Childhood Exposure to the Food Stamp Program: Long-run Health and Economic Outcomes

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Abstract

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 the Food Stamps 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 adult health and economic outcomes to family background and county of residence in early childhood. The identification comes from variation across counties and over birth cohorts in exposure to the food stamp program. Our findings indicate that the FSP has effects decades after initial exposure. Multiplier effects are largest for health in adulthood. We find weaker positive evidence on adult economic outcomes. Overall, our results are consistent with fetal origins literature on long-run health effects of early life interventions.

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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 on causal mechanisms behind the 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 large social welfare program, 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. Both prior work and economic theory suggests that these vouchers were treated the same as cash income (Hoynes & Schanzenbach 2009). Thus, we can utilize the FSP rollout as an identification strategy for 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 2010).

We also build on a growing literature that relates resources *in utero* and during childhood to economic and health outcomes in adulthood. Previous design-based observational studies have generally looked at extreme events, including famines, natural disaster, or disease outbreaks (see Currie 2009 and Almond & Currie 2010 for recent reviews). A point of departure in our analysis is to consider variation in a moderate range. Moderate income variation may be relevant for a larger population than those touched by extreme events. Nevertheless, it is a relatively open question whether such moderate

variation exerts corresponding long-term effects, or whether these linkages are restricted to extreme events. Furthermore, we consider a moderate variation in a *positive* direction, which may map to other public policies which seek to improve resources and target cohorts during early childhood.

The FSP and other public policies presumably passed a cost-benefit criterion based on their contemporaneous or "real time" benefits. Given that empirical evidence on "early origins" is relatively recent, the long-term benefits of such longstanding programs were neither considered nor expected. This raises the possibility that policies targeting early childhood may be sub-optimally small. In other words, while the FSP is a fundamental part of the U.S. safety net, it may be more than a conventional anti-poverty program if it manifests a "multiplier effect" on later-life outcomes.

Our findings indicate that the FSP has effects decades after initial exposure. Multiplier effects are largest for health in adulthood. We find weaker positive evidence on adult economic outcomes. Overall, our results are consistent with fetal origins literature on long-run health effects of early life interventions.

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 Economic Research

That early childhood events can have important vestigial effects has been

documented for a wide range of later-life outcomes, including health status, completed education, wages, and mortality. Observational studies have leveraged short, extreme events experienced in early childhood as identification strategies (see Currie 2009 and Almond & Currie 2010). 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. In considering the long-term effect of *early-life* income changes, the work closest to ours is by Van Den Berg, Portrait, & Lindeboom (2006), who compare Dutch mortality rates among those born during economic downturns to those born during expansions. Those born during expansions lived substantially longer, which it is argued is not due to changes in cohort composition or other potential confounders. Additionally, Sullivan and Von Wachter (2009) consider the effects of job displacement on mortality, finding that even 20 years after job displacement, mortality rates are higher.

A second strand of research has considered the long-term effect of specific, well-

identified programs. Among these studies, the closest is by 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. (2010) find persistent effects of class-size reductions under the Project STAR experiment on initial labor market outcomes. Reyes (2007) and Nilsson (2010) consider the effect of early childhood lead exposure on later life outcomes, focusing on reductions in ambient lead due to elimination of leaded gasoline. Finally, Glied and Neidell (2010) evaluate the long-term impacts of water fluoridation.[1]

3. Scientific Background on Impacts of Early Nutrition and Expected Effects of the FSP

As indicated above, there is little existing research on how income early in life affects subsequent health and economic outcomes. 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.

[1] 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.

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 (Peter Eisinger 1998). A 1968 CBS documentary "Hunger in America" raised national awareness of the problem and possibly influenced the policy debate on the FSP (Berry 1984). Here, we present a brief summary of the linkages between early life nutrition and later life outcomes to give us guidance on which outcomes 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 irreversibly "program" the body's long-term survival outcomes (Gluckman and Hanson 2004). During development, the fetus/baby/child takes 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.[2] 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- or post-natal period, the developing body therefore predicts that the future state of the world will also be nutritionally deprived. The body may then invoke

[2] These are termed "predictive adaptive responses" or PARs.

(irreversible) 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 during adult life, such as obesity, diabetes and/or heart conditions. 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.

Profound 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 a classic study by 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 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.

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) finds that children had lower birth weight if exposed to famine in the third trimester.[3] 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 & Lin 1992).

Almond and Mazumder (2009) and Almond, Mazumder, and Van Ewijk (2010) consider the effect of nutrition *timing* during pregnancy on later-life outcomes, focusing on the Ramadan fast as an identification strategy. Almond and Mazumder (2009) document negative impacts of fasting during pregnancy on birth outcomes, but adult disability status as well. Almond, Mazumder, and Van Ewijk (2010) find that test scores in Britain are lower for cohorts exposed to Ramadan in early pregnancy. Because fasting during Ramadan is confined to daylight hours, the nutritional treatment is relatively mild compared to famine episodes previously considered.

To summarize, the literature has found that lack of nutrition in early life leads to higher incidence of "metabolic syndrome," which is the clustered association between high

[3] In a parallel manner, our earlier work (Almond et al. 2010) finds that children have higher birth weight if

blood pressure (hypertension), type II diabetes, obesity and cardiovascular disease. These impacts have occurred both when the nutritional shock occurred *in utero* and when it occurred in the period shortly after birth. Note two important details about the established relationship. First, the relationships between early nutritional deficits and outcomes have been found with the disease (e.g. hypertension) and not necessarily with the underlying continuous variable (e.g. blood pressure). Second, the long-run health outcomes have been found even in cases in which birth weight itself was not affected.

Most of the prior literature has identified long-term impacts of nutritional shocks based on negative shocks or manipulations (e.g. famine, influenza, experimentally restricting food in animal subjects). Our paper uses a permanent increase in food available to a population that had previously experienced chronic low levels of nutrition. Because here we have a positive shock, we expect that individuals who were exposed to the 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 overall health, diabetes, high blood pressure and measures of obesity – in adult life. We also expect to find better economic outcomes, as measured by education, wages and the like. Because the exact timing required to impact long-term outcomes is ambiguous, we will show a variety of specifications that measure FSP exposure both in utero and during various parts of childhood.

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

exposed to food stamps in the third trimester.

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 FSP 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.[4] 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 the FSP "treatment."

For our identification strategy to yield causal estimates of the program, it is key 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.[5] 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 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

[4] 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.

[5] 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.

indeed implemented the FSP earlier (i.e. consistent with the historical accounts).[6] 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, Autor and Lyle (2004) and Hoynes and Schanzenbach (2009).

5. Data

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

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

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, we use height and weight, and information on whether a doctor has diagnosed the respondent with specific health conditions such as diabetes and high blood pressure. These data are also collected for all heads and wives and have been available since 1999, when the survey became biennial.

The public-use version of the PSID only contains state identifiers, but through special arrangement we have obtained county-level identifiers for each family in each year of the survey. Thus, 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/childhood. The key variable for our analysis is constructed from the month and year that each county introduced a 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 code childhood exposure to the FSP program as whether the program was implemented prior to the individual's attaining a certain age (e.g. age 1 year, age 5 years, birth, 9 months prior to birth, etc.), and use variation across time and geography to isolate the impact of FSP exposure.

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.[7] This is necessary to identify the individual's county of birth.[8] We also use information on the family background of the individual in early life (whether the 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 be impacted by 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. The sample includes one observation for each interview year that the individual 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 2007. Thus given our birth cohorts (1956-1981) the oldest individuals in our sample are 51 at the end of the period.

Finally, we merge on county-level variables from the 1960 Census of Population and Census of Agriculture. These capture characteristics of the county prior to the introduction of the FSP program and are used to control for possible confounders that are associated with introduction of the FSP across areas. The specific measures we use are: 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.

Table 1 presents descriptive statistics on our estimation sample. About 69 percent of the full sample and 60 percent of the 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

[7] In effect, this limits the sample to persons born into original 1968 PSID families.

diabetic. 12 (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:

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

where *i* indexes the individual, *c* the county of birth, *b* the birth year, *s* the state of birth, and *t* the survey year.

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, and the effect of exposure to FSP introduction, 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. In some specifications we also control for family background, including 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.[9] All models are estimated using the PSID sample weights and we cluster standard errors by county of birth.

The validity of our design depends on the exogeneity of the introduction of the FSP

[8] 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.

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 (described above in Section 4). In particular, our model includes interactions between characteristics of the county of birth and linear trends in year of birth ($CB60_c*b$). 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 disentangle the FSP from these other programs, the county by month variation in FSP rollout is key. Further, given that virtually all means tested programs, are administered at the state level our controls for state by year should absorb these program impacts.

The basic identification strategy underpinning equation (1) is different from previous design-based studies in the fetal origins 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". 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.

7. Results

Table 2 presents results for the health outcomes in the full sample. Here we define

[9] These family background measures are averages over the first five years of life, or in the case of the cohorts

FSP exposure as the share of months between ages 0 and 5 that the FSP was available in the individual's county of birth. All models control for year of birth, county and interview year fixed effects, individual demographics and family background, year of birth linear trends by state, and year of birth linear trends by 1960 county of birth characteristics.

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 is 0.041 which implies that increasing one's exposure to FSP in early life by half a year (10 percentage point increase) raises the probability that an individual is in good health by 0.4 percentage points compared to a mean of 69 percent, though this is not statistically significant. Column 2 presents estimates for a work limiting disability and while the coefficient is negative (e.g. an improvement as expected) it is very small and statistically insignificant. Note that the sample size for health status and disability are substantially larger than the subsequent outcomes because these questions have been included in the survey since 1984. These impacts are the average treatment effect, averaging over all persons with a higher or lower likelihood of being affected by food stamps. In reality, only a fraction of the full population participates in food stamps: eligibility requires income be below 130 percent of poverty and thus low-income residents are the only ones eligible to participate. To account for this, we inflate the coefficients by the average FSP participation rate to obtain an estimate of the impact on FSP recipients (i.e. the effect of the treatment on the treated). We calculate the FSP participation rate using families in 1978, after the FSP has been rolled out in all counties.[10] The results (labeled "*Inflated by takeup rate"*) indicate that increasing exposure by 6 months increases the likelihood of good health by 4 percentage points among the treated.

born prior to the beginning of the PSID, the first five years of sample.

We go on to estimate the impact of FSP exposure on various components of "metabolic syndrome". The availability of these measures is more limited in the data, as they were not introduced to the survey until 1999. This decreases the sample size by approximately a factor of 3. The coefficients on diagnosis of diabetes and high blood pressure are both right-signed but insignificant. Note that because the impacted cohorts are still relatively young, the incidence of these diseases is low at 4 percent for diabetes and 12 percent for high blood pressure. We find significant beneficial impacts on body weight as measured by obesity (defined according to the standard as BMI>=30), body mass index (BMI, defined as weight in kilograms divided by height in meters squared), being a "healthy weight" (defined as BMI in the range 18.5 to 25) and the level of body weight in pounds. Increasing FSP exposure by a half a year in early life decreases the likelihood of adult obesity by about 1 percentage point (10 points in the treatment on treated estimate). BMI decreases by 0.2 points, and body weight declines by just over a pound. Finally, we include the "impact" on height as a falsification test. We find no impact of FSP exposure on height in inches.

Table 3 presents the adult economic outcomes for the full sample. The results on economic outcomes are a bit mixed (education and employment increase, family income decreases), but none are close to statistically significant.

In order to better isolate the impact of FSP exposure to groups that were likely treated by the program, in Tables 4 and 5 we limit the sample to individuals born into families with a head of household with low education (a high school education or less). The results are similar for the health impacts--right-signed but imprecise with the exception of the body weight measures. The economic outcomes consistently show a benefit effect of

[10] It is the participation rate of the sample individuals at birth and in early life that is relevant, rather than the

access to the FSP—Table 5 suggests increases in education, earnings, employment and income and a reduction in poverty. However, as with the full sample, none of these outcomes are statistically significant.

Similarly, we can employ placebo tests by limiting the sample to those who are very unlikely to have been impacted by the program. In Table 6, we 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.

To improve the precision of the estimates, we leverage information on differences in FSP participation rates by demographic group. This allows us to use within-cohort comparisons by using a triple difference approach. Defining the demographic group-specific participation rate P_g, we interact this participation measure with FSP exposure FSP_{cb} and estimate the following model:

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

The group specific participation rate is defined by the race, education, and marital status of their family head at birth/early life. The coefficient estimate on ϕ in this model 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.

Results for this specification are presented in Tables 7 and 8. The results in Table 7 show that all of the health outcomes suggest improvements with childhood exposure to the FSP; and many estimates reach statistical significance. In column 1 of Table 7 we find that a 10 percentage point increase in FSP exposure in early life (weighted by the group participation rate) increases the likelihood of being in good health by a statistically

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contemporaneous participation rate.

significant 2 percentage points.[11] The same increase reduces the likelihood of being diagnosed with diabetes by 1.5 percentage points, and reduces the likelihood of a high blood pressure diagnosis by 2 percentage points. The body weight impacts are similar to those found earlier, though here only the obesity rate impact is precisely enough measured to attain statistical significance. Although not shown here, there is again no impact on height, as is expected.[12]

As a robustness check we estimate an event study model on a subsample of individuals who were born into families headed by a single woman. In our event study design we nonparametrically allow for the impact of FSP program to vary with the age since the program was implemented in their county of birth. In particular, we estimate model (1) where the main FSP effect (FSP_{cb}) is replaced with a series of dummies for whether the program was implemented 5 or more years prior to birth, 4 years prior to birth, 3 year prior to birth , …, at age 1, at age 2, and so on up to age 15 or later.[13] The results for being in good health and being obese are presented 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. If the FSP ramped up immediately in counties, then we expect the coefficients to be largest and flat for negative event time. While we do not have a strong prediction about the precise shape of the treatment effects at age 0 and older, our hypothesis is that the impact of exposure to the FSP should decline with age at initial exposure. The results are quite encouraging for our research design. They show that the largest effects of the food stamp treatment are to those who are treated in utero and early

[11] Note that here we do not present coefficients divided by takeup rate, because the coefficient here is already weighted by takeup rate.
[12] We include the main effect on the FSP exposure variable ("FS Share age 0-5") for completeness and as the control in the triple difference model. The interpretation of the main effect is the impact of exposure to the FSP for someone with a predicted participation rate of zero. We would expect these coefficients to be very small and statistically insignificant.

childhood. The results suggest that the adult health impacts of the FSP are minimal if the child is exposed beginning at age 6 or later.

8. Discussion and Conclusion

Surprisingly, reliable evidence on effect of income on health is scant and conflicting. Some have found that income's effect on health argue is perverse (Dehejia & Lleras-Muney, 2004; Snyder & Evans, 2006). Here, we find positive long-term effects of early-life income shocks. This finding most consistent with evidence on long-term effects of more temporary income shocks from recessions (Van Den Berg et al 2006).

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). Behrman and Rosenzweig (2004); Black, Devereux, & Salvanes (2007) and others have argued birth weight is a relatively good proxy for the long-term effect of early life health conditions (it's certainly readily available and commonly used). On the other hand, a reading of the 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 cohort exposed to famine earlier in pregnancy (Painter, Roseboom, & Bleker, 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 FSP exposure after pregnancy, in early childhood. Thus, although in previous work we found that the FSP increased birth weights,

[13] The omitted coefficient is for exposure at age 12, so all results are relative to exposure at that age.

it is unlikely this is the only or primary mechanism by which FSP had a "first stage" effect on early-life health. In this respect, we infer FSP rollout had additional, contemporaneous health benefits not reported Almond, Hoynes, and Schanzenbach (2010). On a practical level, this argues for the reduced form approach we pursue in the paper to evaluate long-term effects.

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

Almond, Douglas and Janet Currie (2010) "Human Capital Development before Age Five". Forthcoming, *Handbook of Labor Economics*, Volume 4: Orley Ashenfelter and David Card, editors.

Almond, Douglas and Bhashkar Mazumder, "Health Capital and the Prenatal Environment: The Effect of Maternal Fasting During Pregnancy" NBER Working Paper No. 14428 (2008)

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, "Inside the War on Poverty: The Impact of Food Stamps on Birth Outcomes," *The Review of Economics and Statistics* (forthcoming)

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

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.

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

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

Chetty, Raj, John Friedman, Nathaniel Hilger, Emmanuel Saez, Diane Schanzenbach, and Danny Yagan (2010) "How Does Your Kindergarten Classroom Affect Your Earnings? Evidence from Project STAR."

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.

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

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

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

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

Glied, Sherry and Neidell, Matthew, "The Economic Value of Teeth", *Journal of Human Resources* (2010), 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, "The Productivity Argument for Investing in Young Children", *Review of Agricultural Economics* (2007), 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.

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

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

McCance (1962)

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

Nilsson, Peter, "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" (2009)

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

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

Van Ewijk, Reyn. "Long-term health effects on the next generation of ramadan fasting during pregnancy." April 2009. CEP Discussion Paper No 926.

Reyes, Jessica Wolpaw, "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, "Separated at Girth: US Twin Estimates of the Effects of Birth Weight", *American Economic Journal: Applied Economics* (2009), pp. 49-85

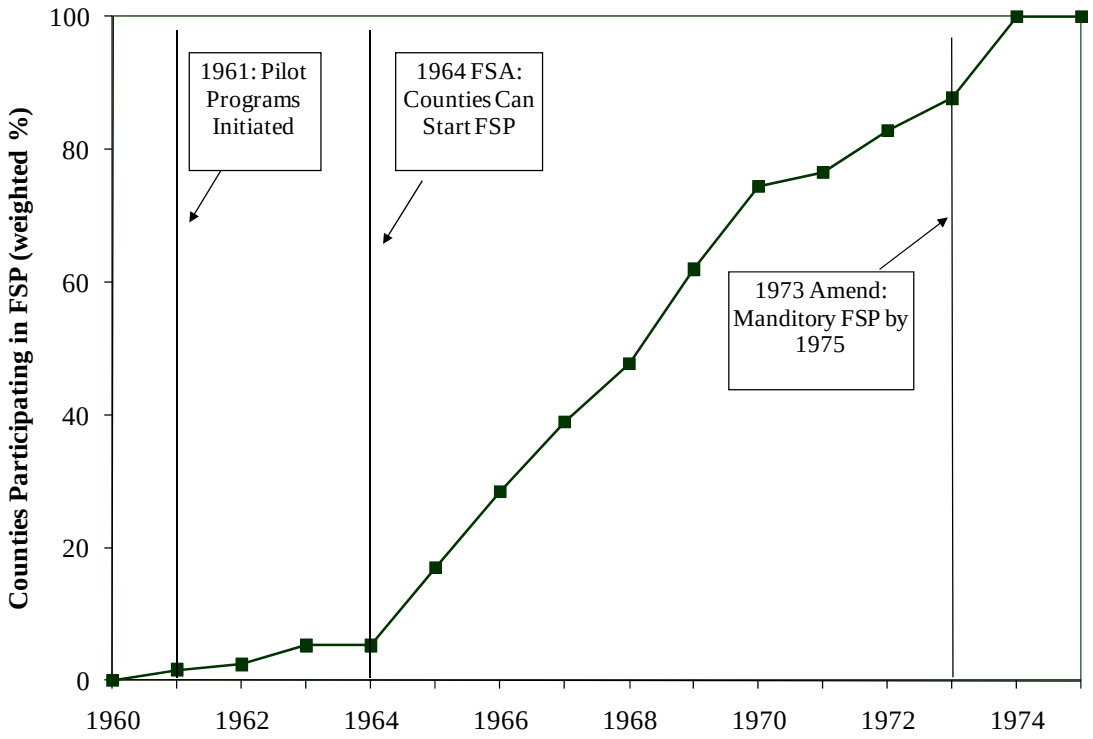
Snyder, Stephen E and Evans, William N, "The Effect of Income on Mortality: Evidence from the Social Security Notch", *Review of Economics and Statistics* (2006), pp. 482-495

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.

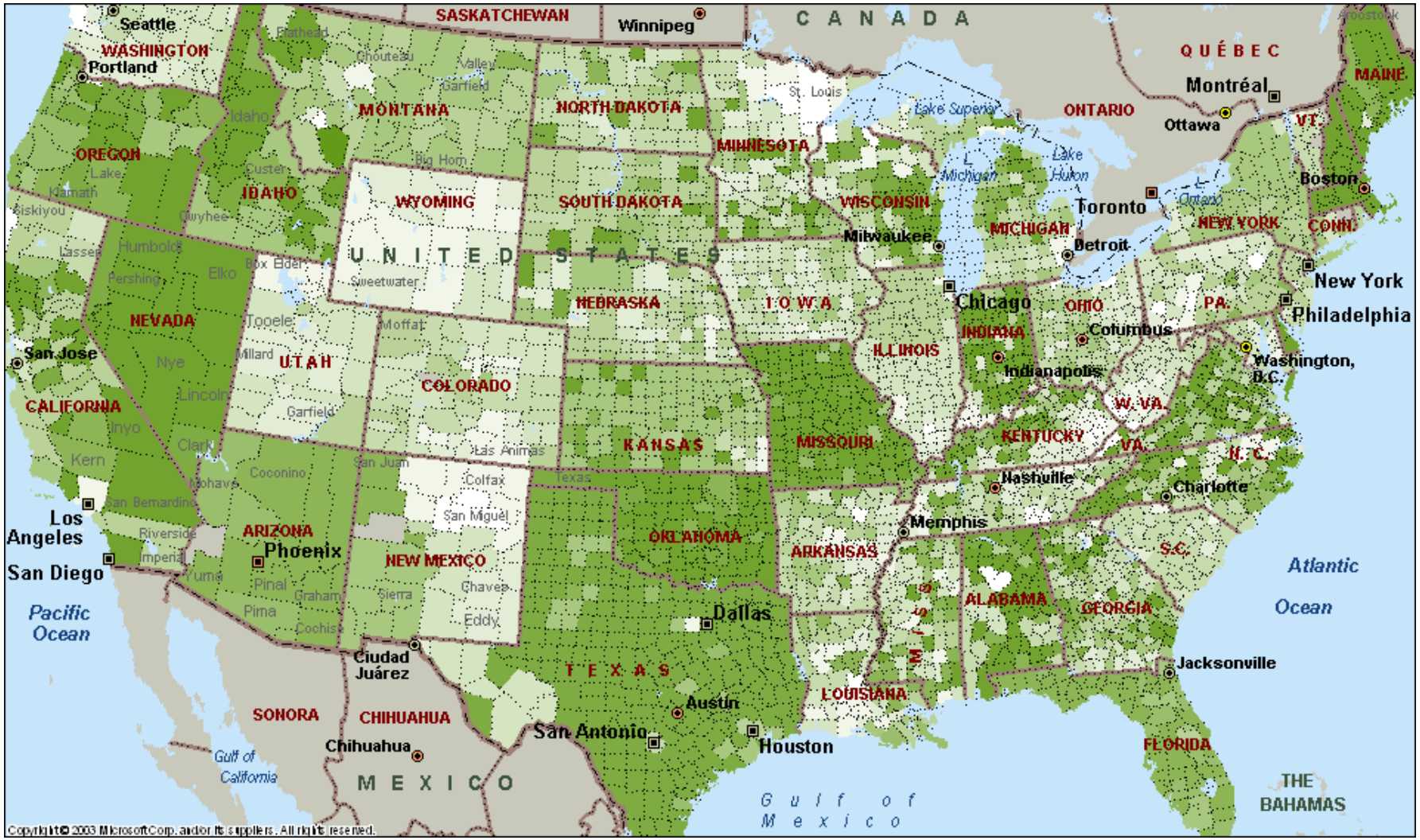
Van Den Berg, Gerard J., Maarten Lindeboom and France Portrait, "Economic Conditions Early in Life and Individual Mortality", *American Economic Review* (2006), pp. 290-302.

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



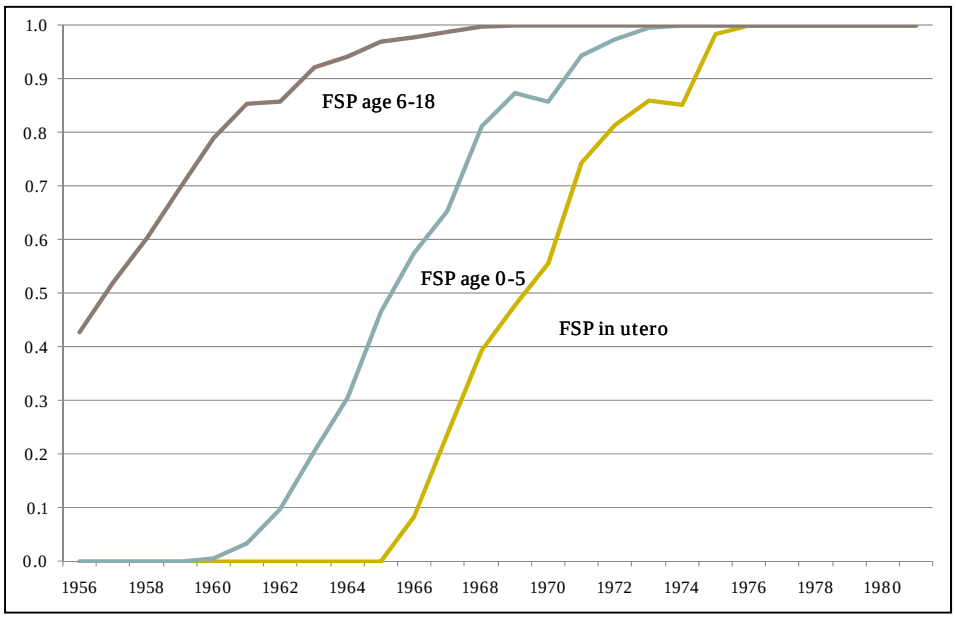
Source: Authors' tabulations of food stamp administrative data (U.S. Department of Agriculture, various years). Counties are weighted by their 1960 population.

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 Adult Health
Impact as a function of year FSP implemented in county of birth

Notes: Each 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 female headed households. The specification also includes all controls given in Table 3.

Table 1
Descriptive Statistics

	Full Sample		Low Education Sample	
	N	Mean	N	Mean
FS share age 0-5	51173	0.339	24157	0.310
Health Outcomes				
In good health =1	51059	0.689	24099	0.594
Disabled =1	51057	0.093	24094	0.113
Diabetes =1	16344	0.037	6720	0.045
High blood pressure =1	16344	0.122	6720	0.173
Obesity =1	17855	0.228	7546	0.309
Healthy weight =1	17855	0.423	7546	0.333
BMI	17855	26.659	7546	28.041
Body weight (pounds)	17860	174.9	7549	182.254
Height (inches)	18185	67.7	7691	67.458
Economic Outcomes				
Education High School Plus	41454	0.903	19625	0.798
Log(Total Fam Income)	41273	10.847	19500	10.519
Earnings (including 0s)	40346	35047	18936	24685
Employed =1	41454	0.864	19625	0.838
Poverty = 1	41273	0.184	19500	0.306
Demographics				
Male	51173	0.457	24157	0.440
Nonwhite	51173	0.167	24157	0.313
High School Grad	51173	0.396	24157	0.506
Greater than High School	51173	0.493	24157	0.275
Age	51173	31.515	24157	31.453
Maried	51173	0.638	24157	0.610
Family Background				
Female headed household	51173	0.090	24157	0.152
Income to needs ratio (5-yr average)	51173	2.381	24157	1.523
Head less than high school education	51173	0.342	24157	1.000
1960 County Characteristics				
Population	51173	555,141	24157	461,137
Fraction of land, farmland	51108	49.7	24097	51.0
Fraction of population, urban	51173	65.7	24157	59.9
Fraction of population, black	51173	9.3	24157	13.6

Notes: Author's tabulations of 1968-2007 PSID. Observations from Alaska are dropped because of missing data on food stamp program start date. For details on sample selection see text.

Table 2
Health Outcomes for Full Sample, Difference in Difference

			"Metabolic Syndrome"						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	In good health =1	**Disabled =1**	**Diabetes =1**	**High blood pressure =1**	**Obesity =1**	**Healthy weight =1**	**BMI**	**Body weight**	**Height**
FS share age 0-5	0.041	-0.003	-0.001	-0.062	-0.088	0.108	-1.90	-12.23	-0.077
	(0.037)	(0.020)	(0.019)	(0.040)	(0.04)**	(0.050)**	(0.71)**	(5.07)**	(0.384)
Inflated by takeup rate	*0.408*	*-0.032*	*-0.008*	*-0.619*	*-0.877*	*1.080*	*-19.0*	*-122.3*	*-0.765*
Y-mean	0.69	0.09	0.04	0.12	0.23	0.42	26.66	174.95	67.73
Observations	50995	50993	16335	16335	17842	17842	17842	17847	18172
R-squared	0.13	0.08	0.11	0.14	0.19	0.22	0.25	0.38	0.62
Demographics	X	X	X	X	X	X	X	X	X
Family Background	X	X	X	X	X	X	X	X	X
Year of Birth fixed effects	X	X	X	X	X	X	X	X	X
County of birth fixed effects	X	X	X	X	X	X	X	X	X
Interview year FE	X	X	X	X	X	X	X	X	X
State * linear time in YOB	X	X	X	X	X	X	X	X	X
cb60 * linear time in YOB	X	X	X	X	X	X	X	X	X

Notes:

Table 3
Economic Outcomes for Full Sample, Difference in Difference

	(1)	(2)	(3)	(4)
	Educational Attainment: High School Plus	**log(Family total income)**	**Employed =1**	**Poverty =1**
FS share age 0-5	0.056	-0.039	0.006	0.000
	(0.045)	(0.071)	(0.028)	(0.032)
Inflated by takeup rate	*0.563*	*-0.394*	*0.063*	*0.005*
Y-mean	0.9	10.85	0.86	0.18
Observations	41397	41863	42047	41863
R-squared	0.24	0.37	0.09	0.23
Demographics	X	X	X	X
Family Background	X	X	X	X
Year of Birth fixed effects	X	X	X	X
County of birth fixed effects	X	X	X	X
Interview year FE	X	X	X	X
State * linear time in YOB	X	X	X	X
cb60 * linear time in YOB	X	X	X	X

Notes:

Table 4
Health Outcomes: Head of Household in Early Childhood has Low Education

			"Metabolic Syndrome"					
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	In good health =1	**Disabled =1**	**Diabetes =1**	**High blood pressure =1**	**Obesity =1**	**Healthy weight =1**	**BMI**	**Body weight**
FS share age 0-5	0.072	0.006	-0.013	-0.083	0.187	-14.07	-2.413	-0.177
	(0.065)	(0.035)	(0.041)	(0.082)	(0.845)	(9.646)	(1.14)**	(0.08)**
Inflated by takeup rate	***0.35***	***0.03***	***-0.06***	***-0.40***	***0.91***	***-68.63***	***-11.77***	***-0.86***
Y-mean	0.59	0.11	0.05	0.17	67.46	182.26	28.04	0.31
Observations	24039	24034	6715	6715	7682	7540	7537	7537
R-squared	0.16	0.13	0.19	0.22	0.64	0.43	0.34	0.26
Demographics	X	X	X	X	X	X	X	X
Year of Birth fixed effects	X	X	X	X	X	X	X	X
County of birth fixed effects	X	X	X	X	X	X	X	X
Interview year FE	X	X	X	X	X	X	X	X
State * linear time in YOB	X	X	X	X	X	X	X	X
cb60 * linear time in YOB	X	X	X	X	X	X	X	X

Notes:

Table 5
Economic Outcomes: Head of Household in Early Childhood has Low Education

	(1)	(2)	(3)	(4)	(5)
	Educational Attainment: High School Plus	**log(Family total income)**	**Earnings**	**Employed =1**	**Poverty =1**
FS share age 0-5	0.169	0.145	1605	0.012	-0.043
	(0.110)	(0.157)	4425	(0.053)	(0.067)
Inflated by takeup rate	*0.824*	*0.706*	*7830*	*0.058*	*-0.212*
Y-mean	0.8	10.52	24654	0.84	0.31
Observations	19572	19650	19065	19776	19650
R-squared	0.27	0.4	0	0.15	0.3
Demographics	X	X	X	X	X
Year of Birth fixed effects	X	X	X	X	X
County of birth fixed effects	X	X	X	X	X
Interview year FE	X	X	X	X	X
State * linear time in YOB	X	X	X	X	X
cb60 * linear time in YOB	X	X	X	X	X

Notes:

Table 6
Placebo: Head of Household in Early Childhood has High Education

		"Metabolic Syndrome"					Economic Outcomes		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	In good health =1	Diabetes =1	High blood pressure =1	Obesity =1	BMI	Body weight	High School Plus	log(Family total income)	Employed = 1
FS share age 0-5	0.006	0.002	-0.061	-0.016	-0.799	-5.4	-0.031	-0.021	0.109
	(0.060)	(0.029)	(0.058)	(0.082)	(1.165)	(9.7)	(0.049)	(0.122)	(0.038)
Y-mean	0.78	0.02	0.07	0.15	25.35	168	0.98	11.15	0.88
Observations	11,555	4,363	4,364	4,638	4,638	4,638	9,534	9,789	9,810
R-squared	0.16	0.22	0.19	0.30	0.40	0.55	0.34	0.35	0.12
Demographics	X	X	X	X	X	X	X	X	X
Year of Birth fixed effects	X	X	X	X	X	X	X	X	X
County of birth fixed effects	X	X	X	X	X	X	X	X	X
Interview year FE	X	X	X	X	X	X	X	X	X
State * linear time in YOB	X	X	X	X	X	X	X	X	X
cb60 * linear time in YOB	X	X	X	X	X	X	X	X	X

Notes:

Table 7
Health Outcomes: Triple Difference Specification

			"Metabolic Syndrome"				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	In good health =1	Disabled =1	Diabetes =1	High blood pressure =1	Obesity =1	BMI	Body weight
FS Share age 0-5 * Pg	0.186**	-0.071	-0.152*	-0.195*	-17.000	-2.469	-0.246**
	(0.091)	(0.056)	(0.093)	(0.100)	(14.605)	(1.779)	(0.120)
FS share age 0-5	0.026	0.002	0.010	-0.047	-11.90**	-1.87**	-0.079*
	(0.040)	(0.022)	(0.019)	(0.041)	(5.449)	(0.756)	(0.046)
Y-mean	0.69	0.09	0.04	0.12	174.91	26.66	0.23
Observations	50,673	50,671	16,251	16,251	17,750	17,745	17,745
R-squared	0.13	0.09	0.11	0.15	0.39	0.26	0.2
Demographics	X	X	X	X	X	X	X
Year of Birth fixed effects	X	X	X	X	X	X	X
County of birth fixed effects	X	X	X	X	X	X	X
Interview year FE	X	X	X	X	X	X	X
State * linear time in YOB	X	X	X	X	X	X	X
cb60 * linear time in YOB	X	X	X	X	X	X	X
Group dummy FE	X	X	X	X	X	X	X

Notes:

Table 8
Economic Outcomes: Triple Difference Specification

	(1)	(2)	(3)	(4)	(5)
	High School Plus	log(Family total income)	Earnings	Employed =1	Poverty =1
FS Share age 0-5 * Pg	-0.086	0.582**	10718	0.038	-0.183*
	(0.108)	(0.254)	(13386.4)	(0.079)	(0.104)
FS share age 0-5	0.083	-0.055	-7634	0.004	0.004
	(0.046)	(0.072)	(4564.0)	(0.028)	(0.031)
Y-mean	0.90	10.85	35131	$0.86	$0.18
Observations	41,115	41,569	40,617	41,750	41,569
R-squared	0.24	0.37	0.11	0.09	0.24
Demographics	X	X	X	X	X
Year of Birth fixed effects	X	X	X	X	X
County of birth fixed effects	X	X	X	X	X
Interview year FE	X	X	X	X	X
State * linear time in YOB	X	X	X	X	X
cb60 * linear time in YOB	X	X	X	X	X
Group dummy FE	X	X	X	X	X

Notes: