(1-R19_{m,y}\right)\left(1-R20_{m,y}\right)*R21_{m,y} \\
&+ \left(1-R18_{m,y}\right)\left(1-R19_{m,y}\right)\left(1-R20_{m,y}\right)\left(1-R21_{m,y}\right)*R22_{m,y} \\
&+ \quad \ldots \\
&+ \left(1-R18_{m,y}\right)\left(1-R19_{m,y}\right)\left(1-R20_{m,y}\right)\left(1-R21_{m,y}\right)\left(1\right. \\
&\left.-R22_{m,y}\right)\left(1-R23_{m,y}\right)\left(1-R24_{m,y}\right)\left(1-R25_{m,y}\right) \\
&*R26_{m,y} \qquad (3)
\end{aligned}
$$

Where R18 denotes risk at age 18 ½ while all other measures denote risk for the full year a person is a given age, for month and year of birth m,y. We multiply induction risk by 100 in all specifications so that all coefficients can be interpreted as the association of the outcome with a one-percentage-point change in risk.

3.5. Methods

We estimate reduced form, ordinary least squares models of the form:

$$H_{it} = \beta_0 + \beta_1 InductionRisk_{m,yob} + \beta_2 X_{it} + \delta_{age} + \gamma_t + \varepsilon_{it} \qquad (4)$$

Where H_{it} is the dependent variable denoting various health outcomes including a continuous variable for BMI and an ordinal variable for self-reported health (1 – 5, with 1 denoting poor and 5 denoting excellent health).[75] We estimate probit models for the binary dependent variables obesity (BMI≥30), fair or poor health (self-reported health ≤ 2), and very good or excellent health (self-reported health ≥ 4) of the following form:

$$Pr(H_{it}) = \phi\left(\beta_1 InductionRisk_{m,yob} + \beta_2 X_{it} + \delta_{age} + \gamma_t + \varepsilon_{it}\right) \qquad (5)$$

For each of these models, X_{it} are demographic and human capital characteristics described below, δ are fixed effects for age at interview, γ are fixed effects for year of

[75] We also estimate regressions with self-reported health as the dependent variable using an ordered probit and find qualitatively similar results; the results are available upon request.

interview, and ε is an error term. In all specifications, we cluster standard errors at the birth year cohort level. We report marginal effects from the probit models.

We run three specifications, adding additional covariates in each specification. The basic specification is limited to the most exogenous regressors: race (non-Hispanic Black, non-Hispanic other race, and Hispanic, with non-Hispanic Whites the omitted reference group), a birth cohort specific trend, defined as birth year minus 1937, and indicator variables for age and year. Specification 2 adds covariates for family size and marital status at the time of NHIS interview. These additional covariates reflect the fact that the Vietnam War has been linked to increased marriage and fertility rates (Kutinova 2009, Hanson 2011). Specification 3 additionally includes years of education past high school, and log income, which are endogenous variables but possible mechanisms through which induction risk may affect health (see e.g. Card and Lemieux 2001). Our primary sample of interest consists of men who did not serve in the military.

As a falsification test, we also estimate our models for male veterans; we hypothesize that veterans' later-life health is uncorrelated with the average induction risk in their birth-month cohort. Male veterans of the Vietnam War certainly experienced stress, but it is unlikely to be correlated with the average induction risk of their cohort. Instead, the stress experienced by veterans is most likely due to their personal wartime experiences, such as participation in combat, wounds, and the deaths of fellow soldiers. A limitation of the NHIS data is that we do not know whether veterans volunteered for service or at what age. Some veterans may never have faced

any induction risk if they volunteered immediately upon coming of age, while others may have been exposed to induction risk prior to service.

As an additional falsification test, we estimate models for women. Women were at zero risk of induction throughout this era, so as a falsification test we assign them the induction risk experienced by men of the same month and year of birth. While women of this age cohort may have experienced stress due to the risk that their brothers, boyfriends, or husbands might be inducted, it is unlikely this risk would be highly correlated with the average risk experienced by men with the same month and year of birth as the women.

For both of these placebo samples, a correlation between induction risk and later-life health that was similar to that of men who did not serve in the military would suggest that the association is due not to a causal effect of stress on later-life health, but instead to trends in unobserved variables. In contrast, a correlation between induction risk and later-life health for male non-Vietnam-veterans, but not for male veterans or women, would be consistent with the hypothesis that the resulting stress had an impact on later-life health. As always, results of falsification tests are suggestive rather than definitive.

3.5.1. Sample Selection

A potential concern with this study is that there will be sample selection on health status in who serves in the military based on the level of induction risk. This selection arises because of potential composition effects caused by variation in military force needs. As the need for more soldiers increases, the induction risk also increases, leading to more inductions and thus potentially culling more of the healthier

individuals into service. In this case, during periods in time in which induction risk was particularly great, the health composition of men not inducted into the armed forces might be slightly lower, which would bias us towards finding a correlation of induction risk on health. We look for evidence of this in the results of the regression models estimated for veterans; if induction takes place in order of healthiness, then when induction risk is higher, it should be associated not only with lower average health among the non-Vietnam-veterans, but also lower average health among the veterans, who just had less healthy individuals join their ranks.

Another possible concern is that those with higher socioeconomic status were more adept at avoiding military service either through family connections or greater resources. In this case, assuming a positive gradient of health across socioeconomic status, those who did not serve will be on average healthier than those who did serve. Thus, there may be influences of both positive and negative health selection into military service.

3.6. Results

We report summary statistics in Table 2. The average risk of military induction in our sample age 18 ½ to 26 is 7.7 percent.[76] Predictably, the men who ultimately served in the military during the Vietnam War were at higher induction risk than those who did not serve, 10.3 compared to 6.9 percent. Over 24 percent of the male NHIS sample served in the military during the Vietnam War. An additional 14 percent of men who did not serve in the military during the Vietnam War era served in the

[76] We assign induction risk of zero for women as women were not eligible for induction into the Armed Forces at this time period. In sensitivity analysis we assign induction risk to women based on their birth month and year, using the same method we use for men.

military at some other point in time.[77] The mean body mass index in the male sample is 26.5, with 19 percent having a BMI classified as obese. Over 70 percent of the sample reported very good or excellent health with only 8.3 percent reporting fair or poor health.

Figure 1 displays total inductions by month over the Vietnam era. Importantly, inductions fluctuate greatly over this time period and move in a non-linear pattern. This makes it unlikely that our measure of induction risk is picking up some sort of birth cohort linear trend that may affect health completely separately from induction risk. We show in Figure 4 induction risk by birth month and year cohort over our study period. This figure presents our actual measure of induction risk for each birth month and year as calculated using equation (3). Table 1 shows the years and ages at which a birth cohort was at risk of induction and provides a clearer picture of the source of variation in risk due to the number of years a given birth cohort was at risk of military induction. It also separately identifies the type of induction system being used during the cohorts age-eligible years, with ages in italics denoting that the draft lottery system was in place from 1970-1972.

3.6.1. Results: Men Who Did Not Serve in Vietnam

Table 3 presents results for men who did not serve during Vietnam controlling for time trends, race and ethnicity, and age and year of survey fixed effects, while Table 4 also includes controls for family size and marital status at the time of interview. Results from these models are similar so we discuss only Table 4 results. Among male non-Vietnam-veterans, a one percentage point increase in induction risk

[77] We remove these individuals who served in the military at any time in a sensitivity analysis presented in section 3.6.3.

between ages 18 ½ and 26 is associated with a 0.05 percentage point (0.3%) increase in obesity, a 0.07 percentage point (0.8%) increase in fair or poor health and a 0.05 percentage point (0.1%) decrease in the probability of being in very good or excellent health. All these results are statistically significant at least at the 10 percent level.

In Table 5 we include controls for years of education completed beyond high school and log income. This is an important change to the model, because college education was a draft-avoidance behavior, and educational attainment is consistently associated with better later-life health (see, e.g., Cutler and Lleras-Muney, 2010). The coefficient on induction risk becomes larger and more statistically significant in this model. A one percentage point increase in induction risk between the ages of 18 ½ and 26 is associated with a 0.17 unit (0.1%) increase in BMI, a 0.15 percentage point (0.8%) increase in the probability of obesity, a 0.005 unit (0.1%) decrease in self-reported health, a 0.1 percentage point (1%) increase in the probability of being in fair or poor health and a 0.2 percentage point (0.3%) decrease in the probability of being in very good or excellent health. Income and education are both associated with better self-assessed health, and education is also associated with a lower probability of obesity (income is negatively correlated with obesity, but it is not statistically significant).

3.6.2. Falsification Tests: Male Vietnam Veterans and Women

In Table 6 we present results for the same models that were estimated using samples of: male Vietnam veterans (results shown in Panel A) and women (Panel B). In the interests of being concise, only the coefficients on induction risk are shown in the table, but full results of the models are available upon request. The results from

these models are substantively different from those for male non-Vietnam-veterans in Tables 3-5. In Panel A for male Vietnam veterans, the coefficient on induction risk for BMI and the marginal effect of induction risk for obesity are very small and not statistically significant. In addition, induction risk is consistently associated with better health among veterans. For example, in specification 2, a one percentage point increase in induction risk is associated with a 0.005 unit (0.1%) increase in self-reported health, a 0.14 percentage point (1.8%) decrease in fair or poor health and a 0.24 percentage point (0.3%) increase in very good or excellent health. Adjusting for family income and educational attainment in specification 3, the estimates on fair poor health and very good or excellent health remain statistically significant although they are only about half as large as in specification 2, while the estimate for self-assessed health overall is both smaller and no longer statistically significant.

Results for women in Panel B of Table 6 are somewhat similar to those for Vietnam veterans from panel A, but very different from those of male non-Vietnam-veterans that were shown in Tables 3-5. In specification 2, a one percentage point increase in induction risk is associated with a 0.0025 unit (0.1%) increase in self-assessed health, a 0.07 percentage point (0.6%) decrease in the probability of being in fair or poor health, and a 0.09 percentage point (0.1%) increase in the probability of being in very good health. In the third specification that controls for education, the only statistically significant correlation is that a one percentage point increase in induction risk is associated with a 0.03 percentage point (0.3%) decrease in the probability of being in fair or poor health.

Overall, the results of these falsification tests suggest that the negative association observed for male non-Vietnam-veterans between induction risk and worse later-life health is not due to a cohort effect or trend in unobserved variables. Although falsification tests cannot be definitive, these are consistent with stress having an adverse impact on later-life health.

3.6.3. Additional Sensitivity Analyses

To investigate whether our results reflect compositional changes among non-Vietnam-veterans, we perform a number of sensitivity analyses. First, we estimate a model for all men (non-Vietnam-veterans and veterans) pooled. The logic is that if compositional changes (such as less healthy men being left behind when induction risk rises) explain the earlier results, then pooling non-Vietnam-veterans and veterans should show no association between induction risk and later-life health. In Appendix Table 1, we reprint our main induction risk results in Panel A and we present the new results for all men pooled in Panel B. The results of specification 3, which control for education, are robust to pooling all men, although they are also smaller in magnitude (as one would expect from adding men to the sample whose stress is not well captured by the average induction risk). Results from specifications 1 and 2 are more fragile.

We also estimate models in which we vary our sample inclusion criteria. These results are presented in Appendix Table 2, with Panel A reprinting our main results on induction risk from tables 4 and 5 for easy comparison.[78] In Panel B, we present results when those who ever served in the military, not just during the Vietnam War era, are dropped from the sample. In Panel C, we include only non-Vietnam-veteran

[78] We do not present estimates on induction risk for specification 1 because these estimates are very similar to those from specification 2.

men who responded without a proxy to the NHIS survey. The purpose for doing this is to eliminate any additional reporting error due to health being proxy-reported instead of self-reported. In Panel D, we include in the sample all birth cohorts of male non-Vietnam-veterans back to 1930, who would have been of military age starting in 1948 and thus subject to the same post World War II draft system as those in our main sample. In Panel E we limit the sample to those born between 1930 and 1936 who did not serve in the military, but were age-eligible to serve during the Korean War. These results are similar to those of the main specification; consistently, exposure to a higher induction risk is associated with a higher probability of obesity and worse self-assessed health. These extensions show that the main results are robust to sample definition, and also show that the relationship between stress and later-life health preceded the Vietnam Era.

We estimate the same models, imposing similar sample restrictions for male veterans in Appendix Table 3. The results generally confirm that veterans do not exhibit the negative correlation between induction risk (for those who did not serve) and later-life health; the exception is that Korean veterans exhibit a positive correlation between induction risk and later-life BMI and obesity.

3.7. Discussion

In this paper we find evidence that stress worsens later-life health; specifically, we find that the risk of military induction for young adult males during the Vietnam War is associated with worse later-life health among non-Vietnam-veterans. These adverse health effects are modest, but are relatively robust, and include a higher risk of obesity and higher probability of reporting being in worse health. Falsification tests

conducted with samples of women and male veterans yield little evidence of an association between average induction risk of men of the same birth month and year, and later-life health; this is suggestive evidence that the relationship found for male non-Vietnam-veterans is not due to cohort effects or trends in unobserved variables. We investigate whether this is due to compositional effects and find that the results are largely robust to pooling veterans with non-Vietnam-veterans.

We recognize several limitations of this study. First, we cannot directly measure stress. No survey that we know of measured stress of individuals using cortisol laboratory measurements at the time of the Vietnam War. However, the measure we use, which we limit to those who did not serve in Vietnam, captures an important source of uncertainty faced by individuals at that time. For those who do not serve, we do not have baseline health measures to investigate whether they did not serve due to preexisting health conditions that would bias these individuals towards worse health later in life. This should not bias our results unless health varies by month and year of birth in a way that is correlated with induction risk, which seems unlikely.

Among veterans, we cannot distinguish those who volunteered immediately for service from those who were inducted after experiencing the stress of the draft. However, this should bias the coefficient on risk in the veterans model towards showing ill effects of draft risk on later-life health, which we do not find for the veteran sample. We also do not know whether a veteran served in combat. Gimbel and Booth (1996) find those service members who experienced combat situations in Vietnam actually had lower cortisol levels and higher testosterone levels as measured

years after their tour in Vietnam. Finally, we are limited in the later-life health outcomes that we can examine, by the questions that were asked in NHIS.

Overall, we find a consistent association between stress and worse later-life health. This study contributes to the literature on the adverse later-life consequences of stress, most of which has focused on the effect of stress or insults at the earliest ages (Almond and Currie, 2011). This paper also contributes to the previous literature on the effects of education on later-life health. Specifically, our finding that induction risk is associated with later-life health even controlling for education, suggests that induction risk may be an invalid instrumental variable for education in models that seek to estimate the effect of education on health.

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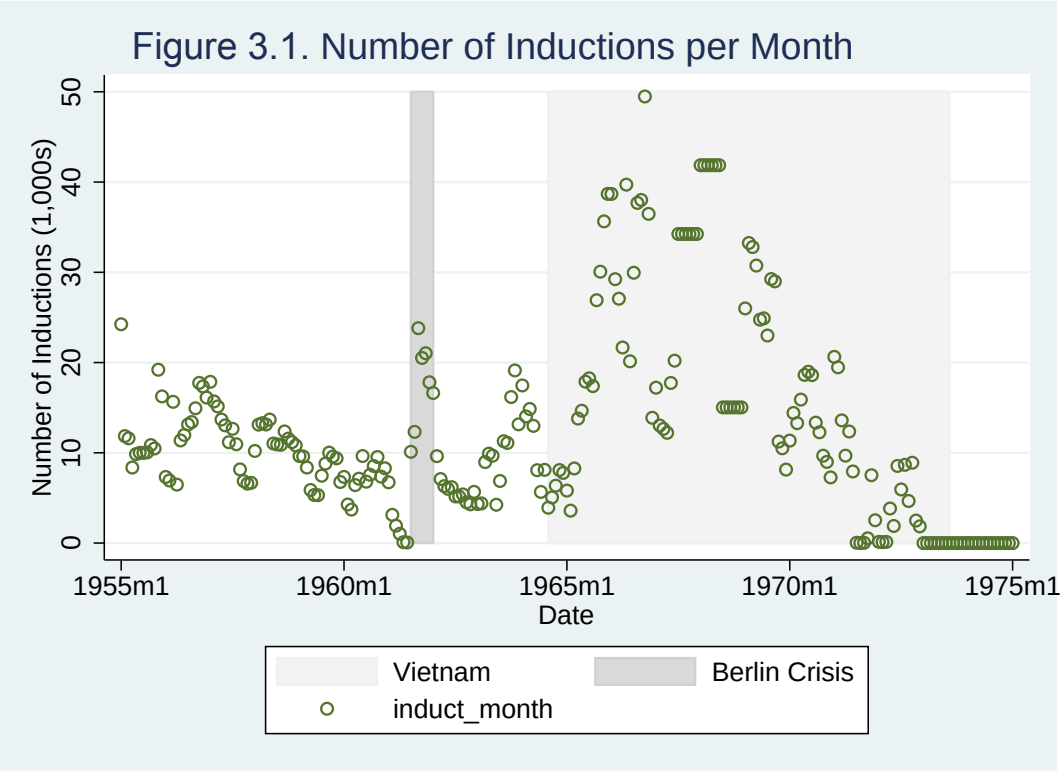


Figure 3.1. Number of Inductions per Month
Number of Inductions (1,000s)
0
10
20
30
40
50
1955m1
1960m1
1965m1
1970m1
1975m1
Date
Vietnam
Berlin Crisis
induct_month

Figure 3.2. Post-World War II through the End of the Vietnam War (1948-1975)

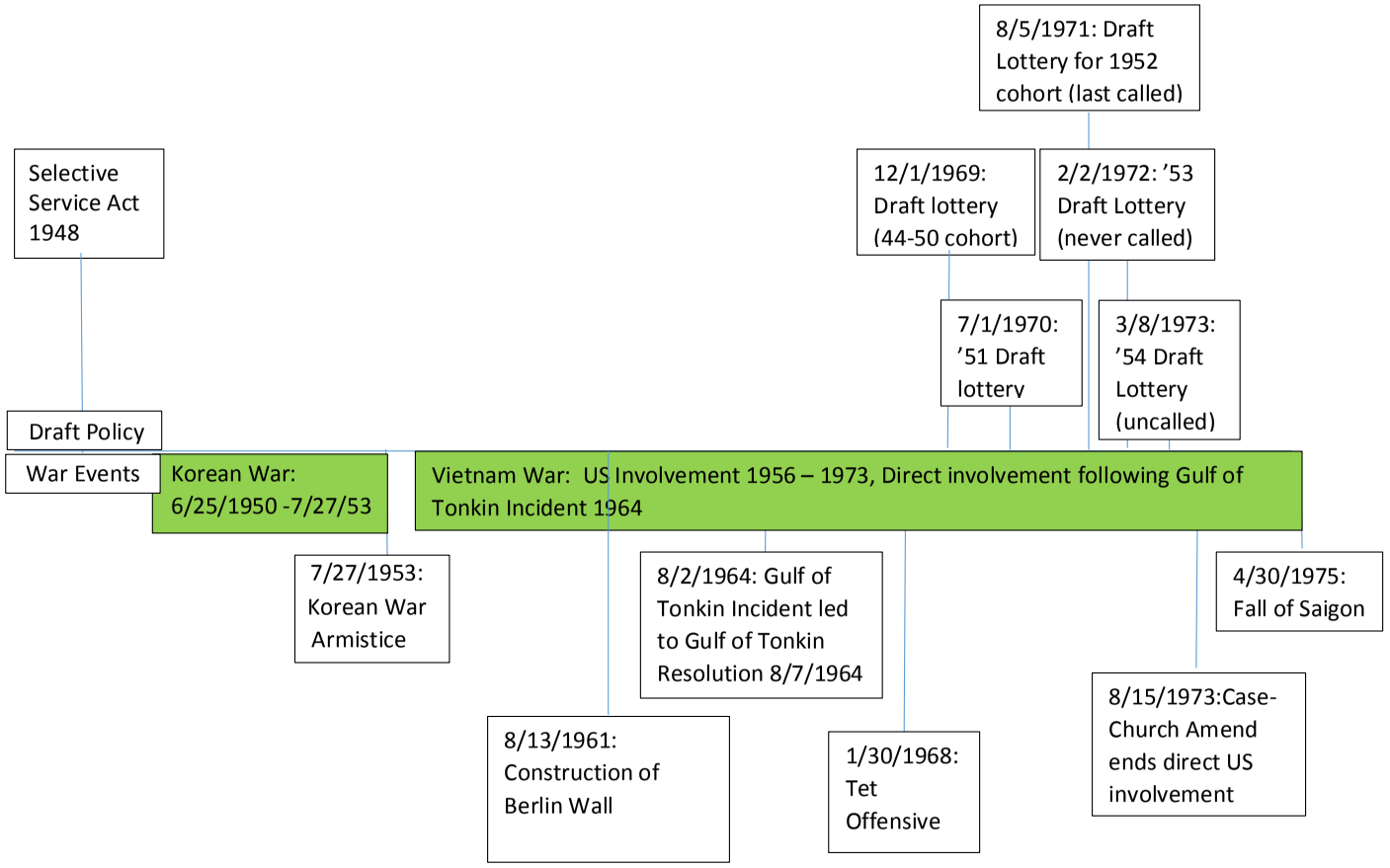
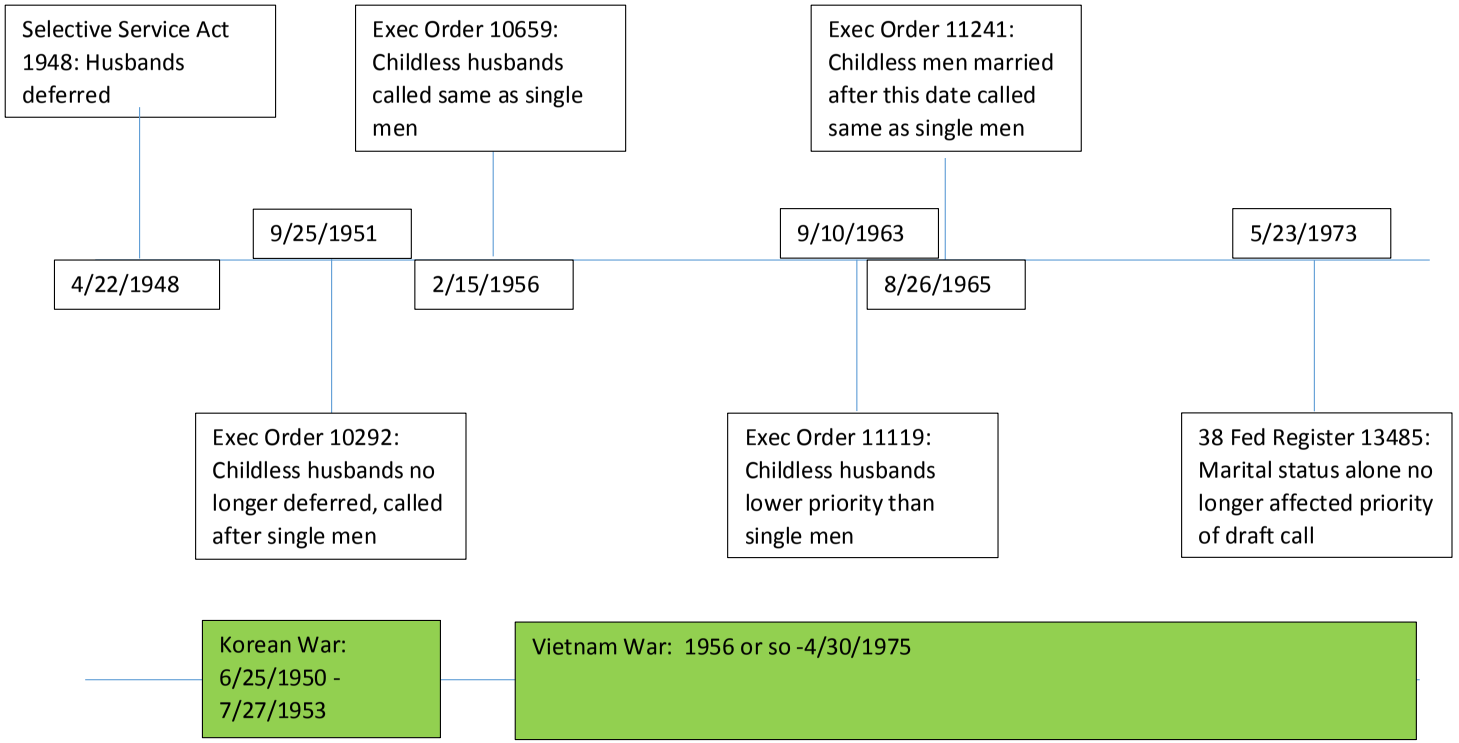


Figure 3.3. Marital Status and Paternity Deferments through the Years (1948-1975)



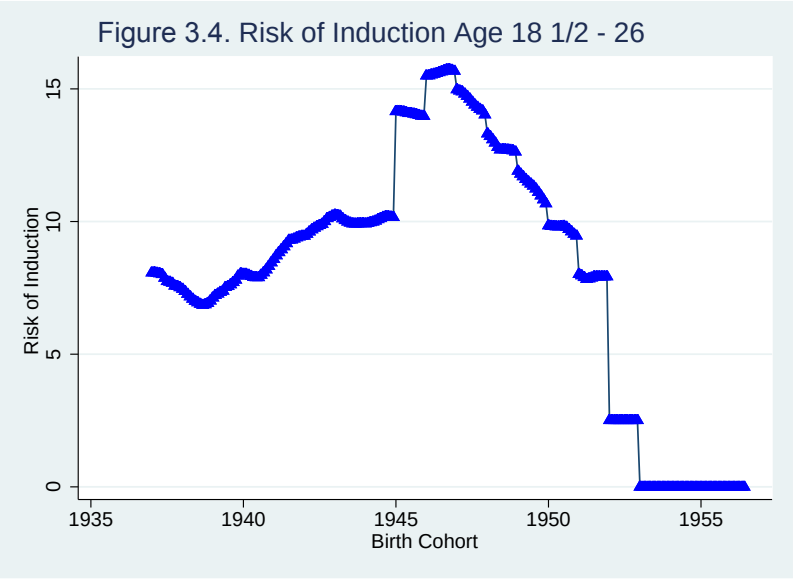


Figure 3.4. Risk of Induction Age 18 1/2 - 26
Risk of Induction
0
5
10
15
1935
1940
1945
1950
1955
Birth Cohort

Table 3.1: Lexis Chart Showing Birth Cohorts and the Ages and Years at which these Cohorts were at Risk of Being Inducted into the US Armed Forces.

	Years Induction Age Eligible																		Age At Survey
Cohort	55	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	1982-1996
1937	18.5	19	20	21	22	23	24	25	26										44-59
1938		18.5	19	20	21	22	23	24	25	26									43-58
1939			18.5	19	20	21	22	23	24	25	26								42-57
1940				18.5	19	20	21	22	23	24	25	26							41-56
1941					18.5	19	20	21	22	23	24	25	26						40-55
1942						18.5	19	20	21	22	23	24	25	26					39-54
1943							18.5	19	20	21	22	23	24	25	26				38-53
1944								18.5	19	20	21	22	23	24	25	*26*			37-52
1945									18.5	19	20	21	22	23	24	*25*			36-51
1946										18.5	19	20	21	22	23	*24*			35-50
1947											18.5	19	20	21	22	*23*			34-49
1948												18.5	19	20	21	*22*			33-48
1949													18.5	19	20	*21*			32-47
1950														18.5	19	*20*			31-46
1951															18.5		*20*		30-45
1952																		*20*	29-44
1953																			28-43
1954																			27-42
1955																			26-41
1956																			25-40

Source: Ages at which the birth cohorts were interviewed in the National Health Interview Survey are displayed in the right-most column. Ages in *italics* denote the draft lottery system was in place at this point in time (1970-1972).

Table 3.2: Summary Statistics

		Male		
		Vietnam Veteran?		
	Men	No	Yes	Women
Induction Risk	7.689	6.873	10.26	7.696
	(5.165)	(5.115)	(4.423)	(5.163)
Body Mass Index (BMI)	26.48	26.42	26.68	25.32
	(4.420)	(4.484)	(4.203)	(5.685)
Obese (BMI>30)	0.190	0.188	0.195	0.185
	(0.392)	(0.391)	(0.396)	(0.388)
Health (1-5)	4.017	4.005	4.053	3.876
	(1.032)	(1.040)	(1.005)	(1.053)
Fair or Poor (Health<3)	0.0827	0.0857	0.0732	0.100
	(0.275)	(0.280)	(0.260)	(0.301)
Very Good (Health>3)	0.708	0.703	0.726	0.649
	(0.454)	(0.457)	(0.446)	(0.477)
Currently Smoke	0.339	0.325	0.384	0.277
	(0.474)	(0.468)	(0.487)	(0.447)
Ever Smoked	0.646	0.621	0.724	0.477
	(0.478)	(0.485)	(0.447)	(0.500)
Black	0.0907	0.0937	0.0812	0.108
	(0.287)	(0.291)	(0.273)	(0.310)
Other Race	0.0355	0.0419	0.0155	0.0374
	(0.185)	(0.200)	(0.123)	(0.190)
Observations	165161	125521	39640	176260

Table 3.2: (Continued)

		Male		
		Vietnam Veteran?		
	Men	No	Yes	Women
Hispanic	0.0697	0.0784	0.0423	0.0724
	(0.255)	(0.269)	(0.201)	(0.259)
Served in Vietnam	0.241	0	1	0.00603
	(0.428)	(0)	(0)	(0.0774)
Ever Served	0.346	0.138	1	0.0127
	(0.476)	(0.345)	(0)	(0.112)
Family Size	3.293	3.296	3.281	3.294
	(1.537)	(1.559)	(1.464)	(1.502)
Married	0.804	0.801	0.815	0.738
	(0.397)	(0.399)	(0.388)	(0.440)
Log Income	10.53	10.51	10.59	10.45
	(0.637)	(0.654)	(0.579)	(0.702)
Education, Years	1.871	1.904	1.767	1.497
Past High School	(2.184)	(2.227)	(2.042)	(2.003)
Educational Attainment	13.38	13.32	13.59	13.03
	(2.986)	(3.168)	(2.306)	(2.751)
Observations	165161	125521	39640	176260

Source: Author's calculation based on National Health Interview Survey data.

The table presents means with standard deviations in parentheses.

Table 3.3: **The Effect of Induction Risk on Health for Men Who Did Not Serve in Vietnam Born between 1937 and 1956: OLS Clustered Results Age 18-26 Risk**

	BMI	Obese	Self-Assessed Health	Health FairPoor	Health VeryGood
Induction Risk	0.0031	0.0004**	–0.0012	0.0007***	–0.0005
	(0.0032)	(0.0002)	(0.0010)	(0.0002)	(0.0003)
trend	0.0379	0.0054**	0.0090**	–0.0010	0.0046**
	(0.0316)	(0.0023)	(0.0039)	(0.0015)	(0.0018)
Black	–0.2060***	–0.0056*	–0.4168***	0.0695***	–0.1675***
	(0.0435)	(0.0029)	(0.0185)	(0.0029)	(0.0043)
Other Race	–2.5488***	–0.1469***	–0.1084***	0.0115***	–0.0592***
	(0.0615)	(0.0065)	(0.0130)	(0.0033)	(0.0065)
Hispanic	0.1083	0.0008	–0.2822***	0.0462***	–0.1268***
	(0.0659)	(0.0051)	(0.0131)	(0.0033)	(0.0052)
Obs	145044	145044	146680	146680	146680
R^2	0.041		0.047		
Dep var mean	26.408	0.187	3.976	0.091	0.691

Source: Authors' estimation of equations (4) and (5) in the text using 25-59 year old men who did not serve in the military from the 1982-1996 NHIS. Each column in the table comes from a separate regression . Estimates for BMI and health are from OLS models while estimates for obese, fair or poor, and very good or excellent health are from probit models and coefficients represent marginal effects. The dependent variable in all equations is the risk of being inducted into the army between the ages 18 1/2 and 26 based on equation 3 in the text. All estimates include age and year of interview fixed effects. Standard errors clustered at the birth cohort level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 3.4: **The Effect of Induction Risk on Health for Men Who Did Not Serve in Vietnam Born between 1937 and 1956: OLS Clustered Results Age 18-26 Risk**

	BMI	Obese	Self-Assessed Health	Health FairPoor	Health VeryGood
Induction Risk	0.0040	0.0005**	–0.0013	0.0007***	–0.0005*
	(0.0030)	(0.0002)	(0.0009)	(0.0002)	(0.0003)
trend	0.0411	0.0055**	0.0099**	–0.0011	0.0048***
	(0.0312)	(0.0023)	(0.0038)	(0.0014)	(0.0017)
Black	–0.1213**	–0.0037	–0.3680***	0.0595***	–0.1484***
	(0.0436)	(0.0029)	(0.0183)	(0.0029)	(0.0042)
Other Race	–2.6244***	–0.1525***	–0.0933***	0.0092***	–0.0532***
	(0.0644)	(0.0066)	(0.0132)	(0.0031)	(0.0064)
Hispanic	0.0267	–0.0057	–0.2545***	0.0420***	–0.1160***
	(0.0655)	(0.0052)	(0.0127)	(0.0032)	(0.0050)
Family Size	0.0949***	0.0080***	–0.0438***	0.0068***	–0.0169***
	(0.0097)	(0.0007)	(0.0029)	(0.0005)	(0.0016)
Married	0.6859***	0.0212***	0.3167***	–0.0598***	0.1216***
	(0.0412)	(0.0037)	(0.0080)	(0.0026)	(0.0037)
Obs	145044	145044	146680	146680	146680
R^2	0.047		0.058		
Dep var mean	26.408	0.187	3.976	0.091	0.691

Source: Authors' estimation of equations (4) and (5) in the text using 25-59 year old men who did not serve in the military from the 1982-1996 NHIS. Each column in the table comes from a separate regression . Estimates for BMI and health are from OLS models while estimates for obese, fair or poor, and very good or excellent health are from probit models and coefficients represent marginal effects. The dependent variable in all equations is the risk of being inducted into the army between the ages 18 1/2 and 26 based on equation 3 in the text. All estimates include age and year of interview fixed effects. Standard errors clustered at the birth cohort level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 3.5: **The Effect of Induction Risk on Health for Men Who Did Not Serve in Vietnam Born between 1937 and 1956: OLS Clustered Results Age 18-26 Risk**

	BMI	Obese	Self-Assessed Health	Health Fair/Poor	Health Very Good
Induction Risk	0.0166***	0.0015***	–0.0048***	0.0010***	–0.0020***
	(0.0033)	(0.0003)	(0.0007)	(0.0002)	(0.0002)
trend	0.0446	0.0066***	0.0098**	–0.0015	0.0042**
	(0.0324)	(0.0024)	(0.0043)	(0.0013)	(0.0020)
Black	–0.1307**	–0.0147***	–0.1531***	0.0167***	–0.0738***
	(0.0513)	(0.0040)	(0.0140)	(0.0030)	(0.0051)
Other Race	–2.3894***	–0.1422***	–0.0926***	0.0049	–0.0570***
	(0.0664)	(0.0071)	(0.0154)	(0.0038)	(0.0065)
Hispanic	–0.0108	–0.0187***	–0.0345**	–0.0001	–0.0351***
	(0.0754)	(0.0063)	(0.0148)	(0.0034)	(0.0068)
Family Size	0.0711***	0.0060***	–0.0322***	0.0059***	–0.0122***
	(0.0094)	(0.0006)	(0.0030)	(0.0004)	(0.0015)
Married	0.5922***	0.0243***	0.1268***	–0.0200***	0.0535***
	(0.0490)	(0.0041)	(0.0162)	(0.0039)	(0.0060)
Log Income	0.2932***	–0.0023	0.3867***	–0.0674***	0.1222***
	(0.0298)	(0.0026)	(0.0226)	(0.0021)	(0.0043)
Education, Years	–0.2047***	–0.0164***	0.0775***	–0.0139***	0.0333***
Past High School	(0.0096)	(0.0008)	(0.0015)	(0.0006)	(0.0007)
Obs	125109	125109	125824	125824	125824
R^2	0.056		0.157		
Dep var mean	26.413	0.187	3.997	0.087	0.700

Source: Authors' estimation of equations (4) and (5) in the text using 25-59 year old men who did not serve in the military from the 1982-1996 NHIS. Each column in the table comes from a separate regression . Estimates for BMI and health are from OLS models while estimates for obese, fair or poor, and very good or excellent health are from probit models and coefficients represent marginal effects. The dependent variable in all equations is the risk of being inducted into the army between the ages 18 1/2 and 26 based on equation 3 in the text. All estimates include age and year of interview fixed effects. Standard errors clustered at the birth cohort level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 3.6: **The Effect of Induction Risk on Health for Male Vietnam Veterans and Women: OLS Clustered Results Age 18-26 Risk**

Sensitivity Sample	BMI	Obese	Self-Assessed Health	Health Fair/Poor	Health Very Good
Panel A: Male Vietnam Veterans					
Specification 1	0.0022	–0.0003	0.0057***	–0.0014***	0.0026***
	(0.0071)	(0.0006)	(0.0014)	(0.0002)	(0.0004)
Specification 2	0.0007	–0.0004	0.0053***	–0.0014***	0.0024***
	(0.0070)	(0.0006)	(0.0014)	(0.0002)	(0.0004)
Specification 3	0.0042	0.0001	0.0020	–0.0007***	0.0012***
	(0.0063)	(0.0005)	(0.0013)	(0.0002)	(0.0005)
Panel B: Women					
Specification 1	0.0001	–0.0002	0.0026***	–0.0007***	0.0010***
	(0.0048)	(0.0003)	(0.0007)	(0.0002)	(0.0003)
Specification 2	0.0010	–0.0002	0.0025***	–0.0007***	0.0009***
	(0.0048)	(0.0003)	(0.0007)	(0.0002)	(0.0003)
Specification 3	0.0058	0.0000	0.0004	–0.0003*	–0.0000
	(0.0047)	(0.0003)	(0.0006)	(0.0001)	(0.0003)

Source: Authors' estimation of equations (4) and (5) in the text using 25-59 year old men who served in the military in Panel A, and women in Panel B from the 1982-1996 NHIS. Each cell in the table comes from a separate regression. Estimates for BMI and health are from OLS models while estimates for obese, fair or poor, and very good or excellent health are from probit models and coefficients represent marginal effects. The dependent variable in all equations is the risk of being inducted into the army between the ages 18 1/2 and 26 based on equation 3 in the text. All estimates include controls for race and ethnicity, time trend, and age and year of interview fixed effects. Specification 2 also includes controls for family size and marital status, while Specification 3 includes log family income and educational attainment controls as well. Standard errors clustered at the birth cohort level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 3.A1: **The Effect of Induction Risk on Health for All Men: OLS Clustered Results Age 18-26 Risk**

Sensitivity Sample	BMI	Obese	Self-Assessed Health	Health Fair/Poor	Health Very Good
Panel A: Main Results, Non-Vietnam Veterans 1937-1955					
Specification 1	0.0031	0.0004**	–0.0012	0.0007***	–0.0005
	(0.0032)	(0.0002)	(0.0010)	(0.0002)	(0.0003)
Specification 2	0.0040	0.0005**	–0.0013	0.0007***	–0.0005*
	(0.0030)	(0.0002)	(0.0009)	(0.0002)	(0.0003)
Specification 3	0.0166***	0.0015***	–0.0048***	0.0010***	–0.0020***
	(0.0033)	(0.0003)	(0.0007)	(0.0002)	(0.0002)
Panel B: Naive Regressions of All Men, 1937-1955					
Specification 1	0.0062**	0.0003	0.0008	0.0000	0.0005**
	(0.0025)	(0.0002)	(0.0006)	(0.0001)	(0.0002)
Specification 2	0.0066**	0.0004	0.0005	0.0001	0.0004*
	(0.0024)	(0.0002)	(0.0005)	(0.0001)	(0.0002)
Specification 3	0.0141***	0.0010***	–0.0020***	0.0005***	–0.0008***
	(0.0025)	(0.0002)	(0.0006)	(0.0001)	(0.0002)

Source: Authors' estimation of equations (4) and (5) in the text using 25-59 year old men who served in the military in Panel A, and women in Panel B from the 1982-1996 NHIS. Each cell in the table comes from a separate regression. Estimates for BMI and health are from OLS models while estimates for obese, fair or poor, and very good or excellent health are from probit models and coefficients represent marginal effects. The dependent variable in all equations is the risk of being inducted into the army between the ages 18 1/2 and 26 based on equation 3 in the text. All estimates include controls for race and ethnicity, time trend, and age and year of interview fixed effects. Specification 2 also includes controls for family size and marital status, while Specification 3 includes log family income and educational attainment controls as well. Standard errors clustered at the birth cohort level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 3.A2: **The Effect of Induction Risk on Health for Men Who Did Not Serve in Vietnam: Comparing Sensitivity Analyses to Main Results, OLS Clustered Results Age 18-26 Risk**

	BMI	Obese	Self-Assessed Health	Health Fair/Poor	Health Very Good
Panel A: Main Results, 1937-1955					
Specification 2	0.0040	0.0005**	–0.0013	0.0007***	–0.0005*
	(0.0030)	(0.0002)	(0.0009)	(0.0002)	(0.0003)
Specification 3	0.0166***	0.0015***	–0.0048***	0.0010***	–0.0020***
	(0.0033)	(0.0003)	(0.0007)	(0.0002)	(0.0002)
Panel B: Non Veterans, 1937-1955					
Specification 2	0.0057	0.0006**	–0.0003	0.0004*	–0.0001
	(0.0033)	(0.0003)	(0.0010)	(0.0002)	(0.0003)
Specification 3	0.0169***	0.0015***	–0.0038***	0.0008***	–0.0016***
	(0.0036)	(0.0003)	(0.0008)	(0.0002)	(0.0002)
Panel C: No Proxy Responses, 1937-1955					
Specification 2	0.0004	0.0001	–0.0007	0.0007**	–0.0003
	(0.0047)	(0.0003)	(0.0012)	(0.0003)	(0.0005)
Specification 3	0.0130**	0.0010***	–0.0049***	0.0011***	–0.0021***
	(0.0056)	(0.0004)	(0.0008)	(0.0002)	(0.0004)
Panel D: All Years, 1930-1955					
Specification 2	0.0033	0.0005*	–0.0004	0.0003	–0.0001
	(0.0034)	(0.0003)	(0.0009)	(0.0002)	(0.0003)
Specification 3	0.0120***	0.0012***	–0.0032***	0.0005**	–0.0013***
	(0.0042)	(0.0003)	(0.0007)	(0.0002)	(0.0003)
Panel E: Korea Only, 1930-1936					
Specification 2	0.0092	0.0017	–0.0105***	0.0033***	–0.0053***
	(0.0170)	(0.0011)	(0.0027)	(0.0009)	(0.0008)
Specification 3	0.0063	0.0006	–0.0077**	0.0009	–0.0050***
	(0.0240)	(0.0017)	(0.0022)	(0.0007)	(0.0017)

Source: Authors' estimation of equations (4) and (5) in the text using 25-59 year old men who did not serve in the military from the 1982-1996 NHIS. Each cell in the table comes from a separate regression. Estimates for BMI and health are from OLS models while estimates for obese, fair or poor, and very good or excellent health are from probit models and coefficients represent marginal effects. The dependent variable in all equations is the risk of being inducted into the army between the ages 18 1/2 and 26 based on equation 3 in the text. All estimates include controls for race and ethnicity, time trend, family size, and marital status and age and year of interview fixed effects. Specification 3 includes log family income and educational attainment controls as well. Standard errors clustered at the birth cohort level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.

Table 3.A3: **The Effect of Induction Risk on Health for Separately for Veteran Men: OLS Clustered Results from Specification 3, Age 18-26 Risk**

Sensitivity Sample	BMI	Obese	Self-Assessed Health	Health Fair/Poor	Health Very Good
Vietnam Veterans,	0.0042	0.0001	0.0020	–0.0007***	0.0012***
1937-1955	(0.0063)	(0.0005)	(0.0013)	(0.0002)	(0.0005)
All Veterans,	0.0085	0.0002	0.0006	–0.0002	0.0007
1937-1955	(0.0053)	(0.0003)	(0.0010)	(0.0002)	(0.0004)
No Proxy Veterans,	0.0050	–0.0003	0.0005	–0.0004	0.0003
1937-1955	(0.0082)	(0.0008)	(0.0019)	(0.0004)	(0.0006)
Veterans,	0.0043	–0.0000	–0.0019	0.0003	–0.0002
1930-1955	(0.0035)	(0.0003)	(0.0011)	(0.0002)	(0.0004)
Korea Veterans,	0.0354**	0.0017*	–0.0051	0.0001	–0.0018
1930-1936	(0.0140)	(0.0010)	(0.0033)	(0.0011)	(0.0013)

Source: Authors' estimation of equations (4) and (5) in the text using 25-59 year old men who served in the military from the 1982-1996 NHIS. Each cell in the table comes from a separate regression. Estimates for BMI and health are from OLS models while estimates for obese, fair or poor, and very good or excellent health are from probit models and coefficients represent marginal effects. The dependent variable in all equations is the risk of being inducted into the army between the ages 18 1/2 and 26 based on equation 3 in the text. All estimates include controls for race and ethnicity, time trend, family size, marital status, log family income, and educational attainment, and age and year of interview fixed effects. Standard errors clustered at the birth cohort level are in parentheses: *** indicates significance at the 1% level, ** indicates significance at the 5% level, and * indicates significance at the 10% level.