

**THE PLACE OF RACE IN HYPERTENSION: HOW FAMILY BACKGROUND AND  
NEIGHBORHOOD CONDITIONS IN CHILDHOOD IMPACT LATER-LIFE HEALTH**

Rucker C. Johnson  
Goldman School of Public Policy  
University of California, Berkeley  
Tel: (510) 643-0169  
E-mail: [ruckerj@berkeley.edu](mailto:ruckerj@berkeley.edu)

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# **THE PLACE OF RACE IN HYPERTENSION: HOW FAMILY BACKGROUND AND NEIGHBORHOOD CONDITIONS DURING CHILDHOOD IMPACT LATER-LIFE HEALTH**

## **ABSTRACT**

This paper investigates the role of family background and neighborhood conditions over the life course, particularly during childhood, in influencing health later in life, with a focus on the case of hypertension. Most of the black-white difference in life expectancy stem from racial differences in mortality rates prior to age 65. Thus, understanding sources of racial health disparities requires the investigation of exposures to socioeconomic conditions and risk factors earlier in the life cycle. Blacks' higher prevalence of cardiovascular disease-related risk factors account for more than half of the racial disparity in life expectancy (Barghaus, Cutler, Fryer, and Glaeser, 2007), with hypertension the leading culprit.

For a US cohort born between 1951 and 1970, I produce nationally representative estimates of the onset of hypertension through mid-life by race/ethnicity, childhood socioeconomic status, and childhood neighborhood poverty. I provide evidence on the consequences of childhood neighborhood poverty on the risks of hypertension; this is the first such study of the full US population. I use nationally representative longitudinal data from the US spanning nearly four decades to estimate hazard models of onset of hypertension. The data set, the Panel Study of Income Dynamics (PSID), has the additional unique feature of allowing analyses of siblings and child neighbors throughout much of their life course. I use the resemblance between neighboring children's subsequent likelihood of hypertension in adulthood in comparison to the similarity between siblings to bound the proportion of inequality in this health condition that can be attributed to disparities in neighborhood and family background. I estimate four-level hierarchical random effects hazard models of the onset of hypertension, which provide a better understanding of the relative importance of family and neighborhood backgrounds. The results demonstrate that both childhood neighborhood conditions and family background influence the disease process and risk of hypertension later in life.

I find childhood neighborhood poverty and its attendant stressors play an influential role in shaping risks of onset of hypertension in middle-age. Other notable neighborhood factors that were shown to influence risks of onset of hypertension in adulthood include childhood neighborhood crime exposure and county per-pupil school expenditures. Notable family background factors include birth weight, parental health status, and parental socioeconomic status. These effects appear linked in part to low intergenerational economic mobility, particularly among blacks. The results indicate that racial differences in these early life neighborhood conditions and family background characteristics play a significant role in explaining racial disparities in hypertension through at least age 50, while contemporaneous economic factors account for relatively little of the racial disparities in this health condition in adulthood.

## **I. INTRODUCTION**

African Americans experience high rates of premature aging. In the US, blacks can expect to live six fewer years than whites, and can expect to live more years with chronic health problems (Hayward and Heron, 1999). Most of the black-white difference in life expectancy stems from racial differences in mortality rates prior to age 65. Thus, understanding sources of racial health disparities requires the investigation of exposures to socioeconomic conditions and risk factors earlier in the life cycle. Blacks' higher prevalence of cardiovascular disease-related risk factors account for more than half of the racial disparity (Barghaus, Cutler, Fryer, and Glaeser, 2007), with hypertension the leading culprit. Hypertension is a major risk factor for heart disease and stroke, the leading causes of death in the US.

Emerging research has sought to identify whether and how early-life differences in exposures to stressful life conditions get under the skin. Recent findings in neuroscience highlight prolonged exposure to stress hormones (e.g., cortisol) can suppress the body's immune response and cause greater vulnerability to chronic health conditions. Early-life experiences of toxic stress, even in the womb, may have profound implications for later-life health (Aizer et al., 2007; Johnson and Schoeni, 2007). Identifying sources of race differences in risk factors, that stem from childhood conditions and lead to elevated levels of adult morbidity and mortality, may shed light on extant inequalities in life chances.

This paper investigates the role of neighborhood conditions over the life course, particularly during childhood, in influencing health later in life, with a focus on the case of hypertension. High blood pressure is the focal point here because it is an important

contributor to the burden of disease, disability, and death in the US, and because there are compelling theoretical reasons to expect chronic stress linked to neighborhood environments to influence hypertension risk. Stressful neighborhood conditions due to high poverty rates, crime, violence, weaker sources of social support, may lead to increased risk of high blood pressure. This analysis examines whether differences across neighborhoods in the prevalence of hypertension, the leading risk factor of cardiovascular-related disease, do in fact reflect causal processes operating over the life course. Given the known lengthy latency periods before health effects like hypertension manifest, it is important to examine whether later-life racial health disparities are rooted in early-life childhood circumstances. Racial differences in rates of hypertension are likely the result of a long-term cumulative process of socioeconomic environmental exposures over the life cycle. The paper seeks to identify causal influences of the life cycle trajectory of inequality in this major cardiovascular disease risk factor. In contrast to cross-sectional evidence linking neighborhoods and health in a static framework, this paper considers the life course residential neighborhood quality histories and their implications for later-life health.

The typical analytical approach used in neighborhood studies is to regress individual level outcomes such as education, criminal activity, or health on neighborhood-level factors such as census tract mean income, poverty rates, or rates of single motherhood. But attempts to estimate causal effects of neighborhood context have faced well-documented challenges of endogeneity (Manski, 1993) and of obtaining accurate measures of neighborhood factors. Few studies have used convincing

identification strategies to overcome these challenges, exceptions being experimental evaluations such as Katz, Kling, Liebman (2001) and Leventhal & Brooks-Gunn (2001).

This paper takes a different approach that largely side-steps these challenges by exploiting a unique feature of the Panel Study of Income Dynamics (PSID). Specifically, the initial PSID sample in 1968 was highly clustered, allowing one to compare the similarity in onset of hypertension between siblings who grew up together, versus unrelated individuals who grew up in the same narrowly defined neighborhood. Instead of performing another typical regression analysis focused on particular neighborhood characteristics, the first goal of the analysis is focused on an overall assessment of the relative contributions of individual, childhood family and neighborhood effects on the onset of hypertension. I use the resemblance between neighboring children's subsequent likelihood of hypertension in adulthood to bound the proportion of inequality in this health condition that can be attributed to disparities in neighborhood background.

The resemblance in the risk of hypertension onset in adulthood among childhood neighbors reflects the lasting and composite influence of factors shared by individuals from the same neighborhood—measured and unmeasured. This approach avoids the difficulty of defining neighborhood quality at the outset and, instead, compares the relative magnitudes of the child neighborhood and family components, placing an upper bound on the neighborhood influence. The magnitude of the overall neighborhood component (from the unconditional random effects hazard model of onset of hypertension) represents an omnibus measure of the overall scope of child neighborhood effects, but is an upper bound because some of the influence may emanate from child neighbors having similar family background characteristics. A central aim of this work is

to disentangle true causal influences of neighborhoods from influences stemming from familial selection into neighborhoods, and to examine whether (and how) child neighborhood context influences subsequent hypertension risk in a way that cannot be reduced to the characteristics of the individuals themselves.

I use nationally representative longitudinal data from the US spanning nearly four decades, and estimate hazard models of onset of hypertension. The data set, the Panel Study of Income Dynamics (PSID), has the unique feature of allowing analyses of siblings and child neighbors 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, and it also contains significant detail on health. The data includes extensive socioeconomic measures in childhood and adulthood and does not have to rely on retrospective reports of early-life economic conditions. I estimate four-level hierarchical random effects hazard models of the onset of hypertension, which provide a better understanding of the relative importance of family and neighborhood backgrounds. I use maximum ‘marginal’ likelihood estimation procedures to allow for the multilevel components at the county, neighborhood, and family levels including measured and unmeasured components at each of these levels.

Upon uncovering a significant overall scope of the influence of childhood neighborhoods on hypertension in this paper, I empirically test the hypothesis that exposure to a factor in early life—childhood neighborhood poverty—influences the disease process and risk of hypertension later in life. For a US cohort born between 1951 and 1970, I produce nationally representative estimates of the onset of hypertension

through mid-life by race/ethnicity, childhood socioeconomic status, and childhood neighborhood poverty. I provide evidence on the consequences of childhood neighborhood poverty on the risks of hypertension; this is the first such study of the full US population. The results highlight the significant role of childhood neighborhood poverty and its attendant stressors in shaping risks of onset of hypertension in middle-age. A subset of models also includes an unusually extensive array of family and neighborhood background factors, including measures of residential segregation, county per-pupil school expenditures, neighborhood crime, child health insurance coverage, parental health status, permanent income, parental education, parental expectations for the educational attainment of the child, family structure, and parental health behaviors. The results indicate that racial differences in these early life neighborhood conditions play a significant role in explaining racial disparities in hypertension through at least age 50.

The results highlight significant roles of both childhood neighborhood conditions and family background on the onset of hypertension over the life course. These effects appear linked in part to low intergenerational economic mobility, particularly among blacks. The results indicate that neighborhood poverty during childhood significantly increases risks of hypertension through mid-life. To probe the robustness of a causal inference, I use a novel empirical approach, recently proposed by Altonji et al. (2005), to gauge how sensitive estimates of the effects of neighborhood poverty are to selection on unobserved variables. I find that even a large amount of selection on unobservable factors does not completely eliminate the significant effect of child neighborhood poverty on hypertension later in life. The ratio of selection on unobservables to selection on

observables would have to exceed 80% in order for one to attribute the entire effect of neighborhood poverty to selection bias.

The remainder of the paper is organized in the following way. I begin with a discussion of how neighborhood and family background may affect an individual's health trajectory in adulthood, and risk of hypertension in particular. I briefly summarize relevant previous research and highlight the relevant theoretical issues that motivate the empirical analyses to follow. Section III lays out the methodological challenges in estimating neighborhood effects. The data are described in section IV. Sections V and VI discuss the econometric model and estimation methods, respectively. The results are presented in section VII, with concluding statements provided in the final section.

## **II. WHY MIGHT NEIGHBORHOOD AND FAMILY BACKGROUND MATTER?**

Family background can have direct effects on health status over the life course through several mechanisms. Transmission of genetic traits from parents to children clearly plays an important role. Parental socioeconomic and demographic factors most likely influence children's health status (Case, Lubotsky, and Paxson, 2002), which in turn carries through to health in adulthood. The transmission of health lifestyle orientation – eating habits, and exercise and smoking behaviors, for example – across generations may also translate into disparities in adult health.

Similarly, it has been hypothesized that neighborhood background can have direct effects on health. Childhood neighborhood factors such as water and air quality, sanitation, pollution and environmental toxins, crime, health care and social services, and public schools most likely have some influence on childhood health. Health lifestyle orientation may also have a neighborhood component as well, with peer groups and role

models within communities or neighborhoods influencing children's opportunities and preferences (Johnson, 2007).

Perhaps equally or more importantly for health dynamics, neighborhood and family background may have indirect effects on health over the life course through their effects on the socioeconomic mobility process. The degree of socioeconomic mobility has direct implications on the resemblance of an individual's childhood and adulthood family characteristics, such as income and education, which may in turn affect health. Since economic status is a major determinant of residential choice, persistence in economic status is likely to lead to persistence in neighborhood quality as well; that is, the lower economic mobility is, the greater the correlation between childhood and adulthood neighborhood characteristics.

### *Stress & SES*

This conceptual framework is consistent with a stress and adaptation perspective on how stressful neighborhood conditions may influence health trajectories. The hypothesis that the differential burden of lifetime stress contributes to racial and socioeconomic disparities in health builds on a solid scientific foundation. Prolonged exposure to stress produces elevated risks of a condition known as allostatic load, which refers to the physiological costs of chronic overactivity or underactivity of systems within the body (e.g., the hypothalamic-pituitary-adrenal axis or the autonomic nervous system) that fluctuate to meet demands of chronic exposure to environmental stressors (McEwen, 1998). Persistent exposures to disadvantaged neighborhood and family conditions may have a cumulative toll in the form of "weathering" (Geronimus, 1992). How people are

affected and adapt to stressful neighborhood environments may depend, in part, on their access to informal sources of social support.

### *Previous Research*

Recent findings in neuroscience indicates that early-life risk factors compound over the life cycle—often-cited examples of the adaptive cost of stress-induced wear and tear (“weathering”) include pushing the endocrine system toward diabetes or the cardiovascular system toward coronary artery disease and hypertension (Halfon and Hochstein, 2002). Blacks appear to face more stress than comparable whites (Geronimus et al., 2006; Cohen et al., 2006), as evidenced in studies that document higher cortisol levels—a hormone that is a marker for stress—among blacks even after accounting for family income (DeSantis et al., 2007).

Recent findings in neuroscience also indicate that developmental health trajectories can be altered more readily during sensitive periods of rapid developmental change than during other periods. Heckman (2007) emphasizes that, “common developmental processes are at work where some cognitive and non-cognitive skills and health capabilities at one stage in childhood cross-fertilize the productivity of investment at later stages”.

A prior study (Fang, Madhavan, and Alderman, 1996) showed that the health status of blacks living in New York City as adults was associated with their place of birth: the northern United States, the southern United States, or the Caribbean. The health status of those from the Caribbean was similar to New York City whites, and better than those from the northern United States; in turn, those from the northern United States had

better health status than those from the southern United States. These findings point to the enduring influence of childhood circumstances, while not identifying what aspects of upbringing matter most.

Chronic health conditions, like hypertension, 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).

Previous research on health effects of neighborhoods and segregation has been criticized for being atheoretical, and identifying causal mechanisms has remained a principal challenge. The empirical regularity that characterizes most cross-sectional neighborhoods research and drives policy discussions may be misinterpreted. The widely

discussed correlation between contemporaneous neighborhood characteristics (e.g., neighborhood poverty) in adulthood and adult health status may arise only because it is *lifetime neighborhood exposures* that have cumulative effects on health/morbidity/mortality, and lifetime neighborhood quality and family resources are strongly positively related to contemporaneous neighborhood environments in adulthood. The multilevel research design taken in this paper aims to explore this, the relationship between low-income families and the socioeconomic profile of the neighborhoods in which they live, as well as to disentangle the effect of housing quality from neighborhood quality. This clarity is imperative for policy prescriptions that address health inequities.

There is a need to "unpack" the specific exposures and pathways through which neighborhood disadvantage leads to poor health outcomes. The innovative research design and unique measures collected on aspects of neighborhood physical, service and social environments may illuminate what lies along the "chain of causation" from poverty to health outcomes over the life course.

No consensus among researchers has been reached on the assessment of the role of neighborhoods and residential segregation patterns in contributing to racial health disparities. Prior work has uncovered puzzling issues about the role of race in explaining spatial differences in morbidity and mortality that have yet to be resolved. In particular, people die younger in cities and states that are more segregated and have a higher fraction of African Americans in their populations, not only because blacks die younger than whites, but because *both* blacks and whites die younger in places where the population is more heavily black and segregated (Deaton and Lubotsky, 2002). We are left with the

puzzle of why the fraction black and extent of residential segregation should increase morbidity and mortality?

Poor black children are less likely to escape poverty than poor white children (Bhattacharya and Mazumder, 2007). In particular, Hertz (2005) finds 17 percent of whites in the bottom decile of family income remain there as adults compared to 42 percent of black children. Differences in childhood neighborhood conditions and school quality may contribute to lower rates of socioeconomic mobility observed among blacks (Card and Rothstein, 2007). Johnson (2008) documents significantly higher rates of persistent exposures to poor neighborhoods from childhood through mid-life among blacks. In particular, my previous work shows that among cohorts born between 1951-1970, the average black child spent about  $\frac{1}{4}$  of their childhood years in high poverty neighborhoods (i.e., neighborhood poverty rates in excess of thirty percent), and about one-third of their early-to-mid adulthood years (ages 30-50) in high poverty neighborhoods, while only roughly 15 percent of these adulthood years were lived in low poverty neighborhoods (i.e., less than ten percent of households in poverty). In contrast, the comparable estimates for the average white child is only three percent of childhood and adulthood years spent in high poverty neighborhoods, while spending 80 percent of childhood years in low poverty neighborhoods and more than half of early-to-mid adulthood years in low poverty neighborhoods. Furthermore, black-white differences in adulthood exposure to neighborhood poverty are largely accounted for by differences in the likelihood of being born into a poor neighborhood, and to a lesser extent by differences in rates of upward and downward socioeconomic mobility over the life

course. This means that blacks are trapped in high poverty neighborhoods for a significant share of the life course to a far greater extent than whites.

### **III. METHODOLOGICAL CHALLENGES IN ESTIMATING NEIGHBORHOOD EFFECTS**

The primary methodological challenge in estimating the causal effects of neighborhoods on health status is that unobserved factors that affect health may also be correlated with neighborhood factors, leading to biased estimates of neighborhood effects. This can arise from the endogeneity of residential location. That is, individuals and families choose where they live based on the characteristics they value (Tiebout, 1956), although constraints such as racial discrimination and exclusionary zoning may be placed on that decision. In this context, families and individuals who care more about their health will be less likely to choose to live in an area with high crime, pollution, or a poor health care system. Furthermore, the set of complex and nuanced characteristics that influence neighborhood choices are not likely to be well measured and accounted for appropriately in econometric models. Oakes (2004) argues that the lack of attention to neighborhood selection and other identification issues implies their resulting estimates “will always be wrong” (p.1941).

However, economists have yet to make much ground on producing more convincing answers to the question. The question of whether and how neighborhood socioeconomic features influence long-run health trajectories is particularly ill-suited for the typical methods by which microeconometricians solve endogeneity problems (e.g., instrumental variables and fixed effect approaches) for several reasons. First, most health outcomes are a product of cumulative exposures to advantaged/disadvantaged environments spanning decades or exhibit long latent periods before problems manifest.

Therefore, the connection between current neighborhood and current health may say little about the overall influence of neighborhoods factors over the life cycle. As well, it may be important to conceptualize neighborhood effects as cumulative and variable over the life course as opposed to isolated and unchanging. Because most methods for overcoming endogenous neighborhood choice are based on small short-run changes in the neighborhood environment, these approaches might be limited to uncovering effects only for rapidly-responding intermediate outcomes such as health behaviors (e.g., smoking/drinking, exercise/diet). An additional issue is that neighborhood variables of the underlying neighborhood feature of interest are notoriously measured with a great deal of noise. The neighborhood attributes of interest change slowly over time, so most year-to-year variations in the characteristic measured are noise.

The most powerful way to address selection is through a randomized trial. But an experimental design where neighborhoods are randomly assigned is rare. A significant exception is the evaluation of the Move to Opportunity (MTO) program, where an experimental design is used to estimate the effects of offering housing assistance that allows individuals to move out of low-income, poor neighborhoods. Evidence from two sites – Boston and New York – demonstrates that MTO had beneficial effects on the health of children and adults (Katz, Kling, Liebman, 2002; Leventhal and Brooks-Gunn, 2002). This evidence is consistent with the claim that neighborhood factors do in fact influence health status, at least in the short-run among poor families. However, neither of two randomized treatment groups that received housing vouchers to move between 1994 and 1997 experienced lower prevalence of hypertension at follow-up in 2002 compared to the control group that received no vouchers (Kling, Liebman and Katz, 2007). It is

important to bear in mind that the treatment effects represent not only changes in neighborhoods, but also the process of moving, which could mask any beneficial effects of leaving a stressful neighborhood.

Among the studies that have tried to address endogeneity and self-selection using non-experimental methods, the most common approach is the use of instrumental variable techniques (e.g., Evans et al., 1992; Case and Katz, 1991; and McLanahan, 1996), where the exclusion restrictions are tenuous. An alternative non-experimental approach is comparing siblings who have been raised in different neighborhoods at different ages because their parents have moved (Aaronson, 1998; Plotnick and Hoffman, 1996). The key assumption is that the family effect is fixed, not time-varying. If, for example, families' preferences change as their children get older, and they become more interested in living in neighborhoods that are less risky for their children's health, then they might move to neighborhoods with less crime or pollution, which may in turn lead to better health outcomes for their kids. But if the underlying change in their preferences towards health outcomes not only caused them to change neighborhoods, but also to spend more time encouraging their children to practice good health behaviors such as eating healthily, exercising, and avoiding high crime areas, then the neighborhood "effect" might actually be representing all of these other factors and not the true causal effects of neighborhoods *per se*. Moreover, it is quite possible that sibling differences may aggravate the endogeneity problem, as has been discussed in the context of the labor market returns to schooling (Griliches, 1979; Bound and Solon, 1999).

Typical neighborhood studies also face the challenge of identifying and measuring relevant factors. The neighborhood qualities that may in fact matter may be

hard to measure, or they may not be measured in enough spatial detail. This issue is analogous to the finding in the family background literature that sibling correlations in socioeconomic status far exceed what has been explained by any particular measured aspects of the siblings' shared background (Corcoran, Jencks, and Olneck, 1976).

Instead of performing another regression analysis focused on particular neighborhood characteristics, in this paper we exploit a unique feature of the PSID and adopt an approach recently used by Solon et al (2000), Page and Solon (2003), and Duncan et al (2001) to examine the role of contextual factors on educational attainment, earnings, child achievement, and delinquency.<sup>1</sup> Specifically, the initial PSID sample in 1968 was highly clustered with most PSID families having several other sample families living on the same block. This survey design allows us to compare the similarity in hypertension risk in early to mid adulthood between siblings who grew up together, versus unrelated individuals who grew up in the same narrowly defined neighborhood. This approach avoids the difficulty of defining neighborhood quality and instead compares the resemblance in the risks of hypertension in adulthood among siblings with that of their childhood neighbors, placing an upper bound on the neighborhood influence and allowing a comparison of the relative sizes of neighborhood versus family effects.

There are four primary reasons why this approach may be able to detect neighborhood effects in ways previous studies have been unable. First, in contrast to the experimental evidence and previous observational studies, I am examining effects over a much longer time horizon. This is particularly important for most health outcomes, as there is likely a longer lag between poor neighborhood quality and the manifestation of health effects. Second, instead of focusing on contemporaneous neighborhood effects, I

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<sup>1</sup> The analysis of Duncan et al. (2001) focuses on adolescent outcomes using the Add-Health Survey data.

analyze the effects of neighborhood origins, which will include indirect effects operating via the economic mobility process as well as cumulative exposure to neighborhood conditions that may vary over the life cycle. Third, I use the census block as our definition of neighborhood, which comprises a much smaller geographic area than previous studies utilize. Finally, I use estimates of the neighborhood random effects component as an omnibus measure of the potential effects of neighborhood quality (including unmeasured characteristics), rather than initially focusing the analysis on particular observable neighborhood attributes.

#### **IV. DATA**

The PSID began interviewing a national probability sample of families in 1968, and it has re-interviewed the members of those families every year since, with bi-annual interviewing beginning in 1997. Most importantly, when children of the 1968 PSID families became adults and left their parents' homes, these children were interviewed themselves in each year.

The PSID used a "cluster sample" when it started in 1968 in order to economize on interviewing costs. This design effect is typically a liability in statistical analyses because one has to account for non-independence across individuals within the same cluster. But for our purposes the clustering provides the unique opportunity to examine health outcomes for adults who were childhood neighbors in 1968. Moreover, because all 1968 children within a given family are followed throughout their lives, we can examine the similarity in health outcomes over the life-course of siblings and childhood neighbors.

In the analyses, I define the neighborhood of upbringing as the census block where the child lived in 1968.<sup>2</sup> Thus, I am able to use a narrower, compact definition of neighborhood than the vast majority of previous studies of neighborhood effects. Typical studies use census tracts to define neighborhoods, and census tracts, which consist of roughly 5,000 families, are much larger than the neighborhood construct we employ. Although the neighboring families in the PSID sample may or may not have been social neighbors in the sense of interacting closely with each other, they did live in close geographic proximity to each other, and this neighborhood construct should capture important environmental influences. In urban areas, neighboring 1968 families in the PSID may have been a city block or even just part of a block. In rural areas, the families were spread further apart, but still were among each other's closest neighbors (Solon et al, 2000). The PSID cluster design is discussed in greater detail in Solon et al (2000).

The 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. Spanning from 1968 through 2005, it is the longest-running nationally representative US sample of households and is one of the premier data sources of income in the world.

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

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<sup>2</sup> The 1968 addresses were geocoded to census block identifiers using GDT geographic mapping technologies. Census blocks are the smallest level of geographic precision reported by the Census Bureau and represent a narrow definition of neighborhood. Census block identifiers are defined for the entire US in 2000.

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, I 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. I then obtain all available information on these individuals for each wave, 1968 to 2005. Therefore, by 2005 the oldest person in the sample is 57 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 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).

The analysis focuses on the prevalence and age of onset of hypertension. 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 hypertension conservative.

I found no evidence that estimates from the adult sample suffer significant bias from health-related attrition due to selective mortality; any potential bias suggests that early mortality will tend to reduce the estimated effect of neighborhood disadvantage during childhood on later outcomes.

To increase the sample size as well as the proportion of poor and black families in the sample, I include both the Survey Research Center (SRC) component and the Survey of Economic Opportunity (SEO) component, commonly known as the “poverty sample,” of the PSID sample. I appropriately apply multi-level sample weights at the neighborhood and family levels to produce nationally-representative estimates.<sup>3</sup> The results are robust to the exclusion of the SEO sample, as estimates that exclude the SEO sample exhibit the same patterns as those reported in the paper (results available upon request).

The ability to conduct analyses within families and between neighboring families is a unique feature of this research. Because this study is among the first to report evidence of sibling similarity in the onset of hypertension, I include all neighborhoods to increase the effective sample size for the sibling comparison estimates. Results on the

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<sup>3</sup> To be eligible for the SEO sample, households had to have income that was below two times the poverty line, which in theory could be problematic for our purposes because two neighboring families could enter that component of the PSID only if they had sufficiently low income. However, due to the significant degree of residential segregation by income, I find evidence that the typical neighbor of a low-income family was also low income; thus, in practice this does not present any significant within-neighborhood sample selection bias problems. In particular, in the 1968 SRC component of the PSID, the average family with income less than two times the poverty line (in that year) lived in neighborhoods in which neighbors' average income was also among the bottom third of the income distribution. Similarly, using larger national samples geocoded to the census block, Hardman and Ioannides (2005) find that among the poorest 30 percent of households, roughly 75 percent live in neighborhoods in which neighbors' median income is also among the poorest 30 percent of households.

sub-sample that is restricted to neighborhoods containing children from at least two different families yielded very similar magnitudes of sibling and child neighbor resemblance in hypertension risk (results available upon request).

The resultant sample consists of 125,164 person-year observations from 2,942 individuals (1,308 males, 1,634 females) from 1,410 families from 991 original neighborhoods in 95 different counties. The mean age at the most recent survey interview is about 44, with age ranging from 30 to 57. The sample is about 41 percent black.

Income is the total for the family in which the child lives, and it is measured from the five-year average for the years 1967-1972. 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. I 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.

The 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.

### **Descriptive analyses**

Using the PSID sample cohort born between 1951 and 1968, I begin by producing nationally-representative estimates of the cumulative risks through middle-age of hypertension by race/ethnicity, child poverty, child health insurance coverage status, and childhood neighborhood poverty. The results are summarized in Table 1 and in Figure 1. Sampling weights are used to produce nationally-representative estimates. I report Nelson-Aalen estimates of the cumulative risk of hypertension onset by various ages over the life course.

#### *Linking childhood neighborhood poverty to hypertension*

The set of results summarized in Table 1 (Figure 1) display striking differences in the age of onset of hypertension depending on whether the individual grew up in a low poverty neighborhood, medium poverty neighborhood, or high poverty neighborhood; depending on low birth weight status and whether average childhood income was below, close to, or well above the poverty line. I tested for the equality of the survivor functions along these lines and the reported differences are statistically significant in the vast majority of cases.

As shown in Table 1, the differences in risks of onset of hypertension by neighborhood poverty rate, low birth weight status, child poverty, health insurance coverage, and race/ethnicity are all stark. I 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).

It is important to note that the magnitudes of these differences in the risks of hypertension tend to become more pronounced as individuals age, a result that mirrors findings in Johnson and Schoeni (2007). The strong associations between neighborhood poverty rate and childhood socioeconomic factors and likelihood of onset of hypertension shown in Table 1 (Figure 1) do not prove that neighborhood 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 childhood neighborhood conditions and family poverty as well as their children's subsequent poorer health in adulthood. We would expect these other factors to affect risks of hypertension, independent of early-life neighborhood and child socioeconomic

characteristics. A portion of the regression analysis in the subsequent section is devoted to attempting to identify whether it is childhood neighborhood poverty itself and its attendant stressors which leads to the higher disease risks, or these other differences in family characteristics that are the main causal factors.

### *Overview of Empirical Strategy*

A primary goal of the analysis is focused on an overall assessment of the relative contributions of individual, family, and neighborhood effects on the onset of hypertension. I analyze the relative contribution of a parsimonious set of measured individual, household, and neighborhood covariates to the total variation from each component, and test specific hypotheses about the effects of specific characteristics of households and neighborhoods.

The strategy for assessing the importance of contextual effects involves estimating the fraction of variation in the risk of hypertension risk that lies between families and neighborhoods, to provide an upper bound on the possible effect of these contexts. The intuition motivating the use of this strategy is that if family background and residential community are important determinants of the onset of hypertension, there will be a strong resemblance between siblings in disease onset, as compared to two arbitrarily chosen individuals. And if the neighborhood where the child grew up is important, it will show up as a strong similarity between neighboring children's subsequent risks of hypertension in adulthood. This is the first study to simultaneously analyze individual-, family-, and child neighborhood-level variation in the onset of hypertension in a nationally representative sample that covers diverse neighborhood environments. By decomposing disparities into within- and between-area components, it

advances our understanding of the possible contribution of residential segregation experienced in childhood to racial and socioeconomic disparities in hypertension in adulthood.

The similarity in health outcomes between siblings capture the effects of all measured and unmeasured factors shared by siblings that may have an impact on these outcomes, such as the socioeconomic status of parents, genetic traits shared by siblings, family structure, as well as neighborhood effects stemming from the quality of neighborhood conditions (e.g., the school quality and availability of social services, housing and environmental conditions, peer group and role model influences, perceived/actual availability of economic opportunities), and sorting of similar family types within neighborhoods. Documenting the resemblance in health between siblings alone cannot identify the separate effects of family and neighborhood origins.

Analyzing the extent to which hypertension risk in adulthood is correlated among siblings versus childhood neighbors enables us to bound the relative importance of family and neighborhood factors. The sibling resemblance can be decomposed into a part arising from shared neighborhood origins and a part related to family background characteristics. I assess the extent to which hypertension risk are correlated among neighboring children above and beyond the correlation that exists because of similar family backgrounds.

#### **IV. ESTIMATION METHODS AND ECONOMETRIC MODEL**

In order to decompose the total variation in hypertension risk into the fraction that lies between neighborhoods, families, and individuals, we estimate a three-level hierarchical random effects discrete-time hazard model to analyze the age onset of

hypertension through mid-life. I also test a subset of models that includes county-, neighborhood-, and family-level random effects. The data are hierarchical because we have data on individuals who are nested within families, which are nested within neighborhoods and counties. Multilevel modeling techniques can accommodate the hierarchical and unbalanced structure of our data, nonindependence of the (sometimes overlapping) pairs of siblings and neighbors, as well as the nonnormality of hypertension onset (Raudenbush and Bryk, 2002).

The hazard function,  $h_{nfst}$ , is the probability that individual  $s$  from family  $f$  in neighborhood  $n$  becomes hypertensive in year  $t$ , given the individual has never had hypertension in any previous year. The hazard is specified in a logit form, where in the baseline model, the explanatory variables include only a gender dummy indicator, a quadratic specification of age, a set of birth cohort dummy indicators, and a neighborhood-level random effect ( $\eta_n$ ) and family-level random effect ( $\phi_{nf}$ ):

$$h_{nfst} = 1 / (1 - \exp[\alpha_1 Age_{nfst} + \alpha_2 BirthYr + \alpha_3 Female + \eta_n + \phi_{nf}])$$

In this model, we are implicitly assuming proportional odds—in particular, we assume the baseline logit hazard curves in the  $J$  neighborhoods are parallel to one another, and the baseline logit hazard curves in the  $K$  families in these neighborhoods are parallel to one another.<sup>4</sup>

In this formulation the random effects, which play the role of additional error terms, are assumed to be normally distributed with mean 0, and  $\text{var}(\eta_n) = \sigma_n^2$  and  $\text{var}(\phi_{nf}) = \sigma_f^2$ . Here  $\varepsilon_{nfs}$  is an individual error term associated with individual  $s$  from

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<sup>4</sup> Maximum-likelihood (ML) estimates based on a numerical integration procedure were computed using the gllamm6 macro in Stata (Rabe-Hesketh et al. 2000).

family  $f$  in neighborhood  $n$  and is assumed to have a standard logistic distribution with mean 0 and variance  $\frac{\pi^2}{3}$  (where  $\pi \approx 3.14$ ).

In this model, individuals from the same neighborhood but not in the same family (i.e., neighbors) are correlated because they share the random effect  $\eta_n$ , and siblings are correlated because they share the random effects  $\eta_n$  and  $\phi_{nf}$ . Because we did not want our baseline random effects components to reflect the influence of age and gender, we adjusted for them in our baseline model.

I then estimate adjusted neighbor correlations, which are net of the resemblance/similarity arising from childhood neighbors having similar family background characteristics (due to sorting influences). To extract the impact of similar family backgrounds out of the neighbor correlation, I first estimate the following regression; for ease of exposition, here I omit the random effects terms that are included in the estimated model:

$$H_{tsfn}^* = \alpha_0 Age_{tsfn} + \alpha_1 Female_{sfn} + \alpha_2' X_{\bullet\bullet fn} + \alpha_3' (\overline{X_{\bullet\bullet n}}) + \varepsilon_{tsfn}, \quad (7)$$

where  $X_{\bullet\bullet fn}$  is a vector of childhood family background characteristics including: average annual family income-to-needs ratio (based on the five-year average as reported in 1967-1972), parental education, parental family structure, race, child health insurance coverage (as reported in 1967-1972), parental annual expenditures on cigarette and alcohol consumption (based on the five-year average in 1967-1972), indicator for birth weight, parental connectedness to informal sources of help, parental expectations for child achievement, and housing plumbing and insulation problems.  $\overline{X_{\bullet\bullet n}}$  is a vector of the 1968 neighborhood-level means of the same above variables.

Inclusion of family-level and neighborhood-level variables measuring the same concepts enables the vector  $\alpha_2$  of coefficient estimates to capture the within-neighborhood effects of these family background characteristics. Using the within-neighborhood estimates of the family background effects of parental income, education, race, family structure, child health insurance coverage, parental health behaviors, birth weight, parental expectations for child achievement, parental connectedness to informal sources of help and housing quality on health in adulthood, will ensure the coefficients ( $\alpha_2$ ) will not be biased by omitted neighborhood variables. This follows from the fact that the neighborhood-level unmeasured factors can only be correlated with the neighborhood-level mean of the covariates. In combination, the resulting estimates of the effects of these family background characteristics can be taken as a conservative estimate of  $\alpha'X_{fn}$  in equation (1).

I then estimate the inter-neighbor variance in  $\hat{\alpha}'X_{fn}$  by estimating a hierarchical random effects model of  $\hat{\alpha}'X_{fn}$  on neighborhood-level, family-level, and individual-level random effects. I then subtract the estimate of the inter-neighbor variance in  $\hat{\alpha}'X_{fn}$  from the estimate of the overall inter-neighbor variance in  $H_{sfn}^*$ . Dividing the resulting quantity by  $\hat{Var}(H_{sfn}^*)$  yields a tighter upper bound on the proportion of  $Var(H_{sfn}^*)$  that can be attributed to child neighborhood effects.

Parental income and neighborhood poverty are dimensions of family and neighborhood background that I give particular emphasis to in the regression analysis. Growing up in a neighborhood with concentrated poverty may have grave consequences above and beyond those of growing up in a poor family because of the absence of

positive role models, social isolation, weakened social institutions, unrelenting stress, inferior health care accessibility, and other factors. I control for parental education, birth order, whether child was low birth weight, born into a two-parent family, and year of birth. I also make use of a unique set of measures of parental expectations of children's educational attainment, county school expenditures per pupil, residential segregation, neighborhood crime (proxied by neighborhood incarceration rate), parental connectedness to informal sources of help, parental aspirations/motivation and long-term planning, parental personality, habits and skills that were collected in the early years of the PSID. These factors may themselves be the product of growing up in a high poverty neighborhood and may represent important pathways through which exposure to depressed neighborhood environments during childhood affect health trajectories later in life. However, controlling for this myriad of ways in which children who grow up in high poverty neighborhoods may differ from children who grow up in affluent neighborhood environments allows us to generate a more conservative estimate of the effect of neighborhood poverty itself, as well as shed light on the factors that affect hypertension risk.

#### *Sensitivity Analysis*

I conduct a sensitivity analysis to test the robustness of the estimated effects of childhood neighborhood poverty to selection bias due to an omitted variable. The goal is to assess how the point estimate and confidence interval of the effect of neighborhood poverty change under the presence of selection bias of varying strengths. I use a novel empirical approach, recently proposed by Altonji et al. (2005) and Krauth (2007), to perform the sensitivity analysis. This analysis allows us to determine the threshold of

selection on unobservables, if any, at which neighborhood poverty during childhood no longer has a significant effect on hypertension risk. The approach uses the statistical relationship between observed explanatory variables in a regression as a guide to generate plausible estimates about the relationship between observed and unobserved variables.

The sensitivity parameter,  $\theta$ , can be defined as

$$\text{corr}(X_k, u) = \theta \text{corr}(X_k, X\beta - X_k\beta_k),$$

where  $\theta$  indexes the magnitude of the correlation between observables and unobservables relative to the analogous correlation among observables themselves. In other words, the correlation between the neighborhood poverty rate and the (effect-weighted) unobservables is proportional to the correlation between the neighborhood poverty rate and the effect-weighted observables. The standard exogeneity assumption is the special case of  $\theta=0$ . This approach provides a way to construct bounds on the effect of neighborhood poverty during childhood on adult health based on the bounds one is willing to place on the sensitivity parameter  $\theta$  (i.e., the relative correlation).

Altonji et al. (2005) argue that if the observable determinants of an outcome are truly just a random subset of the complete determinants, selection on observable characteristics must be equal to selection on unobservable characteristics. Because the PSID was conducted specifically to study family background factors that affect well-being, we would expect selection on observable factors to be greater than selection on unobservable factors; in other words, the extensive measures of family and neighborhood background captured in the PSID are likely to be the most important determinants of adult health. Thus, estimates obtained under the assumption of equal selection will be biased downwards.

## VI. RESULTS

I first present the unadjusted family, neighborhood, and county components in the onset of hypertension, and examine how much of these effects can be explained by the fact that families in a neighborhood tend to be similar. I begin by estimating the aforementioned unconditional four-level (hierarchical) random effects discrete-time hazard model of onset of hypertension, which includes random components at the county-, neighborhood-, and family-levels. About one-third of individuals in the sample became hypertensive at some point during early-to-mid adulthood. The results are presented in Table 2. Estimates of the random effects of the neighborhood ( $\sigma_n$ ) and family ( $\sigma_f$ ) components indicate that neighborhood and family background have large and significant effects on the likelihood of hypertension onset.<sup>5</sup> For example, the estimated  $\sigma_n$  of .502 implies the odds of onset of hypertension in adulthood for children who grow up in neighborhoods that are one standard deviation below average neighborhood quality are 1.7 times [ $\exp(.502)$ ] the corresponding odds of individuals who grow up in neighborhoods of average quality. Similarly, the estimated  $\sigma_f$  of .652 implies the odds of onset of hypertension in adulthood for children who grow up in families that are one standard deviation below average family background have 92 percent higher odds of onset of hypertension relative to individuals who grow up in families of average background. The county-level random components are much smaller in magnitude but are significant as well. The results indicate that children who grow up in counties that are one standard deviation below average have 27 percent higher odds of onset of hypertension relative to individuals who grow up in counties of average quality. The

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<sup>5</sup> Likelihood ratio tests are used to test the statistical significance of the family and neighborhood random effects.

four-level models were estimated as a robustness check to ensure that the childhood neighborhood random effects components were not primarily driven by effects operating at higher geographic levels of aggregation. The three-level hierarchical models yielded fairly similar patterns as the more computationally-intensive four-level models, so for computational simplicity I report only the results from the three-level models in the remainder of the paper.

I next investigate how much of the magnitude of the estimated neighborhood random effects component may arise from childhood neighbors having similar observed family background characteristics as opposed to emanating from neighborhood effects *per sé*. I find that observable family sorting (controlling for a broad array of family background characteristics described above) explain a significant share but not all the resemblance in adulthood hypertension risk among unrelated individuals who grew up in the same neighborhood. I estimated the resultant adjusted neighborhood component after appropriately extracting the part arising from childhood neighbors having similar observed family background characteristics. (These estimates are based in part on within-neighborhood estimates of effects of child family background on hypertension onset). The adjusted between-neighborhood variance (random effects) component is roughly 50% lower than the unadjusted between-neighborhood variance estimate (results available from author upon request). The adjusted neighborhood random effects component estimates imply that the odds of onset of hypertension in adulthood for children who grow up in neighborhoods that are one standard deviation below average neighborhood quality are 1.5 times [ $\exp(.40)$ ] the corresponding odds of individuals who grow up in neighborhoods of average quality.

The remainder of the analysis and presentation of results proceeds in three stages. First, I estimate the raw black-white gap in the risk of onset of hypertension in adulthood (controlling for age, year of birth, and gender). I then attempt to explain the racial disparity in this specific health condition by adding only childhood family and individual-level factors to the baseline model, including parental education, parental income-to-needs ratio, child health insurance coverage, low birth weight, parental smoking and alcohol expenditures, whether born into two-parent family, and birth order. Second, I examine how adjusting for neighborhood context changes estimates of family- and individual-level disparities in the risk of hypertension onset. To accomplish this I estimate a random effects formulation that is equivalent to a neighborhood fixed effect model by centering all family- and individual-level factors by their neighborhood means. This neighborhood-specific centering generates within-neighborhood estimates of these family- and individual-level factors. In the third set of models, I add an extensive set of observable neighborhood-level variables into the hierarchical model, including childhood neighborhood poverty rate, county school expenditures per pupil, measures of residential segregation, parental connectedness to informal sources of help, parental expectations of children's educational attainment, parental aspirations/motivation and long-term planning, parental personality, habits and skills.

In a subset of these models, I also include parental self-reports of neighborhood and housing quality collected in the PSID. These measures include self-reports of whether it is a poor neighborhood for children, whether there exist plumbing problems, housing structural problems, security problems, cockroach or rat problems, insulation problems, neighborhood cleanliness problems, overcrowding, noise, or traffic problems,

burglary, robbery, assault, drug use, or problems related to too few police in the neighborhood in which they live.<sup>6</sup>

In this third set of models, I provide estimates of the distinct effects of neighborhood and family level background variables measuring the same concepts—for example, the effects of family SES conditional on neighborhood SES and vice versa. In addition, explicitly measuring the magnitude of variation in the effects of unmeasured factors allows an assessment of the importance (quasi- $R^2$ ) of the measured variables,  $X$ , in total variation at each level (e.g., measures vs. unmeasured neighborhood characteristics). In a final set of models, I include measures of the individual's own economic status in adulthood into the three-level hierarchical random effects model to examine the extent to which the importance of childhood neighborhood and family background factors operate through their influence on subsequent socioeconomic attainments in adulthood. The results from these models also serve to demonstrate what aspects and sources of current adult health disparities are missed using traditional models that focus more on contemporaneous socioeconomic factors, without the emphasis on earlier life factors. One must use some caution, however, with drawing causal inferences from these coefficient estimates. The estimates are intended instead to summarize the relationships between the risks of hypertension onset in adulthood with various dimensions of neighborhood and family background.

The results are presented in Table 3. As shown in column (2), blacks have twice [ $\exp(.708)$ ] the odds of onset of hypertension in adulthood than whites experience. The

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<sup>6</sup> These measures serve as proxies of neighborhood quality as this information was only collected in the 1975 survey and may not reflect the characteristics of the 1968 neighborhood due to residential mobility over the period. However, as discussed earlier, 1968 families with children in the PSID tended to move to neighborhoods that had observable neighborhood characteristics that were similar to their previous residential location (Kunz et al, 2001).

racial gap is cut in half after inclusion of childhood family and individual-level factors. The most prominent childhood family background factors on hypertension risk include parental education, low birth weight, and child health insurance coverage (parental income was not statistically significant after inclusion of these factors). For example, children with parents who did not graduate from high school have 1.4 times [ $\exp(.361)$ ] the odds of hypertension onset in adulthood as those with parents who were high school graduates (without college attendance); and college-educated parents had children whose odds of hypertension in adulthood was roughly 30 percent lower than that of individuals whose parents were high school graduates (without college attendance). Low birth weight children have, on average, nearly 75 percent higher odds of hypertension onset in adulthood (relative to normal birth weight individuals); and individuals who had access to private health insurance coverage during childhood had about 20 percent lower odds of hypertension onset in adulthood (relative to those who lacked continuous coverage during childhood). These latter findings confirm those reported in Johnson & Schoeni (2007). The patterns of results for the childhood family factors remain after neighborhood adjustments (column (3)) and inclusion of the set of childhood neighborhood factors (column (4)).

The black-white gap is substantially reduced after inclusion of both the childhood neighborhood and family background characteristics—indeed, blacks have just 25 percent higher odds of hypertension onset in adulthood after accounting for these childhood factors (and the race coefficient ceases to be statistically significant at the 10 percent level).

The results indicate that children who grew up in low poverty neighborhoods had about 25 percent lower odds of hypertension onset in adulthood relative to children who spent their childhood living in medium and high poverty neighborhoods. Additionally, growing up in high crime neighborhoods (as proxied by childhood neighborhood incarceration rate) is linked with increased risks of hypertension in adulthood—in particular, a one standard deviation increase in the neighborhood incarceration rate (or roughly a one-percentage point increase) corresponds with roughly a twelve percent increase in the odds of hypertension onset in adulthood.

The results also indicate that a one-thousand dollar increase in county school expenditures per pupil during childhood is related to nearly a 25 percent reduction in the odds of hypertension onset in adulthood. These findings suggest that the linkages may be the result of how they influence the socioeconomic mobility process. Johnson (2008) demonstrates these factors also significantly influence mobility prospects, and explain part of black-white differences in rates of upward mobility from poor families.

It is striking that the lion's share of the enormous racial disparities in hypertension risk can be explained by differences in these childhood factors, particularly neighborhood poverty rate, birth weight, parental education, and child health insurance coverage. In the case of hypertension, the black-white gap remains but its magnitude is a small fraction of the unconditional raw differences after inclusion of these early-life factors. Taken together, the cumulative set of childhood neighborhood and family background factors account for one-half of the neighborhood-level variance in hypertension risk during adulthood (implied quasi- $R^2$  at the neighborhood level); the fact that these measures

account for less of the family-level variance may be the result of the fact that family-level influences include genetic/hereditary risk factors.

A substantial literature has investigated whether contemporaneous economic factors can account for the racial disparities in adult health. In column (6) of Tables 3, I use our models to re-examine this issue and find that sixty-five percent of the black-white gap in hypertension risk remains after accounting for adult socioeconomic factors. The final model includes both the full set of childhood neighborhood and family background factors along with contemporaneous adult socioeconomic status measures (adult neighborhood poverty rate, educational attainment, adult earnings). As shown in column (7) of Tables 3, the results reveal that racial differences in onset of hypertension in adulthood can be accounted for by childhood family and neighborhood factors, while contemporaneous economic factors account for relatively little of this gap. Educational attainment and the proportion of adulthood years living in low poverty neighborhoods were the main adult socioeconomic factors associated with the risks of hypertension onset in adulthood. The coefficient estimates on the childhood neighborhood and family background factors are reduced to some extent with the inclusion of the adult socioeconomic status measures, but the childhood factors remain large and significant. These findings parallel results of Johnson and Schoeni (2007), in which they find that early-life factors can account for the entire black-white gap in general health status, while contemporaneous economic factors account for relatively little of this gap.

*Sensitivity to Selection Bias.* The estimates of the significant effects of neighborhood poverty during childhood on adult health reported in Table 3 are based on models in which exogeneity is assumed. I next evaluate the robustness of these results to

deviations from exogeneity. Figure 6 displays the range of estimated coefficients and confidence intervals on neighborhood poverty as a function of the ratio of selection on unobservables to selection on observables. As the figure shows, the effect of child neighborhood poverty on the risk of onset of hypertension in adulthood remains large and significant even with a reasonably large amount of selection on unobservable factors. The correlation between neighborhood poverty and unobserved outcome-relevant factors would need to exceed 80% of the correlation between neighborhood poverty and observed-relevant factors in order to eliminate the estimated effect. In other words, the ratio of selection on unobservables to selection on observables would have to exceed 80% in order for one to attribute the entire effect of neighborhood poverty to selection bias.

## **CONCLUSION**

This study provides the first evidence on the influence of childhood neighborhood conditions on the onset of hypertension through middle-age for a nationally representative sample of the US population. Hypertension is an important contributor to the burden of disease, disability, and death in the US, with a significant socioeconomic gradient. I find childhood neighborhood poverty and its attendant stressors play an influential role in shaping risks of onset of hypertension in middle-age. Other notable neighborhood factors that were shown to influence risks of onset of hypertension in adulthood include childhood neighborhood crime and county per-pupil school expenditures; and notable family background factors include birth weight, parental health status, and parental socioeconomic status. These effects appear linked in part to low intergenerational economic mobility, particularly among blacks. The results indicate that

racial differences in these early life neighborhood conditions and family background characteristics play a significant role in explaining racial disparities in hypertension through at least age 50.

Most current US health policies focus on individuals and families as the locus of intervention to improve population health, with a traditional emphasis on programs aimed at increasing access to health insurance and provision of medical care services. The similarities between Canada and the US suggest that access to health insurance is not the driver for the increasingly steep gradient in cardiovascular health. Cardiovascular-related disease of which hypertension is the leading modifiable risk factor is traditionally viewed as the outcome of individual choice and medical care. As a result, prevention has focused on individual-centered interventions, including health education and awareness to motivate individuals to change their health behaviors and the early detection of risk factors and the treatment of high blood pressure through the medical system (Roux, 2003). Short-term public health policies focus on smoking, alcohol use, obesity, and exercise. However, influencing health behaviors in isolation may be less effective than modifying the neighborhood conditions that facilitate and promote disease risk (Syme, 1986). The results from this study point to the potential for targeting neighborhood conditions as a means of improving population health and confronting health inequality.

Racial differences in mortality and morbidity in adulthood are large in the US. I find that a few early life factors – neighborhood poverty, birth weight, parental socioeconomic status – can account for the lion's share of the gaps in the prevalence of hypertension in adulthood. While contemporaneous socioeconomic factors have been the

focus of the literature on racial disparities in health, the results suggest that adult health condition disparities may be better explained by early life factors.

Our results highlight the roles of neighborhood poverty and its attendant stressors on the onset of hypertension. 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 stressful neighborhood conditions 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. 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.

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**Table 1. Proportion with Hypertension, by race and child socioeconomic status**

	Proportion ever diagnosed with hypertension by age:			
	25	35	45	50
<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
<i>By Child Neighborhood Poverty Status</i>				
High Poverty Neighborhood	0.0529	0.1601	0.4335	0.7813
Medium Poverty Neighborhood	0.0201	0.1425	0.4062	0.6002
Low Poverty Neighborhood	0.0225	0.0850	0.2339	0.3893
<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

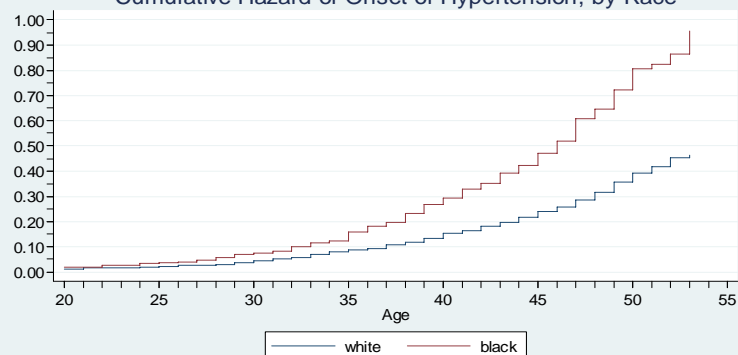
Data: PSID, 1968-2005 (Individuals born between 1950-1968).

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

FIGURE 1

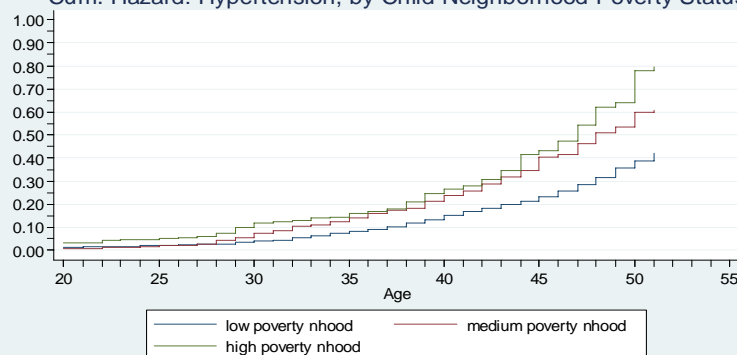
# Cumulative Hazard: Onset of Hypertension

Cumulative Hazard of Onset of Hypertension, by Race



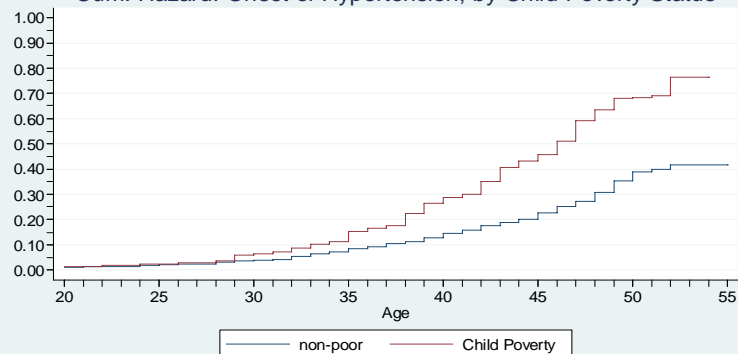
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

Cum. Hazard: Hypertension, by Child Neighborhood Poverty Status



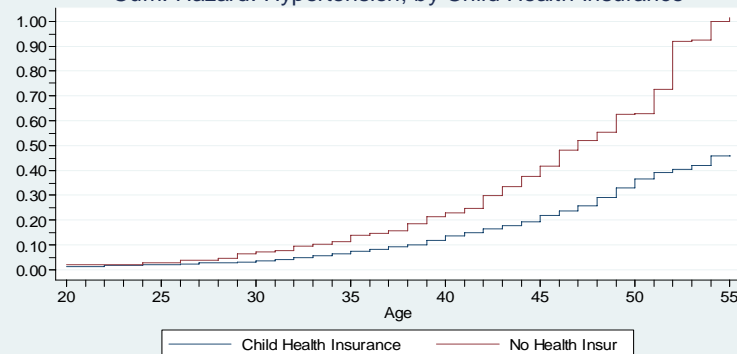
Data: PSID, 1968-2005  
(Individuals born b/w 1950-1968)

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 2. Importance of Child Neighborhood & Family Background on Onset of Hypertension**

**(Discrete-Time Hazard Model of Onset of Hypertension)**

Hierarchical Logit Model	3-Level	4-Level
	(1)	(2)
Age - 40	0.1114*** (0.0071)	0.1103*** (0.0071)
(Age - 40) <sup>2</sup>	-0.0016** (0.0006)	-0.0010 (0.0009)
(Age - 40) <sup>3</sup>	-0.0000 (0.0000)	-0.0000 (0.0000)
Year born - 1960	0.0398*** (0.0105)	0.0344*** (0.0126)
Female	-0.2086** (0.0924)	-0.1950+ (0.1457)
Constant	-4.0253*** (0.0914)	-4.0859*** (0.1151)
<b>Random Effects, Unmeasured (Std Dev)</b>		
Childhood County component		0.2415*** (0.0525)
Childhood Neighborhood component	0.5925*** (0.0952)	0.5020*** (0.1468)
Childhood Family component	0.5267*** (0.1542)	0.6519*** (0.1681)
Log-likelihood	-66753.161	-2939400.6
Number of counties	95	95
Number of neighborhoods	991	991
Number of families	1,410	1,410
Number of individuals	2,942	2,942
Number of person-year observations	125,164	125,164

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

Note: Robust standard errors in parentheses and all standard errors are Huber-corrected, clustered on county.

**Table 3. Race Differences in Onset of Hypertension: Importance of Child Neighborhood & Family Background**

(Discrete-Time Hazard Model of Onset of Hypertension)  
3-Level Hierarchical Logit Model

	Uncond'l model	Raw race gap	No adjustment for neighborhood	Neighborhood adjustment <sup>a</sup>	Controls for Child Nhood + Fam bckgrd	Only Adult Nhood + SES	Child bckgrd + Adult SES
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
<b>Childhood factors</b>							
Black		0.7081*** (0.1082)	0.3553*** (0.1314)	0.3559 (0.5518)	0.2334+ (0.1450)	0.4592*** (0.1199)	0.1894+ (0.1467)
Family income-to-needs ratio (avg during 1967-1972)			-0.0184 (0.0352)	-0.0599 (0.0484)	-0.0081 (0.0369)		0.0177 (0.0369)
High school dropout			0.3612*** (0.1236)	0.2285+ (0.1724)	0.3029** (0.1240)		0.2890** (0.1263)
High school graduate (reference category)							
College-educated			-0.3427** (0.1519)	-0.3914* (0.2082)	-0.3528** (0.1533)		-0.2855* (0.1568)
Private HI coverage in all years 1968 to 1972			-0.2012* (0.1187)	-0.1941 (0.1713)	-0.1780+ (0.1150)		-0.1507+ (0.1150)
Low birth weight			0.5487*** (0.1764)	0.5896*** (0.1746)	0.5400*** (0.1762)		0.5179*** (0.1794)
Birth order			-0.0246 (0.0266)	0.0114 (0.0278)	-0.0208 (0.0259)		-0.0292 (0.0257)
Mother unmarried at child's birth			0.1435 (0.1155)	0.2612** (0.1170)	0.1556+ (0.1171)		0.1428 (0.1177)
Smoked cigarettes at some point, 1968-1972			-0.0764 (0.1190)	-0.1081 (0.1519)	-0.0785 (0.1155)		-0.1233 (0.1149)
Annual cigarette expenditures (in \$100's), 5-year average 1968-1972			0.0036 (0.0099)	0.0077 (0.0135)	0.0042 (0.0099)		0.0056 (0.0101)
<b>Child Neighborhood factors</b>							
Residential segregation dissimilarity index, 1970 (MSA)					0.2446 (0.7622)		-0.0262 (0.7535)
Proportion of childhood yrs lived in low poverty neighborhood					-0.3020* (0.1703)		-0.2312+ (0.1716)
Neighborhood incarceration rate, 1970					0.1169** (0.0527)		0.1275** (0.0524)
County school expenditures per pupil, 1962					-0.2536** (0.1008)		-0.2022** (0.1016)
Parental high expectations for child achievement					-0.1194 (0.1232)		-0.0838 (0.1247)
<b>Adulthood SES</b>							
Proportion of adulthood yrs lived in low poverty neighborhood						-0.4583*** (0.1445)	-0.3547** (0.1520)
Years of completed education						-0.0916*** (0.0242)	-0.0530** (0.0268)
Ln(annual labor earnings)						-0.0775* (0.0431)	-0.0649+ (0.0443)
<b>Random Effects, Unmeasured (Std Dev)</b>							
Childhood Neighborhood component	0.5925*** (0.0952)	0.5079*** (0.1138)	0.4446*** (0.1217)	0.5969*** (0.0983)	0.4172*** (0.1261)	0.4320*** (0.1091)	0.3747*** (0.1281)
Childhood Family component	0.5267*** (0.1542)	0.5107*** (0.1541)	0.4763*** (0.1440)	0.4719*** (0.1515)	0.4492*** (0.1472)	0.4489*** (0.1557)	0.4564*** (0.1413)
Log-likelihood	-66753.161	-66509.944	-65933.001	-66397.549	-65738.665	-65890.274	-65359.924
Number of counties	95	95	95	95	95	95	95
Number of neighborhoods	991	991	991	991	991	991	991
Number of families	1,410	1,410	1,410	1,410	1,410	1,410	1,410
Number of individuals	2,942	2,942	2,942	2,942	2,942	2,942	2,942
Number of person-year observations	125,164	125,164	125,164	125,164	125,164	125,164	125,164

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

Note: All models include a constant and controls for age, age squared, age cubed, year of birth, gender, and columns (3)-(5) & (7) includes indices intended to capture parental connectedness to informal sources of help, parental aspirations/motivation and long-term planning (coefficients suppressed to conserve space).

<sup>a</sup> Neighborhood adjustment refers to centering all covariates around their neighborhood means, which is the analogous random effects formulation of a neighborhood fixed effects model.