Long Run Impacts of Childhood Access to the Safety Net

Hilary W. Hoynes, UC Berkeley and NBER
hoynes@berkeley.edu

Diane Whitmore Schanzenbach, Northwestern University and NBER
dws@northwestern.edu

and

Douglas Almond, Columbia and NBER
da2152@columbia.edu

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Abstract

A growing economics literature establishes a causal link between *in utero* shocks and health and human capital in adulthood. Most studies rely on extreme negative shocks such as famine and pandemics. We are the first to examine the impact of a *positive* and *policy-driven* change in economic resources available *in utero* and during childhood. In particular, we focus on the introduction of a key element of the U.S. safety net, the Food Stamp Program, which was rolled out across counties in the U.S. between 1961 and 1975. We use the Panel Study of Income Dynamics to assemble unique data linking family background and county of residence in early childhood to adult health and economic outcomes. The identification comes from variation across counties and over birth cohorts in availability of the food stamp program. Our findings indicate that the food stamp program has effects decades after initial exposure. Specifically, access to food stamps in childhood leads to a significant reduction in the incidence of "metabolic syndrome" (obesity, high blood pressure, and diabetes) and, for women, an increase in economic self-sufficiency. Overall, our results suggest substantial internal and external benefits of the safety net that have not previously been quantified.

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1. Introduction

There is substantial evidence on the strong intergenerational correlations in health and income. As documented by Case, Lubotsky, and Paxson (2002), health and economic disparities unfold early in life. There is less evidence, however, on causal mechanisms behind this gap. The "early origins" literature offers some guidance toward the causal relationships underlying intergenerational correlations. However, whether public policy may be able to benefit by accessing these "early origins" linkages remains to be been established.

In this paper, we evaluate whether increasing resources available *in utero* and during childhood improves later-life health and economic outcomes. In particular, we focus on the introduction of a key element of the U.S. safety net, the Food Stamp Program (FSP). The FSP was rolled out at a county by county basis between 1962 and 1975, providing low-income families vouchers that could be used at grocery stores to purchase food. Economic theory and prior empirical evidence suggest that these vouchers were treated the same as cash income (Hoynes and Schanzenbach 2009). Thus, we can utilize the FSP rollout as an identification strategy for increases in resource availability early in life. Our analysis builds on previous research finding a positive "first stage" effect of FSP rollout on contemporaneous health, as measured in natality data by birth weight (Almond, Hoynes, and Schanzenbach 2011, Currie and Moretti 2008). The program rollout design links the paper to a growing literature evaluating the introduction of other Great Society programs.[1]

Our analysis also makes two important contributions to the economics literature that relates resources *in utero* and during childhood to economic and health outcomes in adulthood.

[1] Other rollout studies examine Head Start (Ludwig and Miller 2007), Medicare (Almond, Chay and Greenstone, 2007, Finkelstein and McKnight 2008), WIC (Hoynes et al 2012), family planning programs (Bailey 2012), Title I (Cascio et al. 2010), and community health centers (Bailey and Goodman-Bacon 2012).

First, previous design-based observational studies have generally looked at *extreme, negative* events including famines, natural disaster, or disease outbreaks (see Currie 2009, Almond and Currie 2011a, b for recent reviews). Second, our treatment extends beyond in utero exposure; we can test whether the impacts of exposure to Food Stamps vary throughout childhood. A point of departure in our analysis is to evaluate the impact of a *positive* and *policy-driven* event; one that is manifested by an increase in family resources available in utero and during childhood. This income variation may be relevant for a larger population than those touched by extreme events. Nevertheless, it is a relatively open question whether a moderate increase in resources over an extended period of time exerts corresponding long-term effects, or whether these linkages are restricted to extreme (and typically short duration) events.

The Food Stamp program, recently renamed Supplemental Assistance for Needy Families or SNAP, is the fundamental safety net in the U.S. Importantly, it is the only public assistance program that is available to all income eligible families (other programs limit eligibility to particular groups such as female headed households, children, the disabled or the elderly). It is currently the largest U.S. cash or near cash means tested transfer program with spending in 2011 of 70 billion dollars compared to 31 billion for TANF and 59 billion for the federal EITC.[2] The importance of the FSP program is particularly apparent in the current Great Recession, where almost 1 in 7 persons received benefits and lifted more than 5 million families out of poverty[3]. Interestingly, in contrast to the rest of the U.S. safety net, the FSP is a federal program and exhibits little variation across states (Currie 2003). It also has remained largely intact in the

[2] Food stamp participation has increased to historic highs during the Great Recession. In 2007, food stamps, at 31 billion, compared to 48 billion for the EITC and 27 billion for TANF. Program costs are found at U.S. Department of Health and Human Services (2012), Internal Revenue Service (2012), and U.S. Department of Agriculture (2012).
[3] The food stamp participation data refer to 2011 (USDA 2012). The poverty data are based on the supplemental poverty measure (Short 2011) and are for 2010. The anti-poverty effects of the FSP were eclipsed only by the Earned Income Tax Credit, which lifted more than 6 million families out of poverty.

presence of dramatic reforms to other parts of the safety net (Bitler and Hoynes 2010). This lack of variation across states and over time presents significant challenges for evaluating the impacts of the FSP (Currie 2003).

Our main results are for a sample of adults born between 1956 and 1981 who grew up in disadvantaged families (their parent had less than a high school education). We refer to this as the "high-participation sample." We employ a difference-in-difference model where the treatment varies by county of birth and birth cohort, and we include controls for county and year of birth and interview fixed effects, state linear time trends, and county-year of birth controls. We also estimate a triple difference model that extends beyond the low education sample and uses variation across subgroups with varying propensities of being affected by food stamps (Hoynes and Schanzenbach 2009). Our main treatment variable is the share of time between conception and age 5 that a food stamp program was available in the individual's county of birth. We estimate impacts on weight, height, general health status, disability, the incidence of many conditions and diseases (e.g. high blood pressure, diabetes, heart disease, etc.), health behaviors (smoking, drinking), as well as education, earnings, income, and program participation. Because of the many outcome variables, we follow Kling, Liebman and Katz (2007) and Anderson (2008) and estimate summary standardized indices that aggregate information over multiple treatments.

We find that access to the FSP in utero and in early childhood leads to a large and statistically significant reduction in the incidence of "metabolic syndrome" (obesity, high blood pressure, heart disease, diabetes) as well as an increase in reporting to be in good health. We also find for women, but not men, that access to food stamps in early childhood leads to an increase in economic self-sufficiency. Our results are robust to adding a rich set of county controls (possible confounders) and event study models further support the validity of the research

design. Further, we find important results concerning the timing of exposure -- the beneficial health impacts of Food Stamps are concentrated in exposure in the in utero through early childhood period while the effects on economic self-sufficiency relate to exposure in the school years.

Our results not only make a contribution by establishing a link between expanding resources in childhood and long run health outcomes, but we also can speak to the "program evaluation" of FSP. Presumably, the FSP passes a cost-benefit criterion based on the contemporaneous or "real time" benefits (nutrition, anti-poverty). Given that empirical evidence on "early origins" is relatively recent, the long-term benefits of such longstanding programs were neither considered nor necessarily expected. An additional contribution of our work is, therefore, to explore the possibility of internal and external benefits of the safety net that have not previously been quantified. This raises the possibility that policies targeting early childhood may be sub-optimally small. In other words, the FSP generates larger private and social benefits when taking into account the "multiplier effect" on later-life outcomes.

The remainder of our paper is as follows. In Section 2 we summarize the economic literature on long term effects of early life interventions. In Section 3 we summarize the biological science literature in order to provide guidance for which health impacts are expected to be impacted by the FSP treatment. In Section 4 we provide the background on the FSP and in Section 5 we describe our data. In Section 6 we present our empirical model and Section 7 our results. We conclude in Section 8.

2. Background and Prior Research

That *in utero* and early childhood events can have important vestigial effects has been documented for a wide range of later-life outcomes, including health status, test scores, education attainment, wages, and mortality. Prior studies have leveraged short, extreme events experienced *in utero* as identification strategies (see reviews by Currie 2009 and Almond and Currie 2011a,b). Examples include famine (Painter et al. 2005, Susser and Lin 1992), disease (Almond 2006, Barreca 2010), and radiation (Almond et al. 2009). A natural question is how generalizable such linkages are, and in particular whether more routine childhood experiences may also shape health and economic outcomes during adulthood. To date, there has been relatively little research that combines the strength of design-based identification strategies for causal inference with more commonplace treatments/exposures. Those that are amenable to policy are rarer still.

The shortage of previous work stems from the challenge of marrying identification strategies for program evaluation to: a) policies that affect children at a young age and b) policies that can be mapped to data on later-life outcomes in adulthood. Thus, whether "early origins" are more of an empirical curiosity or a general policy avenue remains largely unknown.

A relatively large literature considers the effect of income on health. Much of this literature is concerned with the short-term effects of income changes experienced in adulthood, and there are few studies that considering the long-term effect of *early-life* income changes. Van Den Berg, Lindeboom and Portrait (2006) compare Dutch mortality rates among those born during economic downturns to those born during expansions. Those born during expansions lived substantially longer, which they argue is not due to changes in cohort composition or other potential confounders. Banerjee et al. (2010) consider the a 19th century blight of French

vineyards that created a region by birth cohort varying shock to production and hence income. They find this led to shorter heights in adulthood but no impact on mortality.

A second strand of research has considered the long-term effect of specific, well-identified programs. Among these studies, the closest is Ludwig and Miller (2007), who consider the long-term effect of Head Start program. Their approach uses application assistance provided to poor counties as an instrument for Head Start Program operation, and finds the program reduced childhood mortality rates in affected counties. Furthermore, Ludwig and Miller find that educational attainment is also higher due to Head Start exposure. Chetty et al. (2011) and Dynarski, Hyman and Schanzenbach (2011) find persistent effects of class-size reductions on educational attainment and initial labor market outcomes. Reyes (2007) and Nilsson (2009) consider the effect of early childhood lead exposure on later life outcomes, and Glied and Neidell (2010) evaluate the long-term impacts of water fluoridation.[4] Chay, Guryan, and Mazumder (2012) consider the long-term human capital effect of early childhood health, as proxied by local post-neonatal mortality rates. They find that improved health for young black children during the late 1960s and early 1970s yielded substantially increased NAEP and AFQT test scores during the 1980s. Building on Almond, Chay, and Greenstone (2007), they argue that improved access to hospitals following the Civil Rights Act and Medicare-induced desegregation of hospitals drove the reduction in post-neonatal mortality rates for blacks that subsequently yielded higher test scores. Likewise, more specific medical interventions in the early postnatal period have been found to exert long-term effects, including surfactant and related treatments for very low birth

[4] Duncan, Ziol-Guest, and Kalil (2010) use the PSID to examine the impact of income during childhood on later life economic and health outcomes. Importantly, they explore how income during different stages of childhood (early life, later childhood, etc.) affects outcomes and they explore income's nonlinear impacts. This is an observational design, however, and it is unclear whether they have recovered causal income channels.

weight infants (Bharadwaj, Loken, and Nielson 2012) and breastfeeding-encouragement by hospital nurses following delivery (Fritzsimmons and Vera-Hernandez 2012).

3. Impacts of Early Nutrition and Expected Effects of the FSP

Causal mechanisms by which early childhood events affect later-life are best understood for nutrition. This section reviews specific mechanisms by which early malnutrition can impair development with long-term consequences.

Although FSP was clearly a nutrition program, because most recipients received a Food Stamp benefit below their normal food expenditures, the program is better understood as an income transfer (Hoynes and Schanzenbach, 2009). However, because recipients were by definition poor, a large portion of their FSP benefit was spent on food. Further, at the time FSP was introduced, hunger and nutritional deficiencies were not uncommon among Americans. For example, a survey of low-income families in Texas, Louisiana, Kentucky, and West Virginia in 1968-1970 found that 15 percent of whites and 37 percent of blacks had low hemoglobin levels as well as relatively high rates of deficiencies in vitamin C, riboflavin and protein (Eisinger 1998). The 1968 CBS documentary "Hunger in America" raised national awareness of the problem and possibly influenced the policy debate on the FSP (Berry 1984). Here, by briefly summarizing the linkages between early life nutrition and later life outcomes, we provide predictions about outcomes that may be altered by our FSP treatment.

Some linkages between early life nutrition and later life outcomes are fairly intuitive. For example, severely undernourished children may suffer from anemia and listlessness. This may reduce their ability to invest in learning during childhood and may harm their long-run earnings and other outcomes. Poor early life nutrition may also directly harm long-run outcomes through

altering the body's developmental trajectory. There is an emerging scientific consensus that describes critical periods of development during early life that "program" the body's long-term survival outcomes (Barker, 1992; Gluckman and Hanson 2004). During development, the fetus (and post-natally the child) may take cues from the current environment to predict the type of environment it is expected to face in the long run and in some cases adapts its formation to better thrive in the expected environment (Gluckman and Hanson 2004). A problem arises, however, when the predicted later environment and the actual later environment are substantially different. For example, if nutrients are scarce during the pre-natal (or early post-natal) period, the developing body therefore predicts that the future will also be nutritionally deprived. The body may then invoke (difficult-to-reverse) biological mechanisms to adapt to the predicted future environment. For example, the metabolic system may adapt in a manner that will allow the individual to survive in an environment with chronic food shortages. This pattern is termed the "thrifty phenotype" and is sometimes referred to as the Barker hypothesis. The "problem" arises if in fact there is not a long-run food shortage, and nutrition is plentiful. In that case, the early-life metabolic adaptations are a bad match to the actual environment and will increase the likelihood that the individual develops a metabolic disorder, which can include high blood pressure (hypertension), type II diabetes, obesity and cardiovascular disease. The negative consequences do not usually appear until after reproductive age, which is preferable to the species from an evolutionary perspective (Barker 1992). Note that both pre- and post-natal nutrition can drive this programming.

Lasting impacts of nutrient deprivation in both the pre- and post-natal period have been found on both long-term health and economic outcomes. Much of the experimental work on nutritional programming has been conducted on rats. In McCance (1962), the researchers

experimentally manipulated how much breast milk was available to baby rats during their normal 21-day suckling period. At the end of the experiment, the treatment group (who were fed less than normal) were smaller than the control group (who were fed normally). Subsequently, both groups were fed normally, and the treatment group quickly caught up to normal size. In the longer-run, however, they found that the (former) treatment rats became more obese than the control group even though they were fed the same, normal amount. This set off a number of follow-up studies. In one, the researchers found that if they manipulated the food intake for a different (later) 21-day period, there were no long-run effects. This suggests there is a "critical period" in the early post-natal period during which programming continues to occur.[5]

Some of the strongest research on humans comes from studies of the Dutch Hunger Winter. In World War II, the Nazis imposed strict rationing of food during their occupation of the Netherlands over the 7 month period between November 1944 and April 1945. As a result, the previously well-nourished society experienced an abrupt, severe restriction in available calories. Average caloric intake fell almost overnight from about 1800 calories per day to between 400 and 800. Upon liberation, the food supply returned to normal levels almost instantaneously. Because of the abrupt and severe nature of the period of malnutrition, it is possible to isolate the impact of malnutrition that occurred at different points during development. Painter et al. (2005) find that children had lower birth weight if exposed to famine in the third trimester.[6] Further, when the cohort that was exposed to malnutrition *in utero* hit middle age, they were more likely to be obese, and had higher incidence of heart disease, lower self-reported health status, and worse mental health (Painter et al. 2005, Susser and Lin 1992).

[5] Findings from animal models may need to be interpreted with additional caution if the timing of birth occurs at different stages of development.

[6] In a parallel manner, our earlier work (Almond, Hoynes and Schanzenbach 2011) finds that children have higher birth weight if exposed to food stamps in the third trimester.

Almond and Mazumder (2011) consider the effect of nutrition timing during pregnancy on later-life outcomes, focusing on the Ramadan fast as an identification strategy. They find that children born with in utero exposure to the Ramadan fast experienced large increases in disability in adulthood. Because fasting during Ramadan is confined to daylight hours, the nutritional treatment is relatively mild compared to famine episodes. "Net nutrition" can also be compromised by infectious disease (Bozzoli, Deaton, and Quintana-Domeque 2009), which can increase energy expenditure and reduce consumption and absorption of nutrients. Thus, previous well-identified findings on the long-term effect of the postnatal health environment (e.g. Chay, Guryan, and Mazumder 2012) may also include nutrition as a portion of the underlying mechanism.

To summarize, the literature has found that lack of nutrition in early life leads to higher incidence of metabolic syndrome. These impacts have occurred both when the nutritional shock occurred *in utero* and when it occurred in the period shortly after birth. Importantly, the long-run health outcomes have been found even in cases in which birth weight itself was not affected. Thus the impacts of in utero shocks do not necessarily directly map into birth weight and evaluating the long term impacts requires the analysis of a many-decade past change, such as we do in this paper.

In our setting, we examine a policy-driven increase in resources to a population that had previously experienced chronic low levels of nutrition. Therefore, we expect that individuals who were exposed to the FSP program in early life will be less likely to have mis-adapted to the future environment. As a result, we expect to find that these cohorts experience lower incidence of metabolic syndrome – as measured by high blood pressure, obesity, and diabetes – in adult life. We also expect to find better human capital outcomes, as measured by education, earnings,

income and the like. Because the literature is unclear about the exact timing of post-natal damage, we will explore alternative specifications for the timing of FSP exposure.

4. Introduction of the Food Stamp Program

Today, food stamp benefits are the fundamental safety net in the U.S., being the only public assistance program that is available to all family types (most programs are targeted on female headed households, children, or the elderly). Eligibility requires satisfying income and asset tests and benefits can be used to purchase most grocery store food goods. A family's benefit is equal to the difference between the federally defined maximum benefit level for a given family size and the amount that the family is deemed to be able to afford to pay for food on its own according to the benefits formula (essentially 30 percent of cash income, less some deductions).

The roots of today's Food Stamp Program began with President Kennedy's 1961 announcement of a pilot food stamp program that was to be established in eight impoverished counties. The pilot programs were later expanded to 43 counties in 1962 and 1963. The success of these pilot programs led to the Food Stamp Act of 1964, which gave local areas the authority to start up the FSP in their county. As with the current FSP, the program was federally funded and benefits were redeemable at approved retail food stores. In the period following the passage of the Food Stamp Act, there was a steady stream of counties initiating Food Stamp Programs and Federal spending on the FSP more than doubled between 1967 and 1969 (from $115 million to $250 million). Support for a national FSP grew due to a public spotlight on hunger (Berry 1984). This interest culminated in passage of 1973 Amendments to the Food Stamp Act, which mandated that all counties offer FSP by 1975.

Figure 1 plots the percent of counties with a FSP from 1960 to 1975.[7] During the pilot phase (1961-1964), FSP coverage increased slowly. Beginning in 1964, program growth accelerated; coverage expanded at a steady pace until all counties were covered in 1974. Furthermore, there was substantial heterogeneity in timing of adoption of the FSP, both within and across states. The map in Figure 2 shades counties according to date of FSP adoption (darker shading denotes a later start up date). Our basic identification strategy considers the month of FSP adoption for each county to trigger the beginning of the FSP "treatment."

For our identification strategy to yield causal estimates of the program, it is important to establish that the timing of FSP adoption appears to be exogenous. Prior to the FSP, some counties provided food aid through the commodity distribution program (CDP)—which took surplus food purchased by the Federal government as part of an agricultural price support policy and distributed those goods to the poor. The 1964 Food Stamp Act allowed for counties to voluntarily set up a FSP, but the Act also stated that no county could run both the FSP and the CDP. Thus, for counties which previously ran a CDP, adoption of the FSP implies termination of the CDP.[8] The political accounts of the time suggest that debates about adopting the FSP pitted powerful agricultural interests (who favored the CDP) against advocates for the poor (who favored the FSP, see MacDonald 1977; Berry 1984). In particular, counties with strong support for farming interests (e.g., southern or rural counties) may be late adopters of the FSP. On the other hand, counties with strong support for the low-income population (e.g., northern, urban

[7] Counties are weighted by their 1970 population. Note this is not the food stamp caseload, but represents the percent of the U.S. population that lived in a county with a FSP.

[8] This transition in nutritional assistance would tend to bias downward FSP impact estimates, but we do not think this bias is substantial because of the limited scope of the CDP. The CDP was not available in all counties and recipients often had to travel long distances to pick up the items. Further, the commodities were distributed infrequently and inconsistently, and provided a very narrow set of commodities—the most frequently available were flour, cornmeal, rice, dried milk, peanut butter and rolled wheat (Citizens' Board of Inquiry 1968). In contrast, Food Stamp benefits can be used to purchase a wide range of grocery food items.

counties with large poor populations) may adopt FSP earlier in the period. This systematic variation in food stamp adoption could lead to spurious estimates of the program impact if those same county characteristics are associated with differential trends in the outcome variables.

In earlier work (Hoynes and Schanzenbach 2009), we documented that larger counties with a greater fraction of the population that was urban, black, or low income indeed implemented the FSP earlier (i.e. consistent with the historical accounts).[9] Nevertheless, we found that the county characteristics explain very little of the variation in adoption dates. This is consistent with the characterization of funding limits controlling the movement of counties off the waiting list to start up their FSP (Berry 1984). We view the weakness of this model fit as a strength when it comes to our identification approach in that much of the variation in the implementation of FSP appears to be idiosyncratic. Nonetheless, in order to control for possible differences in trends across counties that are spuriously correlated with the county treatment effect, all of our regressions include interactions of these 1960 pre-treatment county characteristics with time trends as in Acemoglu et al. (2004) and Hoynes and Schanzenbach (2009).

5. Data

Given the county rollout of the FSP, our analysis requires data with information on adult health and economic outcomes as well as county of birth for cohorts that were impacted by the FSP introduction (1963-1975) at birth or during early childhood. We use the Panel Study of Income Dynamics (PSID) which began in 1968 with a sample of approximately 5,000 households, and subsequently followed and interviewed all members and descendants. The

[9] For more detail, see Table 1 in Hoynes and Schanzenbach (2009).

original sample comprises two subsamples: a nationally representative sample of 3,000 households and the "Survey of Economic Opportunity subsample" including 1,900 low-income and minority households selected from an existing sample. To adjust for this nonrandom composition, we conduct all analysis using the PSID weights.

Since the beginning of the survey, the PSID has collected detailed information on economic and demographic outcomes. We use those data to generate adult economic outcomes such as educational attainment, employment, earnings, family income, and poverty. Starting in the 1980s and 1990s, the PSID also began regularly collecting information on health outcomes. We use self-reported general health status (reported on a 5 point scale: excellent, very good, etc.) and disability (physical or nervous condition that limits the type or amount of work), both of which have been asked of heads and wives each year beginning in 1984. In addition, the PSID reports height and weight (hence we can construct obesity[10] and height stunting[11]), and information on whether a doctor has diagnosed the respondent with specific health conditions such as diabetes, high blood pressure, heart attack and heart disease.[12] These health data are collected for all heads and wives and have been available since 1999, when the survey became biennial. These data allow us to test for the prediction that the introduction of food stamps in early life leads to a reduction in the incidence of metabolic syndrome. In addition, we use information on health behaviors (smoking and drinking) and health care utilization (hospitalization).

[10] Obesity is defined as having a body mass index (weight in kilograms divided by height in meters squared) of 30 and above.

[11] Height stunting is measured as being below the 5th percentile in the nationally normed height distribution (Lewit and Kerrebrock 1997). We use a gender-specific height distribution for those aged 30-50 in 2003-2006 reported in McDowell et al. (2008).

[12] Other health conditions measured in the PSID include stroke, arthritis, asthma, cancer, psychiatric problems, lung disease, and mental ability.

We use a restricted version of the PSID allowing for identification of county of residence for each year of the survey (the public-use version of the data identifies state). Because of the longitudinal and dynastic nature of the data, for each individual, we can assign their county of residence at birth or, for those born prior to the beginning of PSID data collection, their county of residence in 1968 when their family is first observed in the data. We merge the PSID data to FSP program information based on this county of birth. The key variable for our analysis is constructed from the month and year that each county introduced the FSP, which we collected from USDA annual reports on county FSP caseloads (USDA, various years). With this, and using the month and year of birth (measured for each person in the survey), we construct measures of childhood exposure to the FSP. In our main specification, we use the share of time between conception and age 5 that FSP is available in county of birth.[13]

Our sample includes individuals born between the years 1956 and 1981. Importantly, this yields cohorts that span the entire food stamp rollout period (as well as several cohorts both pre- and post-rollout) to identify the impact on adult outcomes. In addition, we only include individuals whose family is observed at the individual's birth or in early life.[14] This is necessary to identify the individual's county of birth.[15] We also use information on the family background of the individual in early life (whether the child was born into a family is headed by a single woman, education of head, and family income) as control variables and to identify groups more and less likely to participate in the FSP. We limit the sample to persons age 18 and above for the health outcomes and age 25 and over for the economic outcomes (to facilitate completed education). The sample includes one observation for each interview year that the individual

[13] We assume a 9-month gestation, so month of conception is 9 months prior to birth month.
[14] In effect, this limits the sample to persons born into original 1968 PSID families.
[15] Because of the possibility of nonrandom migration, we calculate childhood exposure to the FSP using county of birth, rather than the time varying county of residence.

satisfies these age restrictions, and is a head or wife (recall that the health measures are only asked if a head or wife). We use the PSID data through interview year 2009. Thus given our birth cohorts (1956-1981) the oldest individuals in our sample are 53 at the end of the period. We focus on a "high participation" sample, the sample of adults who grew up in disadvantaged families (their parent had less than a high school education).

We augment the PSID data with additional county variables to control for possible county confounders. First, we use county-level variables from the 1960 Census of Population and Census of Agriculture including: the percent of the 1960 county population that lives in an urban area, is black, is younger than 5, is older than 65, has income less than $3000 (in 1959 dollars), the percent of land in the county used for farming, and log county population. Second, we measure for each county and year the number of hospital beds and hospitals per capita (from the American Hospital Association[16]), real (non-FSP) government transfers per capita (from the REIS[17]), and whether or not the county has a community health center.[18] We use the AHA and REIS data and construct averages for the first five years of life (using county and year of birth). We use the community health center data to measure the share of months between conception and age 5 that there was a community health center present.

[16] The AHA data provides annual data that allows for us to measure county variables for the first five years of life for all birth cohorts (the "Hospitals: Guide Issue" publication goes back to 1948). We collapse the hospital level data to county-year and then convert to per capita measures using historical county population data. We then prepare a simple average over the first five years of life. We thank Amy Finkelstein and Martin Gaynor for the pre-1976 data, Jean Roth of the NBER for the 1976 and on data, and Martha Bailey and Andrew Goodman-Bacon for providing code to clean the data.

[17] The REIS data is available for 1959 and 1962 and then annually beginning in 1965. We construct three measures for real per capita transfers that can be consistently measured throughout this period: cash public assistance benefits (Aid to Families with Dependent Children, Supplemental Security Income and General Assistance), medical spending (Medicare and military health care), and cash retirement and disability payments (OASI, DI, other). We interpolate to fill in the gaps (1960, 1961, 1963, 1964). Analyses with these controls must drop birth cohorts before 1960 due to missing data.

[18] The information on community health centers provide the year that the first center established in a county, which occurred between 1965 and 1974 (Bailey and Goodman-Bacon 2011). We thank Martha Bailey and Andrew Goodman-Bacon for sharing this data.

Table 1 presents descriptive statistics on our estimation sample. About 69 percent of the full sample and 60 percent of the high participation (parent low education) sample report to be in excellent or very good health. About 10 percent report a work disability and less than 5 percent are diabetic. Twelve (17) percent of the full sample (low education subsample) has high blood pressure and 23 percent (31 percent) are obese.

6. Empirical Model

Our basic specification is a difference-in-differences model, where we compare adult outcomes for those with early childhood exposure to FSP in their county of birth to those born earlier (and therefore without childhood FSP exposure). We estimate:

$$y_{icb} = \alpha + \delta FSP_{cb} + X_{icb}\beta + \eta_c + \lambda_b + \gamma_t + \theta_s * b + \varphi CB60_c * b + \varepsilon_{icb} \qquad (1)$$

where *i* indexes the individual, *c* the county of birth, *b* the birth year, *s* the state of birth, and *t* the survey year. *FSP* is a measure of food stamp availability in early life. In our base case models we measure the share of months between conception and age 5 that food stamps is available in the adult's birth county.

Because counties adopted FSP at different times, we compare those with or without FSP access in early childhood by virtue of their county and date of birth. Thus, we can allow for unrestricted cohort effects at the national level λ_b , unrestricted county effects η_c, unrestricted interview year effects γ_t and state specific linear year of birth trends $\theta_s * b$. The parameter of interest is δ, the effect of exposure to FSP, which is identified from variation across counties and birth cohorts. We also control for individual-level covariates X_{icb} (including gender, marital status, race, and a quadratic in age) and family background (whether you were born into a female headed household, the education attainment of the head of household, and the family's income to

needs ratio[19]). All models are estimated using the PSID sample weights and we cluster standard errors by county of birth.

Because of the many outcome variables, we follow Kling, Liebman and Katz (2007) and Anderson (2008) and estimate summary standardized indices that aggregate information over multiple treatments. In particular, we form two indices: metabolic syndrome and economic self-sufficiency. As discussed by Kling et al. (2007) aggregating multiple measures in a given area (e.g. metabolic syndrome) improves statistical power. The summary index is the simple average across standardized z-score measures of each component. The z-score is calculated by subtracting the mean and dividing by the standard deviation.[20] In the case of metabolic syndrome, all components are "bads" (obesity, high blood pressure, diabetes, heart disease, heart attack) and so an increase in metabolic syndrome index indicates a worse outcome. For economic self-sufficiency, we convert each component of the index so that a higher score is a better outcome (e.g. convert "poor" to "not poor"). Economic self-sufficiency includes seven measures: high school graduate, employed, not poor, not on TANF, not on food stamps, earnings, and family income.[21]

The validity of our design depends on the exogeneity of the introduction of the FSP across counties. We address this in two ways. First, following Hoynes and Schanzenbach (2009) we control for trends in the observable determinants of FSP adoption by including interactions between characteristics of the county of birth and linear trends in year of birth ($CB60_c*b$).

[19] These family background measures are averages over the first five years of life, or in the case of the cohorts born prior to the beginning of the PSID, the first five years of sample.
[20] Kling et al. (2007) analyze a randomized experiment and use the control group mean and standard deviation in calculating the z-score. In our quasi-experimental design, we mimic this approach by using the mean and standard deviation of the cohorts born before food stamp rollout began (cohorts born before 1961).
[21] The top-coding of earnings and income changes over the course of the survey. We trim the sample and drop those (relative few observations) with earnings or income in excess of $300,000.

Further, this period of FSP introduction took place during a period of tremendous expansion in cash and noncash transfer programs as part of the War on Poverty and Great Society. To explore these possible confounders, we directly control for several characteristics of county of birth (community health centers, hospitals and hospital beds per capita, and non-FSP government transfers per capita), measured as averages over the first five years of life.

The basic identification strategy underpinning equation (1) is different from previous design-based studies in the fetal origins literature and other papers in the "rollout" literature. Typically, natural experiments induced by famines, disease outbreaks, etc., are episodic: they turn on and then turn off. In contrast, once the FSP starts operating in a given county, it keeps operating and does not "turn off." An analysis of short-term impacts of the policy, such as maternal exposure and impacts on birth weight (as in Almond, Hoynes and Schanzenbach 2011) leads to a 0/1 treatment variable for "FSP introduction." However, in the current setting, we have a much longer period of potential exposure (through childhood). This restricts the set of cohort comparisons that can be made. For example, we will never observe a birth cohort exposed in early childhood (e.g., up to age five), but without exposure in later childhood (after age 5). Instead, comparisons are "from above": we observe cohorts with the addition of exposure prior to age five, but this comes on top of exposure at older ages. So, comparisons are inherently about additional FSP exposure earlier in childhood, conditional on "already" having it later in childhood. To illustrate the variation we have, Figure 3 shows average FSP exposure by birth cohort for three measures of food stamp availability: in utero, share of months between birth and age 5, and share of months between 6 and age 18.

7. Results

High Participation Sample

We begin with our "high participation" sample, adults born between 1956 and 1981 that grew up in disadvantaged families (i.e., their parent had less than a high school education). Table 2 presents estimates for metabolic syndrome for the high participation sample. We define FSP exposure as the share of months between conception and age 5 that the FSP was available in the individual's county of birth ("FS share IU-5" in the table). The "metabolic syndrome index" is the equal weighted average of the z-score of five dichotomous variables: obese, diabetic, high blood pressure, heart disease and heart attack. This, and all subsequent specifications, include individual demographics, family background controls, and fixed effects for year of birth, year of interview, county, state linear trends (in cohort), and 1960 county characteristics linear trends (in cohort). The effect of access to food stamps in childhood on metabolic syndrome, as shown in column 1, is -0.294 and is statistically significant at the 1 percent level. The magnitude of the coefficient implies that increasing the share from 0 to 1 (from no exposure to full exposure *in utero* to age 5) reduces metabolic syndrome by 0.3 standard deviations.

The remaining columns of Table 2 show the regressions for the individual components of metabolic syndrome index. While individually only obesity reaches statistical significance, each of the point estimates indicate an improvement in adult health with the food stamp treatment in childhood.

The estimates in Table 2 are intent-to-treat estimates, averaging across persons with higher and lower likelihoods of being affected by food stamps.[22] Of course, the food stamp

[22] This is a reduced form model, rather than an instrumental variables approach with the FSP rollout instrumenting for the household's FSP participation. We do not estimate the IV because participation is measured only beginning in 1968, the first year of the PSID survey.

participation rate is not 100 percent, even in this high participation sample. We estimate that for families where heads have less than a high school degree, about a quarter participate in food stamps at some point in their child's life. Thus to convert these estimates to the treatment on the treated, one should divide the treatment effects by 0.24.[23]

Table 3 presents estimates for other health outcomes for the high participation sample. Column 1 presents results for being in "good health" defined as one if the individual reports being in excellent or very good health (as opposed to good, fair or poor health). The coefficient equals 0.11, which implies that going to full exposure between conception and age 5 leads to an 11 percentage point increase compared to a mean of 59 percent, though this is not statistically significant. Column 2 presents estimates for a work limiting disability and while the coefficient is negative (i.e., an improvement as expected) it is very small and statistically insignificant.[24] The third column indicates that access to the food stamp program leads to a reduction in the risk of stunting (below the 5th percentile of the nationally-normed distribution of height).[25] The final two columns of the table present results for health behaviors—dichotomous variables for whether the person ever smoked and whether they drink (at all). Both suggest an improvement but neither is statistically significant.

We go on to analyze the economic outcomes for the high participation sample in Table 4. The first column presents estimates for the "economic self-sufficiency index." This is an equal

[23] It is the participation rate of the sample individuals *at birth and in early life* that is relevant, rather than their contemporaneous (adult) participation rate. To calculate the 0.24 FSP participation rate, we calculate the share of families with children who ever report receiving food stamps (in the period when they have children in the household). We limit the sample to 1978 and later, after the FSP has been rolled out in all counties.

[24] Note that the sample size for health status and disability are substantially larger than those in Table 2 because these questions have been included in the survey since 1984.

[25] Table 3 shows that the mean of the stunting measure is below 0.05 which is consistent with the results in Andreski, McGonagle and Schoeni (2009) finding that the height measures in the PSID are somewhat higher other health surveys. The qualitative results are the same if we define stunting as below the 10th percentile.

weighted average of seven items where, for each, the variables are converted (if needed) such that an increase in the outcome represents a better outcome. The components are: educational attainment is high school or higher, not poor, not on food stamps, not on TANF, employed, earnings and the log of family income. [26] The coefficient on the food stamp treatment is 0.182, implying that full FSP access to age 5 leads to a 0.2 standard deviation improvement in economic self-sufficiency (p-value is 0.14). The remainder of the columns provides the estimates for the individual components of the economic self-sufficiency index. All coefficients with the exception of employment status suggest that exposure to food stamps leads to an improvement in economic well-being: increases in education, earnings, and income and a reduction in poverty and participation in public assistance programs. However, only the coefficient on educational attainment reaches statistical significance.

Interestingly, the interpretation of the coefficient on adult food stamp receipt (column 4 of Table 4) is potentially more complicated than the biological theories discussed above. Observational data shows a relatively strong degree of intergenerational transmission of "welfare use" (Bane and Ellwood 1994). Obtaining causal estimates for intergeneration transmission is difficult, however, given the strong persistence in other factors associated with economic success. In any case, the intergeneration transmission story would imply a positive effect of exposure to food stamps in early life on adult participation in the program. While the results here are not conclusive given the lack of precision in the estimate, the point estimate (positive on *not* participating in food stamps) suggests that on net the biological story dominates.

[26] Note that in Table 4, the mean of the self-sufficiency index is not zero. As described above, we use the full sample of individuals born before 1962 to create the z-scores of each component. The mean here is lower due to our "high participation" subsample (which is lower SES) and due to our sample being younger (and thus lower earnings etc.) than the pre-1962 sample.

In Table 5 we present the main results in the high participation sample by gender of the adult.[27] We find quite striking evidence that the effects for economic self-sufficiency is large and statistically significant for women, compared to the smaller and insignificant results for men. This is consistent with the several studies that find larger impacts of post-natal, early life interventions among girls (Anderson 2008, Bleakley 2007, Dahl and Lochner 2012, Field et al. 2009, Kling et al. 2007, Maccini and Yang 2009, Milligan and Stabile 2011). The estimates for the effect of food stamps on metabolic syndrome are significant for both groups, but larger for men than for women. The larger effects for men are consistent with the biological evidence that males are more subject to harm in utero than females (Almond and Currie 2011a).[28] Interestingly, the gender differences in self-reported health status show a different pattern, with significant positive effects concentrated among women. In interpreting these gender differences, it is important to point out that there is no evidence that pre-natal exposure to food stamps affects live births or infant mortality, overall or by gender (Almond, Hoynes and Schanzenbach 2011); such an effect, if present, suggests selective mortality which would cloud the interpretation of these results.[29]

Identification in the model comes from variation in food stamp rollout across counties and birth cohorts. Importantly, the food stamp program was expanded in the midst of the Great Society, a time when many health and human capital programs were expanding across the U.S.

[27] Table 5 shows that we have more women in our sample than men which is a result of the fact that our sample includes individuals in survey years when they are a head or spouse. This gender imbalance is known and has been documented by Andreski et al. (2009).

[28] Males tend to suffer higher mortality rates in response to adverse events than females. In addition to higher mortality, males could also exhibit larger long-term effects if males suffer a more pronounced (unobserved) health shock than females in response to the same event (e.g. Food Stamps).

[29] Aside from biological factors, it is possible that post-natal treatment differs between girls and boys, and that this might change with food stamp treatment. Interestingly, Lhila and Simon (2008) find that families with girls are more likely to take up the WIC nutrition program post-natally.

Much of that policy variation resulted from state rather than county implementation. However, in Table 6, we examine the sensitivity of our core health and economics outcomes, to adding controls for county programs and resources available between ages 0 and 5. The first three columns examine metabolic syndrome, where the first column repeats the main estimates from Table 2, column 1. In the second column we add controls for access to health care resources (hospitals per capita, hospital beds per capita, presence of community health centers). In the third column we add controls for real per capita government transfers.[30] We then repeat the three specifications for the economic self-sufficiency index in columns 4 to 6. The table shows that our results are highly robust to adding these controls.

An additional test for the validity of the design is to estimate the model by limiting the sample to those who are unlikely to have been impacted by the program. In Table 7, we employ this placebo test and limit to only those individuals from families with high levels of head's education (more than a high school education). These results show small, imprecise and generally wrong-signed impacts for both health and economic outcomes. This adds support to our approach.

The results so far show that in the high participation sample, we find that childhood exposure to the food stamp program leads to robust and significant improvement in metabolic syndrome (obesity, high blood pressure, heart disease and diabetes) for adult men and women. We also find that childhood exposure to the safety net improves economic outcomes for adult women, but not for men.

Full Sample Triple Difference

[30] The number of observations declines when we add the county controls for government per capita transfers. This is because the REIS data begins in 1959 and we therefore have to drop all observations with a year of birth 1958 or earlier.

In choosing our preferred sample for this analysis, we face a tradeoff between sample size (using the full sample of adults) and targeting (using the smaller high participation sample). To build on the findings for the high participation sample, here we use the full sample of adults in our PSID sample, but use a triple-difference specification that accounts for different probabilities of being affected by food stamps. In particular, we augment model (1) above and estimate:

$$y_{icb} = \alpha + \varphi FSP_{cb} + \delta FSP_{cb}P_g + X_{icb}\beta + \eta_c + \lambda_b + \gamma_t + \mu_g + \theta_s * b + \varphi CB60_c * b + \varepsilon_{icb} \quad (2)$$

To capture the varying risks of being treated we multiply the FSP treatment by a group level food stamp participation rate (Bleakley 2007, Hoynes and Schanzenbach 2009). The group food stamp participation rate P_g is defined for 12 groups using education (<12, 12, >12), race (white, nonwhite), and marital status (married, not married) based on the family background of the adult (e.g., their parents' characteristics). We calculate the participation rate in the same fashion as discussed above (to convert intent-to-treat to treatment-on-treated). In addition to the variables in the model (1), we add a main effect for food stamp treatment, fixed effects for each group μ_g, and (although not shown in (2)), interactions of P_g with demographics, year of birth and interview fixed effects, and 1960 county characteristic trends. The coefficient on the main effect for food stamp treatment, φ, represents the impact for a participation rate of zero; therefore we expect this coefficient to be zero. In this triple difference model, the maintained assumption is that there are no differential trends for high participation versus low participation groups within early versus late implementing counties.

Results for this specification are presented in Table 8. Note that the main treatment variable, *FS Share IU-5,* is interacted with the participation rate and therefore the coefficient represents the impact of FSP exposure on health and economic outcome for someone who takes

up the program. Thus, these are treatment-on-the-treated estimates and should be compared to the inflated estimates in the high participation sample.

The results in Table 8 show that full exposure to food stamps through age 5 leads to a 0.4 standard deviation reduction in metabolic syndrome and a 30 percentage point increase in reporting good health. Economic self-sufficiency is improved, but not significantly. The magnitudes are similar but somewhat smaller than the comparable results for the high participation sample. Further, as expected the main effect on food stamp exposure (effect implied for a group with a participation rate of 0) is very small and insignificant.

Putting the Effect Size into Context

The magnitude of effects we estimate is generally large (though with a wide confidence interval). We have argued that analyzing long-term effects of policy-induced variation in early-childhood circumstance constitutes a contribution of our paper. This contribution circumscribes the set of existing studies to which we might compare effect magnitudes, especially those that include metabolic syndrome or its components as the outcome.

That said, famine studies offer one benchmark. Conditional on duration, famine is clearly a more extreme health experience than receiving Food Stamps: famine's long-term effects may therefore be larger. On the other hand, it is not known how the long-term impacts of chronic moderate food deprivation compare to acute famine. Additionally, selection into "surviving cohorts" may be more of an issue with famines because of famine-induced early childhood mortality and selection into childbirth. In our earlier work, we found no significant impacts on number of births or on sex ratios among those exposed to the food stamp program (Almond et al. 2011). In general, famine studies find that those giving birth nine months after famine tend to be fewer in number and positively selected. For example Stein et al. (1975) find that among the

cohorts conceived around the Dutch Hunger Winter, father's occupation quality "improves" markedly with famine exposure. Selective mortality is likely to understate the true impacts of famine.

Despite the greater scope for positive selection, famine's estimated long-term effects tend to be quite large. Painter et al. (2005) found that coronary heart disease tripled for the cohort *in utero* during the Dutch famine, jumping from 3 to 9 percent. They also found the number self-reporting "poor health" doubled from 5 to 10 percent. Ravelli et al. (1976) found a near doubling of obesity for the cohort exposed to Dutch famine during the first 2 trimesters of pregnancy. Likewise, large long-term health effects have been found following the 1959-61 famine in China. Luo, Mu, and Zhang (2006) find that female obesity rates in the most impacted cohorts are approximately 50 percent higher measured at ages 30-40. Note that while the famine literature is generally concerned primarily with in utero exposure, our estimates are based on exposure over a broader time period both in utero and during childhood.

Estimated magnitudes from non-famine studies are also quite sizeable. During Ramadan, Muslims worldwide engage in a daytime fast lasting one lunar month, which falls on different dates in different years. Van Ewijk (2011) considered prenatal exposure to Ramadan in Indonesia, and finds a roughly 0.1 standard deviation increase in pulse pressure (systolic minus diastolic blood pressure) and a 0.16 standard deviation increase in the probability of chest pain. Almond and Mazumder (2008) find a 20 percent increase in the probability of being disabled among those with prenatal exposure to Ramadan in Uganda.[31]

[31] Additionally, Almond, Currie, and Herman (2012) consider improvements in post-neonatal mortality in 1960s and 1970s United States, which were driven by various factors including access to medical care. Focusing on how these improvements differed by race, they found that improvements in early life disease environment decreased the probability of diabetes during pregnancy (i.e. when the exposed cohort reached adulthood) by almost 30 percent for Blacks. Almond (2006) found a roughly 20 percent increase in the physical disability rate more than sixty years after a very-brief exposure to influenza.

Additionally, relatively little is understood about how impacts vary over the life cycle. Evidence from the Dutch Hunger Winter finds that BMI impacts are larger among women at age 50 than they were at age 20, but grow smaller among men as they age (Ravelli et al., 1999). It is possible that our outcomes, generally measured in the respondent's 30s and 40s, are capturing the impacts when they are the largest and that the impacts – especially those on health – may decline as the cohort continues to age.

In sum, long-term famine effects tend to be very large, frequently much larger the effects we find for Food Stamps. Shorter and less extreme natural experiments have also been found to have large long-term effects. Our findings are broadly consistent with the patterns in the literature.

Does the Timing of Treatment Matter?

All the results thus far measure food stamp treatment as the share of time between conception and age 5 that food stamps is in place in your county of birth. There are two reasons to explore alternative specifications for exposure to the food stamps rollout. First, as discussed above, the nature of our treatment is such that the policy turns on and does not turn off. Therefore, when a child is treated in early life (e.g. age 0-5) they are also treated in later childhood. Our estimates, therefore, may reflect exposure beyond age 5; exploring the timing of impacts may help in interpreting the magnitudes of the effects. Second, the biological and economic literature is not clear on when exposure to the safety net matters. Thus exploring alternative specifications for food stamp exposure can provide new evidence on this important issue. For this and the remainder of the results we return to the high participation sample.

To explore the timing of food stamp exposure more systematically, and to evaluate the validity of the research design, we also estimate an event study model. In particular, these

estimates allow us to explore non-parametrically the relationship between age at initial rollout and adult outcomes. In addition, we can use these results to rule out the presence of pre- (or post-) trends that could lead to spurious findings. Specifically, we allow for the impact of FSP program to vary with the *age at FSP introduction* in their county of birth. For example, a person born in 1970 whose birth county implemented food stamps in 1975 would have an event time of 5. They would have event time of -5 if FSP was implemented in their birth county in 1965 (and thus they were exposed during their entire childhood).

We estimate a version of model (1) where the main FSP effect (FSP_{cb}) is replaced with a series of dummies for whether the program was first implemented 5 or more years prior to birth, 4 or 3 years prior to birth, 2 or 1 year prior to birth, at birth or at age 1, at age 2 or 3, age 4 or 5, and so on up to age 12 or later.[32] We present results for metabolic syndrome index in Figure 4. Note that these are the reverse of a typical event study graph, in that negative "event time" is the case where a person was fully treated (food stamps was in place in their county prior to birth). Further, treatment (exposure to the program) increases as we move from the right (treated in later life) to the left (treated in early life).

While we do not have a strong prediction about the precise shape of the treatment effects, our hypothesis is that the impact of the FSP treatment should decline as age at initial exposure increases. Or to state the reverse, the younger the initial age of exposure the larger the (cumulative) effect of the FSP. If exposure in later childhood does not matter, then the event study coefficients should be flat on the right end of the graph (suggesting no "pre-trend"). Eventually, once we hit the point in early childhood when exposure matters, a movement left (towards earlier initial exposure) should reduce the metabolic syndrome index. Eventually, the

[32] The omitted coefficient is for exposure at age 10-11, so all results are relative to exposure at that age.

event study should be flat once exposure is "complete" (exposure is prior to conception or an event time of -1 or before).

The results in Figure 4 are highly consistent with these predictions and quite encouraging for our research design. They show that the largest effects of the food stamp treatment (in this case a reduction in metabolic syndrome is good and so a beneficial effect is represented by a negative impact) are to those who are treated in utero and early childhood. The improvement in health is steepest with additional exposure between conception and age 4 or 5. The results suggest that the adult health impacts of the FSP are minimal if the child is exposed after age 5. It is notable that for negative event time (fully exposed) the line is flat (and similarly that it is flat across older ages)—this is an important result that can rule out that our estimates are identified by cohort trends within counties.

Table 9 explores the issue of timing in the regression setting. Column 1 repeats the base specification for metabolic syndrome, from Table 2 column 1. In the second column, we add a second exposure variable for the share of months between age 6 and 18 that the adult was exposed to food stamps. Consistent with the results in Figure 4, the 6-18 coefficient is small and statistically insignificant; and its addition does not change the magnitude of the coefficient on the early child exposure. In the third column, we include a dummy for whether food stamps was available when the adult was in utero (specifically, we look whether the program is in place at conception). The biological and economic literature predicts that we should see benefits to exposure in utero. The estimate on in utero exposure is -0.11, implying that exposure in utero reduces metabolic syndrome. These results are not conclusive (the p-value is 0.13) and, along with the basic trends illustrated in Figure 1, suggest that we don't have the power to separately identify the in utero and share of childhood impacts. The remaining three columns repeat these

specifications for economic self-sufficiency. The results in column 5 suggest that food stamp exposure in both early and later childhood is beneficial to adult economic outcomes. This is consistent with a story whereby children with better nutrition gain more from school, which in turn translates into higher economic self-sufficiency. The basic patterns—effects on metabolic syndrome result from pre- and early post-natal exposure whereas the effects on economic self-sufficiency result from childhood exposure—are an important result from the analysis.

8. Conclusion

In this paper, we present new evidence that expanding resources in utero and in early childhood can lead to significant improvement in adult health. In particular, we use the rollout of the most important cash or near cash safety net in the U.S., the food stamp program. We find that access to food stamps in utero and in early childhood leads to significant reductions in metabolic syndrome conditions (obesity, high blood pressure, heart disease, diabetes) in adulthood and, for women, increases in economic self-sufficiency (increases in educational attainment, earnings, income, and decreases in welfare participation). Further, we provide new evidence on when exposure to additional resources matters—the gains are large an increasing with exposure to age 5. Beyond that point the additional resources do not translate into improved adult health outcomes. These results pass several robustness tests including controlling for other county-year of birth controls for the Great Society period, a placebo test, and event study models.

Given the near-cash nature of food stamp vouchers (Hoynes and Schanzenbach 2009), the exact biological mechanisms that lead to the long run improvement in health and human capital is not clear. The availability of food stamps leads to more food consumption (Hoynes and Schanzenbach 2009, Currie 2003) and thus one clear channel is through an increase in nutrition

in the critical in utero and early life period. Additionally, recent work suggests that additional income can lead to reductions in cortisol in mothers, reducing biological harm due to persistent stress (Aizer, Stroud, and Buka, 2009, Evans and Garthwaite 2011).

Because "long-term impacts can only be estimated for cohorts treated a long time ago," an intrinsic challenge is how to "generalize estimates of long-term effects to current policies" (Ludwig and Miller, 2007). Some have argued birth weight is a relatively good proxy for the long-term effect of early life health conditions (Behrman and Rosenzweig 2004, Black, Devereux, and Salvanes 2007). If birth weight was a "sufficient statistic" for early life exposure, this would facilitate more timely evaluations of shocks and treatments. However, evidence from Dutch famine suggests otherwise: the cohort showing the largest birth weight decrease was exposed to famine later in pregnancy, while the larger long-term morbidity effects appear for the cohort exposed to famine earlier in pregnancy (Painter et al. 2005). Similarly, Kelly (2009) and Royer (2009) find less consistent long-term effects of birth weight. In other words, birth weight may be an unreliable metric of long-term health, and that from an empirical perspective an important component of this effect remains latent early in life.

Moreover, our analysis finds effects for food stamp exposure through early childhood. Thus, although in previous work we found that the FSP increased birth weight (Almond, Hoynes and Schanzenbach 2011), the evidence presented here show that it is unlikely this is the only or primary mechanism by which food stamps affects long run outcomes.

References

Acemouglu, Daron, David Autor and David Lyle," Women War and Wages: The Impact of Female Labor Supply on the Wage Structure at Mid-Century", *Journal of Political Economy* (2004), pp. 497-551.

Aizer, Anna, Laura Stroud, and Stephen Buka. 2009. "Maternal Stress and Child Well-Being: Evidence from Siblings." Working paper.

Almond, Douglas. 2006. "Is the 1918 Influenza Pandemic Over? Long-Term Effects of in utero Influenza Exposure in the Post-1940 U.S. Population." Journal of Political Economy, 114(4): 672–712.

Almond, Douglas, Kenneth Chay and Michael Greenstone. 2007. "Civil Rights, the War on Poverty, and Black-White Convergence in Infant Mortality in the Rural South and Mississippi." *MIT Department of Economics Working Paper No. 07-04*.

Almond, Douglas and Janet Currie (2011a) "Human Capital Development before Age Five". *Handbook of Labor Economics,* Volume 4b: Orley Ashenfelter and David Card, editors.

Almond, Douglas and Janet Currie (2011b). "Killing Me Softly: The Fetal Origins Hypothesis," Journal of Economic Perspectives, 25, 153-172.

Almond, Douglas, Janet Currie, and Mariesa Hermann (2012). "From Infant to Mother: Early Disease Environment and Future Maternal Health." *Labour Economics*, 19(4) August, 475-483.

Almond, Douglas, Lena Edlund, Hongbin Li, and Junsen Zhang (2010). "Long-Term Effects of the 1959–61 China Famine: Mainland China and Hong Kong." In The Economic Consequences of Demographic Change in East Asia, NBER-EASE Vol. 19, ed. T. Ito and A. Rose, 321–50. Chicago: University of Chicago Press.

Almond, Douglas, Lena Edlund, and Mårten Palme (2009). "Chernobyl's Subclinical Legacy: Prenatal Exposure to Radioactive Fallout and School Outcomes in Sweden." Quarterly Journal of Economics, 124(4): 1729–72.

Almond, Douglas and Bhashkar Mazumder (2011). "Health Capital and the Prenatal Environment: The Effect of Ramadan Observance During Pregnancy," American Economic Journal: Applied Economics, 3 (October 2011), 56-85.

Almond, Douglas, Bhashkar Mazumder and Van Ewijk (2010) "Prenatal Ramadan Fasting and Academic Outcomes in Britain"

Almond, Douglas, Hilary W. Hoynes and Diane Whitmore Schanzenbach (2011). "Inside the War on Poverty: The Impact of Food Stamps on Birth Outcomes," *The Review of Economics and Statistics*, Vol. 93, No. 2: 387-403.

Anderson, Michael (2008). "Multiple Inference and Gender Differences in the Effects of Early Intervention: A Reevaluation of the Abecedarian, Perry Preschool," *Journal of the American Statistical Association*, Vol. 103, No. 484.

Andreski, Patricia, Katherine McGonagle, and Bob Schoeni (2009). "An Analysis of the Quality of the Health Data in the Panel Study of Income Dynamics," PSID Technical Series Paper #09-02.

Bailey, Martha (2012). "Reexamining the Impact of U.S. Family Planning Programs on Fertility: Evidence from the War on Poverty and the Early Years of Title X," AEJ Applied Economics 4 (2): 62-97.

Bailey, Martha and Andrew Goodman-Bacon (2012). "The War on Poverty's Experiment in Public Medicine: Community Health Centers and the Mortality of Older Americans," working paper, University of Michigan.

Bane, Mary Jo and David Ellwood (1994). *Welfare Realities: From Rhetoric to Reform*, Harvard University Press.

Banerjee, Abhijit, Esther Duflo, Gilles Postel-Vinay, and Tim Watts (2010). "Long-Run Health Impacts of Income Shocks: Wine and Phylloxera in Nineteenth-Century France." Review of Economics and Statistics, 92(4): 714–28.

Barker (1992). *Fetal and infant origins of adult disease*. London: British Medical Journal.

Barreca, Alan (2010). "The Long-Term Economic Impact of In Utero and Postnatal Exposure to Malaria." Journal of Human Resources, 45(4): 865–92.

Behrman, Jere R. and Mark R. Rosenzweig, "Returns to Birthweight", *The Review of Economics and Statistics* (2004), pp. 586-601

Berry, Jeffrey M. 1984. *Feeding Hungry People: Rulemaking in the Food Stamp Program*. New Brunswick, NJ: Rutgers University Press.

Bharadwaj, Prashant, Katrine Loken and Chrstopher Neilson 2012. Early-Life Health Interventions and Academic Achievement. *American Economic Review*, forthcoming.

Bitler, Marianne and Hilary Hoynes (2010). "The State of the Safety Net in the Post-Welfare Reform Era," *Brookings Papers on Economic Activity* Fall 2010, pp. 71-127.

Black, Sandra E., Paul J. Devereux and Kjell G. Salvanes (2007). "From the Cradle to the Labor Market? The Effect of Birth Weight on Adult Outcomes", *Quarterly Journal of Economics*, pp. 409-439

Bleakley, Hoyt. 2007. "Disease and Development: Evidence from Hookworm Eradication in the American South." Quarterly Journal of Economics, 122(1): 73–117.

Bozzoli, Carlos, Angus Deaton, and Climent Quintana-Domeque (2009). "Adult Height and Childhood Disease" *Demography*. 46(4): 647-649.

Cascio, Elizabeth, Nora Gordon, Ethan Lewis, and Sarah Reber (2010). "Paying for Progress: Conditional Grants and the Desegregation of Southern Schools." Quarterly Journal of Economics 125(1), 445-482, February 2010.

Case, Anne, Darren Lubotsky and Christina Paxson (2002). "Economic Status and Health in Childhood: The Origins of the Gradient", *American Economic Review* pp. 1308-1334

Chay, Kenneth, Jonathan Guryan, and Bhashkar Mazumder (2012). "Birth Cohort and the Black-White Achievement Gap: The Role of Access and Health Soon After Birth" Federal Reserve Bank of Chicago Working Paper 2008-20.

Chetty, Raj, John Friedman, Nathaniel Hilger, Emmanuel Saez, Diane Schanzenbach, and Danny Yagan (2011) "How Does Your Kindergarten Classroom Affect Your Earnings? Evidence from Project STAR." Quarterly Journal of Economics 126(4): 1593-1660, 2011

Currie, Janet (2003). "U.S. Food and Nutrition Programs," in Means-Tested Transfer Programs in the United States, Robert Moffitt (ed.). Chicago: University of Chicago Press.

Currie, Janet (2009). "Healthy, Wealthy, And Wise: Is There A Causal Relationship Between Child Health And Human Capital Development?" *Journal of Economic Literature*, XLVII(1):87–122, March 2009.

Currie, Janet and Enrico Moretti (2008). "Did the Introduction of Food Stamps Affect Birth Outcomes in California?'' in R. Schoeni, J. House, G. Kaplan, and H. Pollack, (Eds.), *Making Americans Healthier: Social and Economic Policy as Health Policy*, (New York: Russell Sage Press).

Cutler, David M., Angus S. Deaton and Adriana Lleras-Muney (2006). "The Determinants of Mortality," *The Journal of Economic Perspectives*, pp. 97-120

Dahl, Gordon and Lance Lochner 2012. "The Impact of Family Income on Child Achievement: Evidence from Changes in the Earned Income Tax Credit," American Economic Review Vol. 102, No. 5, pp. 1927-1956.

Dehejia, Rajeev and Adriana Lleras-Muney (2004). "Booms, Busts, and Babies' Health", *The Quarterly Journal of Economics*, pp. 1091-1130

Duncan, Greg, Kathleen M. Ziol-Guest, and Ariel Kalil (2010). "Early-Childhood Poverty and Adult Attainment, Behavior, and Health", *Child Development*, Volume 81, Number 1, Pages 306–325.

Dynarski, Susan, Joshua M. Hyman, and Diane Whitmore Schanzenbach (2011). "Experimental Evidence on the Effect of Childhood Investments on Postsecondary Attainment and Degree Completion" NBER working paper 17533.

Eisinger, Peter (1998). *Toward an End to Hunger*. Washington, D.C.: Brookings Institution Press.

Evans, William and Craig Garthwaite. 2011. Giving Mom a Break: The Effect of Higher EITC Payments on Maternal Health, working paper.

Field, Erica, Omar Robles, and Maximo Torero (2009). "Iodine Deficiency and Schooling Attainment in Tanzania," AEJ Applied.

Finkelstein, Amy, and Robin McKnight. 2008. "What Did Medicare Do? The Initial Impact of Medicare on Mortality and Out of Pocket Medical Spending." Journal of Public Economics, 92(7):1644–68.

Fritzsimons, Emla and Marcos Vera-Hernandez (2012) "Breastfeeding and Children's Development." Manuscript, University College London.

Glied, Sherry and Neidell, Matthew (2010). "The Economic Value of Teeth", *Journal of Human Resources*, pp. 468-496

Gluckman, Peter and Mark Hanson (2004). *The Fetal Matrix: Evolution, Development and Disease.* Cambridge: Cambridge University Press.

Heckman, James J. and Dimitriy V. Masterov (2007). "The Productivity Argument for Investing in Young Children", *Review of Agricultural Economics*, pp. 446-493

Hoynes, Hilary and Diane Whitmore Schanzenbach (2009) "Consumption Reponses to In-Kind Transfers: Evidence from the Introduction of the Food Stamp Program," *American Economic Journal: Applied Economics* Vol. 1, No. 4, October 2009, pp. 109-139.

Hoynes, Hilary, Marianne Page, and Ann Stevens. 2011. "Can Targeted Transfers Improve Birth Outcomes? Evidence from the Introduction of the WIC Program." Journal of Public Economics, 95(7–8): 813–27.

Internal Revenue Service (2012). http://www.eitc.irs.gov/central/eitcstats/, accessed 9/1/12.

Kling, Jeffrey, Jeffrey Liebman and Lawrence Katz (2007). "Experimental Analysis of Neighborhood Effects," *Econometrica*, Vol. 75, No. 1 (January, 2007), 83–119.

Kelly, Elaine (2009). "The scourge of Asian Flu: in utero exposure to pandemic influenza and the development of a cohort of British children", Institute for Fiscal Studies.

Lewit, Eugene and Nancy Kerrebrock (1997). “Population-Based Growth Stunting,” The Future of Children Vol. 7, No. 2, Summer/Fall 1997.

Lhila, Aparna and Kosali Simon (2008). “Prenatal Health Investment Decisions: Does the Child’s Sex Matter?” Demography, 45(4): 885–905.

Ludwig, Jens and Douglas L. Miller (2007). “Does Head Start Improve Children's Life Chances? Evidence from a Regression Discontinuity Design”, *Quarterly Journal of Economics*, pp. 159-208

Luo, Zhehui, Ren Mu, and Ziaobo Zhang (2006). “Famine and Overweight in China.” *Review of Agricultural Economics*. 28(3): 296-304.

Maccini, Sharon, and Dean Yang. 2009. “Under the Weather: Health, Schooling, and Economic Consequences of Early-Life Rainfall.” American Economic Review, 99(3): 1006–26.

MacDonald, Maurice (1977). *Food, Stamps, and Income Maintenance* (Madison, WI: Institute for Poverty Research).

McDowell, Margaret, Cheryl Fryar, Cynthia Ogden, and Katherine Flegal (2008). “Anthropometric Reference Data for Children and Adults: United States, 2003–2006”, National Health Statistics Reports, Number 1, October 22, 2008.

Milligan, Kevin and Mark Stabile (2011). “Do Child Tax Benefits Affect the Wellbeing of Children? Evidence from Canadian Child Benefit Expansions.” American Economic Journal Economic Policy, Vol. 3, No. 3, pp. 175-205.

Nilsson, Peter (2009). “The Long-term Effects of Early Childhood Lead Exposure: Evidence from Sharp Changes in Local Air Lead Levels Induced by the Phase-Out of Leaded Gasoline.”

Painter, Rebecca C., Tessa J. Roseboom and Otto P. Bleker (2005). “Prenatal exposure to the Dutch famine and disease in later life: An overview”, *Reproductive Toxicology*, pp. 345-352

Paxson, Christina, Elisabeth Donahue, C. Tracy Orleans and Jeanne Ann Grisso (2006). “Childhood Obesity: Introducing the Issue”, *The Future of Children*, pp. 3-18

Ravelli, ACJ, JHP van der Meulen, RPJ Michels, C Osmond, DJP Barker, CN Hales, OP Bleker, MD (1998). “Glucose tolerance in adults after prenatal exposure to famine”
The Lancet, Volume 351 (9097), 17 January 1998, 173–177.

Ravelli GP, Stein ZA, and Susser MW (1976). “Obesity in young men after famine exposure in utero and early infancy.” *New England Journal of Medicine.* Aug 12;295(7):349-53.

Ravelli, A, van der Meulen J, Osmond C, Barker D, and Bleker O (1999). “Obesity at the age of 50 y in men and women exposed to famine prenatally.” American Journal of Clinical Nutrition 70(5): 811-816.

Reyes, Jessica Wolpaw (2007). “Environmental Policy as Social Policy? The Impact of Childhood Lead Exposure on Crime”, *B.E. Journal of Economic Analysis and Policy*, 7:1 (Contributions), October 2007 (2007).

Royer, Heather (2009). “Separated at Girth: US Twin Estimates of the Effects of Birth Weight”, *American Economic Journal: Applied Economics*, pp. 49-85

Short, Kathleen "The Research Supplemental Poverty Measure: 2010." Current Population Reports P60-241. U.S. Census Bureau, November 2011.

Snyder, Stephen E and Evans, William N (2006). “The Effect of Income on Mortality: Evidence from the Social Security Notch”, *Review of Economics and Statistics*, pp. 482-495

Stein, Zena, M. Susser, G. Saenger, and F. Marolla. 1975. *Famine and Human Development: The Dutch Hunger Winter of 1944–1945*. New York: Oxford Univ. Press.

Sullivan, Daniel and Till Von Wachter (2009). “Job Displacement and Mortality: An Analysis using Administrative Data”, August 2009, *Quarterly Journal of Economics*.

Susser, ES and SP Lin (1992), “Schizophrenia after Prenatal Exposure to the Dutch Hunger Winter of 1944-1945,” *Archives of General Psychiatry* 49(12): 983-8.

U.S. Department of Agriculture, ‘‘Food Stamp Program, Year-End Participation and Bonus Coupons Issues,’’ technical report, Food and Nutrition Service, (various years).

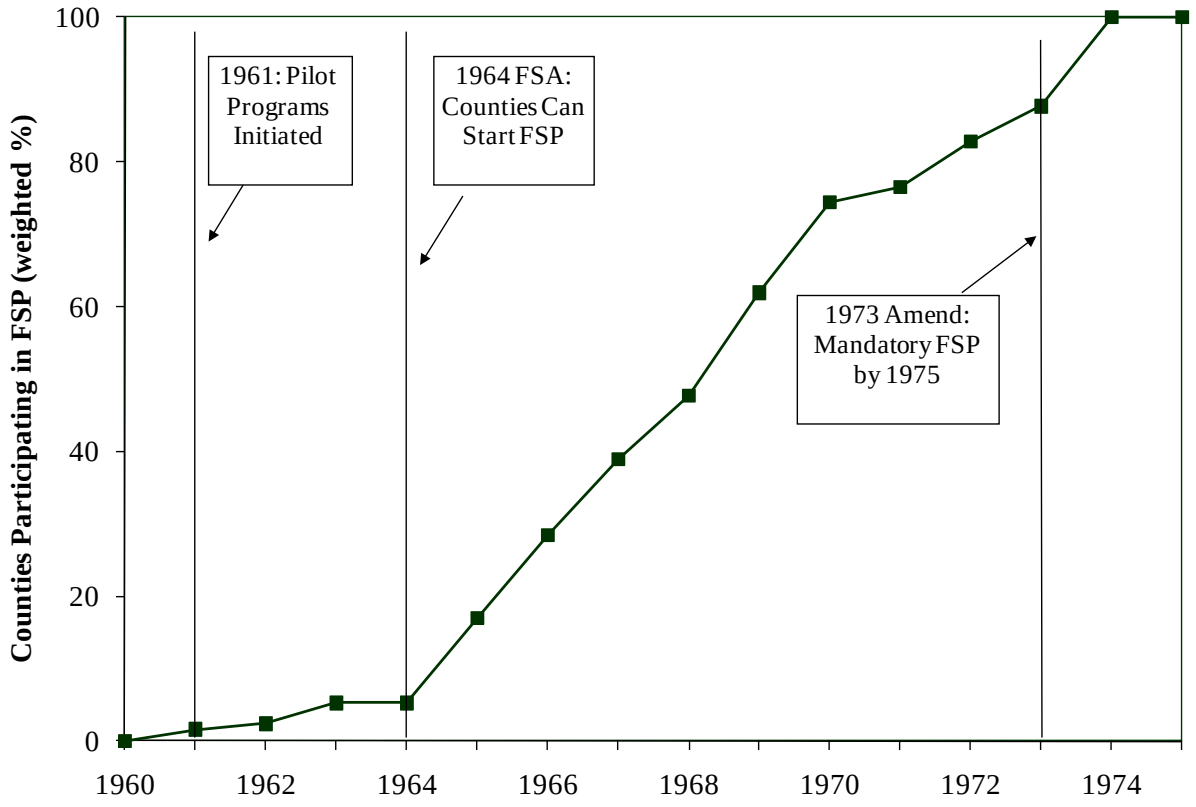
U.S. Department of Agriculture (2012), Food and Nutrition Service, http://www.fns.usda.gov/pd/SNAPsummary.htm, accessed 9/1/12.

U.S. Department of Health and Human Services (2012), Assistant Secretary for Planning and Evaluation, www.acf.hhs.gov/programs/ofs/data/index.html, accessed 9/1/12.

Van Den Berg, Gerard J., Maarten Lindeboom and France Portrait (2006). “Economic Conditions Early in Life and Individual Mortality”, *American Economic Review*, pp. 290-302.

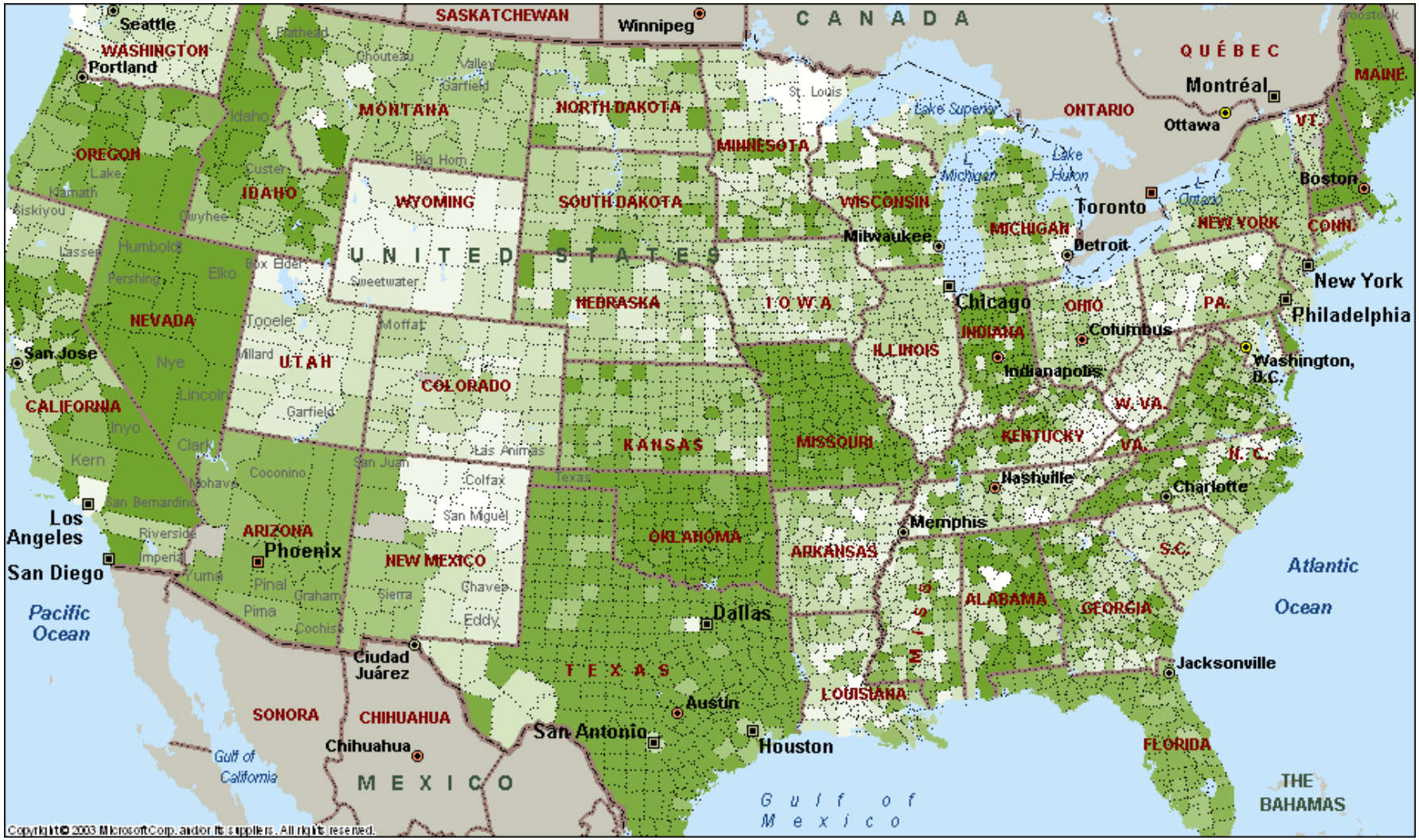
Van Ewijk, Reyn, 2011. "Long-term health effects on the next generation of Ramadan fasting during pregnancy," *Journal of Health Economics*, Elsevier, vol. 30(6), 1246-1260.

Figure 1: Weighted Percent of Counties with Food Stamp Program, 1960-1975



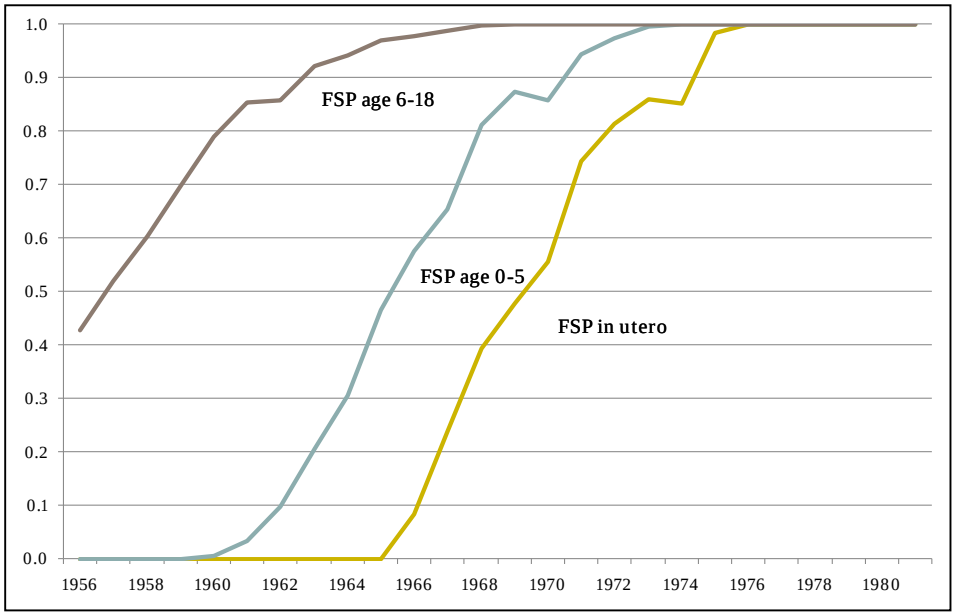
Source: Authors' tabulations of food stamp administrative data (U.S. Department of Agriculture, various years). Counties are weighted by their 1960 population.

Figure 2: Food Stamp Program Start Date, By County (1961-1975)



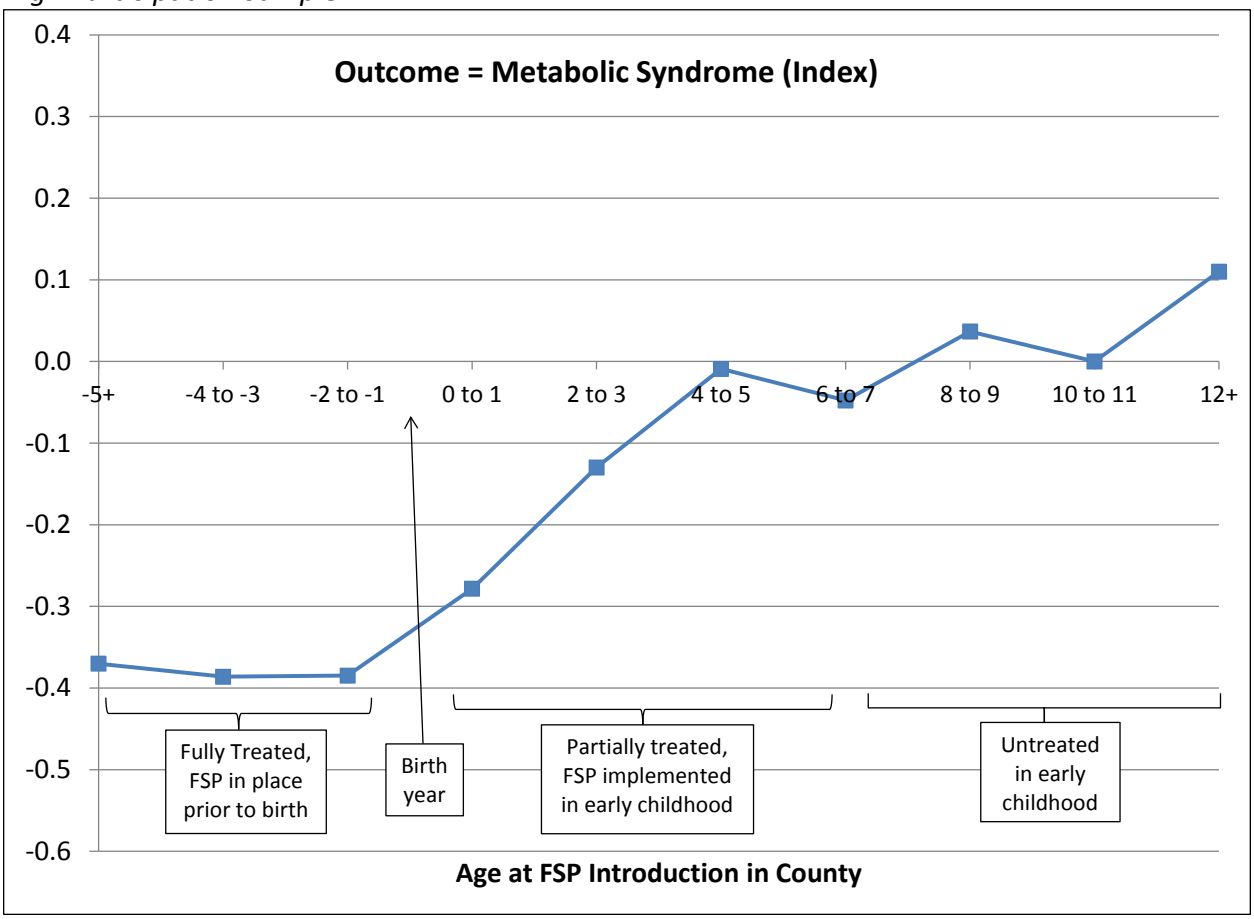
Note: Authors' tabulations of food stamp administrative data (U.S. Department of Agriculture, various years). The shading corresponds to the county FSP start date, where darker shading indicated later county implementation.

Figure 3: Food Stamp Exposure in Early Life, Variation by Birth Cohort



Note: Authors' tabulations of food stamp administrative data (U.S. Department of Agriculture, various years) and PSID sample.

Figure 4
Event Study Estimates of the Impact of FSP Exposure on Metabolic Syndrome Index
High Participation Sample



Notes: The figure plots coefficients from an event-study analysis. Event time is defined as age when FSP is implemented in the birth county. The models are estimated for the sample of individuals born into families where the head has less than a high school education. Age 10-11 is the omitted year so estimates are relative to that point. See the text for a description of the model.

Table 1
Descriptive Statistics

	Full Sample		High Impact Sample	
	N	Mean	N	Mean
FS share age IU-5	60782	0.370	28808	0.338
Health Outcomes				
Metabolic Health Index	22070	-0.079	9097	0.010
In good health =1	60757	0.679	28833	0.581
Disabled =1	60753	0.096	28827	0.118
Diabetes =1	22546	0.041	9321	0.054
High blood pressure =1	22544	0.133	9319	0.187
Obesity =1	24127	0.240	10209	0.322
Heart Disease = 1	22543	0.019	9320	0.028
Heart Attack = 1	22548	0.006	9323	0.008
Healthy weight =1	24127	0.408	10209	0.322
BMI	24127	26.862	10209	28.255
Body weight (pounds)	24645	193.148	10461	202.688
Height (inches)	24589	67.760	10428	67.427
Height below 5th pctile	24589	0.011	10428	0.016
Economic Outcomes				
Economic Outcome Index	57585	-0.051	27303	-0.304
Education High School Plus	60106	0.903	28663	0.786
Log(Total Fam Income)	60599	10.847	28706	10.435
Earnings (including 0s)	59136	35047	27862	23473
Employed =1	60843	0.864	28881	0.739
Poverty = 1	60599	0.184	28706	0.339
Food stamp receipt	60665	0.085	28759	0.157
TANF Receipt	60839	0.033	28873	0.061
Health Behaviors				
Ever Smoked	22548	0.447	9318	0.522
Drink Any	22493	0.703	9300	0.600
Demographics				
Male	60898	0.462	28905	0.442
Nonwhite	60777	0.171	28823	0.317
High School Grad	60106	0.390	28663	0.500
Greater than High School	60106	0.502	28663	0.286
Age	60898	32.135	28905	32.126
Maried	60897	0.585	28904	0.542
Family Background				
Female headed household	60898	0.094	28905	0.161
Income to needs ratio (5-yr average)	60898	2.365	28905	1.512
Head less than high school education	60496	0.345	28905	1.000
1960 County Characteristics				
Population	60882	593,051	28905	514,635
Fraction of land, farmland	60882	48.4	28905	49.4
Fraction of population, urban	60882	67.0	28905	61.4
Fraction of population, black	60882	9.6	28905	13.8
Retirement Transfers Per Capita, 0-5 Avg	48009	928.29	21984	876.26
Medicare Transfers Per Capita, 0-5 Avg	47568	149.71	21661	125.15
Other Public Assistance Per Capita, 0-5 Avg	47568	195.03	21661	194.66
Number of Hospital Beds, 0-5 Avg	58098	4.608	27274	4.494
Number of Hospitals, 0-5 Avg	58098	0.036	27274	0.040
CHC Share, Age 0-5	60898	0.100	28905	0.073

Notes: Author's tabulations of 1968-2009 PSID. Sample consists of heads and wives born between 1956-1981. Observations from Alaska are dropped because of missing data on food stamp program start date. For details on sample selection see text.

Table 2
Metabolic Syndrome Index for High Participation Sample

		Components of metabolic syndrome index				
	Metabolic syndrome (index)	Diabetes	High blood pressure	Obesity	Heart disease	Heart attack
FS share IU-5	-0.294***	-0.032	-0.13	-0.159*	-0.053	-0.031
	(0.107)	(0.048)	(0.086)	(0.086)	(0.027)	(0.019)
Mean of dep var	0.01	0.05	0.19	0.33	0.03	0.01
Observations	8,246	8,431	8,430	9,217	8,430	8,432
R-squared	0.26	0.19	0.22	0.26	0.13	0.08

Table 3
Additional Health Outcomes for the High Participation Sample

	Other health outcomes			Health behaviors	
	In good health	Disabled	Height below 5th perc.	Ever smoked	Drink any
FS share IU-5	0.110	-0.004	-0.060**	-0.056	-0.023
	(0.074)	(0.039)	(0.026)	(0.064)	(0.049)
Y-mean	0.59	0.12	0.02	0.44	0.70
Observations	25,738	25,731	9,398	20,946	20,896
R-squared	0.16	0.13	0.22	0.19	0.18

Notes to Tables 2 and 3: Each parameter is from a separate regression of the outcome variable on FSP exposure (share of months between conception and age 5 that FSP is in the county). The sample comes from the 1968-2009 PSID and includes heads and wives born between 1956-1981 who are between 18 and 53 (or 24-53 for economic outcomes). The high participation sample includes those born into families where the head had less than a high school education. Estimates are weighted using the PSID weights and clustered on county of birth. The models control for individual demographics, family background, and fixed effects for year of birth, year of interview, county, state specific linear cohort, and 1960 county characters interacted with linear cohort. Standard errors are in parentheses and ***, **, and * indicate that the estimates are significant at the 1%, 5% and 10% levels.

Table 4
Economic Self-Sufficiency in the High Participation Sample

		Components of economic self sufficiency index						
	Economic self sufficiency (index)	High school plus	Not Poor	Not on food stamps	Not on TANF	Employed	Earnings	log(family income)
FS share IU-5	0.182	0.184*	0.052	0.032	0.023	-0.008	3610	0.247
	(0.124)	(0.108)	(0.067)	(0.052)	(0.026)	(0.056)	(5,064)	(0.165)
Y-mean	-0.25	0.80	0.70	0.86	0.95	0.76	24495	10.52
Observations	20,115	21,197	21,209	20,115	21,347	21,348	20,529	21,160
R-squared	0.38	0.29	0.30	0.38	0.16	0.18	0.34	0.37

Table 5
Metabolic Syndrome and Economic Self-Sufficiency in the High Participation Sample, by Gender

	Women			Men		
	Metabolic syndrome (index)	Good Health	Economic self sufficiency (index)	Metabolic syndrome (index)	Good Health	Economic self sufficiency (index)
FS Share IU-5	-0.312**	0.336***	0.306*	-0.526**	-0.077	0.005
	(0.130)	(0.100)	(0.164)	(0.251)	(0.112)	(0.168)
Mean of Dependent Var	0.03	0.53	-0.37	-0.01	0.66	-0.11
Observations	5,062	15,702	12,208	3,184	10,036	7,907
R-squared	0.37	0.22	0.43	0.32	0.18	0.46

Notes: Each parameter is from a separate regression of the outcome variable on FSP exposure (share of months between conception and age 5 that FSP is in the county). The sample comes from the 1968-2009 PSID and includes heads and wives born between 1956-1981 who are between 18 and 53 (or 24-53 for economic outcomes). The high participation sample includes those born into families where the head had less than a high school education. Estimates are weighted using the PSID weights and clustered on county of birth. The models control for individual demographics, family background, and fixed effects for year of birth, year of interview, county, state specific linear cohort, and 1960 county characters interacted with linear cohort. Standard errors are in parentheses and ***, **, and * indicate that the estimates are significant at the 1%, 5% and 10% levels.

Table 6
Metabolic Syndrome and Economic Self-Sufficiency in the High Participation Sample, Adding County Controls

	Metabolic syndrome (index)			Economic Self Sufficiency (index)		
FS share IU-5	-0.294***	-0.200**	-0.209**	0.182	0.171	0.210
	(0.107)	(0.079)	(0.081)	(0.124)	(0.125)	(0.150)
Y-mean	0.01	0.01	-0.02	-0.25	-0.27	-0.26
Observations	8,246	7,737	6,561	20,115	18,992	13,268
R-squared	0.26	0.27	0.26	0.38	0.38	0.37
Hospitals, beds per capita		X	X		X	X
Community health center		X	X		X	X
REIS real per capita transfers			X			X

Notes: Each parameter is from a separate regression of the outcome variable on FSP exposure (share of months between conception and age 5 that FSP is in the county). The sample comes from the 1968-2009 PSID and includes heads and wives born between 1956-1981 who are between 18 and 53 (or 24-53 for economic outcomes). The high participation sample includes those born into families where the head had less than a high school education. Estimates are weighted using the PSID weights and clustered on county of birth. The models control for individual demographics, family background, and fixed effects for year of birth, year of interview, county, state specific linear cohort, and 1960 county characters interacted with linear cohort. The additional county controls are annual averages from birth to age 5. Standard errors are in parentheses and ***, **, and * indicate that the estimates are significant at the 1%, 5% and 10% levels.

Table 7
Placebo Test: Metabolic Syndrome and Economic Self-Sufficiency for High Education Group

	Metabolic syndrome (index)	Economic Self Sufficiency (index)
FS share IU-5	-0.013	0.073
	(0.060)	(0.087)
Y-mean	-0.17	0.22
Observations	5,398	10,180
R-squared	0.24	0.33
"right" signed components	obesity, high blood pressure	employed, earnings, TANF
"wrong" signed components	good health, disability, diabetes, heart disease	education, family income, food stamps

Notes: Each parameter is from a separate regression of the outcome variable on FSP exposure (share of months between conception and age 5 that FSP is in the county). The sample comes from the 1968-2009 PSID and includes heads and wives born between 1956-1981 who are between 18 and 53 (or 24-53 for economic outcomes). The sample includes those born into families where the head had a high school education or more. Estimates are weighted using the PSID weights and clustered on county of birth. The models control for individual demographics, family background, and fixed effects for year of birth, year of interview, county, state specific linear cohort, and 1960 county characters interacted with linear cohort. Standard errors are in parentheses and ***, **, and * indicate that the estimates are significant at the 1%, 5% and 10% levels.

Table 8
Triple Difference Estimates for Metabolic Syndrome and Economic Self-Sufficiency, Full Sample

	Metabolic syndrome (index)	Good Health	Economic self sufficiency (index)
FS Share IU-5 * Pg	-0.438**	0.292**	0.400
	(0.204)	(0.133)	(0.323)
FS share IU-5	-0.032	-0.021	-0.045
	(0.073)	(0.051)	(0.083)
Mean of Dependent Var	-0.08	0.68	0.69
Observations	19,948	54,787	43,117
R-squared	0.20	0.13	0.35

Notes: Each parameter is from a separate regression of the outcome variable on FSP exposure (share of months between conception and age 5 that FSP is in the county) interacted with a group specific FSP participation rate. The sample comes from the 1968-2009 PSID and includes heads and wives born between 1956-1981 who are between 18 and 53 (or 24-53 for economic outcomes). The sample includes those born into families where the head had a high school education or more. Estimates are weighted using the PSID weights and clustered on county of birth. The models control for individual demographics, family background, and fixed effects for year of birth, year of interview, county, state specific linear cohort, and 1960 county characters interacted with linear cohort. Standard errors are in parentheses and ***, **, and * indicate that the estimates are significant at the 1%, 5% and 10% levels.

Table 9
Metabolic Syndrome and Economic Self-Sufficiency in the High Participation Sample, Alternative Specifications for Timing of Exposure

	Metabolic syndrome (index)			Economic Self Sufficiency (index)		
FS share IU-5	-0.294***	-0.279**	-0.241**	0.182	0.383**	0.159
	(0.107)	(0.127)	(0.111)	(0.124)	(0.185)	(0.116)
FS share 6-18		0.060			0.729*	
		(0.270)			(0.428)	
FS in 1st trimester			-0.107			0.069
			(0.070)			(0.080)
Y-mean	-0.01	-0.01	-0.01	-0.25	-0.25	-0.25
Observations	8,246	8,246	8,246	20,115	20,115	20,115
R-squared	0.26	0.26	0.26	0.38	0.38	0.38

Notes: Each parameter is from a separate regression of the outcome variable on FSP exposure (share of months between conception and age 5 that FSP is in the county) interacted with a group specific FSP participation rate. The sample comes from the 1968-2009 PSID and includes heads and wives born between 1956-1981 who are between 18 and 53 (or 24-53 for economic outcomes). The sample includes those born into families where the head had a high school education or more. Estimates are weighted using the PSID weights and clustered on county of birth. The models control for individual demographics, family background, and fixed effects for year of birth, year of interview, county, state specific linear cohort, and 1960 county characters interacted with linear cohort. Standard errors are in parentheses and ***, **, and * indicate that the estimates are significant at the 1%, 5% and 10% levels.