

Health Disparities among Middle-Class African Americans: Exposure to Childhood
Disadvantage or Diminished Returns to Socioeconomic Status

ABSTRACT

Although the existence of racial disparities in health is well documented, much less is known about the underlying mechanisms that create and sustain these divergent trajectories across the lifecourse, especially among nonpoor subpopulations. Using 20 consecutive waves of data from the Panel Study of Income Dynamics (PSID), we seek to determine the extent to which suboptimal levels of self-rated health among college-educated, working-age (18-65) African Americans as opposed to Whites can be attributed to childhood disadvantage or the nonequivalence of socioeconomic status (SES) across race. Findings from multilevel logistic regression models suggest that early life hardship predicts subsequent health trajectories in adulthood, but does little to explain the Black/White gap in the association between family income and self-rated health over time. We conclude that racial disparities in health among the Black middle-class are more likely to be the result of restricted returns to upward mobility than exposure to childhood disadvantage.

In the United States, the health of African Americans lags well behind those of most other racial/ethnic groups (Williams and Sternthal 2010; Williams 2012). Compared to their White counterparts, Black men and women face significantly higher risks of chronic illnesses, infectious diseases, and injuries - all of which to serve to shorten their average life expectancy by as much as 6 years (Murphy, Xu, and Kockanek 2013). However, Black/White disparities in physical wellbeing do not simply take root at advanced ages. Rather, they emerge at birth and fluctuate according to predictable patterns across the lifecourse, with infancy and midlife being the time periods during which racial inequalities are most pronounced (Williams 2005; Williams et al. 2010). These divergent health trajectories translate into distinctly different life experiences for African Americans and Whites, in terms of survival as well as health-related quality of life.

Although the existence of stark racial disparities in physical wellbeing is well documented, much less is known about the underlying mechanisms that create and sustain these divergent trajectories. Socioeconomic status (SES) is known to be one of the most powerful predictors of subsequent morbidity and mortality (Pavalko and Caputo 2013) , so much so that it is commonly referred to as a fundamental cause of population health disparities (Phelan, Link, and Tehranifar 2010). The association between SES and health is generally thought to be positive and monotonic, such that individuals who occupy higher positions on the socioeconomic gradient can expect to live healthier and longer lives than those who occupy positions below them (Adler and Rehkopf 2008; Adler et al. 1994; Elo and Preston 1996). However, the empirical evidence regarding the extent to which the Black/White gap in physical wellbeing is due to racial differences in SES has been mixed, with a majority of studies reporting that disparate levels of

educational attainment, occupational prestige, family or per capita income, and even wealth explain only a portion of racial inequalities in health outcomes (Braveman et al. 2010; Franks, Gold, and Fiscella 2003).

Moreover, the extent to which the health of African Americans lags behind that of Whites is not consistent across levels of SES. Black/White disparities in morbidity and mortality tend to be more pronounced at higher levels of SES (Williams and Sternthal 2010). For example, among adults with incomes below 100% of the federal poverty threshold, 11% more nonHispanic Blacks than Whites rate their health as fair or poor. Among adults whose family incomes are at or above 200% of poverty, 35% more nonHispanic Blacks than Whites describe their overall health as being fair or poor (Centers for Disease Control and Prevention 2012). This represents more than a two-fold increase in the Black/White gap in self-rated health at the upper end of the income distribution compared to the lower end. These findings suggest that the ways in which race and SES interact to produce disparate health outcomes across the lifecourse are complex and deserve further study.

To this end, we examine the patterning of racial disparities in physical wellbeing among a subpopulation that has been vastly understudied – middle-class African Americans (Jackson and Stewart 2003; Jackson 2005; Landry and Marsh 2011). Specifically, we seek to determine the extent to which the Black/White gap in self-rated health among middle-class Americans can be explained by two competing lifecourse mechanisms – early exposure to childhood disadvantage or the nonequivalence of socioeconomic status across race. The first mechanism suggests that middle-class Whites exhibit better health outcomes than their African American counterparts because the

latter are more likely to have spent their formative years with limited access to social and economic resources. Given what we know concerning the latent and long-lasting effects of childhood poverty (Case, Lubotsky, and Paxson 2002; Duncan et al. 1998; Duncan, Ziol-Guest, and Kalil 2010) as well as the cumulative impact of negative social exposures on health throughout the lifecourse (Geronimus et al. 2006; Hayward and Gorman 2004; Walsemann, Geronimus, and Gee 2008), the consequences of this early life disparity might not be fully expressed until mid-life or beyond. The second mechanism proposes a different pathway, whereby the impact of gains in SES over the lifecourse (ie. upward socioeconomic mobility) will have a substantially muted effect on the health of middle-class African Americans compared to middle-class Whites.

BACKGROUND

Lifecourse SES and Health: Theoretical Perspectives

Examining health disparities from a lifecourse perspective encourages researchers to envision the population patterning of morbidity and mortality as resulting from a set of dynamic processes rather than static exposures. In so doing, these theoretical conceptualizations serve as important reminders that experiences occurring early on during one's life can influence health outcomes years, even decades, later. This makes them a particularly useful theoretical lens when trying to assess why the Black/White gap in health is more pronounced among nonpoor as opposed to poor subpopulations, even though members of the middle-class typically have access to adequate socioeconomic resources.

Researchers who examine the population patterning of health across the lifecourse typically invoke one of two explanatory mechanisms to conceptually ground empirical approaches. The first emphasizes the role that early life exposures to disadvantageous social and economic circumstances play in the production of health and wellbeing later in life, while the second highlights the cumulative nature and impact of these health eroding insults over time. Existing lifecourse frameworks have emerged simultaneously from two different disciplinary perspectives – social epidemiology and medical sociology. There is much theoretical overlap; therefore, we will draw conceptual connections between the two subfields by discussing similar models in tandem.

Early Life Exposures

The first theoretical framework we consider suggests that encountering social and economic hardship early in life, especially during developmentally sensitive (ie. critical) periods, will negatively impact an individual's health status as she ages. Most notably, this conceptual approach allows for the possibility that a substantial amount of time may pass between early life exposures and the expression of adult onset disease or disability. This latency period is likely to be clinically unremarkable and offer few, if any, clues to the health deterioration that is about to commence at mid-life or beyond.

The most commonly referenced, empirically tested, and methodologically contested early origins framework is Barker's Fetal Origins Hypothesis (FOH). Barker hypothesizes that individuals exposed to unfavorable intrauterine environments during specific stages of fetal development, primarily resulting from inadequate maternal nutritional intake, will develop certain metabolic characteristics and eventually face

increased rates of chronic illness, particularly diabetes and cardiovascular disease, in adulthood. Barker traces this latent expression of disease back to physiological changes that occur at the cellular level, especially those that are involved in regulating blood pressure and insulin, as a direct result of fetal growth restriction (Barker 1998, 2003). The FOH emphasizes three essential points. First, conditions experienced *before birth* can have potentially long lasting health impacts over the lifecourse. Second, negative outcomes may only be noticeable following a latency period which is characterized by a lack of physical symptoms or negative clinical outcomes. Finally, excess adult morbidity and mortality is driven by biological programming that occurs at the cellular, or possibly the genomic, level (Almond and Currie 2011; Geronimus 2013).

The most convincing empirical evidence in support of the FOH comes from research that exploits natural experiments during which maternal nutritional intake was highly restricted. These efforts go beyond most of Barker's work that is primarily correlational in nature and is likely to suffer from selection bias, reverse causality, and other methodological shortcomings (Almond and Currie 2011; Paneth and Susser 1995). Examples of such studies have explored the long-term health impacts of children born to women who were pregnant during the Dutch Hunger Winter of World War II (Stein et al. 2006, 2007; Susser and Stein 1994), the 1959-1961 Famine in China (Chen and Zhou 2007), as well as Muslim women who fasted during Ramadan (Almond and Mazumder 2011). Results from this line of research suggest that children exposed to limited caloric intake during gestation are significantly more likely to be obese, exhibit unhealthy fat distribution, have high blood pressure, and face a greater risk of schizophrenia as adults

(Chen and Zhou 2007; Hoek, Brown, and Susser 1998; Ravelli, Stein, and Susser 1976; St Clair et al. 2005; Stein et al. 2006, 2007).

Theoretical conceptualizations that emphasize the role of early life exposures in the subsequent development of health later in life are not without proponents in the corresponding fields of Sociology, Demography, and Economics. Researchers have noted that, in particular, the years between birth and 6 years of age when most children begin formal schooling are particularly important for long-term outcomes, health and otherwise (Case, Fertig, and Paxson 2005; Case and Paxson 2008, 2010; Conti, Heckman, and Urzua 2010; Currie 2008; Duncan et al. 2010; Hayward and Gorman 2004; Heckman, Pinto, and Savelyev 2012; Palloni 2006).

Cumulative Disadvantage/Advantage

The concept of cumulative (dis)advantage has a long tradition in social stratification research and can be traced back to the work of Merton (1968) in which he described how early professional success among scientists garnered increased access to resources and rewards which grew exponentially over time, resulting in divergent career pathways. It was also invoked by Blau and Duncan (1967) in their studies of status attainment to explain why African American men experienced less occupational mobility relative to White men. In this application, the notion of cumulative disadvantage was used to highlight both the direct and indirect processes by which race influenced outcomes at different lifecourse stages, thus allowing for the possibility that Blacks experienced diminishing returns to gains in SES over time.

More recently, the concept of cumulative (dis)advantage has gained theoretical traction in the field of social epidemiology and led to the development of two distinct frameworks, each of which describes the specific, time-dependent pathways through which health is produced and maintained over time. The first, the accumulation of risk model, predicts that the longer an individual is exposed to socioeconomic hardship, the worse her health will be. Thus, the relationship between SES and physical wellbeing across the lifecourse is thought to be relatively straightforward, with increases in SES over time resulting in concomitant improvements in health. The second framework, the chains of risk model, more directly draws upon the Mertonian concept of cumulative (dis)advantage in that it emphasizes the ways in which exposures to hardships earlier in the lifecourse can place an individual on a trajectory that is likely to result in the expression of poor health outcomes over time. This perspective highlights the importance of transitions between lifecourse stages, specifically the probability that exposures to social conditions during a specific time period will determine subsequent life chances, including but not limited to health outcomes (Hertzman and Power 2003; Pavalko and Caputo 2013).

Both cumulative (dis)advantage approaches – the accumulation of risk and the chains of risk models – have been criticized for not adequately incorporating the experiences of African Americans (Colen 2011; Geronimus 2013). Proponents of these approaches typically do not consider how occupying another disadvantaged social status, such as being a member of a racial minority, might impact the seemingly straightforward association between SES and health. Therefore, these theoretical models may have

limited applicability when investigating why Black/White disparities in health are more pronounced among middle-class as opposed to lower-class subpopulations.

Instead, we rely on conceptual framework put forth by Colen (2011) to explain why African Americans are less likely to benefit, with respect to their health, from gains in SES across the lifecourse than similarly situated Whites. Although a detailed discussion of this model is beyond the scope of the current paper and can be found elsewhere (Colen 2011), it is outlined in Figure 1. Drawing on prior work by Blau & Duncan (1967), Farley and Frey (1994), as well as Farmer & Ferraro (2005) the restricted returns to mobility model carefully delineates the specific mediating mechanisms through which the health returns typically associated with either intra- or intergenerational upward mobility are likely to be muted for Blacks compared to Whites. These barriers stem primarily from racial inequalities that operate on multiple and distinct levels – structural (macro), interpersonal (mezzo), and intrapersonal (micro).

Insert Figure 1 About Here

These two theoretical approaches to studying the distribution of health across the lifecourse – the early origins framework and the cumulative (dis)advantage framework - suggest different explanatory mechanisms for why the health of middle-class African Americans remains remarkably disadvantaged relative to their White counterparts. The early origins framework places an emphasis on the exposure to socioeconomic disadvantage during childhood. Thus, we would expect the association between SES and health during adulthood to be similar for Blacks and Whites once differential exposures to early life adversity are taken into account. Alternatively, the diminishing returns hypothesis predicts that African Americans will experience fewer health benefits to

subsequent to improvements in their socioeconomic status over time, regardless of if their spent their childhoods in or near poverty. Thus, we would expect the association between socioeconomic status and health to be significantly more pronounced for Whites than Blacks even after accounting for differential early life exposures.

Previous Research on Race, Lifecourse SES, and Health

Previous research examining racial disparities in the relationship between SES and health across the lifecourse has produced mixed results, with some studies providing evidence that early life exposures to social and economic disadvantage, particularly during childhood, shape subsequent health trajectories and contribute to the emergence of Black/White inequalities in adult health (Haas and Rohlfen 2010; Hayward et al. 2000; Warner and Hayward 2006) while others lend credence to the diminishing returns hypothesis (Farmer and Ferraro 2005; Hudson et al. 2013; Kahn and Fazio 2005).

Haas and Rohlfen (2010) find that childhood socioeconomic deprivation significantly predicts the timing of the subsequent onset of functional disability, largely through health-related mechanisms, but cannot explain Black/White disparities in this critical indicator of physical wellbeing. On the other hand, Farmer & Ferraro (2005) provide the most convincing empirical support for the notion that African Americans may be less likely to benefit, with respect to their health, from gains in SES across the lifecourse by demonstrating that increases in educational attainment resulted in better self-rated health for White but not Black adults aged 25-74. Kahn & Fazio's work (2005) echoes these findings using household income instead of education and expands the

choice of health outcome measures to include chronic diseases and functional impairment.

However, the generalizability of these findings is limited by two important shortcomings. First, many studies rely on samples of older adults, often in conjunction with retrospective data, to examine how the expression of health and wellbeing across the lifecourse differs by race. This is especially problematic when comparing health outcomes among African Americans as opposed to Whites, since selection pressures at older ages (Masters 2012; Willson, Shuey, and Elder, Jr. 2007) in conjunction with substantially different average life expectancies (Geronimus et al. 2001; Harper et al. 2007) may produce patterns of population health that are unique to older individuals and do not adequately capture trends among younger cohorts.

Second, much of the research examining racial disparities in health using a lifecourse perspective fails to consider how these overarching patterns and the mechanisms that drive them may not be consistent across other key social statuses, such as socioeconomic status. Most notably, the experiences of middle-class African Americans are assumed to be similar to those of their poorer counterparts. There have been a few recent exceptions (Colen et al. 2006; Hudson et al. 2013) but their limited numbers fail to provide a deep or thorough understanding of the complex, intersecting processes associated with both race and SES that are likely to produce health disparities over time. Using data from the Coronary Artery Risk Development in Young Adults (CARDIA) Study, Hudson et al. (2013) report that a measure of lifecourse socioeconomic position (SEP) was positively and significantly associated with self-rated health scores for Whites but failed to reach statistical significance ($p < 0.05$) for Blacks. Colen et al.

(2006) find similar results combining nationally representative data from the National Longitudinal Survey of Youth (NLSY 1979) for three generations of women to examine the association between lifecourse trajectories of family income on the risk of giving birth to a low birthweight baby.

This study seeks to directly address these shortcomings by using 20 waves of nationally representative prospective data from the Panel Study of Income Dynamics (PSID) from 1985 to 2011. Our overarching research question seeks to determine whether suboptimal health outcomes among middle-class African Americans compared to middle-class Whites stem from a higher likelihood of being exposed to adverse childhood conditions or are more likely to result from restricted returns to intragenerational gains in SES.

DATA & METHODS

Description of the Data

The PSID is a nationally representative, panel survey of American families begun in 1968. Face-to-face interviews were conducted annually from 1968 to 1997 and then biennially after that. The original PSID sample, which oversampled low-income African Americans, contained detailed information on approximately 5,000 families. By 2011, the number of families participating in this study was 8,907 and included 24,661 individuals. Information is collected on every person living in the household during a given survey year although much of it focuses on the head and his/her spouse or cohabiting partner (PSID Main Interview User Manual: Release 2012.1 2013)

The unique structure and comprehensive content of the PSID make it one of the best secondary data sources with which to examine the long-term effects of lifecourse socioeconomic status on health outcomes among a demographically diverse, nationally representative population. Members of the original sample have been followed over an extended period of time (more than 40 years) during which they have established their own households, completed their educations, and pursued occupational goals. Moreover, this dataset contains detailed information along several dimensions of SES including education, occupation, income, as well as wealth. Yearly response rates consistently fall between 95% and 98% (PSID Main Interview User Manual: Release 2012.1 2013).

We restrict our study sample to middle-class, working-age (18-64 years old), nonHispanic White and nonHispanic Black PSID heads and wives (including cohabiting partners). We classified PSID respondents as middle-class if they completed more than 12 years of formal education. However, in a subsequent set of sensitivity analyses, we operationalized middle-class status according to two different sets of criteria: (1) if the respondent's main occupation could be characterized as a primarily white collar position (ie. sales or clerical, managerial or administrative, or professional) or (2) if the respondent's family income for the year prior to data collection fell within the highest three quintiles of the income distribution. Regression results did not qualitatively differ according to definitional specifications. Due to space limitations, we only discuss, indepth, findings in which we define middle-class based on educational attainment.

Measures

For the purpose of this study, an individual's physical wellbeing is assessed using a measure of self-rated health. PSID respondents were asked to rate their health, in general, as being excellent, very good, good, fair, or poor. This outcome measure has been shown to be a particularly powerful indicator of physical wellbeing and predicts subsequent mortality more accurately than physician diagnosis (Jylhä 2009). Moreover, it is one of the most common ways to operationalize general health status (Ferraro and Farmer 1999); thus, its utilization allows us to compare our findings to results from other extant studies on related topics. We collapsed the original response categories to create a dichotomous indicator in which 0 represents good, very good, or excellent health and 1 represents poor or fair health. This was primarily done to achieve convergence in our multilevel models and to foster ease of interpretation. Subsequent sensitivity analyses indicated that regression results did not qualitatively differ according to the number of response categories used to capture self-rated health.

The main independent variable of interest, lifetime SES, is captured using a continuous measure of family income that combines earnings and income transfers from each adult member of the current household obtained during the year prior to the current survey year. We adjust this variable for inflation using the CPI-U index and report all values in 2010 dollars. Due to the skewed nature of its distribution, we transform this variable by taking the natural log of its value for all multivariate regression analyses.

The decision to use family income to depict changes in SES across the lifecourse was intentional and guided by previous research (Duncan et al. 2002) suggesting it is an optimal way to accurately capture the association between SES and health, especially as it fluctuates over time. By relying on this particular a measure of lifetime SES, we are

able to accomplish two important empirical objectives. First, we are able to avoid problems that derive from changes in how upward mobility is characterized over time. For example, during the 1970s and 1980s, when PSID data was first being collected, having a college degree meant something different than it does today, largely because it is more common and comes with fewer social and economic rewards (Aaronson and Mazumder 2008; Goldin and Katz 1999; Moretti 2008). Second, the inclusivity of an income measure captured at the household as opposed to the individual level reflects socioeconomic resources available to an individual regardless of other demographic characteristics they possess. This is particularly advantageous when trying to assess SES trajectories over time, during which someone is likely to experience changes in household composition, marital status, or employment status.

Similarly, the decision to focus on intragenerational as opposed to intergenerational mobility was based on important conceptual and methodological considerations. First, little is known how the association between socioeconomic mobility and health differs across race. Thus, we determined that it would be prudent to begin with a more straightforward approach and incorporate more sophisticated indicators of mobility, such as intergenerational transfers of SES, as the body of evidence concerning this particular research question increases. Second, capturing fluctuations in family income across multiple generations demands that respondents participate in a particular study for elongated periods of time. Since racial minorities and low-income individuals are more likely to drop-out of longitudinal surveys (Lillard and Panis 1998; Radler and Ryff 2010), we were concerned that our results would be biased by differential loss-to-follow-up across race and SEP.

Respondent's race was determined using a self-reported measure and coded as 0 or 1 if he/she was nonHispanic White or nonHispanic Black, respectively. All individuals who identified as Hispanic were excluded from the analyses that form the basis of the current study. In the small number of cases (about 300, or 3% of the sample) where reported race changes across waves, the most recent report from 1997 or 2005 – when background variables were re-asked – is used for all person-years contributed to the sample.

We include additional covariates in all multivariate regression models to adjust for potential confounders that have been found to be associated with both socioeconomic status and self-rated health such as respondent's age (in years), age squared, sex (male/female), relation to household head (self, wife or husband, cohabiting partner), marital status (married or cohabiting, never married, widowed, divorced, or separated), employment status (working, temporarily laid-off, unemployed, or otherwise not working), and number of children in the household (under the age of 18).

In a subset of regression analyses, we also control for health insurance coverage (private, public, none) as well as multiple health behaviors to determine if our results are robust to Black/White differences in access to health care and reliance on individually based efforts at health promotion. Current smoker (yes/no) is coded as 0 if the PSID respondent did not smoke cigarettes at the time of interview and 1 if he/she did. Alcohol consumption is based on the following question, "On average, do you have less than one drink a day, one or two drinks in a day, three to four drinks a day, or five or more drinks a day?" (1999-2003) and "In the last year, on the days you drank, about how many drinks did you have?" (2005-2011). PSID participants are placed into three categories based on

their answer – 0 drinks per day, 1-2 drinks per day, 3+ drinks per day. Similar to other studies (Cerdá, Johnson-Lawrence, and Galea 2011), we refer to these classifications as abstainers, moderate drinkers, and heavy drinkers. The PSID contains information regarding the frequency with which a respondent typically engages in either light or heavy exercise. Light exercise is defined as activities such as walking, dancing, gardening, golfing, and bowling, while heavy exercise is defined as vigorous physical activity or sports such as heavy housework, aerobics, running, swimming, or bicycling. Both measures adhere to the following coding schema: 0 if the PSID participant indicates he/she never exercises; 1 if he/she exercises 1-2 times per week; 2 if he/she exercises 3-4 times per week; and 3 if he/she exercises 5 or more times per week.

Analytic Strategy

We employ multilevel logistic regression models with random intercepts to estimate the association between family income and self-rated health across the lifecourse and determine the extent to which this relationship differs by race (Skrondal and Rabe-Hesketh 2004; Snijders and Bosker 2011). The composite statistical model can be written as follows:

$$\text{logit}[\text{Pr}(y_{it} = 1|\mathbf{x}_{it}, \zeta_t)] = (\beta_1 + \zeta_t) + \beta_2 x_{2it} + \dots + \beta_n x_{nit} + \epsilon_{it}$$

where y_{it} indicates whether or not individual i is in poor or fair health at time t , β_1 represents the average intercept, ζ_t indicates the individual-specific error component that remains constant across time periods, ϵ_{it} stands for the time-specific error component, $\beta_2 - \beta_n$ are regression coefficients for individual i at time t , and $x_2 - x_n$ are the values of each

predictor for individual i at time t . The random intercepts are assumed to be independent and normally distributed across individuals and independent of the covariates x_{it} .

Multilevel models parse variation in the dependent variable in two ways - within individuals across panels and between individuals as they age over time. In our case, the data have a hierarchical structure wherein multiple observations collected at time t are nested within each individual i . Because it would be unrealistic to assume that the error terms (ε_{it} and ε_{it+1}) are uncorrelated over time, our regression models estimate random intercepts—that is, the model provides an average intercept, as well as a variance parameter that summarizes individual-specific deviations from the average intercept. As such, our models parse within-individual and between-individual variation in self-rated health, conditional on the variables included in the model. Given the nonindependence of observations within individuals and the likelihood of heteroskedasticity, we calculated robust standard errors using the Huber/White correction method and clustered them at the highest level of aggregation - the original PSID family. All analyses were based on maximum likelihood estimation with adaptive quadrature and conducted using Stata/MP 13.1.

To handle issues of missing data, we rely on multiple imputation techniques to generate values for all covariates included in descriptive and multivariate analyses. Evidence from the relationships of missing data with individual time varying and time invariant characteristics suggests the data are not missing completely at random (unconditional on the observed covariates) which makes typical approaches such as listwise deletion inappropriate (Allison 2001). In the imputation phase, the procedures use a diverse set of predictors to estimate five sets of plausible values for each missing

value. The imputed values in the five data sets include a random component based on draws from the posterior predictive distribution of the missing data under a posited Bayesian model and, under the missing-at-random assumption - a more plausible assumption than is made by listwise deletion - provide unbiased estimates of variance (Allison 2001). Following von Hippel (2007), we impute values for all variables in a given model and then delete observations with missing data on self-rated health. Most notably, the independent variables of interest, race and family income, did not have any missing values.

RESULTS

Descriptive Results

Table 1A presents descriptive statistics for college-educated PSID respondents between the ages of 18 and 64, stratified by race. Black/White disparities in self-rated health are clearly evident. 14.77% of African Americans but only 7.35% of Whites described their health as being fair or poor. At the opposite end of the spectrum, 67.83% of Whites but only 49.45% of African Americans characterized their health as very good or excellent.

Insert Table 1A About Here

Compared to their Black counterparts, White PSID respondents who attended college have substantially lower earning potential. Mean family incomes, adjusted for inflation and reported in 2010 dollars, for Whites and African Americans in our study sample are \$76,000 and \$46,500, respectively. College-educated Whites tend to have fewer children under 18 years of age present in the household than college-educated Blacks (0.89 vs. 1.22), so their incomes serve to support fewer people. Whites maintain a

more even sex distribution (52% vs.48%), while women are overrepresented among Blacks (41% vs. 59%). This is likely attributed to the fact that African American women have historically exhibited stronger attachment to the labor force than White women (Browne and Misra 2003; Browne 2000; Jones 2009; Willson 2003). Regarding racial differences in marital status, 79% of White respondents but only 52% of Black respondents were currently married or cohabiting. Given this stark racial disparity in percent married, it is not surprising that almost one-fifth (19%) of Whites but almost one-half (45%) of African Americans reported being single, separated, or divorce. Despite their educational attainment, 8% of Black college attendees but only 3% of White college attendees are unemployed.

Considering racial disparities in health insurance and health behaviors among college-educated respondents, fewer African Americans than Whites are covered under private health insurance plans (80% vs. 73%) while more are covered under public plans (14% vs. 18%) or report having no insurance at all (6% vs. 8%). However, these differences are relatively small in magnitude, likely as a result of restricting our sample to PSID respondents with at least some college. A similar proportion of middle-class Whites and Blacks report being current smokers (21% vs. 22%) and Whites smoke, on average, significantly more cigarettes per day (15 vs. 12). 51% of White college attendees identify as moderate drinkers compared to 45% of Black college attendees, while the opposite pattern is discernable among abstainers. The only health behavior that favors Whites over African Americans is physical activity. 12% and 17% of college-educated Whites and Blacks, respectively, report never engaging in light exercise, while 30% and 36%, respectively, describe never participating in heavy exercise.

The racial patterning of childhood disadvantage reveals itself quite clearly in Table 1B. Not surprisingly, college-educated African Americans are more likely to have encountered adverse social and economic conditions in childhood. 45% of middle class Blacks but only 28% of middle class Whites report that when they were growing up, their parents were poor. This stands in contrast to 53% and 35% of Whites and Black college attendees, respectively, who characterize their parents as being of average social standing. Similarly, 30% of Whites in our sample but only 11% of Blacks had fathers who attended at least some college. These differences in SES during childhood are further reinforced by significant racial disparities in the proportion of college-educated PSID respondents who lived with both parents at 16 years of age (83% of Whites vs. 62% of Blacks).

Insert Table 1B about Here

Multilevel Regression Results

Results from multilevel regression models, stratified by race, are presented in Tables 2 and 3. Model 1 displays the association between family income and self-rated health controlling for age, age-squared, sex, relation to head, marital status, employment status, and number of children. In Model 2, we add indicators of childhood disadvantage including parental poverty status, living arrangements at age 16, father's educational attainment, and mother's educational attainment. Model 3 takes into account the respondent's access to health care and health behaviors in a given year by further controlling for health insurance coverage, cigarette smoking, alcohol consumption, as well as frequency of engagement in light and heavy exercise.

Insert Tables 2 and 3 about Here

According to results for Model 1, for every 10% increase in family income, the odds of being in fair or poor health decrease by 6.3% for Whites but only 4.7% for African Americans. This represents a 25% racial disparity in the association between family income and self-rated health across the lifecourse. Using post-estimation testing, we sought to determine if the difference in regression coefficients was statistically significant and found evidence that it was large enough not to be attributed to random chance alone ($t = -2.43$). Thus, it appears that compared to college-educated Whites, similarly educated Blacks adults experience diminished health returns to intragenerational gains in family income.

Once we control for the fact that middle-class African Americans are more likely to experience social and economic disadvantage early in life (Model 2), regression coefficients for both Blacks and Whites decrease slightly, with the change being somewhat more pronounced among White respondents. However, the Black/White gap in the association between family income and self-rated health remains sizeable and statistically significant ($t = -2.08$). After taking into account racial disparities in exposure to childhood disadvantage, we find that for every 10% increase in family income over time, college-educated Whites and Blacks can expect the odds of being in fair or poor health to decrease by 6.0% and 4.6%, respectively.

It is also interesting to note that for Whites, all aspects of childhood disadvantage seem to increase the odds of being in fair or poor health in adulthood even after controlling for current SES, at least that which is captured by family income. For example, college-educated Whites who resided with both parents at age 16 were 27% less

likely to be in fair or poor health as an adult. However, for African Americans, living arrangements during childhood, father's educational attainment, and, to a large extent, mother's educational attainment do not predict self-rated health trajectories in adulthood. The one exception to this overarching pattern is parental poverty status. Compared to college-educated Blacks whose parents were poor, similarly educated Blacks whose parents were average or well-off were 35% and 24%, respectively, less likely to be in fair or poor health as an adult.

Our third set of regression findings demonstrate the extent to which restricted health returns to intragenerational gains in family income can be attributed to Black/White differences in health care access and health behaviors. Even after including 5 additional potential confounders in Model 3 – including health insurance coverage, cigarette smoking, alcohol consumption, as well as frequency of light and heavy physical activity - race-specific regression coefficients for the family income variable remain remarkably consistent (-0.579 for Whites and -0.446 for Blacks). More importantly, racial disparities in the association between family income on self-rated health across the lifecourse remain largely unexplained.

DISCUSSION

The objective of this paper was to determine the extent to which Black/White disparities in health among the middle-class are attributable to differential exposure to disadvantaged social and economic conditions in childhood or to restricted health returns to intragenerational gains in SES over the lifecourse. Taken as a whole, our findings lend credence to the diminishing returns as opposed to the early origins hypothesis to explain

the patterning of racial inequalities in self-rated health among nonpoor, working-age adults in the United States. Although childhood disadvantage was significantly associated with the probability of being in fair or poor health as an adult, it only slightly attenuated the association between family income and self-rated health trajectories. These results stand in contrast to those presented by others (Haas and Rohlfen 2010; Hayward et al. 2000; Shuey and Willson 2008). Unlike previous empirical efforts, our study focuses solely on racial disparities in the association between income and self-rated health across the lifecourse among members of the Black middle-class, which is a vastly understudied subpopulation (Jackson and Stewart 2003; Landry and Marsh 2011) that, due to its growing numbers (Hunt and Ray 2012; Marsh et al. 2007) whose experiences deserve more attention and careful investigation.

Moreover, a wider variety of early life hardships seem to negatively impact adult health for college-educated Whites compared to college-educated Blacks. For Whites, parental poverty status, childhood living arrangements, as well as father's and mother's educational attainment all predict subsequent adult health trajectories. For African Americans, however, only parental poverty status and whether or not their mother graduated high school was associated with the likelihood of being in poor or fair health across the prime working ages. These racial differences in the importance of specific early life predictors could be attributable to African Americans' greater propensity to grow up in female headed households (Cherlin 2010; McAdoo 2007; Teachman, Tedrow, and Crowder 2000) and/or with mother's who have traditionally exhibited a greater attachment to the labor force than their White counterparts (Browne 2000; Browne and Misra 2003; Willson 2003; Jones 2009).

The results presented here suggest that while childhood social and economic conditions are important predictors of adult health, they are not sufficient to explain why the health of middle-class African Americans still lags behind that of similarly situated Whites. On the contrary, it appears that even after accounting for several key indicators of childhood disadvantage, middle-class Blacks should expect to reap significantly fewer health benefits as they experience intragenerational upward mobility. This is a sobering thought and is likely to have important consequences for social policies often designed to improve the life chances of racial minorities.

First and foremost, our findings provide evidence that racial disparities in health are not simply a reflection of differential access to social and economic resources. Instead, they suggest a more nuanced and complicated relationship between SES and health that is likely to vary across different racial and ethnic (Kaufman, Cooper, and McGee 1997; Williams and Sternthal 2010). If, as a society, we want to work toward eliminating racial disparities in health, we will need to recognize that anti-poverty programs are important but not sufficient to achieve this lofty goal. Furthermore, we will need to address at least some of the fundamental causes, such as residential segregation (Williams and Collins 2001), differential wealth accumulation (Oliver and Shapiro 1995; Shapiro 2004), and continued exposure to interpersonal racial discrimination (Gee and Walsemann 2009; Williams and Mohammed 2009; Williams, Neighbors, and Jackson 2003), that undergird suboptimal health outcomes among African Americans of all class backgrounds.

Second, over the previous decade, lifecourse researchers have presented convincing evidence that exposures in childhood are key drivers of health in adulthood

(Campbell et al. 2014; Haas 2008; Hayward and Gorman 2004; Pudrovska 2014) and policy recommendations have begun to follow suit (Barnett and Belfield 2006; Forrest and Riley 2004; Hertzman et al. 2010; Irwin, Siddiqi, and Hertzman 2007). While our results do not contradict these findings, especially for Whites, they suggest that early life factors cannot fully explain racial disparities in health over the lifecourse. In order to reduce the Black/White gap in health outcomes, we will need to design and implement social policies that help working-age adults *in addition* to their children. This is likely to be an especially unpopular policy recommendation in an era that has witnessed recent efforts to reduce already paltry welfare and food stamp benefits, initiate mandatory drug testing for participants in federal and state benefit programs, and further limit access to reproductive health care, especially for low-income and minority women.

Expanding health insurance coverage is one area where the United States has seen some movement in this direction. In 1997, the State Children's Health Insurance Program (SCHIP) was established to provide health insurance coverage to children in families whose incomes were too high to qualify for Medicaid but too low to purchase health insurance through private means (Center for Medicaid and CHIP Services 2014b). The Patient Protection and Affordable Care Act, (ACA), which was signed into law in 2010, further expanded Americans' ability to gain access to health insurance through the establishment of health insurance exchanges operating at the federal or state level (Center for Medicaid and CHIP Services 2014a). The results presented here, however, suggest that improved access to health care, at least that which is facilitated by health insurance coverage, does little to reduce the Black/White gap in the association between family

income on self-rated health. Instead, we need to look toward public health programs and policies that go beyond simply increasing access to care.

Finally, our findings provide additional evidence that racial disparities in health are not primarily the result of minority populations engaging in worse health behaviors. To the contrary, in our study sample, college-educated African Americans consume alcohol less frequently and smoke cigarettes at similar rates to their White counterparts. It also appears that Blacks may be less likely to benefit from adhering to healthier behaviors than Whites. The magnitude of regression coefficients for all health behaviors examined in the current study was smaller among African American than White respondents. These findings echo those presented by Jackson et al. (2010) and Mezuk et al. (2010) and lend additional support for the proposition that racial inequalities may prevent African Americans from the full health benefits of engaging in health behavior changes strategies such as smoking cessation efforts or physical activity regiments.

Even in an era of improving socioeconomic status and increasing political power among racial minorities, racial disparities in health, especially those among African Americans, remain entrenched. In order to reduce, and hopefully eventually eliminate, the Black/White gap in morbidity and mortality, we need to gain a deeper understanding of how race and SES interact to produce health outcomes across the lifecourse. This is not likely to be simple relationship – one in which the health of African Americans improves steadily or similarly to Whites as they ascend the socioeconomic hierarchy. Instead, it will take careful empirical inquiry to demonstrate how structural level racial inequalities as well as continued exposure to racial discrimination erode the health returns to upward mobility we have come to expect as an integral part of the “American Dream.”

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Figure 1 Mechanisms likely to restrict health returns to upward mobility for African Americans

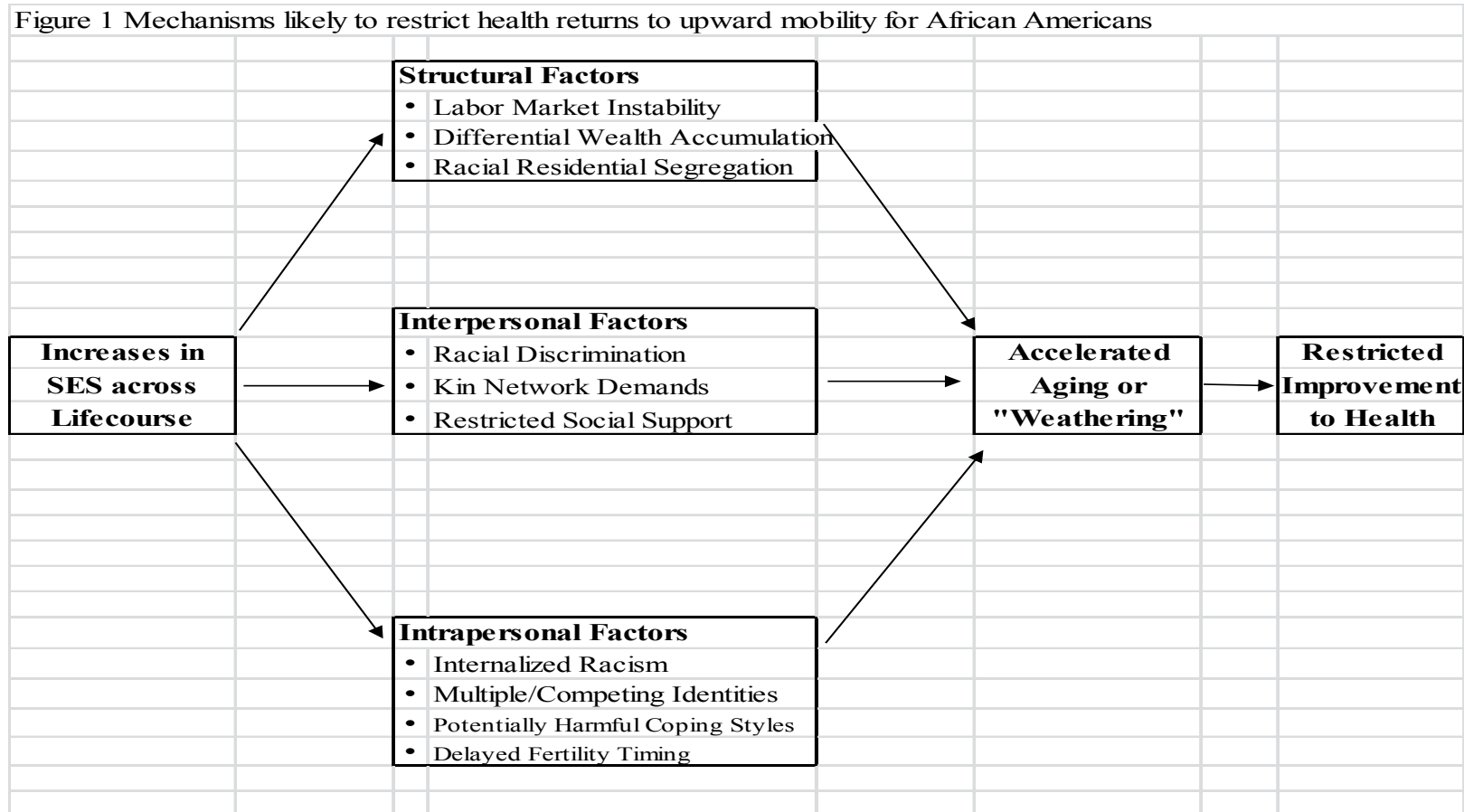


Table 1A. Descriptive Statistics for College Educated NonHispanic White and NonHispanic Black Working-Age PSID Respondents (1985-2011), by Race

	White	Black	<i>p</i> Value for Difference
<i>Self Rated Health</i>			
Excellent	28.84	19.08	***
Very Good	38.99	30.37	
Good	24.81	35.75	
Fair	5.75	12.27	
Poor	1.60	2.50	
Mean Family Income (2010 \$)	76,010	46,522	***
Mean Age	39.42	38.79	***
Mean No. Children in Household	0.89	1.22	***
<i>Sex</i>			
Men	48.04	40.74	***
Women	51.96	59.26	
<i>Relation to Head</i>			
Head	58.09	70.84	Ref
Wife/Husband	40.07	26.30	***
Cohabitor	2.86	1.85	*
<i>Marital Status</i>			
Married/Cohabiting	79.05	52.06	Ref
Single	6.49	20.65	***
Widowed	1.96	3.19	***
Divorced	10.91	17.28	***
Separated	1.58	6.82	***
<i>Employment Status</i>			
Working	80.69	75.16	Ref
Temporarily Laid Off	0.73	1.18	***
Unemployed	2.80	8.30	***
Otherwise Not Working	15.78	15.36	***
Mean Hours Worked/Week	29.96	25.30	***

Source: Panel Study of Income Dynamics, 1985-2011

^aTests for difference are based on two-tailed t-tests generated from bivariate OLS, logit, ordered, or multinomial logit regression models. All models were estimated using robust standard errors clustered at the level of the household.

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.10$

Table 1B. Descriptive Statistics for College Educated NonHispanic White and NonHispanic Black Working-Age PSID Respondents (1985-2011), by Race

	White	Black	<i>p</i> Value for Difference
<i>Health Insurance</i>			
Private	80.27	73.27	Ref
Public	13.76	18.25	***
None	5.97	8.48	***
Current Smoker	20.77	21.87	***
<i>Drinks Alcohol</i>			
None	37.36	43.20	***
1-2 Drinks Per Day	50.84	45.46	
3+ Drinks Per Day	11.80	11.35	
<i>Light Exercise</i>			
Never	11.55	16.92	***
1-2 Times Per Week	21.57	21.55	
3-4 Times Per Week	18.85	16.63	
5+ Times Per Week	48.02	44.90	
<i>Heavy Exercise</i>			
Never	29.74	36.49	***
1-2 Times Per Week	28.47	25.85	
3-4 Times Per Week	20.69	18.02	
5+ Times Per Week	21.10	19.64	
<i>Parents Poor</i>			
Poor	27.62	44.94	***
Average	53.17	35.04	
Well Off	19.21	20.02	
Lived with Both Parents	82.58	61.94	***
<i>Father's Education</i>			
Less Than High School	21.76	39.82	***
Some High School	10.62	15.09	
High School Graduate	38.08	33.80	
Some College	10.28	5.50	
College Graduate	12.08	4.22	
Graduate School	7.18	1.57	
<i>Mother's Education</i>			
Less Than High School	13.39	24.24	***
Some High School	10.80	22.54	
High School Graduate	50.85	39.01	
Some College	11.81	8.48	
College Graduate	9.44	4.28	
Graduate School	3.72	1.45	

Source: Panel Study of Income Dynamics, 1985-2011

^aTests for difference are based on two-tailed t-tests generated from bivariate OLS, logit, ordered, or multinomial logit regression models. All models were estimated using robust standard errors clustered at the level of the household.

****p* < 0.001; ***p* < 0.01; * *p* < 0.05; + *p* < 0.10

Table 2. Results from Multilevel Logistic Regression Models Predicting Fair or Poor Health for College Educated, Working Aged NonHispanic White PSID Respondents (1985-2011)

	Model 1 ^b			Model 2 ^b			Model 3 ^b		
	b		SE ^a	b		SE ^a	b		SE ^a
Ln Family Income	-0.633	***	0.043	-0.597	***	0.045	-0.579	***	0.045
<i>Parents Poor</i>									
Poor				Ref			Ref		
Average				-0.364	***	0.086	-0.359	***	0.086
Well Off				-0.050		0.105	-0.053		0.104
Lived with Both Parents				-0.316	***	0.087	-0.303	**	0.086
<i>Father's Education</i>									
Less Than High School				Ref			Ref		
Some High School				-0.085		0.107	-0.084		0.106
High School Graduate				-0.226	**	0.081	-0.225	**	0.083
Some College				-0.368	*	0.143	-0.353	*	0.143
College Graduate				-0.414	*	0.164	-0.390	*	0.163
Graduate School				-0.803	***	0.206	-0.778	***	0.206
<i>Mother's Education</i>									
Less Than High School				Ref			Ref		
Some High School				-0.055		0.102	-0.066		0.102
High School Graduate				-0.286	**	0.103	-0.287	**	0.104
Some College				-0.344	*	0.170	-0.333	+	0.170
College Graduate				-0.454	*	0.183	-0.442	*	0.183
Graduate School				-0.149		0.213	-0.140		0.210
<i>Health Insurance</i>									
Private							Ref		
Public							0.139	*	0.060
None							0.047		0.097
Current Smoker							0.150	*	0.056
<i>Drink Alcohol</i>									
None							Ref		
Moderate							-0.138	**	0.045
Heavy							-0.051		0.092
<i>Light Exercise</i>									
Never							Ref		
1-2 Times Per Week							-0.196	**	0.068
3-4 Times Per Week							-0.264	**	0.070
5+ Times Per Week							-0.299	**	0.080
<i>Heavy Exercise</i>									
Never							Ref		
1-2 Times Per Week							-0.210	*	0.074
3-4 Times Per Week							-0.282	***	0.053
5+ Times Per Week							-0.270	***	0.061
N (Person-Years)			109,351			109,351			109,351
N (Individuals)			11,777			11,777			11,777

Source: Panel Study of Income Dynamics, 1985-2011

Notes: All multilevel regression models are estimated with random intercepts for each PSID respondent.

^a Robust standard errors are calculated with the Huber/White correction method and clustered at the family level.

^b Additional control variables include: age, age², sex, relation to head, marital status, employment status, hours worked & number of children in the household.

***p < 0.001; **p < 0.01; * p < 0.05; + p < 0.10

Table 3. Results from Multilevel Logistic Regression Models Predicting Fair or Poor Health for College Educated, Working Aged NonHispanic Black PSID Respondents (1985-2011)

	Model 1 ^b			Model 2 ^b			Model 3 ^b		
	b		SE ^a	b		SE ^a	b		SE ^a
Ln Family Income	-0.474	***	0.046	-0.457	***	0.047	-0.446	***	0.047
<i>Parents Poor</i>									
Poor				Ref			Ref		
Average				-0.427	***	0.097	-0.429	***	0.098
Well Off				-0.277	**	0.102	-0.291	**	0.101
Lived with Both Parents				-0.092		0.080	-0.086		0.080
<i>Father's Education</i>									
Less Than High School				Ref			Ref		
Some High School				-0.015		0.090	-0.020		0.090
High School Graduate				-0.127		0.086	-0.138		0.087
Some College				-0.125		0.161	-0.127		0.160
College Graduate				0.059		0.203	0.056		0.203
Graduate School				-0.057		0.323	-0.052		0.325
<i>Mother's Education</i>									
Less Than High School				Ref			Ref		
Some High School				-0.036		0.089	-0.039		0.090
High School Graduate				-0.217	*	0.091	-0.216	*	0.092
Some College				-0.197		0.142	-0.192		0.141
College Graduate				0.022		0.174	0.033		0.171
Graduate School				-0.515	+	0.272	-0.505	+	0.271
<i>Health Insurance</i>									
Private							Ref		
Public							0.148	*	0.065
None							-0.069		0.088
Current Smoker							0.098	*	0.047
<i>Drink Alcohol</i>									
None							Ref		
1-2 Drinks Per Day							-0.082		0.071
3+ Drinks Per Day							0.066		0.070
<i>Light Exercise</i>									
Never							Ref		
1-2 Times Per Week							-0.116		0.088
3-4 Times Per Week							-0.193		0.112
5+ Times Per Week							-0.155	*	0.069
<i>Heavy Exercise</i>									
Never							Ref		
1-2 Times Per Week							-0.185	**	0.058
3-4 Times Per Week							-0.221	*	0.081
5+ Times Per Week							-0.219	***	0.053
N (Person-Years)			48,409			48,409			48,409
N (Individuals)			6,400			6,400			6,400

Source: Panel Study of Income Dynamics, 1985-2011

Notes: All multilevel regression models are estimated with random intercepts for each PSID respondent.

^a Robust standard errors are calculated with the Huber/White correction method and clustered at the family level.

^b Additional control variables include: age, age², sex, relation to head, marital status, employment status, hours worked & number of children in the household.

***p < 0.001; **p < 0.01; * p < 0.05; + p < 0.10