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Early Childhood Poverty and Inflammation in Adulthood

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**Abstract**

Research on the association between childhood poverty and adult cardiovascular illness has suggested that inflammation may be a key mediator explaining the differences in cardiovascular risk. A limitation of this literature is that studies have primarily examined childhood economic status retrospectively or at only one point in time. This study uses data (n = 919) from the US Panel Study of Income Dynamics (PSID) to examine the relationship between poverty in childhood and adult reports around the age of 30-37 of hypertension. It further examines the mediating role of contemporaneous health characteristics and adult socioeconomic status. Results from logistic regressions, which include extensive controls (including parents' health conditions) demonstrate that duration of poverty in childhood is significantly associated with hypertension and diabetes in adulthood. Moreover, we show that, for low-income children, higher income in the years birth to five is associated with a significantly lower likelihood of hypertension and arthritis in adulthood. These results suggest that the stresses associated with childhood poverty may induce a premature aging of the immune response.

## Early Childhood Poverty and Inflammation in Adulthood

### Introduction

Inflammation is an integral part of the human stress and immune response. The immune system is the two-pronged mechanism by which the human body protects itself from foreign microbes. The primary component, or the innate immune system, serves as a non-specific defense system against pathogenic or malignant particles. The adaptive immune system acts as the second component and has evolved to specifically target individual antigenic threats to the host system. Tight regulation of these processes is required in order to effectively protect the host system from potentially harmful cells.

Researchers had until recently described inflammation as a single, acute process involving the immune system and characterized by heat, pain, swelling, and redness. Yet recent data have shown that inflammation can also comprise a chronic process, albeit operating at lower than acute levels (NIA, 2004). Shifts in homeostasis can occur due to infections (e.g. human immunodeficiency virus), but can also occur as a function of the social environment (Cohen et al, 2004; McEwen & Dhabhar, 2002; Miller & Chen, 2007; Prescott et al, 1999; Ramsay et al, 2007). More specifically, recent research in psychoneuroimmunology suggests that psychological stress associated with poverty may act to induce dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, thereby resulting in attenuation and activation in the innate and/or adaptive immune systems.

Inflammatory processes have been implicated in the development of a range of chronic diseases and conditions of aging. Although most studies demonstrating associations with inflammation have focused primarily on cardiovascular disease, recent investigations have shown links between inflammation and osteoarthritis, osteoporosis, Alzheimer's disease, muscle

wasting and frailty, cancer, insulin resistance and diabetes, and rheumatoid arthritis – diseases or conditions which are primarily associated with aging (NIA, 2004). Life-long inflammatory burden may shape later life patterns of aging and mortality (Crimmins & Finch, 2006). It is thus critically important to identify the factors that contribute to inflammatory processes in adulthood, and there is reason to believe that early life conditions, especially those that impose chronic environmental demands, play an important role. Chief among such early life conditions is poverty.

### **Conceptual framework**

Early childhood income is a key predictor of later life human capital outcomes (Duncan, Ziol-Guest, & Kalil, 2009; Evans & Schamberg, 2009; Ziol-Guest, Duncan, & Kalil, 2010). However, it is uncertain whether low-income during this childhood period, versus other stages of childhood, plays a uniquely important role in predicting health conditions in adulthood. If we can identify the specific associations between income in childhood and adult health, there may be a greater chance of implementing targeted interventions for low-income children in the United States.

Why might early childhood (including the prenatal years) income predict inflammation-related conditions in adulthood? Several theoretical frameworks support this association. The “fetal origins hypothesis” posits a programming process whereby stimulants and insults during the prenatal period have long-lasting implications for physiology and disease risk (Strauss, 1997). Chronic stress from growing up poor could also play a role in dysregulation across multiple physiological systems whose effects persist (or possibly compound) into adulthood (Evans & Schamberg, 2009). For example, Evans and Schamberg (2009) showed that childhood poverty increases children’s allostatic load, a biological index of the cumulative wear and tear on

the body. Moreover, the longer the children had lived in poverty, the higher their allostatic load (Evans & Schamberg, 2009). Allostatic load is caused by the mobilization of multiple physiological systems in response to chronic stresses in the environment. In this fashion, childhood poverty may actually “reset” the immune system such that important processes, such as inflammation, become dysregulated, resulting in higher levels and prolonged production of proinflammatory cytokines.

Other pathways could include the impact of low-income in childhood on educational attainment or occupational status. Low-income children attain less education as adults, and education is an important determinant of health in part through knowledge of healthy diet and health-inducing behaviors (Duncan et al, 1998). Low-income children are also more likely as adults to work in lower-status occupations, where social norms about healthy eating and exercise habits may be less strong, or where opportunities for on-site exercise and healthy eating are less prevalent (McLaren, 2007). Low income in childhood may also be related to the early development and persistence of different social norms of eating, exercise, and overweight.

Finally, low income in early childhood has been linked to poor physical and mental health in adulthood. Clearly, physical limitations may constrain the physical activity necessary for good health. Poor mental health, such as depression, may lead to overeating, physical inactivity, or stress, all of which are associated with the development of disease. Stress and depression can directly alter inflammatory cytokine production (Kiecolt-Glaser, cite). Conversely, physical activity may help attenuate chronic inflammatory processes. Finally, childhood poverty may influence adult inflammation through its influence on behavioral risk factors such as poor diet or high body mass index, smoking, and alcohol consumption, all of which affect inflammatory responses (Kiecolt-Glaser, cite).

## Data and measures

This study uses data from the Panel Study of Income Dynamics (PSID), which has followed a nationally representative sample of about 5000 families and their children since 1968 (<http://psidonline.isr.umich.edu>). Annually from 1968 to 1996 and biennially from 1997 to 2005, all members of the original household in the study, including children born into the study, were surveyed. The PSID is the longest-running longitudinal study of income in the United States and collects detailed economic and demographic information across the lifecourse.

Our target study sample consisted of the 1,296 individuals who were (1) heads of households or the wives in households in the 2005 PSID as only heads and wives responded to health questions in the 2005 survey, and (2) were born into the PSID households between 1968 and 1975 and are thus considered PSID sample members. The 1968 to 1975 birth cohorts were chosen so that lifetime household economic status could be observed between the prenatal year and into adolescence, and so that respondents could be observed well-into adulthood. The analytical sample consisted of 919 respondents who participated in the 2005 survey, had valid responses for at least one of the inflammation questions, and were not missing data on control variables or income between the prenatal year and age 15. All analysis is weighted using the PSID attrition-adjusted weights, eliminating the difference between the sample of children born between 1968 and 1975 who were not observed in 2005 and the analysis sample.

### *Dependent Variables*

The PSID provides data on several adult diseases and conditions in which inflammatory processes have been implicated. Two of these variables relate to the presence of reported hypertension. Respondents are asked whether or not a doctor has ever told them that they have high blood pressure or hypertension. Respondents then report how much this condition limits

their normal daily activities. Based on these questions we classify respondents in two ways. First, we use an indicator for whether or not they have hypertension. Second, we classify respondents as having daily limiting hypertension if they report that high blood pressure limits their normal daily life a lot, somewhat or just a little. Those without hypertension and those who indicate that their high blood pressure does not affect their daily life are considered not having a limiting condition.

In addition, respondents report on the presence of a diagnosis of arthritis and diabetes. Specifically, they indicate if a doctor has ever told them they have (1) arthritis or rheumatism and (2) diabetes or high blood sugar. We create two indicators that signify whether or not they have arthritis or diabetes.

#### *Childhood socioeconomic status*

We measure children's socioeconomic status in several different ways. For all computations we used the PSID's high-quality edited measure of annual total family income, inflated to 2005 levels using the Consumer Price Index. We examine both the respondent's prenatal to age 15 socioeconomic status; as well as experiences across three periods: the prenatal year through the calendar year in which the child turned five; ages six to ten; and ages 11 to 15.

First, we assessed the experience of poverty during childhood by using the provided income as well as needs (calculated based on family size) to create an income-to-needs measure. We then determined whether or not the respondent was in poverty (income-to-needs less than one) in each year of life; and average the number of years they are poor in each of the childhood periods as well as across childhood.

Second, we averaged the annual income measures provided by the PSID in the prenatal to age 15 period, as well as across three childhood periods. To account for a possible differential

impact of increments to low as opposed to higher family income, in our multivariate models we also allowed the coefficients on average income within each childhood period to have distinct linear effects for average incomes up to \$25,000 and for incomes \$25,000 and higher.

### *Demographics*

To avoid attributing to income what should be attributed to correlated determinants of both childhood income and our outcomes of interest, several important demographic characteristics taken around the time of the child's birth are included. We include dummy variables for seven of the eight birth years, a dichotomous variable for race (*non-white=1*), a dichotomous variable for gender (*male=1*), a dichotomous variable for whether the child's mother was married at the time of his or her birth (*married=1*), and a dichotomous variable representing whether the child was born in the south (*south=1*). Age of the mother is a continuous variable ranging from 14 to 45, and the number of siblings is the total number of births to the child's mother minus one and ranges from zero to 12. A dichotomous variable representing whether the child was the first birth of the mother (*first=1*) was also included. Finally three characteristics of the head of the household (usually the father in two-parent households or the mother in single-parent households) in which the child was born are included. These measures include years of completed schooling, the head's score on a sentence completion test administered in the 1972 interview, and an indicator of whether the head has a physical or nervous condition that limits the kind or amount of work he can do.

### *Parent Health Variables*

We also include several measures that account for the health of the respondent's parents also in 2005. Doing so may allow us to control for genetically-mediated transmission of health status across generations that are independent of children's early poverty. These measures for

parents include body mass index, heart disease, and hypertension. We use the respondent's mother's information if available, and if not then the father's information. If neither parent participated in the 2005 survey we recode these variables to zero and include flags for those missing parental reports.

### *Adult Characteristics*

We also include several measures of current behavior and health status through which the association between childhood income and adult hypertension may operate. These include four dichotomous behavior measures including exercising, smoking, drinking, and obesity. The data are coded to indicate whether or not the respondent reported doing any vigorous physical activities, any light or moderate physical activities, or any physical activities to strengthen muscles such as lifting weights (*exercise=1*). Whether or not the respondent smokes is ascertained from one question noting whether they smoke cigarettes, and respondents are coded as drinking alcohol if they report on drinking in the past year. Additionally, a respondent's body mass index in 2005 was coded as obese ( $>30$ ) or not. Finally, a control for current socioeconomic status is included. This measure is the natural log of total household income from all sources.

### *Analysis plan*

Three models were estimated with logistic regressions to test the association between poverty and income throughout childhood and adult inflammation. Model 1 includes the demographic control variables, parental health, and the varying measures of poverty. Model 2 includes the demographic control variables, parental health, and the income measures in the childhood periods instead of poverty. Model 3 includes the demographic control variables, parental health, and the spline models. Model 4 introduces the adult behaviors, and finally Model 5 includes the current socioeconomic status.

All of our logistic regressions were run in STATA 10.0 SE, use the PSID's weights to adjust for differential sampling fractions and attrition, and adjust for origin-family clustering on the mother using Huber-White methods. To facilitate their interpretation, logistic regression coefficients and standard errors are expressed in the tables in the form of marginal effects (and their associated standard errors) on the probabilities of the given event occurring. These are computed using the MFX command in STATA.

## **Results**

### *Sample Description*

Means or proportions and standard deviations of each of our control and outcome variables are presented in Table 1. These summary statistics are weighted using the 2005 PSID-provided weight. Descriptive statistics are presented for children whose prenatal-to-age-5 incomes averaged: i) below the official poverty line; ii) between one and two times the poverty line; and iii) more than twice the poverty line. The final column of the table provides information on the statistical significance (at  $p < .05$  or below, two-tailed test) of the mean differences across the three groups.

As shown in Table 1, there are significant differences in the control variables measured around the birth depending on whether childhood income prior to age six was below, close to, or well above the poverty line. Compared with children whose families had incomes of at least twice the poverty line during their early childhood, poor children are more likely to be nonwhite, born to an unmarried mother, born in the south, and have younger mothers and more siblings. Similarly, poor children's head of household scored significantly lower on the IQ test in 1972, had lower educational attainment, and was more likely to report a work-limiting disability. Not

surprisingly, children with average annual incomes below poverty in the earliest period have lower average income in all three periods compared with the other two groups.

Among the parental health measures, only body mass index differed across the groups, with the near poor experiencing the highest BMI. The majority of respondents across all three groups reported at least some exercise, although those in poverty in early childhood were less likely to report exercising. Further, the poorest were also least likely to report drinking alcohol as well. Current socioeconomic status also differed depending on the respondents poverty status in the prenatal to age 5 period, with the poorest respondents earning \$50,000 less on average than their non-poor counterparts 30 years later.

Those in poverty, on average, between their prenatal and birth year, spent 72% of their childhood (between prenatal and age 15) in poverty, whereas 17% and 2% of the near-poor and non-poor did respectively. Similarly, those who were poor on average in the prenatal to age 5 period were likely to spend the majority of the other periods also in poverty. On average, those who were poor in the first childhood period had family incomes of \$25,000 per year on average between prenatal year and age 15, compared to an average of \$78,000 among the non-poor.

The outcomes differed depending on whether the child experienced poverty in early childhood, as those who did had significantly higher reported levels of hypertension and arthritis. They did not differ in reports of diabetes or whether hypertension limits the ability to work.

### *Multivariate Results*

Table 2 presents the findings from Model 1, which controls for the birth characteristics, the parental health measures, and the poverty measures. Findings suggest that males are more likely to report hypertension, and that higher parental body mass index is also associated with greater likelihood of hypertension. In addition, poverty in childhood is also associated with

hypertension. Specifically, spending a larger proportion of childhood in poverty is associated with higher rates of hypertension, and this association is strongest for poverty experienced in early childhood. However, the current behavior and income conditions do not appear to mediate the association between number of early childhood years in poverty and adult hypertension.

The findings for arthritis point to the role of maternal age, as well as head of household characteristics around the time of birth. Poverty is not significantly associated with arthritis in these models. Being born in the south and higher household head education are associated with reduced rates of diabetes, whereas being born to a married mother is associated with increased rates.

Poverty in childhood is significantly associated with diabetes, although the findings for the three separate childhood periods do not indicate that experiences in any one childhood period are driving this result. The current behavior and income conditions appear to account for about one-third of association between number of years in poverty across all childhood years and adult diabetes. Finally, experiences with poverty across childhood (but again, not in any one period) is associated with increased reports that hypertension is limiting in adulthood. Here again, the current behavior and income conditions appear to account for about one-third of association between number of childhood years in poverty and limiting hypertension.

We next turn from an analysis of the association of the duration of poverty in childhood to an analysis of the association of income in different childhood periods with later life health measures. In these analyses we examine income in a linear fashion and also whether the role of income is different at different places in the income distribution. Table 3 presents the findings from Model 2, specifically controlling for the characteristics around the birth, the parental health characteristics, and income measured in the prenatal to age 15 period (both continuous and

logged) and Table 4 presents the findings from Model 2, but controls for income measured in the three different childhood periods (both continuous and logged). There is very little in the way of consistent findings across the models within each outcome.

Table 5 presents the findings from Models 3, 4, and 5 for each outcome. Although not presented in the tables, all models control for demographic characteristics around the birth as well as the parental health measures. Findings for hypertension suggest that higher levels of income in the earliest childhood period for low-income children are associated with significantly lower likelihood of hypertension in adulthood. These findings are essentially unchanged when current health behaviors and current socioeconomic status is included in the model, although obesity and smoking are independently associated with increased hypertension.

As in the regression for hypertension, higher levels of income in the earliest childhood period for low-income children are significantly associated with a lower risk of arthritis. Inclusion of current health behaviors reduces the size of the effect by about 50%. The inclusion of current income does little to explain the association once current health behaviors are accounted for, although higher income in adulthood is also associated with a lower risk of arthritis.

Findings for diabetes do not support our hypothesis for the role of early childhood income; rather, these findings suggest that increases to income in late childhood, and only for non-poor children, are associated with a reduced risk of diabetes. Current behaviors and income do not mediate this relationship, however. Finally, there are no significant findings for activity-limiting hypertension.

## **Discussion**

Acute inflammation, which involves a number of feedback mechanisms regulating homeostasis, is hypothesized to confer necessary short-term benefits at the expense of long-term damage; aging represents the long-term accumulation of damage from repeated episodes of acute inflammation. The outcomes we have examined here (hypertension, diabetes, arthritis) are typically considered diseases of old age. Yet, we have shown that 25% of adults ages 30-37 who were poor in early childhood had hypertension; almost 7% suffered from arthritis and over 4% had diabetes. These rates are at least twice the rate of those who were not poor in early childhood. In short, it may be that the stresses associated with early childhood poverty induce a premature aging of the immune response.

Our multivariate analysis suggests that most of these associations hold up in the face of a rich set of controls, including similar health conditions of respondents' parents, which may be a proxy for hereditary risk. Specifically, we found that the number of years of childhood spent in poverty is associated with a significantly increased risk of hypertension, work-limiting hypertension, and diabetes. This set of results did not show a particular role for years in poverty in any one childhood period.

However, our analyses that examined the role of increases to income at different points in the income distribution tell a more nuanced story. Here, we find that for low-income children (but not their higher-income counterparts), higher income during early childhood (but not other stages of childhood) is related to a lower risk of adult hypertension and arthritis. This corresponds to a number of recent studies showing that income plays a more potent role in later life outcomes for those at the bottom end of the income distribution (e.g., Duncan et al., 2010; Loken, Mogstad, & Wiswald, 2010).

Our analysis of the pathways through which early childhood poverty is associated with inflammation in adulthood was only partially revealing. We were not able to account for any of the association between level of income in early childhood (for poor children) and later-life hypertension with any of our measures of current behavior or current income. This suggests that such a channel may operate at the physiological realm, whereby elevated chronic stress in childhood contributes to cumulative physiological wear and tear on the body, leading to the early occurrence of a disease typically associated with aging. Our finding of the particular importance of income during the early years for adult hypertension and arthritis is consistent with the hypothesis that these years represent a sensitive period during which social processes become embedded in biology and that epigenetic modifications could be responsible for these associations.

In contrast, with respect to the association between income in early childhood (again, for poor children) and later-life arthritis, we find that approximately half of this association is accounted for by current measures of exercise and obesity. Our previous work has shown a significant association between early childhood income and obesity (Ziol-Guest et al., 2009); obesity is a known risk factor for the development of arthritis (and this association can be seen in our data also). Our analyses here thus fill in an additional piece of information in this chain of events.

Finally, regarding the mediators of the duration of exposure to poverty across childhood, we found that measures of current behavior and income accounted for about one-third of the effect on diabetes and limiting hypertension, but none of the effect on risk of hypertension itself. These findings, too, support a hypothesis that the association of childhood economic conditions

with a diagnosis of hypertension in adulthood may operate through physiological, rather than behavioral, factors.

Our data offers many advantages relative to existing studies. A major limitation of many existing studies in this area is their reliance on retrospective measures of family income, small or non-representative samples, or indicators of childhood economic conditions (such as “fathers’ occupation”) that are not obviously amenable to public policy intervention. The prospective and high-quality measures of income in the PSID thus represent a major advantage. At the same time, although the PSID is well suited to estimating the unique contribution of early childhood income to adult health conditions, it lacks key mediational measures during childhood, such as childhood health conditions, physical activity, stress exposure, health risk behaviors, health status of the mother at the time of the child’s birth, and breastfeeding practices, all of which might help provide an understanding of the process by which early economic conditions matter for later life health. Despite this limitation, existing literature about the role of these childhood measures finds that they are associated with children's health but do not explain the relationship between socioeconomic status and health.

A second limitation reflects the age of the respondents in the PSID at the time we observe their outcomes. Hypertension, arthritis, and diabetes are typically considered diseases of a population older than the 30-37-year olds we observe here. We thus may have lacked power to detect the true associations. If this is the case, then our results can be taken as a lower-bound estimate. Moreover, it is likely that these and other physiological markers of adaptation to environmental demands are inter-related and co-occurring within individuals. Their combined effect is likely to be more predictive of morbidity and mortality; however, such disease risk profiles would be difficult to detect in a sample as relatively young as ours.

All else equal, our results suggest that raising the average income of low income children by \$10,000 per year during each year from birth to age five would decrease their risk of hypertension in adulthood by about 9 percentage points. Although this would be a sizeable income increase given the average income of poor families, the reduction in risk for hypertension would also be sizeable (i.e., 36%), given the 25% rate of hypertension among those adults who were poor in early childhood.

These findings thus point to the potential importance of income transfers, particularly investments in early childhood, on influencing eventual health. In particular, our findings indicate that we should focus our efforts on those who are the most economically disadvantaged. Virtually all countries have a variety of tax and transfer programs that redistribute income. In the United States, the Earned Income Tax Credit pays up to \$4400 per year to low-income working families, the Temporary Assistance for Needy Families program provides cash grants to low-income families for limited periods, and the child tax credit grants a nonrefundable credit of up to \$1000 per year per child (information from the 2006 tax year). Taking as a realistic example an income transfer on the order of \$3,000, the resulting decrease in the risk of hypertension would be 3 percentage points, or about a 12% decrease. Targeting these transfers, or similar programs, to families with the youngest children may offer the largest benefit for later-life health and well-being, and this may be especially important in an era of rapidly-rising health care costs.

Table 1  
*Weighted Descriptive Statistics by Prenatal to Age 5 Poverty Status*

	Poor (<100%)		Near Poor (100%-200%)		Not Poor (>200%)		p-value
	Mean or %	SD	Mean or %	SD	Mean or %	SD	
<i><u>Variables around birth</u></i>							
Non-white	65.66%	0.48	24.99%	0.43	10.34%	0.30	$p < .001$
Male	49.03%	0.50	46.61%	0.50	52.20%	0.50	$p = .224$
Born to married mother	47.79%	0.50	82.65%	0.38	94.76%	0.22	$p < .001$
Born in the south	45.31%	0.50	36.16%	0.48	27.21%	0.45	$p < .001$
Age of mother at birth	22.95	6.95	24.04	5.64	25.53	5.16	$p < .001$
Number of siblings	3.99	3.32	2.39	1.43	1.75	1.16	$p < .001$
First born	37.92%	0.49	30.92%	0.46	43.41%	0.50	$p < .001$
Head of household IQ score (1972)	7.66	2.26	9.43	1.90	10.26	1.83	$p < .001$
Head of household education	8.99	3.39	11.09	2.50	13.41	2.39	$p < .001$
Head of household work limiting disability	33.88%	0.48	13.15%	0.34	6.21%	0.24	$p < .001$
<i><u>Parental health in 2005 (non-missing cases)</u></i>							
Body mass index	28.92	5.71	29.34	6.79	27.02	5.56	$p < .01$
Hypertension	42.93%	---	47.66%	---	38.61%	---	$p = .530$
Heart disease	20.07%	---	13.28%	---	8.67%	---	$p = .856$
<i><u>Adult characteristics in 2005</u></i>							
Exercise	86.38%	---	83.16%	---	95.06%	---	$p < .001$
Smoke	31.00%	---	26.91%	---	21.82%	---	$p = .547$
Drink alcohol	53.78%	---	64.93%	---	79.18%	---	$p < .001$
Obese	45.05%	---	31.93%	---	25.63%	---	$p = .006$
Income (10,000 2005\$)	3.32	2.65	5.68	5.20	8.59	8.50	$p < .001$
<i><u>Childhood socioeconomic status</u></i>							
<i>Poverty (% years)</i>							

Prenatal to age 15	71.85%	0.23	17.14%	0.20	2.20%	0.07	$p < .001$
Prenatal to age 5	80.07%	0.15	15.93%	0.20	1.13%	0.04	$p < .001$
Age 6 to age 10	68.68%	0.37	18.32%	0.28	2.34%	0.10	$p < .001$
Age 11 to age 15	63.50%	0.39	17.67%	0.29	3.55%	0.14	$p < .001$
<i>Income (10,000 2005\$)</i>							
Prenatal to age 15	2.52	1.27	4.35	1.55	7.84	3.93	$p < .001$
Prenatal to age 5	2.42	1.23	3.68	1.10	7.06	3.47	$p < .001$
Age 6 to age 10	2.58	1.67	4.62	2.11	8.01	4.78	$p < .001$
Age 11 to age 15	2.60	1.65	4.99	2.67	8.76	5.34	$p < .001$
<i>Outcomes</i>							
Hypertension	24.50%	---	9.75%	---	9.40%	---	$p < .001$
Arthritis	6.70%	---	6.63%	---	3.42%	---	$p < .05$
Diabetes	4.14%	---	5.88%	---	1.77%	---	$p = .128$
Hypertension limits ability to work	4.07%		2.01%		1.79%		$p = .665$

*Note:* Parental health measures are the mother's measures. If mother's measure is not available, then father's measure is used.

Table 2  
*Logistic Regressions on 2005 Heads and Wives: Poverty Measures*

					Hypertension					
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>
<i><u>Variables around birth</u></i>										
Non-white	0.019		0.036		0.014		0.035		0.018	
Male	0.081	**	0.023		0.079	**	0.024		0.074	***
Born to married mother	-0.038		0.053		-0.035		0.052		-0.022	
Born in the south	-0.011		0.024		-0.010		0.024		-0.009	
Age of mother at birth	-0.001		0.003		-0.001		0.003		0.000	
Number of siblings	0.000		0.007		-0.001		0.007		0.001	
First born	0.026		0.026		0.026		0.026		0.022	
Head of household IQ score (1972)	-0.002		0.005		-0.002		0.006		-0.001	
Head of household education	0.005		0.005		0.005		0.005		0.007	
Head of household work limiting disability	0.020		0.039		0.020		0.038		0.019	
<i><u>Parental health in 2005</u></i>										
Body mass index	0.005	**	0.002		0.005	**	0.002		0.003	+
Hypertension	0.009		0.025		0.009		0.025		0.012	
Heart disease	-0.018		0.030		-0.016		0.030		-0.013	
<i><u>Current Mechanisms</u></i>										
Exercise	---		---		---		---		0.007	
Smoke	---		---		---		---		0.064	+
Drink alcohol	---		---		---		---		-0.020	
Obese	---		---		---		---		0.070	*
Income (ln 2005\$)	---		---		---		---		-0.002	
<i><u>Poverty</u></i>										
Prenatal to age 15 (% years in	0.114	*	0.049		---		---		0.110	*

poverty)									
Prenatal to age 5 (% years in poverty)	---	---	0.101	+	0.054	---	---	0.096	* 0.048
Age 6 to 10 (% years in poverty)	---	---	-0.041		0.064	---	---	-0.052	0.062
Age 11 to 15 (% years in poverty)	---	---	0.069		0.061	---	---	0.082	0.058

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

					Arthritis						
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	
<i><u>Variables around birth</u></i>											
Non-white	0.013	0.015	0.013	0.014	0.010		0.015	0.009	0.013		
Male	-0.014	0.010	-0.014	0.010	-0.014	+	0.008	-0.015	0.008	+	
Born to married mother	-0.004	0.016	-0.003	0.017	-0.003		0.012	-0.002	0.012		
Born in the south	-0.010	0.008	-0.010	0.008	-0.013	*	0.006	-0.013	0.006	*	
Age of mother at birth	0.002	+	0.001	0.001	0.001	+	0.001	0.001	0.001		
Number of siblings	-0.002	0.003	-0.002	0.003	-0.002		0.002	-0.002	0.002		
First born	-0.005	0.010	-0.005	0.010	-0.007		0.008	-0.007	0.008		
Head of household IQ score (1972)	0.007	**	0.003	0.007	**	0.003	0.005	*	0.002	0.005	*
Head of household education	-0.006	**	0.002	-0.006	**	0.002	-0.004	**	0.001	-0.004	**
Head of household work limiting disability	-0.008	0.009	-0.008	0.009	-0.005		0.007	0.005	0.007		
<i><u>Parental health in 2005</u></i>											
Body mass index	0.001	0.001	0.001	0.001	0.000		0.001	0.000	0.001		
Hypertension	-0.002	0.010	-0.002	0.010	0.002		0.009	0.002	0.009		
Heart disease	0.041	0.033	0.041	0.033	0.040		0.029	0.041	0.029		
<i><u>Current Mechanisms</u></i>											
Exercise	---	---	---	---	0.015	***	0.005	0.016	0.005	***	
Smoke	---	---	---	---	0.004		0.010	0.004	0.009		
Drink alcohol	---	---	---	---	-0.001		0.009	-0.001	0.009		
Obese	---	---	---	---	0.015	+	0.009	0.014	0.009	+	
Income (ln 2005\$)	---	---	---	---	-0.007	**	0.003	-0.007	0.003	*	
<i><u>Poverty</u></i>											
Prenatal to age 15 (% years in poverty)	0.015	0.022	---	---	0.007		0.015	---	0.015	---	
Prenatal to age 5 (% years in poverty)	---	---	0.014	0.029	---		---	0.013	0.020		

Age 6 to 10 (% years in poverty)	---	---	-0.004	0.029	---	---	-0.010	0.023
Age 11 to 15 (% years in poverty)	---	---	0.006	0.029	---	---	0.007	0.021

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

					Diabetes							
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>		
<i><u>Variables around birth</u></i>												
Non-white	0.006	0.012	0.006	0.011	0.003	0.008	0.003	0.007				
Male	0.003	0.008	0.004	0.008	0.000	0.006	0.000	0.005				
Born to married mother	0.018	***	0.005	0.018	***	0.005	0.013	***	0.004	0.013	**	0.004
Born in the south	-0.015	+	0.008	-0.015	+	0.008	-0.012	*	0.005	-0.011	*	0.005
Age of mother at birth	0.001		0.001	0.001		0.001	0.001		0.001	0.001		0.001
Number of siblings	-0.003		0.003	-0.003		0.003	-0.002		0.002	-0.002		0.002
First born	0.015		0.009	0.017	+	0.009	0.010		0.007	0.010		0.007
Head of household IQ score (1972)	0.003		0.003	0.003		0.003	0.002		0.002	0.002	*	0.002
Head of household education	-0.002	*	0.001	-0.003	*	0.001	-0.002	*	0.001	-0.002		0.001
Head of household work limiting disability	0.003		0.015	0.003		0.014	-0.001		0.008	-0.001		0.008
<i><u>Parental health in 2005</u></i>												
Body mass index	0.000		0.001	0.000		0.001	0.000		0.000	0.000		0.000
Hypertension	0.008		0.008	0.008		0.008	0.003		0.005	0.003		0.005
Heart disease	-0.007		0.010	-0.007		0.010	-0.005		0.007	-0.005		0.007
<i><u>Current Mechanisms</u></i>												
Exercise	---	---	---	---		0.001	0.009		0.001	0.009		0.009
Smoke	---	---	---	---		-0.004	0.005		-0.004	0.005		0.005
Drink alcohol	---	---	---	---		-0.005	0.008		-0.005	0.008		0.008
Obese	---	---	---	---		0.029	0.014	*	0.029	0.014	*	0.014
Income (ln 2005\$)	---	---	---	---		0.002	0.003		0.002	0.003		0.003
<i><u>Poverty</u></i>												
Prenatal to age 15 (% years in poverty)	0.045	*	0.020	---	---	0.031	0.016	*	---	---		---
Prenatal to age 5 (% years in poverty)	---	---	0.019	0.021	---	---	0.014		---	0.014		0.014

---

Age 6 to 10 (% years in poverty)	---	---	0.026	0.023	---	---	0.017	0.013
Age 11 to 15 (% years in poverty)	---	---	-0.003	0.015	---	---	-0.002	0.012

---

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

			Limiting Hypertension					
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>
<i><u>Variables around birth</u></i>								
Non-white	-0.001	0.002	0.000	0.002	0.000	0.001	0.000	0.001
Male	0.004	0.003	0.003	0.003	0.002	+	0.001	0.002
Born to married mother	-0.016	0.013	-0.015	0.012	-0.006	0.005	-0.006	0.005
Born in the south	0.002	0.002	0.002	0.002	0.001	0.001	0.001	0.001
Age of mother at birth	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Number of siblings	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
First born	-0.001	0.002	-0.001	0.002	-0.001	0.001	-0.001	0.001
Head of household IQ score (1972)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Head of household education	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Head of household work limiting disability	0.003	0.004	0.002	0.003	0.001	0.002	0.001	0.001
<i><u>Parental health in 2005</u></i>								
Body mass index	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Hypertension	0.001	0.001	0.002	0.003	0.002	0.002	0.002	0.002
Heart disease	0.002	0.003	0.000	0.001	0.000	0.001	0.000	0.001
<i><u>Current Mechanisms</u></i>								
Exercise	---	---	---	---	0.001	0.001	0.001	0.001
Smoke	---	---	---	---	0.003	0.003	0.002	0.002
Drink alcohol	---	---	---	---	-0.001	0.001	-0.001	0.001
Obese	---	---	---	---	-0.001	0.001	-0.001	0.001
Income (ln 2005\$)	---	---	---	---	0.000	0.000	0.000	0.000
<i><u>Poverty</u></i>								
Prenatal to age 15 (% years in poverty)	0.008	*	0.004	---	---	0.005	*	0.002
Prenatal to age 5 (% years in poverty)	---	---	-0.003	0.004	---	---	-0.001	0.002

Age 6 to 10 (% years in poverty)	---	---	0.006	0.003	---	---	0.002	0.002
Age 11 to 15 (% years in poverty)	---	---	0.003	0.003	---	---	0.003	0.002

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

Table 3

*Logistic Regressions on 2005 Heads and Wives: Prenatal to Age 15 Income Measures*

	Hypertension				Arthritis					
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>		
<i>Variables around birth</i>										
Non-white	0.036	0.037	0.031	0.036	0.011	0.013	0.011	0.013		
Male	0.077	**	0.023	0.079	**	0.023	-0.013	0.009	-0.013	0.009
Born to married mother	-0.063	0.061	-0.046	0.058	-0.001	0.014	0.000	0.014		
Born in the south	-0.013	0.024	-0.011	0.025	-0.008	0.008	-0.009	0.008		
Age of mother at birth	-0.001	0.003	-0.001	0.003	0.002	0.001	0.002	*	0.001	
Number of siblings	0.006	0.007	0.005	0.007	-0.002	0.002	-0.002	0.003		
First born	0.023	0.026	0.024	0.026	-0.004	0.008	-0.004	0.009		
Head of household IQ score (1972)	-0.003	0.005	-0.002	0.005	0.007	**	0.002	0.007	0.003	
Head of household education	0.004	0.005	0.006	0.005	-0.005	*	0.002	-0.005	**	0.002
Head of household work limiting disability	0.029	0.041	0.025	0.039	-0.008	0.007	-0.009	0.008		
<i>Parental health in 2005</i>										
Body mass index	0.005	*	0.002	0.005	*	0.002	0.000	0.001	0.000	0.001
Hypertension	0.009	0.026	0.008	0.026	-0.002	0.009	-0.002	0.010		
Heart disease	-0.018	0.030	-0.017	0.030	0.034	0.029	0.037	0.030		
<i>Income</i>										
Prenatal to age 15 (10,000 2005\$)	0.114	0.005	---	---	-0.004	*	0.002	---	---	
Prenatal to age 15 (ln)	---	---	-0.045	+	0.026	---	---	-0.017	+	0.010

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

	Diabetes				Limiting Hypertension					
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>		
<i>Variables around birth</i>										
Non-white	0.006	0.008	0.006	0.009	0.000	0.002	0.000	0.002		
Male	0.002	0.005	0.003	0.006	0.004	0.003	0.004	0.002		
Born to married mother	0.011	**	0.004	0.016	0.004	-0.026	0.019	-0.016	0.014	
Born in the south	-0.010	+	0.005	-0.012	0.006	0.003	0.002	0.003	0.002	
Age of mother at birth	0.009		0.001	0.001	0.001	0.000	0.000	0.000	0.000	
Number of siblings	-0.002		0.002	-0.002	0.002	0.000	0.000	0.000	0.000	
First born	0.010		0.006	0.012	0.007	-0.001	0.002	-0.001	0.002	
Head of household IQ score (1972)	0.002		0.002	0.003	0.002	0.000	0.000	0.000	0.000	
Head of household education	-0.001		0.001	-0.001	0.001	0.000	0.000	0.000	0.000	
Head of household work limiting disability	0.001		0.009	0.002	0.011	0.005	0.005	0.003	0.004	
<i>Parental health in 2005</i>										
Body mass index	0.000		0.000	0.000	0.001	0.000	0.000	0.000	+ 0.000	
Hypertension	0.004		0.005	0.005	0.006	0.002	0.002	0.001	0.002	
Heart disease	-0.003		0.007	-0.005	0.008	0.002	0.003	0.001	0.003	
<i>Income</i>										
Prenatal to age 15 (10,000 2005\$)	-0.005	**	0.002	---	---	0.000	*	0.000	---	---
Prenatal to age 15 (ln)	---		---	-0.026	0.008	---	---	-0.003	*	0.002

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

Table 4  
*Logistic Regressions on 2005 Heads and Wives: Childhood Period Income Measures*

	<u>ME</u>	<u>Hypertension</u>				<u>Arthritis</u>				
		<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>		
<i><u>Variables around birth</u></i>										
Non-white	0.037	0.035	0.031	0.035	0.010	0.012	0.011	0.013		
Male	0.075	**	0.023	0.077	**	0.023	-0.012	0.008	-0.013	0.009
Born to married mother	-0.048	0.050	-0.042	0.054	-0.003	0.014	-0.001	0.015		
Born in the south	-0.011	0.023	-0.011	0.024	-0.008	0.008	-0.009	0.008		
Age of mother at birth	-0.001	0.003	-0.001	0.003	0.002	*	0.001	0.002	*	0.001
Number of siblings	0.004	0.007	0.004	0.007	-0.001	0.002	-0.001	0.002		
First born	0.023	0.025	0.022	0.026	-0.003	0.008	-0.004	0.008		
Head of household IQ score (1972)	-0.003	0.005	-0.003	0.005	0.006	**	0.002	0.007	**	0.002
Head of household education	0.005	0.005	0.006	0.005	-0.005	**	0.002	-0.005	**	0.002
Head of household work limiting disability	0.031	0.041	0.024	0.040	-0.008	0.007	-0.009	0.007		
<i><u>Parental health in 2005</u></i>										
Body mass index	0.005	**	0.002	0.005	*	0.002	0.000	0.001	0.000	0.001
Hypertension	-0.015	0.030	0.007	0.026	-0.003	0.008	-0.003	0.009		
Heart disease	0.007	0.025	-0.016	0.031	0.032	0.024	0.038	0.028		
<i><u>Income</u></i>										
Prenatal to age 5 (10,000 2005\$)	-0.007	0.005	---	---	-0.002	0.002	---	---		
Age 6 to 10 (10,000 2005\$)	-0.009	+	0.005	---	---	-0.003	0.002	---	---	
Age 11 to 15 (10,000 2005\$)	0.009	+	0.005	---	---	0.001	0.002	---	---	
Prenatal to age 5 (ln)	---	---	-0.039	0.032	---	---	-0.019	0.011		
Age 6 to 10 (ln)	---	---	0.036	0.040	---	---	-0.004	0.013		
Age 11 to 15 (ln)	---	---	-0.042	0.029	---	---	0.002	0.013		

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Note: \*\*\*  $p < .001$ ; \*\*  $p < .01$ ; \*  $p < .05$ . All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

	Diabetes				Limiting Hypertension					
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>		
<i>Variables around birth</i>										
Non-white	0.006	0.007	0.005	0.008	0.001	0.002	0.000	0.002		
Male	0.002	0.005	0.002	0.006	0.004	0.003	0.003	0.002		
Born to married mother	0.011	**	0.004	0.016	***	0.004	-0.018	0.013	-0.019	0.016
Born in the south	-0.009	+	0.005	-0.011	*	0.006	0.002	0.002	0.002	0.002
Age of mother at birth	0.001	0.000	0.001	0.001	0.000	0.000	0.000	0.000	0.000	
Number of siblings	-0.002	0.002	-0.002	0.002	0.000	0.000	0.000	0.000	0.000	
First born	0.009	0.006	0.011	0.007	-0.001	0.002	0.000	0.002		
Head of household IQ score (1972)	0.002	0.002	0.002	0.002	0.000	0.000	0.001	0.000		
Head of household education	-0.001	0.001	-0.001	0.001	0.000	0.000	0.000	0.000		
Head of household work limiting disability	0.001	0.009	0.001	0.011	0.004	0.004	0.003	0.003		
<i>Parental health in 2005</i>										
Body mass index	0.000	0.000	0.000	0.001	0.000	0.000	0.000	0.000		
Hypertension	0.003	0.005	0.004	0.006	0.001	0.002	0.002	0.002		
Heart disease	-0.003	0.007	-0.004	0.009	0.002	0.003	0.003	0.003		
<i>Income</i>										
Prenatal to age 5 (10,000 2005\$)	-0.003	0.002	---	---	-0.001	*	0.000	---	---	
Age 6 to 10 (10,000 2005\$)	0.001	0.001	---	---	0.001	0.000	---	---		
Age 11 to 15 (10,000 2005\$)	-0.003	0.001	---	---	0.000	0.000	---	---		
Prenatal to age 5 (ln)	---	---	-0.019	*	0.008	---	---	-0.004	0.003	
Age 6 to 10 (ln)	---	---	0.007	0.010	---	---	-0.001	0.003		
Age 11 to 15 (ln)	---	---	-0.015	+	0.009	---	---	0.001	0.002	

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, and heart disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on mother id). ME are marginal effects.

Table 5  
*Logistic Regressions on 2005 Heads and Wives: Current Characteristics*

	Hypertension						Arthritis									
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>					
<i>Splines</i>																
Prenatal to age 5 (<25k)	-0.085	*	0.037	-0.089	*	0.039	-0.089	*	0.039	-0.033	*	0.013	-0.023	*	0.012	-0.021
Prenatal to age 5 (>25k)	-0.004		0.005	-0.004		0.005	-0.004		0.005	0.000		0.002	-0.001		0.002	-0.001
Age 6 to 10 (<25k)	0.048		0.040	0.056		0.039	0.056		0.040	0.019	+	0.011	0.016	+	0.009	0.019
Age 6 to 10 (>25k)	0.008	+	0.005	0.006		0.004	0.006		0.004	-0.004		0.002	-0.003		0.002	-0.003
Age 11 to 15 (<25k)	-0.034		0.039	-0.045		0.034	-0.044		0.034	0.007		0.012	0.005		0.009	0.003
Age 11 to 15 (>25k)	-0.008	+	0.005	-0.006		0.005	-0.006		0.005	0.001		0.002	0.001		0.002	0.001
<i>Current Mechanisms</i>																
Exercise	---	---		0.014		0.028	0.014		0.029	---	---		0.013	**	0.004	0.012
Smoke	---	---		0.068	+	0.037	0.068	+	0.037	---	---		0.006		0.009	0.004
Drink alcohol	---	---		-0.020		0.023	-0.020		0.023	---	---		0.003		0.006	0.002
Obese	---	---		0.071	*	0.029	0.071	*	0.030	---	---		0.016	*	0.008	0.011
Income (ln 2005\$)	---	---	---	---	---	---	0.000	---	0.010	---	---	---	---	---	---	-0.005

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on ME are marginal effects).

	Diabetes						Limiting Hypertension					
	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	<u>SE</u>	<u>ME</u>	
<u>Splines</u>												
Prenatal to age 5 (<25k)	-0.004	0.007	-0.001	0.006	-0.002	0.006	-0.003	0.003	-0.003	0.003	-0.003	
Prenatal to age 5 (>25k)	-0.002	0.002	-0.002	0.002	-0.002	0.001	0.000 +	0.000	0.000	0.000	0.000	
Age 6 to 10 (<25k)	-0.003	0.009	-0.003	0.008	-0.004	0.007	-0.002	0.002	0.000	0.001	-0.001	
Age 6 to 10 (>25k)	0.001	0.001	0.001	0.001	0.001	0.001	0.000 +	0.000	0.000	0.000	0.000	
Age 11 to 15 (<25k)	0.005	0.009	0.002	0.008	0.002	0.007	0.000	0.002	-0.001	0.001	0.001	
Age 11 to 15 (>25k)	-0.003 *	0.001	-0.002 *	0.001	-0.002 *	0.001	0.000	0.000	0.000	0.000	0.000	
<u>Current Mechanisms</u>												
Exercise	---	---	0.000	0.006	0.000	0.005	---	---	0.001	0.001	0.001	
Smoke	---	---	-0.003	0.004	-0.002	0.004	---	---	0.004	0.003	0.004	
Drink alcohol	---	---	-0.004	0.006	-0.004	0.006	---	---	-0.001	0.001	-0.001	
Obese	---	---	0.016 +	0.009	0.016 +	0.009	---	---	-0.001	0.001	0.000	
Income (ln 2005\$)	---	---	---	---	0.003	0.003	---	---	---	---	0.000	

Note: \*\*\* p <.001; \*\* p <.01; \* p <.05. All regressions control for individual controls around the time of birth, and parent bmi, hypertension, disease in 2005. All regressions are weighted using the 2005 weight, and adjust the standard errors for the presence of siblings (clustering on ME are marginal effects).