

**Early-Life Origins of Adult Disease:  
The Significance of Poor Infant Health and Childhood Poverty**

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**Abstract:** The fetal origins hypothesis posits that under-nutrition in utero leads to adaptive changes in growth and development that may be beneficial in the short-run, but produces susceptibility to coronary heart disease and the related disorders hypertension, stroke, and diabetes later in life. This study provides the first evidence on the influence of low birth weight and child income on the onset of asthma and six fatal chronic health conditions through middle-age for a nationally representative sample of the US population. We find that poor health at birth and limited parental resources (including low income and lack of health insurance) interfere with healthy development and lead to increased risks of onset of asthma, hypertension, diabetes, coronary heart disease, and stroke or heart attack. These effects are substantial, and the overall pattern of results is robust to the inclusion of sibling fixed effects and an extensive set of controls. The results reveal that being born low weight increases the odds of asthma by two-thirds, increases the odds of hypertension by roughly 50 percent, and more than doubles the odds of a stroke or heart attack during middle-age. We find that a few early life factors – birth weight, family income, and health insurance coverage – can account for the lion’s share of the racial disparities in the prevalence of health conditions in adulthood. The general pattern of results across the various health condition outcomes indicates that low birth weight and parental income during childhood years is a very critical period that shapes opportunities and influences subsequent risks of onset of disease over the life course.

## **I. Introduction**

The fetal origins hypothesis, developed by David Barker and colleagues, proposes that when nutritional intake of a fetus is limited, the body's physiology and metabolism are changed fundamentally, and some of the consequences of these changes would become visible much later in life. The hypothesis of in utero "programming" posits that under-nutrition in utero leads to adaptive changes in growth and development that may be beneficial in the short-run, but produces susceptibility to coronary heart disease and the related disorders hypertension, stroke, and diabetes later in life. In 1995, studies in Britain showed for the first time that low birth weight was associated with increased risks of coronary heart disease and the disorders related to it: stroke, non-insulin dependent diabetes, raised blood pressure, and the metabolic syndrome. Over the past decade, a voluminous empirical literature in epidemiology document similar associations that support Barker's theory, drawing largely on data from the United Kingdom. (See Barker, 1998, for a review). Yet, there remains controversy over the nature and source of the statistical association. Uncovering the specific nature of the mechanisms underlying the association between low birth weight and risks of cardiovascular diseases later in life has proven elusive.

An alternative explanation is that this statistical association is spurious and instead reflects the lasting influence of hereditary risk factors and childhood socioeconomic conditions that are correlated with both low birth weight and onset of chronic disease. The evidence to date remains inconclusive in distinguishing between these competing explanations, and there is on-going debate regarding the source of reported associations between low birth weight and risks of cardiovascular diseases later

in life (see e.g., Huxley et al., 2002 *Lancet*). Assessing the relative importance of these competing explanations has implications for our understanding of the early-life developmental origins of adult disease. This research carries with it important policy implications that may lead to valuable insights that enable the development of effective early-life interventions to ameliorate the burden of disease and reduce the economic burden on the health care system.

In this paper, we empirically test the hypothesis that exposure to two factors in early life—low birth weight and childhood poverty—influences the disease process later in life. We use nationally representative longitudinal data from the US spanning nearly four decades, and estimate hazard models of onset of chronic health conditions that are among the leading causes of mortality and disability. The data set, the Panel Study of Income Dynamics (PSID), has the additional unique feature of allowing analyses of siblings throughout much of their life course. Moreover, most prior studies of the connection between early life health and economic status and adult health have relied on health surveys that have very limited economic data. The PSID is one of the premier income surveys in the world, while at the same time collecting significant detail on health. The data contains extensive socioeconomic measures in childhood and adulthood and does not have to rely on retrospective reports of early-life economic conditions.

In particular, we provide evidence on the consequences of low birth weight and childhood poverty on the risks of hypertension, asthma, stroke, heart disease, heart attack, and diabetes; this is the first such study of the full US population. We use sibling comparisons in which we relate sibling differences in birth weight and childhood stage-specific income to differences in their subsequent likelihood of onset of specific health

conditions in adulthood. The sibling fixed effect models explicitly control for unobserved persistent family background factors (parental health status, permanent income, parental education, family structure, parental health behaviors) and shared genetic factors.

For a U.S. cohort born between 1951 and 1970, we produce nationally representative estimates of chronic disease onset through mid-life by race/ethnicity, childhood socioeconomic status, and birth weight. Our results highlight the significant role of poor infant health and childhood poverty in shaping risks of onset of major chronic diseases in middle-age. We find that low birth weight significantly increases the likelihood of asthma, hypertension, stroke or heart attack, and diabetes. These results are robust to sibling fixed effects and the inclusion of an extensive set of controls. We also find that limited parental resources (childhood poverty, lack of child health insurance coverage) significantly increase many of these disease risks in mid-adulthood. Furthermore, racial differences in these early life conditions play a dominant role in explaining racial disparities in chronic health conditions through at least age 50.

The paper is organized as follows. The next section provides a brief review of the relevant literature. Section III describes the data, lays out our conceptual framework and identification strategy, and discusses econometric model specification issues. Section IV provides descriptive analysis of our outcomes of interest. Section V presents our regression results. Section VI concludes with a summary discussion of the results.

#### *Theoretical considerations*

The fetal origins hypothesis emphasizes that adverse conditions in utero trigger biological responses (e.g., damage to organ systems) that increase the risk of fatal chronic

conditions later in life. Alternatively, health shocks early in life, even in the womb, may have immediately visible effects on health that last from birth through adulthood and old-age. In either scenario, events in the earliest stages of life have effects that unfold over subsequent stages of life. Childhood is a sensitive life-cycle period in which disruptions in biological development can have far-reaching negative consequences for health and mortality in adulthood.

Recent evidence from human and animal studies highlight the critical importance of early childhood for brain development and for setting in place the structures that will shape future cognitive, social, emotional, and health outcomes (Shonkoff and Phillips, 2000). Limited parental resources (including child poverty and lack of health insurance) and its attendant stressors have the potential to shape the neurobiology of the developing child in powerful ways, which may lead directly to worse health later in life. Childhood poverty can also reduce investments in children's learning and development. Lack of child health insurance can discourage the use of medical care, particularly in the early and more treatable stages of a health problem (e.g., Rice and Winn, 1990). Such a lack of resources in the family environment can compound and amplify the neurobiological disadvantages that many poor children already face (Duncan et al., 2007).

Chronic health conditions typically grow out of socioeconomic conditions over a lengthy life-cycle period rather than from circumstances at a single point in time. Socioeconomic status and health status are highly correlated. This strong association holds for a variety of health status measures, is true in countries with varying levels of economic development and government-sponsored medical care, and has existed as far back in time as data are available. The association also holds across the entire life course,

although the gap appears to widen with age through about age 60, and then declines (Smith, 2004). The direction of causality between health and economic status is unclear. While it is most likely the case that health causally affects economic status, and economic status causally affects health, the magnitude of each effect is uncertain (Smith, 1999). But if causality runs in both directions, then a life course model would imply that health problems early in life could affect health later in life because the problem is chronic, because the health shock damaged health stock making it more susceptible to deterioration later in life, and because the health problem affects socioeconomic outcomes such as education which in turn influences health later in life (Kuh and Wadsworth, 1993).

The conceptual framework emphasizes the fact that the aging process begins at conception and evolves over the life course in response to health shocks, biological deterioration, and investments in and uses of health capital. This study examines the long-run consequences of poor birth outcomes and parental resources on the onset of fatal chronic conditions in adulthood. These early-life conditions are a critical period of rapid development with potential consequences for life chances (including both longevity and years lived in good health).

Low birth weight infants account for large public health expenditures —studies show that more than one third of the total dollar amount spent in the US on health care during the first year of life can be attributed to low birth weight even though low birth weight infants account for less than 10% of all births in the US (Lewitt et al., 1995). Birth weight is a complex outcome that is influenced by factors that vary from one pregnancy to the next for the same mother (e.g., child-specific genetic component, timing

of prenatal take-up, parental socioeconomic economic conditions, maternal smoking, exercise, diet) and those that can be considered to be fixed maternal characteristics.

*Sources of Birth Weight Differences.* Birth weight is an indicator of the prenatal cause, not the cause itself. The biomedical and epidemiological literatures estimate that environmental influences (e.g., intrauterine nutrition, maternal smoking and alcohol consumption during pregnancy, quantity and quality of prenatal care, maternal stress, neighborhood conditions) account for about 25 percent of the variation in birth weight, and family background and genetic influences account for between 40 to 75 percent (reference from birth weight symposium article). These studies have been criticized for attempting to disentangle environmental and genetic factors without taking into account the degree of assortative mating in the population and the genetic-environmental covariance that has arisen endogenously due to intergenerational factors (e.g., environmental factors are correlated across generations and play a role in determining poor birth outcomes of one generation and subsequently represent a hereditary risk factor for the succeeding generation's birth outcomes) (see Goldberger 1977, 1979).

Birth weight is the product of length of gestation and intrauterine growth of the fetus. Previous research suggests/indicates that variability in gestation length account for roughly 60 percent of the overall cross-sectional variation in birth weight (Almond et al., 2005; other cites). Although most studies have indicated that it is growth for gestational age rather than duration of gestation itself that is associated with adult health, few have the statistical power to look at the separate effects. Leon et al. (2000), the single study that has been able to distinguish between these effects, finds that, among Swedish young men, higher blood pressure was associated with both reduced growth for gestational age

and reduced gestational age—i.e., the highest blood pressures occurred among those who grew less well in utero and were delivered early.

### **III. Data and measures**

The Panel Study of Income Dynamics (PSID) is among the best equipped data sources to investigate the linkages between health and economic status in the initial stages of life and the subsequent onset of specific health conditions, as it is the longest-running nationally representative US sample of households and is one of the premier data sources of income in the world.

The PSID began interviewing a national probability sample of families in 1968. These families were re-interviewed each year through 1997, when interviewing became biennial. The data we use in this paper spans the period 1968 through 2005. All persons in PSID families in 1968 have the PSID “gene,” which means that they are followed in subsequent waves. In addition, anyone born to or adopted by PSID sample members acquires the PSID “gene” themselves and therefore is followed. When children with the “gene” become adults and leave their parents’ homes, they become their own PSID “family unit” and are interviewed in each wave. This sample of “split offs” has been found to be representative (Fitzgerald, Gottschalk and Moffitt, 1998). Moreover, the genealogical design implies that the PSID sample today includes numerous adult sibling groupings and parent-child groupings who have been members of PSID-interviewed families for nearly four decades.

The sample chosen for the focus of the study consists of PSID sample members who were children when the study began and who have been followed into adulthood. Specifically, we choose PSID sample members born between 1951 and 1968, which

consists of children 0-17 years old in the first wave of interviewing in 1968. We then obtain all available information on these individuals for each wave, 1968 to 2005. Therefore, by 2005 the oldest person in the sample is 55 and the youngest is 37. The ages of these respondents by the end of the sample period correspond to the years of life (50s) in which both socioeconomic differences in health and birth weight differences in adult health status begin to approach peak levels, and rates of deterioration in health begin to accelerate for disadvantaged persons (House et al., 1994; Johnson and Schoeni, 2007).

Our analysis focuses on the prevalence and age of onset of the major fatal chronic diseases: hypertension, heart disease, stroke, heart attack, and diabetes. These diseases are among the leading causes of death among individuals in their 50s. We also analyze asthma. Respondents in the 1999-2005 PSID survey interviews were asked whether a doctor ever told them they have a particular disease, and if so, at what age the initial diagnosis occurred. It is important to bear in mind that this self-report measure may be affected by differential access and interaction with the health care system. In particular, poor families and African-Americans lower levels of health care use relative to affluent and white families, with the exception of emergency room care, is expected to bias the socioeconomic gradient and racial health gaps toward equality, making our estimates of these differences in health conditions conservative.

The key birth outcome variable examined in both the adult and child samples is birth weight. For the adult sample, mothers reported in 1985 whether their child (i.e., the adult) was born low birth weight, defined as less than 5.5 pounds.<sup>1</sup> Information collected

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<sup>1</sup> Although the PSID low birth weight information for the adult sample is based on retrospective maternal reports, previous validation studies have demonstrated that comparisons of maternal birth weight reports and those from vital records show high rates of agreement (Baker et al., 1993; Klebanoff and Graubard, 1986).

in the PSID on the age of onset of a variety of specific health conditions suggests that very few of the low birth weight individuals in our sample experienced birth defects. Thus, it is unlikely that the presence of birth defects drives the underlying relationships between low birth weight and adult health conditions analyzed in this paper. We also found no evidence that estimates from the adult sample suffer significant bias from health-related attrition due to selective mortality among individuals born at low weight; any potential bias suggests that early mortality will tend to reduce the estimated effect of birth weight on later outcomes.

Income is the total for the family in which the child lives, and it is measured at various points in childhood (noted in each table). All dollar values are expressed in 1997 dollars using the CPI-U. The parental income measure is specified as the income-to-needs ratio and we explore nonlinearities in effects at the bottom of the income distribution (child poverty).

Child health insurance coverage is measured through information collected in the first five waves of the PSID (1968-1972) on whether the parent (head of household) had access to private health insurance coverage and if so, whether the entire family was covered. We define three categories of child health insurance coverage: continuously covered by private health insurance coverage in childhood years during 1968-1972; intermittent coverage during those years, and lacking private health insurance coverage in all of these years. Lack of private health insurance may discourage preventive medical care use. For those who lacked private coverage for their children, the data suggest that public health insurance coverage was utilized to some extent, but there were not enough individuals in our sample who persistently lacked public and private insurance during

these childhood years to define “no public or private insurance during childhood” as a reference category.

Our parental health status measures are the proportion of years spent when the parent was in their 50s in which they were in problematic health (based on self-reports of work-limiting conditions and/or general health status). These ages correspond to years in life when rates of health deterioration typically begin to accelerate.

The PSID in 1999-2005 asked adults to recall their health in childhood (i.e., ages less than 17) and rate it as excellent, very good, good, fair, or poor. We investigate the effects of childhood health on the onset of health conditions, and we examine the extent to which birth weight influences adult health through its effects on health in childhood.

#### *Empirical Strategy*

The central methodological question (which any work in this area must confront) is whether the estimated long-run effects of poor infant health (child poverty) are caused by low birth weight (low family income) *per se*, or are instead the result of a broader set of factors such as the quality of family life, schools, and neighborhood environments that impact poor families with young children. These indeed are likely not mutually exclusive explanations, but the appropriate policy response requires the identification of the mechanisms through which early-life health and child poverty thwarts life chances and years without health problems. Empirically, it has proven difficult to disentangle the effects of poor birth outcomes and low family income from poor parents, disadvantaged neighborhoods, and inferior school quality. The concern is that the estimated effects of birth weight and parental income on children’s subsequent onset of health conditions in adulthood may be spurious and instead capture (at least in part) unmeasured “true” causal

factors such as parental ability, diligence, father involvement, and parent’s valuing needs of their children’s development above their own. Hereditary risk factors may play a role in influencing temperament, cognitive ability, and health.

We begin by using a random effects discrete-time hazard model to analyze the age of onset of specific health conditions, including asthma, hypertension, diabetes, stroke, heart disease, and heart attack. Given the different disease etiologies, different sets of determinants may be more salient for a particular disease, so these models are estimated separately for each health condition. The hazard function,  $h_{fsr}(t)$ , is the probability that individual  $s$  from family  $f$  obtains specific health condition  $r$  at age  $t$ , given the individual has never had the condition in any previous year. The hazard is specified in a logit form, where in the baseline model, the explanatory variables include only a set of age dummy variables and a family-level random effect ( $u_{fr}$ ):

$$h_{fsr}(t) = \beta_r X_{fs}(t) + \Gamma_r \text{AgeDummies} + u_{fr} + \varepsilon_{fsr}(t)$$

In this model, we are implicitly assuming proportional odds—in particular, we assume the baseline logit hazard curves in the  $K$  families are parallel to one another.

The key methodological strength of the empirical approach to isolate the impact of low birth weight and child poverty is the model’s inclusion of an extensive array of childhood family background measures. The unusually rich set of family and child-specific controls included in the models minimize potential omitted variable bias. These controls include age, gender, race, parental health status, parental education, parental health behaviors (smoking and alcohol use), birth order, maternal age at birth, birth cohort dummies, pregnancy intentions, and an indicator for whether the child was born into a two-parent family. This set of child family characteristics provide stronger

evidence on whether the estimated effects of low birth weight and parental income on subsequent disease onset can be attributable to poor birth outcomes and childhood poverty itself, as opposed to the broader effects of growing up in poor neighborhood, school, and home environments. For each specific health condition, we examined alternative functional forms of the key explanatory variables to best fit the data. In all regression models, standard errors are clustered at the person level.

We first estimate this version of the hazard model of disease onset, which specifies unobserved family heterogeneity as a random effect, and then estimate a sibling fixed-effect Cox proportional hazard model to examine the effects of explicitly controlling for unobserved family background factors on the onset of specific health conditions.

#### *Sibling Fixed-Effect Cox Proportional Hazard Model*

We use sibling comparisons in which we relate sibling differences in birth weight and childhood stage-specific income to differences in their subsequent likelihood of onset of specific health conditions in adulthood. The sibling fixed effect models more fully account for family background factors (e.g., parental health status, permanent income, parental education, family structure, persistent parental health behaviors (smoking and alcohol habits), fixed parental healthy lifestyle orientation), and shared genetic factors. Although within-family analyses explicitly control for all persistent parental family background factors, they are unable to account for possible effects of unshared genetic factors. Full biological siblings share on average 50 percent of their genetic make-up, so genetic differences between non-identical siblings remain and can be a source of bias.

A family's resources in childhood may have a lasting impact on a child's health trajectory, which we investigate. The sibling fixed effects models identify the effect of family income from differences in family income between siblings at the same life stage, i.e., ages 13-16. It is important to note that if parents' permanent income matters most for their children's adult health status, this sibling fixed effect specification represents a very stringent (and perhaps inappropriate) test of the importance of family income. This is because the identification of the income effects relies on transitory changes in parental income to generate between-sibling differences at the same stage in childhood. The family's income-to-needs ratio is used as our measure of parental income (where a value of 1 is equivalent to family income equal to the poverty line).

To be more formal, suppose that from family  $f$  we have  $n_f$  siblings (ordered by their increasing length of time without obtaining the health condition) and that the duration of each sibling's condition-free spell is denoted  $t_{fs}$ , where  $s$  stands for the particular sibling. Assuming all individuals from the same family are independently distributed given a family heterogeneity parameter, we can write the hazard functions as

$$\lambda_{fs}(t) = \exp(\beta'X_{fs}(t) + \alpha_f) \lambda_{f0}(t), \quad s = 1, \dots, n_f; f = 1, \dots, N. \quad (1)$$

Then it can be shown that the partial log-likelihood function is equal to<sup>2</sup>

$$L_p = \sum_{f=1}^N \sum_{s=1}^{n_f-1} \ln \left( \frac{\exp(\beta'X_{f(y)}(t_{f(s)}))}{\sum_{k=s}^{n_f} \exp(\beta'X_{f(k)}(t_{f(s)}))} \right), \quad (2)$$

where the denominator corresponds to the risk set of family  $f$ . Note that both  $\alpha_f$  and  $\lambda_{f0}$  do not appear in equation (2). Although all biases caused by unobserved persistent

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<sup>2</sup> See Lancaster (1990), pp. 268-71. Note that a censored spell must be at least as long as the smallest completed spell in order to contribute anything to the likelihood function.

family heterogeneity are removed using Chamberlain's extension of Cox's partial likelihood method, the problem of biases caused by unobserved child-specific heterogeneity (or time-varying unobserved family factors) remains.

Due to small sample sizes and relatively low incidence rates prior to age 50, we combined stroke and heart attack to increase the precision of our estimates in our sibling fixed effect model specifications.

#### **IV. Descriptive analyses**

Using our PSID sample cohort born between 1951 and 1968, we begin by producing nationally-representative estimates of the cumulative risks through middle-age of asthma and six major fatal chronic health conditions—hypertension, diabetes, stroke, heart attack, heart disease—by race/ethnicity, low birth weight, child poverty, and child health insurance coverage status. The results are summarized in Tables 1-7 and in Figures 1-7. Sampling weights are used to produce nationally-representative estimates. We report Nelson-Aalen estimates of the cumulative risk of disease onset by various ages over the life course.

##### *Linking low birth weight and childhood poverty to adult health conditions*

The set of results summarized in Tables 1-7 (Figures 1-7) display striking differences in the age of onset of each these specific health conditions depending on low birth weight status and whether average childhood income was below, close to, or well above the poverty line. We tested for the equality of the survivor functions along these lines and our reported differences are statistically significant in the vast majority of cases. We discuss the descriptive findings of each specific health condition in turn.

*Asthma.* Asthma currently accounts for around 9 million office visits and around 1.9 million visits to emergency departments each year, and is the leading cause of child disability in the US. As shown in Table 1, asthma has a socio-economic gradient, striking poor children with greater frequency than wealthier children. By age 45, we find that low birth weight individuals were nearly twice as likely to have asthma, relative to individuals born at normal weight (20 percent compared with 11.7 percent). We see that individuals who grew up without access to private health insurance coverage were about six percentage points more likely to have asthma by age 45, and the black-white gap in asthma prevalence at age 45 is of similar magnitude (18.3 percent vs. 12.5 percent). All of these differences are statistically significant. Racial differences in asthma prevalence and morbidity seem to persist after attempts to control for socioeconomic status and access to care. The observed disparities have led researchers to question whether differences may be due to environmental, physiological, or genetic variations. Poor birth outcomes are an under-explored source of these differences that we return to in the regression analysis.

*Hypertension.* High blood pressure is often called the ‘silent killer’ because it usually has no noticeable warning signs or symptoms until other serious problems arise. Hypertension is a major risk factor for heart disease and stroke, the leading causes of death in the US. As shown in Table 2, the differences in risks of onset by low birth weight status, child poverty, health insurance coverage, and race/ethnicity are all starkest for this health condition. We find that by age 45, forty-two percent of low birth weight individuals had hypertension (compared with one-quarter of normal birth weight individuals); 45.7 percent of individuals raised in poverty had hypertension (compared

with 22.6 percent of individuals with parental incomes during childhood that were at least two times the poverty line); 41.7 percent of individuals who lacked access to private health insurance coverage during childhood had hypertension (compared with 22 percent among those with access); and 47 percent of African-Americans had hypertension (compared with less than one-quarter of whites).

*Diabetes.* Diabetes is the sixth leading cause of death in the US, increases the risk of heart disease, stroke, and pregnancy complications, and is the leading cause of blindness and End Stage Renal Disease. As shown in Table 3, we find that by age 45 individuals with one of these early markers of childhood disadvantage—low birth weight, child poverty, lack of health insurance coverage—have a 50-100 percent greater likelihood of having diabetes.

*Heart Disease, Stroke, Heart Attack.* Heart disease is the leading cause of death in the US and is a major cause of disability. Almost 700,000 people die of heart disease in the US each year. That is about 29% of all US deaths. Tables 4-7 summarize the same set of differences in the percent who had suffered a stroke, heart attack, or had heart disease. While incidence rates prior to age 50 are relatively low, the differences by our measures of early child disadvantage are equally striking on the order of 50-100 percent greater disease onset by age 45, depending on the relevant childhood disadvantage dimension (i.e., low birth weight status, child poverty status, child health insurance coverage status, and race).

It is important to note that the magnitudes of these differences in the risks of these various health conditions tend to become more pronounced as individuals age, a result that mirrors findings in Johnson and Schoeni (2007). The strong associations between

low birth weight and childhood socioeconomic factors and likelihood of onset of adult health conditions shown in Tables 1-7 (Figures 1-7) do not prove that low birth weight or child poverty itself is the cause of these differences. Of course, families who exhibit different patterns of disease onset are different from one another in a multitude of ways that may also contribute to the raw differences in their children's adult health status outcomes that we observed. Family economic conditions in adulthood may be what really matters. Or perhaps some third factor, such as inadequate parental education, parental health status, is the cause of poor birth outcomes and family poverty as well as their children's subsequent poorer health in adulthood. We would expect these other factors to affect risks of chronic health conditions, independent of early-life health and child socioeconomic characteristics. The remainder of this analysis aims to identify whether it is low birth weight and child poverty itself which leads to the higher disease risks, or these other differences in family characteristics that are the main causal factors.

## **V. Regression Results**

Table 8 presents the exponentiated coefficient results of our Cox-proportional hazard models of the effects of low birth weight and child socioeconomic factors on the onset of specific health conditions, including asthma, hypertension, diabetes, stroke, heart attack, and heart disease. Even after the inclusion of an extensive set of controls (including gender, race, parental health status, parental education, parental health behaviors, birth order, mother's age at birth, whether born into a two-parent family, birth year), we find that low birth weight significantly increases the likelihood of onset of asthma and hypertension each by 62.4 percent, increases the odds of diabetes by 70 percent, more than doubles the risk of a stroke, and increases the odds of heart disease by

75.6 percent. The results indicate that a one-unit increase in the parental income-to-needs ratio decreases the odds of experiencing a heart attack by 26 percent and decreases the odds of having heart disease by 7.5 percent.

Lack of health insurance can discourage the use of medical care, particularly in the early and more preventable stages of a health problem. We find that a lack access to private health insurance coverage during childhood is associated with a roughly 40 percent increase in the odds of both hypertension and diabetes, and those who were only intermittently covered by private health insurance coverage in childhood had 19 percent greater odds of having hypertension, relative to individuals who were continually covered in childhood (1968-1972).

It is equally striking that the lion's share of the enormous racial disparities in these specific health conditions can be explained by differences in these childhood factors, particularly birth weight, parental income, and child health insurance coverage. The black coefficient ceases to be significant after inclusion of early-life factors across the broad set of health conditions; only in the case of hypertension does the black-white gap remain significant and its magnitude is a small fraction of the unconditional raw differences. These findings parallel results of Johnson and Schoeni (2007), in which they find that the inclusion of a single childhood variable – family income-to-needs during childhood – can account for the entire black-white gap in general health status, while contemporaneous economic factors account for relatively little of this gap.

*Sibling Fixed-Effect Hazard Results.* We next test whether these results are robust to the inclusion of sibling fixed effects. The exponentiated coefficients from the sibling fixed-effect Cox proportional hazard models are presented in Table 9. We find

that the general pattern of results of the effects of low birth weight are qualitatively unchanged when using sibling comparisons for the identification of effects, though as expected the coefficient estimates are much less precise (see non-FE and FE column estimates in Table 9). Thus, we do not find any evidence in these results that support the view that our previous hazard model estimates were driven by unobserved persistent family background factors. In these models, we also analyze the effects of child health status and examine whether the birth weight effects manifest early and operate principally through diminished child health. In particular, we find that child health status has large, significant effects on the onset of asthma, hypertension, and stroke or heart attack; and, these patterns of results persist in the sibling fixed effect models (with the exception of stroke or heart attack, due to small sample sizes and relatively low incidence rates that preclude definitive evidence). However, the evidence does not suggest that the birth weight effects on the onset of these chronic conditions operate primarily through diminished child health, but rather these health problems appear to manifest much later in life.

For the sibling (family fixed-effects) models relating sibling differences in childhood-stage specific income *differences* to sibling differences in likelihood of disease onset, we found that sibling variability was not sufficient to produce reasonably precise estimates of parental income effects.

## **VI. Discussion**

This study provides the first evidence on the influence of low birth weight and child income on the onset of asthma and six fatal chronic health conditions through middle-age for a nationally representative sample of the US population. The health

conditions considered are among the leading causes of death and disability in the US. We find that poor health at birth and limited parental resources (including low income and lack of health insurance) interfere with healthy development and lead to increased risks of onset of asthma, hypertension, diabetes, coronary heart disease, and stroke or heart attack. These effects are substantial, and the overall pattern of results is robust to the inclusion of sibling fixed effects and an extensive set of controls. The results reveal that being born low weight increases the odds of asthma by two-thirds, increases the odds of hypertension by roughly 50 percent, and more than doubles the odds of a stroke or heart attack during middle-age. The general pattern of results across the various health condition outcomes indicates that low birth weight and parental income during childhood years is a very critical period that shapes opportunities and influences subsequent risks of onset of disease over the life course.

Racial differences in mortality and morbidity in adulthood are large in the US. We find that a few early life factors – birth weight, family income, and health insurance coverage – can account for the lion’s share of the gaps in the prevalence of health conditions in adulthood. While contemporaneous socio-economic factors have been the focus of the literature on racial disparities in health, our results suggest that adult health condition disparities may be better explained by early life factors (Johnson and Schoeni, 2007).

Our results highlight the roles of poor infant health and childhood poverty and provide insights into the developmental origins of chronic disease. Other related evidence contained in Johnson and Schoeni (2007) highlight the profound influence of childhood economic conditions. Taken together, these findings suggest that early-life

interventions have great promise as cost-efficient approaches to promote human capital development and the quantity of years lived without health problems.

Future work is needed to identify the mechanisms along the causal chain linking poor infant health and child poverty to chronic health conditions later in life. This research may enable more effective policy interventions to be implemented to ameliorate the burden of disease and the economic burden to the health care system. The economic drain may be reduced by greater investment in early life interventions, particularly those that lower risks of low birth weight. This work can assist in shifting the goal from secondary symptom amelioration to primary disease prevention. Our findings suggest that the seeds of vulnerability to chronic health conditions are planted early in life, possibly in utero.

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**Table 1. Proportion with Asthma, by birth weight and child socioeconomic status**

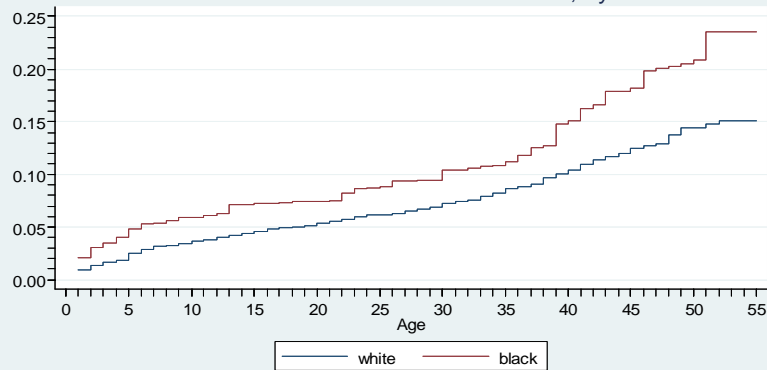
	Proportion ever diagnosed with asthma by age:			
	25	35	45	50
<i>By Birth weight</i>				
Low birth weight	0.1370	0.1474	0.1998	0.2375
Normal birth weight	0.0567	0.0772	0.1168	0.1368
<i>By Child Poverty Status</i>				
Child Poverty	0.0511	0.0854	0.1675	0.2181
Non-poor	0.0648	0.0883	0.1300	0.1494
<i>By Child Health Insurance Status</i>				
No Childhood Health Insurance	0.0788	0.1048	0.1894	0.2143
Health Insurance	0.0632	0.0870	0.1270	0.1428
<i>By Race</i>				
Black, non-Hispanic	0.0879	0.1122	0.1826	0.2086
White, non-Hispanic	0.0614	0.0867	0.1248	0.1440

Data: PSID, 1968-2005 (Individuals born between 1950-1968).

Notes: Sampling weights are used to provide nationally-representative estimates.

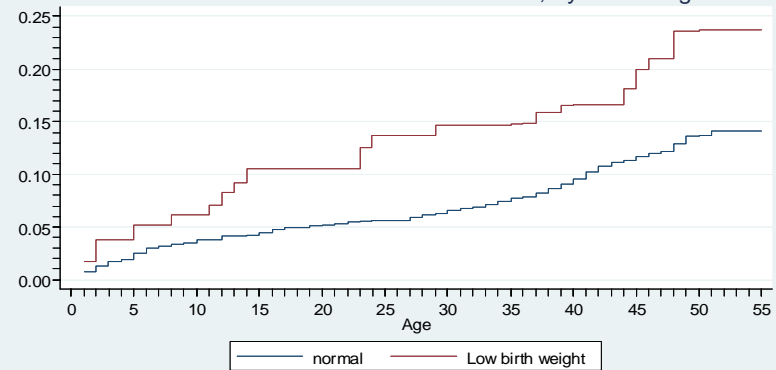
# Cumulative Hazard: Onset of Asthma

### Cumulative Hazard of Onset of Asthma, by Race



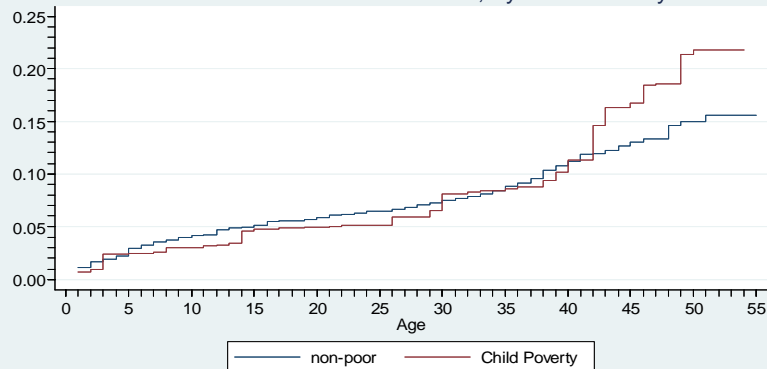
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

### Cumulative Hazard of Onset of Asthma, by Birth weight



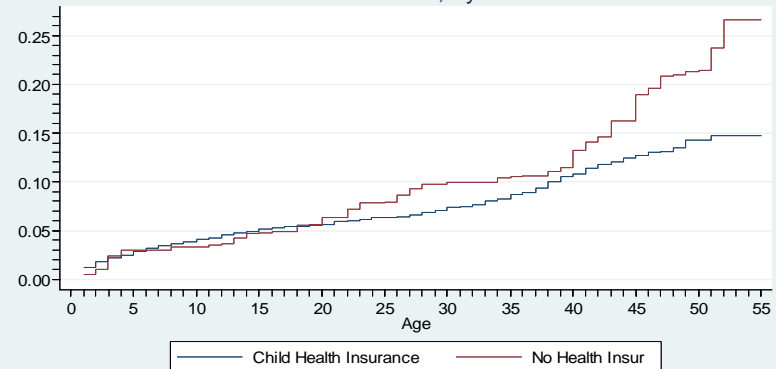
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

### Cumulative Hazard: Onset of Asthma, by Child Poverty Status



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

### Cum. Hazard: Onset of Asthma, by Child Health Insurance



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

**Table 2. Proportion with Hypertension, by birth weight and child socioeconomic status**

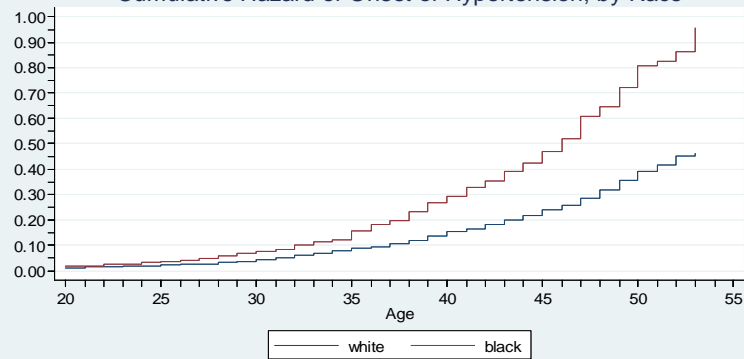
	Proportion ever diagnosed with hypertension by age:			
	25	35	45	50
<i>By Birth weight</i>				
Low birth weight	0.0232	0.1627	0.4197	0.6360
Normal birth weight	0.0228	0.0905	0.2502	0.4038
<i>By Child Poverty Status</i>				
Child Poverty	0.0259	0.1520	0.4566	0.6835
Non-poor	0.0201	0.0841	0.2262	0.3906
<i>By Child Health Insurance Status</i>				
No Childhood Health Insurance	0.0290	0.1380	0.4169	0.6283
Health Insurance	0.0219	0.0755	0.2198	0.3651
<i>By Race</i>				
Black, non-Hispanic	0.0380	0.1568	0.4705	0.8078
White, non-Hispanic	0.0223	0.0893	0.2386	0.3908

Data: PSID, 1968-2005 (Individuals born between 1950-1968).

Notes: Sampling weights are used to provide nationally-representative estimates.

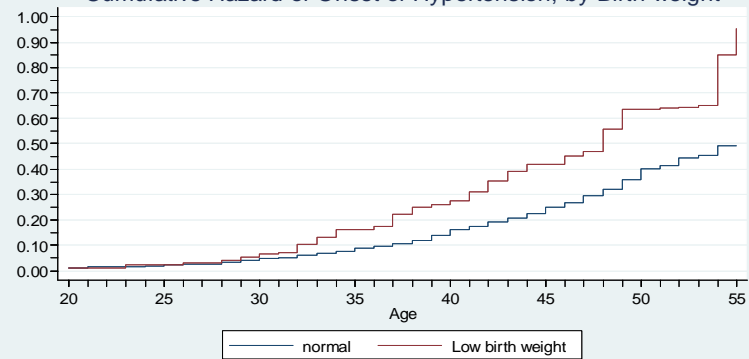
# Cumulative Hazard: Onset of Hypertension

Cumulative Hazard of Onset of Hypertension, by Race



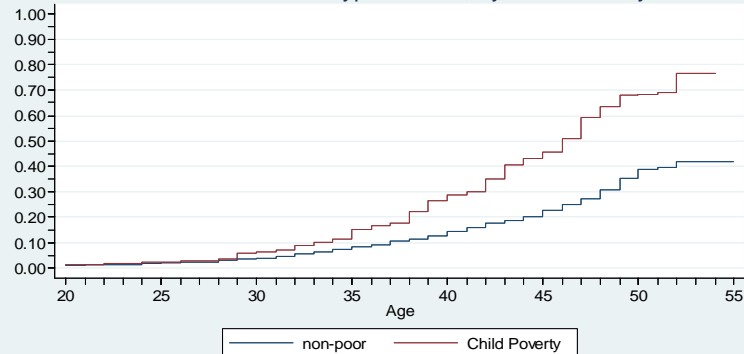
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cumulative Hazard of Onset of Hypertension, by Birth weight



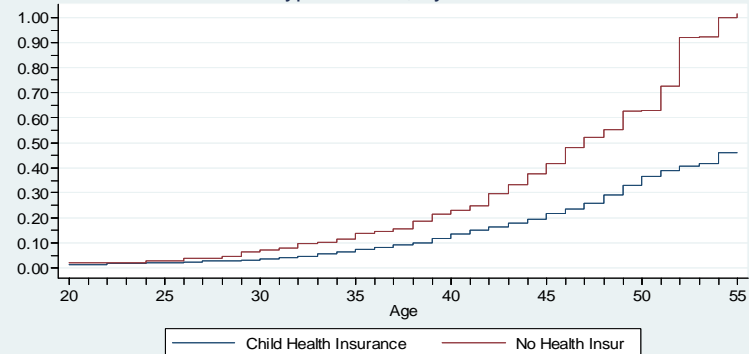
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Onset of Hypertension, by Child Poverty Status



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Hypertension, by Child Health Insurance



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

**Table 3. Proportion with Diabetes, by birth weight and child socioeconomic status**

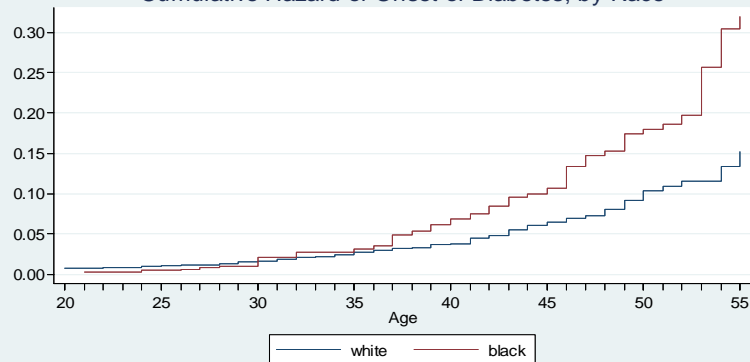
	Proportion ever diagnosed with diabetes by age:			
	25	35	45	50
<i>By Birth weight</i>				
Low birth weight	0.0000	0.0244	0.1079	0.1639
Normal birth weight	0.0109	0.0291	0.0682	0.1025
<i>By Child Poverty Status</i>				
Child Poverty	0.0186	0.0377	0.0913	0.1987
Non-poor	0.0105	0.0297	0.0613	0.1083
<i>By Child Health Insurance Status</i>				
No Childhood Health Insurance	0.0101	0.0421	0.1280	0.1973
Health Insurance	0.0086	0.0237	0.0605	0.0944
<i>By Race</i>				
Black, non-Hispanic	0.0054	0.0310	0.1067	0.1799
White, non-Hispanic	0.0107	0.0272	0.0649	0.1034

Data: PSID, 1968-2005 (Individuals born between 1950-1968).

Notes: Sampling weights are used to provide nationally-representative estimates.

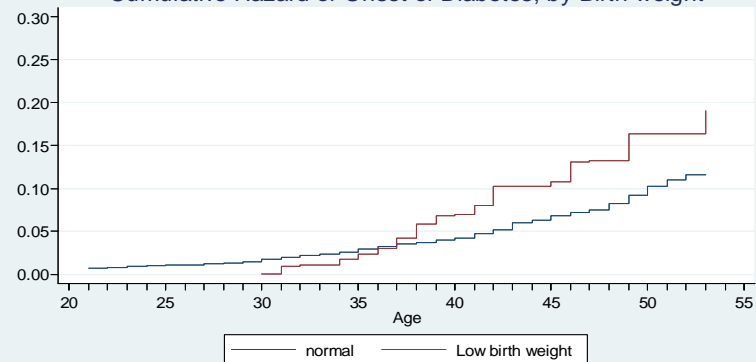
# Cumulative Hazard: Onset of Diabetes

### Cumulative Hazard of Onset of Diabetes, by Race



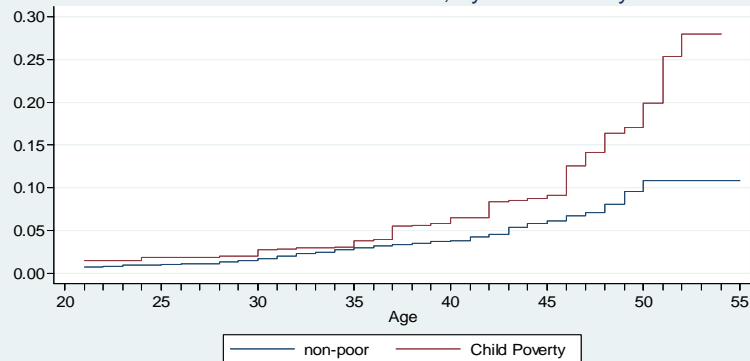
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

### Cumulative Hazard of Onset of Diabetes, by Birth weight



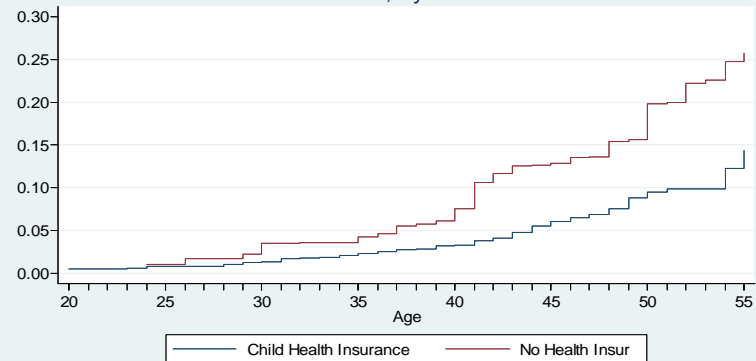
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

### Cum. Hazard: Onset of Diabetes, by Child Poverty Status



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

### Cum. Hazard: Diabetes, by Child Health Insurance



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

**Table 4. Proportion ever had a Stroke, by birth weight and child socioeconomic status**

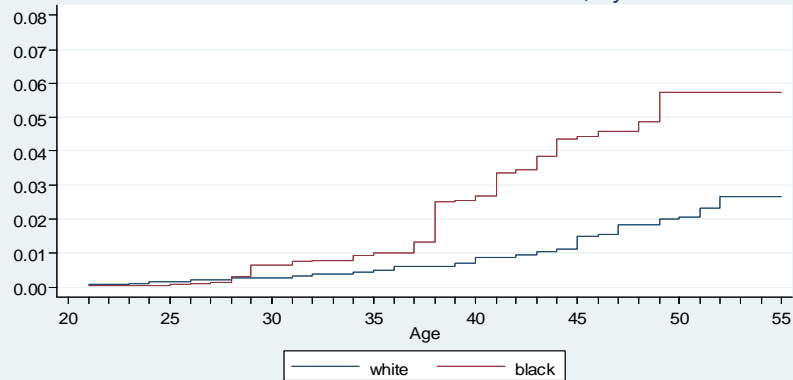
	Proportion ever had a stroke by age:			
	25	35	45	50
<i>By Birth weight</i>				
Low birth weight	0.0000	0.0168	0.0465	0.0595
Normal birth weight	0.0020	0.0043	0.0152	0.0195
<i>By Child Poverty Status</i>				
Child Poverty	0.0017	0.0073	0.0596	0.0759
Non-poor	0.0012	0.0044	0.0101	0.0122
<i>By Child Health Insurance Status</i>				
No Childhood Health Insurance	0.0011	0.0071	0.0243	0.0561
Health Insurance	0.0009	0.0035	0.0122	0.0167
<i>By Race</i>				
Black, non-Hispanic	0.0031	0.0123	0.0467	0.0598
White, non-Hispanic	0.0017	0.0049	0.0148	0.0206

Data: PSID, 1968-2005 (Individuals born between 1950-1968).

Notes: Sampling weights are used to provide nationally-representative estimates.

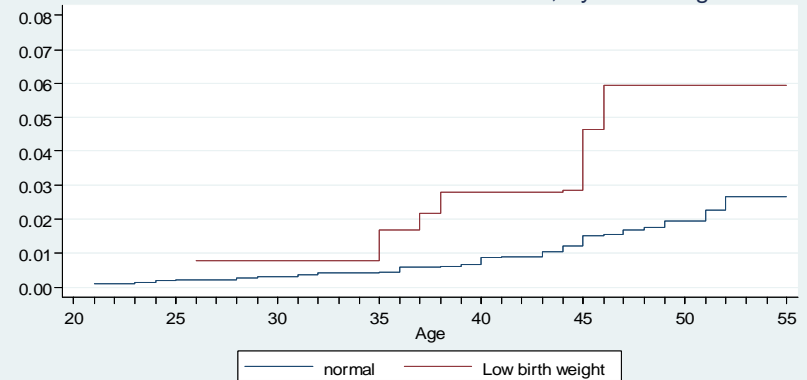
# Cumulative Hazard: Onset of Stroke

## Cumulative Hazard of Onset of Stroke, by Race



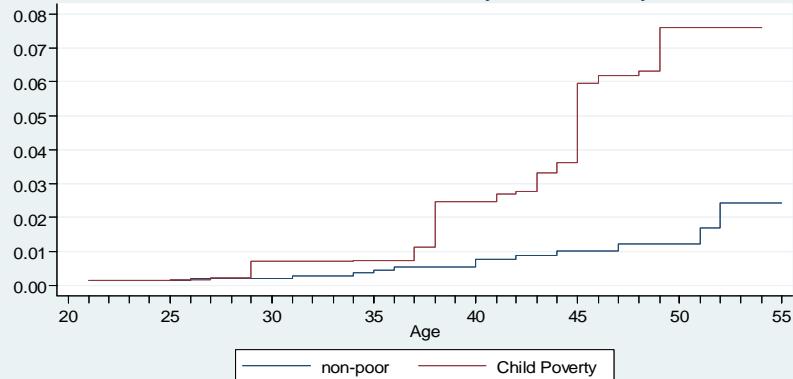
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

## Cumulative Hazard of Onset of Stroke, by Birth weight



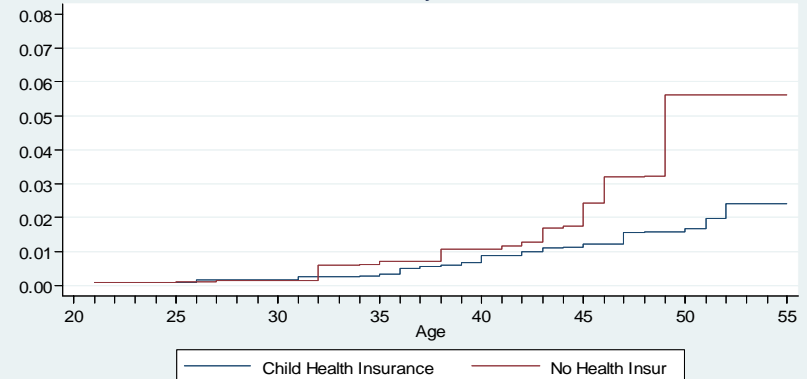
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

## Cum. Hazard: Onset of Stroke, by Child Poverty Status



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

## Cum. Hazard: Stroke, by Child Health Insurance



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

**Table 5. Proportion ever had a Heart Attack,  
by birth weight and child socioeconomic status**

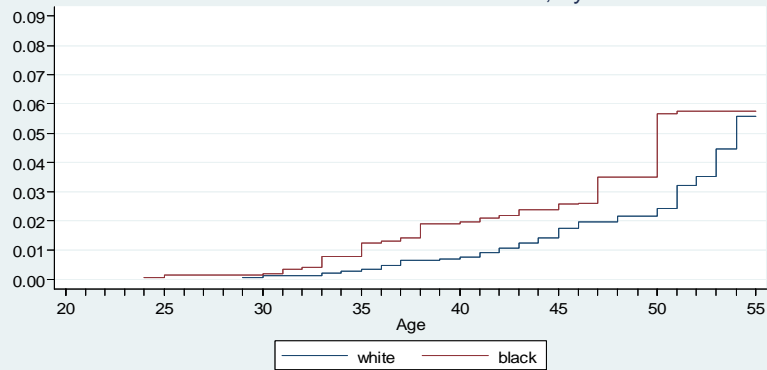
	Proportion ever had a heart attack by age:			
	25	35	45	50
<i>By Birth weight</i>				
Low birth weight	0.0000	0.0143	0.0278	0.0278
Normal birth weight	0.0008	0.0038	0.0153	0.0218
<i>By Child Poverty Status</i>				
Child Poverty	0.0008	0.0031	0.0211	0.0252
Non-poor	0.0000	0.0040	0.0118	0.0165
<i>By Child Health Insurance Status</i>				
No Childhood Health Insurance	0.0003	0.0060	0.0346	0.0457
Health Insurance	0.0008	0.0041	0.0184	0.0251
<i>By Race</i>				
Black, non-Hispanic	0.0014	0.0123	0.0258	0.0568
White, non-Hispanic	0.0005	0.0040	0.0179	0.0247

Data: PSID, 1968-2005 (Individuals born between 1950-1968).

Notes: Sampling weights are used to provide nationally-representative estimates.

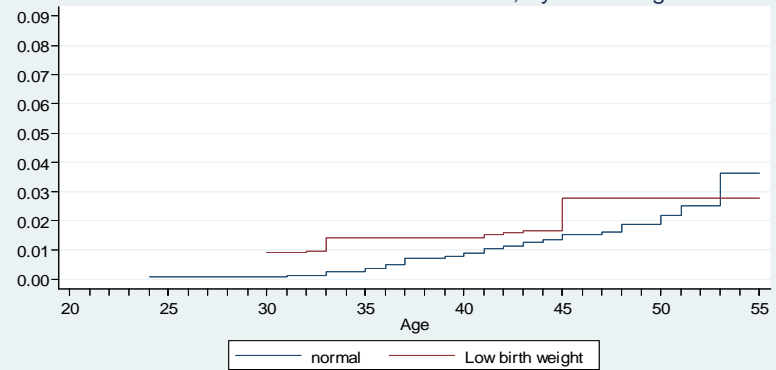
# Cumulative Hazard: Heart Attack

Cumulative Hazard of Heart Attack, by Race



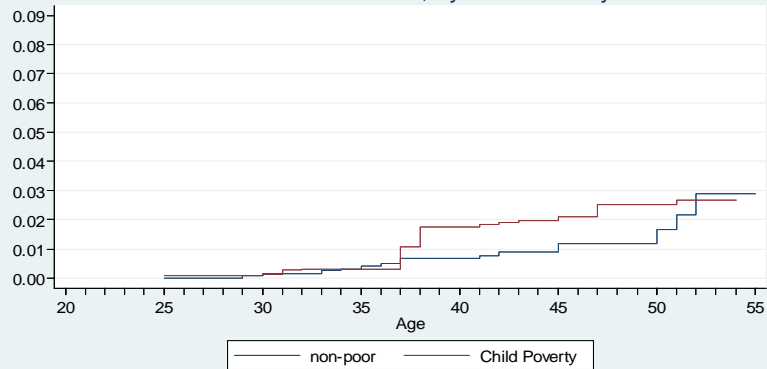
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cumulative Hazard of Heart Attack, by Birth weight



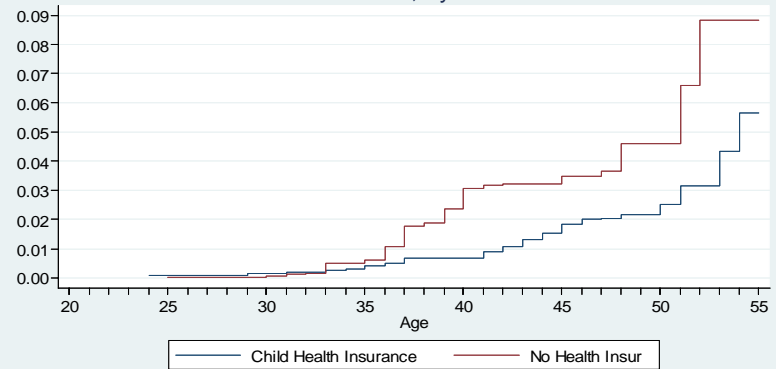
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Heart Attack, by Child Poverty Status



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Heart Attack, by Child Health Insurance



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

**Table 6. Proportion ever had a Stroke or Heart Attack,  
by birth weight and child socioeconomic status**

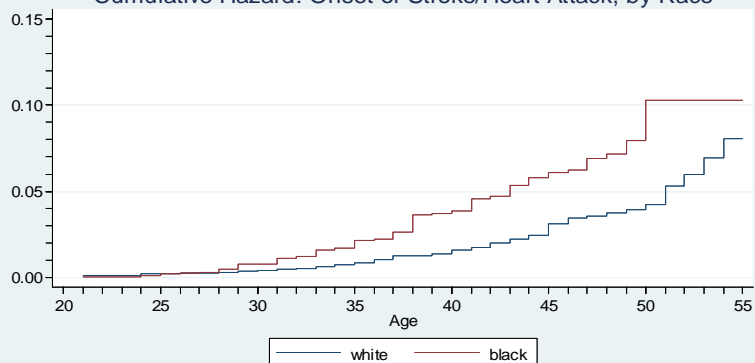
	Proportion ever had a stroke or heart attack by age:			
	25	35	45	50
<i>By Birth weight</i>				
Low birth weight	0.0000	0.0312	0.0691	0.0822
Normal birth weight	0.0028	0.0080	0.0287	0.0396
<i>By Child Poverty Status</i>				
Child Poverty	0.0025	0.0101	0.0732	0.0937
Non-poor	0.0013	0.0084	0.0220	0.0288
<i>By Child Health Insurance Status</i>				
No Childhood Health Insurance	0.0014	0.0120	0.0486	0.0920
Health Insurance	0.0017	0.0075	0.0294	0.0391
<i>By Race</i>				
Black, non-Hispanic	0.0045	0.0238	0.0632	0.1051
White, non-Hispanic	0.0022	0.0089	0.0315	0.0430

Data: PSID, 1968-2005 (Individuals born between 1950-1968).

Notes: Sampling weights are used to provide nationally-representative estimates.

# Cumulative Hazard: Onset of Stroke/Heart Attack

Cumulative Hazard: Onset of Stroke/Heart Attack, by Race



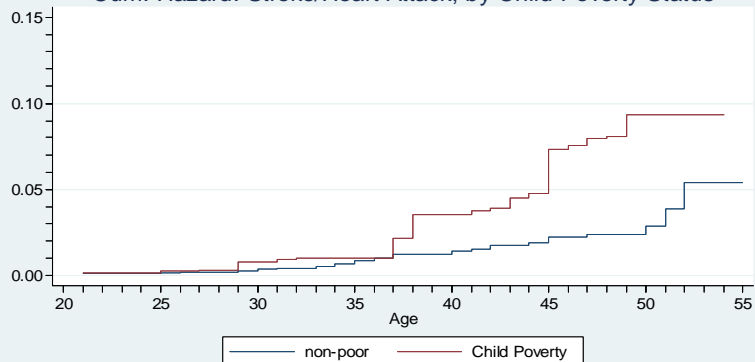
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Stroke/Heart Attack, by Birth weight



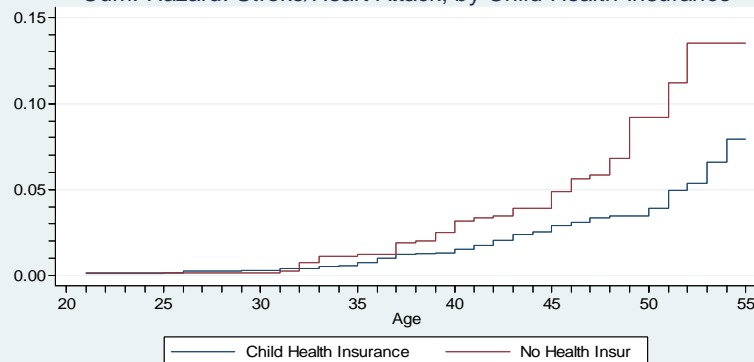
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Stroke/Heart Attack, by Child Poverty Status



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Stroke/Heart Attack, by Child Health Insurance



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

**Table 7. Proportion with Heart Disease, by birth weight and child socioeconomic status**

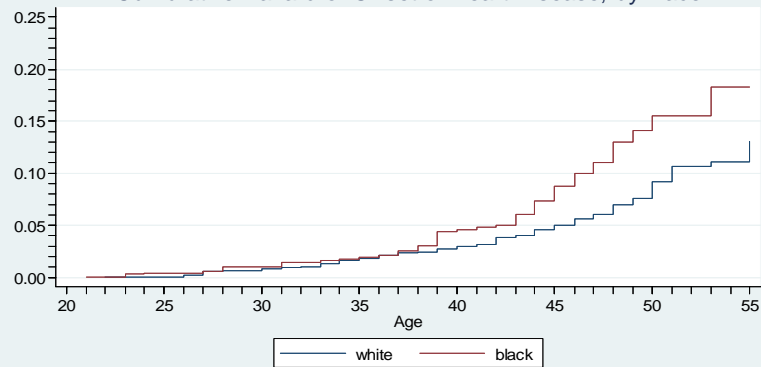
	Proportion ever diagnosed with heart disease by age:			
	25	35	45	50
<i>By Birth weight</i>				
Low birth weight	0.0089	0.0403	0.0815	0.2345
Normal birth weight	0.0057	0.0213	0.0532	0.0804
<i>By Child Poverty Status</i>				
Child Poverty	0.0115	0.0196	0.1049	0.1342
Non-poor	0.0048	0.0257	0.0552	0.0959
<i>By Child Health Insurance Status</i>				
No Childhood Health Insurance	0.0192	0.0368	0.1054	0.1830
Health Insurance	0.0056	0.0239	0.0516	0.0879
<i>By Race</i>				
Black, non-Hispanic	0.0085	0.0238	0.0919	0.1601
White, non-Hispanic	0.0052	0.0230	0.0548	0.0966

Data: PSID, 1968-2005 (Individuals born between 1950-1968).

Notes: Sampling weights are used to provide nationally-representative estimates.

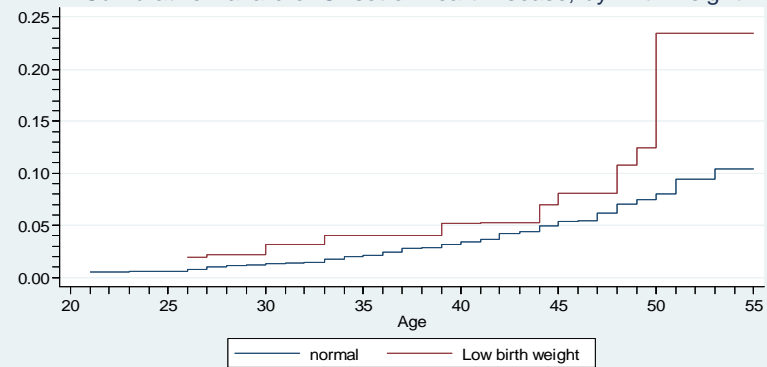
# Cumulative Hazard: Onset of Heart Disease

Cumulative Hazard of Onset of Heart Disease, by Race



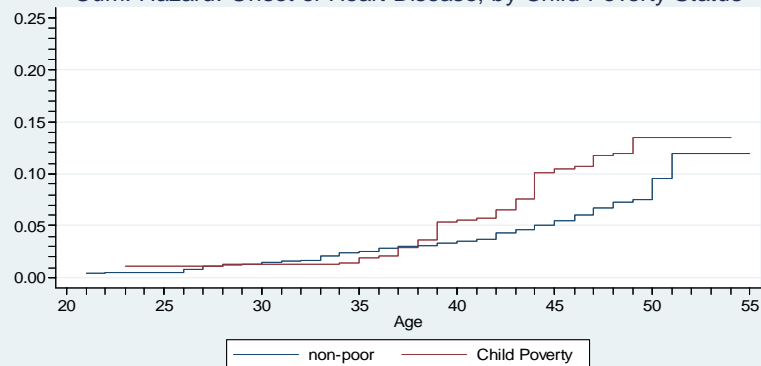
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cumulative Hazard of Onset of Heart Disease, by Birth weight



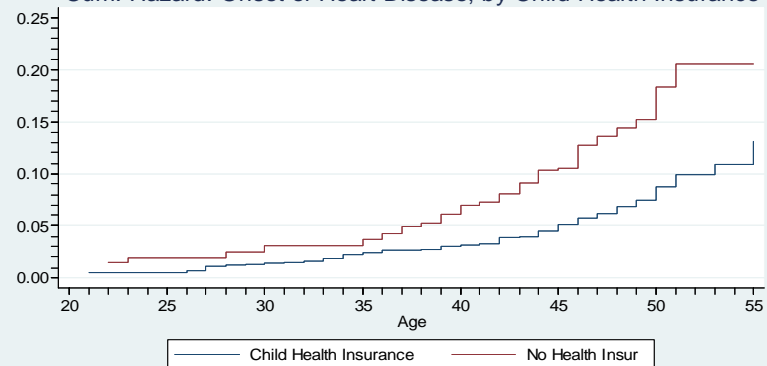
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Onset of Heart Disease, by Child Poverty Status



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Onset of Heart Disease, by Child Health Insurance



Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

**Table 8. Effects of Low Birth Weight and Child Socioeconomic Factors on the Onset of Specific Health Conditions**

	Cox-Proportional Hazard Models of Specific Health Conditions (hazard ratios)					
	Asthma	Hypertension	Diabetes	Stroke	Heart Attack	Heart Disease
	(1)	(2)	(3)	(4)	(5)	(6)
Low birth weight	1.6237** (0.3889)	1.6236*** (0.2624)	1.7012** (0.4401)	2.3775* (1.1132)	1.2576 (0.8054)	1.7560* (0.5096)
Parental income-to-needs ratio at ages 13-16	1.0309 (0.0287)	0.9781 (0.0295)	1.0014 (0.0479)	0.8432 (0.1235)	0.7396* (0.1253)	0.9246* (0.0545)
Health insurance coverage during childhood:						
Private HI coverage in all years 1968 to 1972 (reference)						
No private HI coverage, 1968-1972	1.1909 (0.2597)	1.3658** (0.1921)	1.4432* (0.3815)	0.7965 (0.3844)	0.7738 (0.3931)	1.2154 (0.3284)
Intermittent health insurance coverage, 1968-1972	0.9243 (0.1502)	1.1914* (0.1280)	1.1188 (0.2101)	1.1862 (0.4133)	0.5333 (0.2082)	1.0544 (0.2297)
Female	1.6541*** (0.2192)	0.8185** (0.0686)	1.1955 (0.1891)	1.7860* (0.5800)	0.2683*** (0.0812)	1.0327 (0.1736)
Black, non-Hispanic	1.0984 (0.2220)	1.4094*** (0.1588)	1.2275 (0.2738)	1.6243 (0.6047)	1.0742 (0.4120)	1.0281 (0.2350)
Parental health status, age 50-59						
Mother: proportion of 50s in problematic health	1.3758* (0.2572)	1.2328* (0.1538)	1.7240** (0.3657)	2.0986* (0.8738)	1.6392 (0.7462)	2.1373*** (0.5241)
Father: proportion of 50s in problematic health	1.1333 (0.2484)	1.0745 (0.1605)	0.8564 (0.2454)	1.5816 (0.7951)	1.8227 (0.8797)	1.2080 (0.3384)
Parent's (head's) education:						
High school dropout (reference group)						
High school degree	0.8078 (0.1393)	0.7452*** (0.0802)	0.9160 (0.1803)	1.1805 (0.3930)	0.9547 (0.3754)	0.8682 (0.1878)
Some college	1.1950 (0.2248)	0.5737*** (0.0789)	0.6626* (0.1704)	0.9519 (0.4393)	0.5175 (0.2826)	1.0692 (0.2829)
Parental health behaviors:						
Smoked cigarettes at some point, 1968-1972	1.0538 (0.1859)	0.9755 (0.1173)	1.2759 (0.2934)	2.0069* (1.0455)	1.1047 (0.5432)	1.0846 (0.2650)
Annual cigarette expenditures (in \$100's), 5-year average 1968-1972	1.0006 (0.0123)	0.9992 (0.0093)	0.9997 (0.0156)	1.0200 (0.0335)	1.0277 (0.0320)	1.0057 (0.0168)
Number of families	1,415	1,415	1,415	1,415	1,415	1,415
Number of individuals	2,951	2,951	2,951	2,951	2,951	2,951
Person-year observations	125,947	125,274	132,185	133,883	133,958	132,093

Robust standard errors (clustered on individual) in parentheses

\*\*\* p<0.01, \*\* p<0.05, \* p<0.10 (one-tailed test)

Note: All models include controls for birth order, mother's age at birth, whether born into two-parent family, and birth year (coefficients suppressed in Table to conserve space).

**Table 9. Sibling Fixed-Effect Estimates of Effects of Low Birth Weight and Childhood Health on the Onset of Specific Health Conditions**

	Cox-Proportional Hazard Models of Specific Health Conditions (hazard ratios)					
	Asthma		Hypertension		Stroke or Heart Attack	
	non-FE	FE	non-FE	FE	non-FE	FE
	(1)	(2)	(3)	(4)	(5)	(6)
Low birth weight	1.5768*	1.6663*	1.5438***	1.3820*	1.5963	6.5304**
	(0.3823)	(0.6364)	(0.2542)	(0.3089)	(0.6617)	(5.6843)
Parental income-to-needs ratio at ages 13-16	1.0402	1.1017	0.9779	0.9982	0.8135*	1.0321
	(0.0296)	(0.0671)	(0.0298)	(0.0602)	(0.0895)	(0.2652)
Childhood health status:						
Excellent (reference group)						
Very Good	1.4814**	1.3511*	1.0164	0.8135	0.8802	0.4888*
	(0.2297)	(0.2832)	(0.1049)	(0.1128)	(0.2607)	(0.1793)
Good	1.8762***	1.3598*	1.2014*	0.9239	1.9698**	0.8065
	(0.3537)	(0.3079)	(0.1456)	(0.1487)	(0.6321)	(0.3367)
Fair/Poor	2.7216***	3.0660***	1.7163**	2.0854***	2.1121*	1.0735
	(0.7923)	(1.2176)	(0.3720)	(0.5609)	(0.9563)	(0.7009)
Female	1.5897***	1.4742**	0.8077**	0.8025**	0.5972**	0.8528
	(0.2145)	(0.2484)	(0.0682)	(0.0860)	(0.1307)	(0.2384)
Sibling fixed effects?	no	yes	no	yes	no	yes
Number of families	1,415	1,415	1,415	1,415	1,415	1,415
Number of individuals	2,951	2,951	2,951	2,951	2,951	2,951
Person-year observations	125,947	125,274	132,185	133,883	133,958	132,093

Robust standard errors (clustered on individual) in parentheses

\*\*\* p<0.01, \*\* p<0.05, \* p<0.10 (one-tailed test)

Note: All models include controls for birth order, mother's age at birth, whether born into two-parent family, birth year, and the non-fixed effect models include child health insurance coverage, parental education, parental health status, parental smoking behavior, race, as in Table 8 (coefficients suppressed to conserve space).