Life Course Pathways to Racial Disparities in Cognitive Impairment among Older Americans[*]

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Abstract

Blacks are especially hard hit by cognitive impairment at older ages compared to whites. Here, we take advantage of the Health and Retirement Study (1998–2010) to assess how this racial divide in cognitive impairment is associated with the racial stratification of life course exposures and resources over a 12-year period among 8,946 non-Hispanic whites and blacks aged 65 and older in 1998. We find that blacks suffer from a higher risk of moderate/severe cognitive impairment at baseline and during the follow-up. Blacks are also more likely to report childhood adversity and to have grown up in the segregated South, and these early-life adversities put blacks at a significantly higher risk of cognitive impairment. Adulthood socioeconomic status is strongly associated with the risk of cognitive impairment, net of childhood conditions. However, racial disparities in cognitive impairment, though substantially reduced, are not eliminated when controlling for these life course factors.

In 2002, approximately 13.9% of Americans aged 71 and older had dementia, and 22.2% had cognitive impairment without dementia (Plassman et al. 2007; Plassman et al. 2008), with the prevalence rates rising sharply with age. For example, about 5% of those aged 71–79 were estimated to have dementia, compared to 24% of those aged 80–89 and 37% of those aged 90 and older (Plassman et al. 2007). The national economic cost of informal home care for older adults with mild, moderate, and severe cognitive impairment was estimated to be more than $18 billion in 1993 (Langa et al. 2001). More recent estimates of dementia care (direct costs and informal care) for 2009 were as high as $133 billion in the United States (Wimo, Winblad, and Jönsson 2010).

Blacks are especially hard hit by cognitive impairment and dementia. Estimates of dementia prevalence rates in the older population are substantially higher for blacks than for whites (Manly and Mayeux 2004; Shadlen et al. 2006; Taylor, Sloan, and Doraiswamy 2004), prompting the Alzheimer's Association to identify Alzheimer's disease as an emerging public health crisis among older blacks (Alzheimer's Association 2010a). According to the latest estimates from 2006, the prevalence of cognitive impairment among Americans aged 65 and older was 8.8 percent for whites and 23.9 percent for blacks (Alzheimer's Association 2010b). Blacks also have a higher prevalence of vascular dementia than whites (Froehlich, Bogardus, and Inouye 2001). However, despite the racial gap in prevalence, there are surprisingly few population-level longitudinal studies that have investigated the origins and mechanisms through which the racial gap in cognitive impairment is produced.

Here, drawing on seven waves of the Health and Retirement Study (HRS), we investigate how racial differences in the onset of moderate/severe cognitive impairment among older Americans are tied to the racial stratification of socioeconomic conditions and health over the life course. Our life course approach of incorporating both childhood and adulthood conditions allows us to assess potential pathways through which racial disparities are produced.

BACKGROUND

Childhood Conditions, Adult Socioeconomic Status(SES), and Cognitive Impairment in Later life

The life course perspective has often been invoked to understand the origins of SES and racial disparities in adult health and mortality (O'Rand and Hamil-Luker 2005; Pavalko and Caputo 2013; Warner and Hayward 2006). In recent years, researchers have also turned to this perspective to examine the determinants of cognitive function in later life as well as racial and ethnic differences in cognitive aging (Glymour and Manly 2008; Luo and Waite 2005). The usefulness of the life course approach is rooted in the age patterning of cognitive function over the life course wherein cognitive function shows "rapid growth and development in the early years rising to a peak or plateau at maturity, and then a gradual decline with age" (Kuh 2007: 718). The brain reserve obtained at maturity and the rate of decline ultimately determine when the "threshold" for cognitive impairment is crossed.

A growing body of literature provides empirical evidence that cognitive impairment in later life is rooted at least in part in childhood (Deary et al. 2004; Scazufca et al. 2008; Zhang, Gu, and Hayward 2008). Although the exact biological, behavioral, and psychosocial mechanisms linking childhood conditions and late-life cognitive impairment remain unclear, two general mechanisms have been offered to explain the association. One mechanism posits that early-life adversity may have direct effects on late-life cognition because of impairments in brain development. The brain grows and develops most during the prenatal period and the first few years of life and continues to grow in childhood and adolescence (Lupien et al. 2009). Thus, insults such as poverty, hunger, poor nutrition, chronic stress, and poor health that occur during early childhood and adolescence are particularly harmful because they may impair the development of many regions of the brain and have long-term ramifications including "less myelin, less branching of dendrites, and less developed connectivity

patterns" in the brain (Moceri et al. 2000:415). In other words, early-life factors may strongly influence the brain reserve or biological capacity acquired at maturity.

Compelling evidence from research on childhood SES and brain development using behavioral, electrophysiological, and neuroimaging methods shows that in early childhood, there are already SES disparities in neurocognitive function (Hackman and Farah 2009).Recent birth cohort studies in Britain found that childhood cognitive ability was negatively associated with cognitive decline in midlife as well as the risk of late-onset dementia, suggesting that cognitive deficits in early life may put individuals at higher risk of cognitive impairment in later life (Richards et al. 2004; Whalley et al. 2000). Consistent with this idea, research in both developed and developing countries has reported that early-life nutritional deprivation (e.g., measured by hunger and shorter stature) and other childhood socioeconomic disadvantages are associated with cognitive impairment and dementia in later life, net of adulthood socioeconomic achievement (Huang et al. 2008; Melrose et al. 2015; Zhang, Gu, and Hayward 2010).

An alternative yet complementary perspective posits that early-life circumstances may indirectly influence cognitive impairment via educational attainment, occupation, income, health behaviors, and chronic diseases. Education can promote brain growth and enable the brain network to operate more efficiently. Thus, individuals with a higher level of education may enter old age with a greater number of synapses, which would provide protection against cognitive decline in later life and delay the onset of cognitive impairment (Alley, Suthers, and Crimmins 2007; Fritsch et al. 2007). Higher levels of education often lead to occupations that involve cognitive challenges and practice, which could further enhance cognitive function in adulthood (Schooler 1987). Education also shapes health behaviors throughout the life course. For example, the well educated are more likely to exercise, to drink moderately, and to avoid smoking, all of which are associated with good cognitive health in later life (Cutler and Lleras-Muney 2010; Lee et al. 2010; Peters et al. 2008). Not surprisingly, many studies have shown that a greater number of years of education and good quality of schooling (e.g., literacy and reading ability) are associated with better cognitive function, slower cognitive decline, and a lower risk of dementia in later life (Alley et al. 2007; Jefferson et al. 2011; Reuser, Willekens, and Bonneux 2011; Shadlen et al. 2006).

Higher income and wealth can reduce older adults' exposure to stress associated with financial strains (Kahn and Pearlin 2006; Lynch, Kaplan, and Shema 1997), and perceived stress has been associated with faster cognitive decline and a higher risk of dementia (Aggarwal et al. 2014; Chiao, Botticello, and Fuh 2014; Johansson et al. 2010). A few studies have found that self-reported household income and financial inadequacy predicted cognitive function, cognitive decline, and dementia among older adults (Chiao et al. 2014; Luo and Waite 2005; Yaffe et al. 2013; see Gonzalez et al. 2013 for an exception). In addition, poor early-life conditions can increase the risk of hypertension, heart disease, and stroke in adulthood, which in turn adversely affects late-life cognitive function (O'Rand and Hamil-Luker 2005;Turrell et al. 2002).

It is important to point out that the determinants of cognitive health in later life are multifactorial. Early-life environment, genetic predisposition, adulthood socioeconomic

resources, diet, illness, and social engagement are associated with cognitive function in later life (Deary et al. 2004). The two conceptual models for the effects of childhood conditions on late-life cognition are not mutually exclusive and may operate together in complex ways. It is possible that some childhood conditions may affect cognition directly, while others may act indirectly via factors in adulthood (Karp et al. 2004; Zhang et al. 2010).

Racial Differences in Childhood Conditions and Adulthood SES and Health

Throughout U.S. history, race has been one of the most important social stratification systems that puts blacks at a substantial socioeconomic disadvantage throughout the life course (Hayward et al. 2000; Zsembik and Peek 2001). Older blacks are more likely to report that they experienced poverty and poor health in their childhood than older whites in national surveys (Warner and Hayward 2006). While growing up in the early 1900s and in the South, blacks lived in vastly different social and physical environments from whites as a result of discrimination, residential segregation, and restricted access to education (Barnes et al. 2012). Recent research has suggested that having lived in the South while growing up may be particular harmful for older blacks' cognition, in part because of the very different schooling experiences for blacks and whites until the Supreme Court's Brown vs. Board of Education decision of 1954, which declared state laws upholding school segregation unconstitutional. Before the legally imposed desegregation, blacks not only attended schools with much poorer funding and lower quality than those attended by whites in the South but also spent fewer days at schools. School terms for white children were 50–100% longer than those for black children in segregated states (Glymour and Manly 2008). These disadvantages of schooling for black children in the South might have negatively affected their cognitive development (Lee 1951; Ceci 1991).

As a consequence of poverty, discrimination, and restricted access to medical care, there is a large racial gap in childhood health throughout the 20th century (Costa 2004). For example, low birth weight rate and infant mortality rate among blacks were approximately twice as high as they were among whites in the first third of the 20th century (Costa 2004; Guyer et al. 2000).

Entering early adulthood, blacks are more likely than whites to complete fewer years of schooling, to have marginal jobs, to earn lower income, and to have higher unemployment rates due to residential segregation and occupational discrimination (Hayward et al. 2000; Williams and Sternthal 2010). In particular, there is an enormous gap in wealth accumulation between blacks and whites (Shapiro, Meschede, and Sullivan 2010). According to estimates in the 1990s, mean household wealth for whites is more than three times that of blacks. In addition, at every education and income quartile level, blacks accumulate far less wealth than their white counterparts, due in part to their lower income, poorer health, and smaller inheritance as well as the limited appreciation of home equity in segregated neighborhoods (Choudhury 2003; Colen 2011). Wealth is an important component of economic well-being because it offers people some protection against life events that cause financial stress, such as periods of unemployment, business losses, marital losses, and bouts of major illness. Recent research shows that both income and wealth are

significant predictors of morbidity and mortality (Huie et al. 2003; Hummer and Chinn 2011; Pollack et al. 2007).

In addition to wide racial gaps in SES, research shows that there are also significant racial differences in health behaviors such as smoking, drinking, and sedentary behavior (Dubowitz et al. 2011; Kurian and Cardarelli 2007) and that blacks have earlier onset and higher prevalence of major chronic diseases such as hypertension, heart disease, stroke, and diabetes than whites (Geronimus 2001; Hayward et al. 2000).

Taken together, the current literature shows that compared to whites, blacks are exposed to substantial socioeconomic disadvantages in childhood and throughout their adulthood (Warner and Hayward 2006; Williams and Sternthal 2010). It is important to understand how early disadvantages for blacks in comparison to whites shape significant racial differences in cognitive impairment in later life. Specifically, we are interested in finding out how racial differences in childhood conditions, adulthood SES, adult health and heath behaviors influence the racial gap in cognitive impairment.

Guided by the life course perspective and drawing on prior research on the racial gap in cognitive impairment, we examine the following hypotheses: (1) Compared to whites, blacks are more likely to suffer from a higher risk of cognitive impairment at baseline and during the 12-year follow-up; (2) Blacks' higher risk of cognitive impairment at baseline and during follow-up will be significantly reduced when childhood health and adversity are controlled for; (3) Blacks' higher risk of cognitive impairment will be further reduced when adulthood socioeconomic achievement, chronic disease, and health behaviors are controlled for in addition to childhood conditions.

DATA AND METHODS

We used data from 7 waves of the HRS (1998–2010) to investigate life course origins and pathways of the racial gap in cognitive impairment in later life. The 1998 wave of the HRS used a nationally representative sample of noninstitutionalized adults older than 50 and included information from 21,384 respondents. It also oversampled blacks and Hispanics. The HRS first asked all respondents about childhood conditions in 1998. The survey collected detailed information on cognitive, physical, economic, work, and family conditions as well as health behaviors approximately every two years, either by telephone or in person. About 10% of the interviews were done through proxies (spouses or children) for sample members who could not complete the survey (Langa et al. 2008). We have retained respondents with proxy reports in our analysis. Our analytic sample included 9,044 non-Hispanic blacks (hereafter, blacks) and non-Hispanic whites (hereafter, whites) aged 65 and over who lived in the community in 1998. Many respondents had missing data for mother's (13%) or father's education (16%). In order to retain these cases, we imputed the values and details were described below. We excluded 98 respondents (1%) who had missing values on childhood health, adult health, or health behaviors, which resulted in 8,946 respondents. Among the 8,946 respondents at baseline, 8,552 respondents were considered cognitively unimpaired and we followed them until they experienced the onset of cognitive impairment,

died, or dropped out of the study. From 1998 to 2010, 1,438 individuals (16.8%) experienced the onset of cognitive impairment, 3,345 (39.1%) died, and 1,319 (15.4%) dropped out.

Measures

The measurement of cognitive status in the HRS, our dependent variable, differed for self- and proxy respondents. Cognitive tests were administered in all 7 waves of the HRS to self-respondents and can be used to track the cognitive transitions of respondents over time. We used the summary measure of cognitive function, which was based on the modified version of the Telephone Interview for Cognitive Status (TICS), to classify respondents' cognitive status. The modified version of TICS included date identification, object naming, naming of the President and Vice President, serial subtraction of 7s, and tests of immediate and delayed recall of a list of 10 words. The summary score ranged from 0 (severely impaired) to 35 (highly functioning). A small percentage of respondents in each wave refused to participate in tests of immediate and delayed recall and serial subtraction of 7s, and the HRS has developed a multiple imputation strategy that imputed cognitive variables for all waves (Ofstedal, Fisher, and Herzog 2005). We used the imputed cognitive variables released by the HRS in our analysis. Following previous research (Freedman, Aykan, and Martin 2002; Herzog and Wallace 1997; Reuser et al. 2011), we classified respondents as having moderate/severe cognitive impairment if they scored an 8 or lower out of 35 at baseline. In additional analysis, we examined cut-off points of 7 and 9, and the results (not shown) were similar. For follow-up surveys (2000–2010), we increased the cut-off point by 1 and classified those who scored 9 or less as moderately/severely impaired. Previous research suggested that a higher cut-off point for cognitive impairment was appropriate for HRS follow-up surveys to account for practice effects (Lièvre, Alley, and Crimmins 2008; Reuser et al. 2011).

For sample members who were unable to participate in the cognitive tests, proxies were asked to report on a list of symptoms of cognitive impairment. Five symptoms were consistently asked about from 1998 to 2010: got lost in a familiar environment; wandered off and did not return by himself/herself; could not be left alone for an hour; had hallucinations; and had poor memory. In a recent paper, Crimmins et al. (2011) found that difficulty with eating and difficulty with managing money were significantly associated with clinical diagnosis of dementia in the Aging, Demographics, and Memory Study (ADAMS) We thus constructed a summary score ranging from 0 (no symptoms of cognitive impairment) to 7 (severely impaired) based on reports of the above-mentioned five symptoms of cognitive impairment and of difficulties with the two daily activities of eating and managing money. Respondents who had two or more of these symptoms were identified as moderately/ severely cognitively impaired. In sum, for those previously identified as free of cognitive impairment, we defined the onset of cognitive impairment as scoring 9 points or less or having two or more symptoms of cognitive impairment.

Independent Variables

Our key independent variables included self-reported race (black=1, white=0), childhood conditions, adulthood SES, health, and health behaviors.

Childhood conditions—Childhood condition indicators included childhood health, childhood SES, and birth region. All variables were created based on respondents' answers to questions on their health and family circumstances when they were growing up (from birth to age 16). Childhood health was based on the respondent's rating of his or her health as a child on a 5-point scale ranging from poor to excellent. We dichotomized the measure into poor (fair and poor) vs. good (good/very good/excellent). Previous research has shown that the retrospective measure of overall childhood health was valid and showed good reliability over time (Haas 2007; Smith 2009). Cumulative childhood adversity was assessed with an index that included four items: father's education (1=less than 8 years), mother's education (1=less than 8 years), father's occupation (1=blue collar work including farming/ fishing/forestry, sales, operators of machines, mechanics/repair, clerical support, personal services, and don't know), and subjective assessment of family's financial situation (1=the family was poor). The index ranged from 0 (most advantaged) to 4 (most disadvantaged). A significant proportion of respondents had missing data for mother's or father's education. Following the practice of other researchers (Luo and Waite 2005; Montez and Hayward 2014), we imputed the missing data as less than 8 years of education. Our preliminary analysis also showed that respondents who had missing data on parental education were similar on other indicators of family economic circumstances in childhood to those adults whose parents had less than 8 years of education. Finally, we used a Southern birth variable (1=Southern) to classify those respondents whose self-reported states of birth were defined as part of the Southern region by the U.S. Census. All childhood variables came from 1998 HRS.

We created three blocks of variables to evaluate the direct and indirect pathways of early-life environment on racial gaps in late-life cognitive impairment.

Adulthood SES—Adulthood SES was measured using education, household income, and net household wealth. Education was measured in four categories: less than 8 years, 8–11, 12, and 13 or more years. Household income, a time-varying variable, measured the total household income in the year prior to each interview wave. Net household wealth was also measured as a time-varying variable representing the value of household assets minus debts. Both household income and household wealth were adjusted by adding constants to eliminate zero income and negative wealth; the values were then divided by the square root of household size and logged (Glymour et al. 2008; Zhang and Hayward 2006). The household income and wealth data were provided by the RAND Center for the Study of Aging (version L) which consistently imputed missing income and wealth data across waves.

Adult health—Chronic disease was measured with a time-varying dichotomous variable. It compared those respondents who reported having heart disease, hypertension, diabetes, or stroke with those who had none of these diseases. These conditions were included because of prior studies pointing to racial differences in these chronic diseases (Hayward et al. 2000; LaVeist et al. 2009; Mensah et al. 2005), which put older adults at higher risk of cognitive impairment (Singh-Manoux and Marmot 2005; Tilvis et al. 2004).

Health behaviors—We examined three health behaviors: exercise, smoking, and drinking. Exercise was coded as a dummy variable which compared respondents that participated in vigorous physical activity or exercise three times a week or more over the past 12 months (reference) with those who did not. Because the question on exercise changed over waves, we only included exercise at baseline in the analysis. We coded smoking into three categories: never smoked (reference), former smoker, and current smoker. Drinking was coded into four categories: never drank, former drinker, current light drinker (reference), and current heavy drinker. Light drinker referred to those who on average drank less than 7 cups of alcoholic beverages per week during the past three months. This definition was based on the recommendation of the National Institute of Alcohol Abuse and Alcoholism (Lin, Guerrieri, and Moore 2011).

Control Variables—We controlled for demographic variables including age, gender (women=1), and marital status. Age was a continuous variable. Marital status had four categories: married/cohabiting (reference), divorced/separated, widowed, and never married. We also created a dummy variable to indicate whether a proxy respondent was used for an individual who was unable to participate in the cognitive tests.

Analytic Strategy

We began by looking at racial differences in the prevalence of cognitive impairment at baseline and examining the role of childhood conditions and education in explaining the racial disparity. Then we analyzed racial difference in the incidence of cognitive impairment from 1998 to 2010. When we examined the incidence of cognitive impairment, the risk set was persons free of cognitive impairment. The three possible ways out of the risk set were being observed at follow-up in one of three states: cognitively impaired, dead, or lost to follow-up. The non-mortal outcomes were assumed to be absorbing, and thus we did not examine transitions from cognitive impairment to normal cognition. The cognitive impairment process is age-dependent, and we employed a discrete-time event history modeling approach to characterize this process using the HRS data. Person-interval record files were created for the two-year intervals from 1998 to 2010, and a multinomial logit modeling approach was used for the discrete-time event history analysis. An individual could potentially contribute between 1 and 6 person-intervals to the analysis. We first estimated the risk of cognitive impairment as a function of race to obtain the total effect of race, net of demographic variables. We then introduced explanatory variables in a series of nested models to evaluate whether and by what mechanisms early-life conditions contributed to racial differences in the onset of cognitive impairment across the 12-year period. The sets of early-life characteristics and adulthood SES, health, and health behaviors were added sequentially. All time-varying independent variables were lagged by one wave in the prospective analyses. By comparing changes in the coefficients of race across the nested models, we can assess the role of early-life characteristics as well as adulthood conditions in accounting for the racial gap in cognitive impairment (Warner and Hayward 2006). We estimated all models using SAS 9.2 and adjusted for the complex sampling design of the HRS. All models were based on weighted data using 1998 HRS sampling weights.

RESULTS

We present weighted sample means by race at baseline in Table 1 to show the extent of differences in social and economic resources between blacks and whites in childhood and adulthood. As expected, blacks, on average, report growing up in more disadvantaged socioeconomic environments than whites. On the index of cumulative childhood adversity (0–4), blacks' average score is 2.84, compared to 2.06 for whites. About 87.54% of blacks were born in the South, compared to about 26.65% of whites. Although slightly more blacks (7.58%) report poor health in childhood than whites (6.05%), the difference is not statistically significant. In terms of adulthood economic achievement, the black-white gaps appear wider. Blacks are more likely to have lower levels of education. About 28.45 % percent of blacks have 0–7 years of schooling compared to 4.69% among whites. Blacks have roughly one-half of whites' household income, and one-fifth of whites' household net wealth. Also, more blacks (75.38%) report having at least one of the major chronic diseases than whites (64.67%). The profile of health behaviors for blacks is mixed compared with that of whites. Blacks are less likely to exercise vigorously and more likely to smoke currently. However, blacks are less likely to drink heavily and more likely never to have drunk any alcohol in their lives.

As is shown in Table 1, the prevalence of cognitive impairment for blacks is more than three times that for whites (10.58% vs. 3.09%) at baseline, and a higher proportion of blacks (12.54%) had proxy reports than whites (7.17%). Table 2 presents the estimated prevalence of cognitive impairment by age and gender for blacks and whites in 1998. At each age listed, black men and black women have much higher prevalence of impairment than their white counterparts. Moreover, the prevalence rates for blacks at each age are not reached for whites until roughly ten years later. For example, the prevalence of cognitive impairment for black men at age 70 is 5% whereas the prevalence for white men at age 80 is 4%.

What factors contribute to the higher prevalence of cognitive impairment among blacks at baseline? We examined the roles of childhood conditions and education in Table 3. As is shown in Model 1, the odds of impairment for blacks are 3.69 times the odds for whites, controlling for demographic variables and proxy reports. Introducing childhood conditions in Model 2 reduces the odds of impairment for blacks from 3.69 to 2.77. We find that being born in the South is associated with higher odds of cognitive impairment. In Model 3, we add education. In accordance with the results of previous studies, a lower level of education is associated with higher odds of impairment. Although the race gap is further reduced, the odds of impairment for blacks are still 2.36 times the odds for whites.

Next, we turn to a series of nested multinomial logistic regression models to examine: (a) whether there are racial differences in the onset of cognitive impairment from 1998 to 2010 among those whose cognition is considered unimpaired at baseline, and (b) the extent to which the racial gap in impairment is explained by childhood and adulthood circumstances. Table 4 summarizes the results from our six models. First, it is evident in Model 1 that the odds of experiencing the onset of cognitive impairment for blacks are approximately 2.52 times the odds for whites, net of control variables. Note that in Table 3 we showed that

blacks are already significantly more likely to be impaired at baseline. The incidence results show the persistence of blacks' disadvantage in the 12-year follow-up.

When childhood conditions are added in Model 2 of Table 4, the odds of impairment for blacks are reduced from 2.52 to 1.86. This reduction primarily stems from racial differences in cumulative childhood adversity and birth region, which are significantly associated with odds of cognitive impairment. With each additional point on the cumulative childhood adversity index, the odds of impairment increase by 11%. To put it another way, the odds of impairment for those who are most disadvantaged in childhood (i.e., those with a score of 4 on the cumulative childhood adversity index) are 1.52 times the odds for the most advantaged (i.e., those with a score of 0). Being born in the South is also significantly associated with higher odds of impairment.

In Model 3, the odds of cognitive impairment for blacks are further reduced when education is introduced into the model. Consistent with previous research, we find that low levels of education are associated with a higher risk of impairment. Moreover, after adding education, the effect of cumulative childhood adversity on impairment is reduced and no longer statistically significant; accounting for education also slightly attenuates the effect of Southern birth, though it remains statistically significant.

When we add adulthood income and wealth in Model 4, the odds of cognitive impairment for blacks are reduced slightly. Those with higher household income have a significantly lower risk of impairment. Overall, Models 3–4 suggest that adult SES in the form of educational attainment and household income are strongly and negatively associated with the incidence of impairment, net of childhood conditions. Nonetheless, even after accounting for racial gaps in childhood conditions and adulthood SES, blacks are still more likely to experience the onset of cognitive impairment than whites.

When the chronic disease measures are introduced into Model 5, the racial gap barely changes, from an odds ratio of 1.45 to 1.43. This finding suggests that once childhood conditions and adult SES are controlled for, chronic disease accounts for little of blacks' higher risk of impairment. Since adulthood SES and chronic disease are intertwined, we estimated another model (results not shown) that included demographic controls, proxy reports, childhood conditions, and chronic disease. Compared to the results in Model 2 that controlled for childhood conditions only, the odds of impairment for blacks changed from 1.86 to 1.83 (results not shown) when we added chronic disease in this alternative model. Clearly, racial differences in adulthood SES, rather than racial disparity in chronic disease, accounts for blacks' higher risk of cognitive impairment in later life, once childhood conditions are controlled.

The final model (Model 6) introduces three health behavior measures. Similar to what happens when controlling for chronic disease status, the racial gap in cognitive impairment does not change when current health behaviors are controlled for. In additional analyses (results not shown), we add each health behavior separately, and the results are similar to those in Model 5. Note that the effect of Southern birth on impairment is slightly reduced in Model 6 compared to Model 5 but remains statistically significant. The effects of education

and household income on the risk of impairment also remain strong and robust in Model 6. With regard to the pattern of associations between cognitive impairment incidence and the health behaviors, we find the expected relationships. Smoking and nondrinking are associated with a higher risk of impairment, whereas vigorous exercise is associated with a lower risk of impairment. The results in Table 4 show that once childhood conditions and adulthood SES and health are controlled for, health behaviors explain very little of the remaining racial gaps in the onset of cognitive impairment.

The eighth column of Table 4 shows the odds of death relative to being cognitively unimpaired from 1998 to 2010. Cognitively unimpaired blacks are significantly less likely to die than their white counterparts, controlling for other variables in Model 6. While childhood conditions have no independent effects on mortality, adulthood SES indicators such as household income and wealth exert powerful effects on the odds of death, net of controls. As expected, chronic disease, health behaviors, demographic variables, and proxy reports are strongly associated with mortality. The last column of Table 4 shows the odds of loss to follow-up relative to being cognitively unimpaired. Only a few variables are statistically significant. Respondents with higher scores on the cumulative childhood adversity index are more likely to drop out of the study than those who are less disadvantaged. The more wealthy respondents are more likely to drop out. Those who have chronic disease and proxy reports are also more likely to drop out.

DISCUSSION

As stated earlier, we have two goals in this study. One is to examine whether there is a racial gap in the risk of cognitive impairment in later life; the second is to investigate childhood conditions and adulthood SES, health, and health behaviors as important pathways by which the racial gap is generated. We hypothesized that the higher risk for blacks of cognitive impairment was largely explained by their childhood disadvantages and subsequent lower socioeconomic status and poorer health in adulthood compared to whites.

All three of our hypotheses are supported. We find that blacks suffer from a higher risk of moderate/severe cognitive impairment both at baseline and during the 12-year follow-up (Hypothesis 1). Our findings are consistent with most of the previous studies on racial differences in dementia or Alzheimer's disease based on regional and nonrepresentative samples (e.g., Demirovic et al. 2003; Tang et al. 2001; Yaffe et al. 2013). As expected, we find that blacks are more likely to report childhood adversity than whites and to have grown up in the segregated South, and these early-life adversities put them at a significantly higher risk of cognitive impairment in later life. The racial gap is reduced after the adjustment of childhood conditions (Hypothesis 2). Thus, our results suggest that the racial gap in cognitive impairment can be explained in part by the racial difference in childhood conditions.

As for hypothesis 3, we find that adulthood SES, educational attainment in particular, is associated with the risk of cognitive impairment, net of childhood conditions; and the wide gap in adulthood SES between older blacks and whites accounts for a significant part of the racial gap in cognitive impairment. In addition, once childhood conditions and adulthood

SES are controlled for, racial differences in adult health and health behavior explain very little of the remaining differences between blacks and whites, although health and health behaviors are associated with cognitive impairment.

Taken together, we contribute to the ongoing debate on racial differences in cognitive aging by providing evidence from a national longitudinal data set that there is a gap in the onset of moderate/severe cognitive impairment between older blacks and whites. Furthermore, our results point to racial differences in childhood social and economic adversity and adulthood SES as major pathways through which racial gap in cognitive impairment is produced. Our results provide support for the recent findings of the effects of life-course SES (education, literacy, income, and financial adequacy) on black-white disparities in more serious cognitive impairment such as dementia from regional studies (Shadlen et al. 2006; Yaffe et al. 2013). In this research, we pay careful attention to the longitudinal attrition through mortality and non-response by employing competing-risks models. One of the important findings is that those who used proxies are a very disadvantaged group of people who not only have higher risks of cognitive impairment and mortality but also are more likely to drop out. Since blacks are more likely to have proxy respondents, studies that exclude proxy interviews may underestimate racial differences in cognitive impairment.

The present study has several strengths, including the use of a nationally representative data set, the 12-year follow-up of blacks and whites to study the onset of moderate/severe cognitive impairment, the exploration of both childhood and adulthood conditions as pathways to the racial gap in cognitive impairment, the rich measures of adulthood socioeconomic resources, and the inclusion of both self- and proxy respondents. However, researchers should consider several limitations when interpreting our results. First, it is important to emphasize that the medical diagnosis of cognitive impairment often relies on a comprehensive clinical evaluation, and using cognitive tests and proxy respondents to detect cognitive impairment, as we do in this study, has its limitations. For example, researchers found that using the cognitive tests for self-respondents in the HRS, they could correctly classify about 74% of a selected group of HRS subjects into clinical diagnosis categories of normal; cognitively impaired, not demented (CIND); or demented. For proxy respondents, 86% of the sample was correctly classified (Crimmins et al. 2011). Previous research also showed that cognitive tests used in large population surveys had the best screening accuracy when they were used to differentiate the demented from the nondemented (Crimmins et al. 2011; Manly et al. 2011). Therefore, in this study we focused on the moderately/severely cognitively impaired and tried to minimize the danger of misclassification. We also acknowledge that respondents' education level, and the quality of education they receive in particular, may influence their performance on the cognitive tests. A related challenge is whether community-based screening tools for cognitive impairment and dementia including cognitive tests and informant reports of cognitive function operate differently for blacks and whites due to unmeasured educational and cultural factors, and whether different cut-off points should be established for older blacks and whites (Glymour and Manly 2008; Manly 2005; Potter et al. 2009). We were unable to address these issues in our paper due to the lack of clinical data on cognitive impairment in the HRS. Future research should collect more clinical data and produce screening instruments that take into consideration the quality of education the respondents have received (e.g., literacy) and the different cultural attitudes

toward cognitive impairment as recent studies show that black informants may be less likely to report mild cognitive change (Burns et al. 2006; Potter et al. 2009).

Second, although we are able to examine childhood conditions as one of the major pathways through which racial differences are produced, our measures of childhood conditions are based on retrospective self-reports. As in other studies that have used retrospective reports (e.g., Hayward and Gorman 2004; Luo and Waite 2005), there is the potential problem of recall bias. Third, our measures of childhood conditions are limited, and future research should tap into potential racial differences in childhood nutrition and growth, childhood trauma and maltreatment such as abuse, neglect, discrimination, and family stressors such as parental conflicts and parental loss (Barnes et al. 2012; Dik et al. 2003; Shonkoff and Garner 2012), which may contribute to racial differences in cognitive impairment. Fourth, recent studies showed that social relationship and emotional support in adulthood may protect against cognitive decline (Ellwardta et al. 2013; Hakansson et al. 2009) and therefore documented racial differences in marital status, kin support, and relationship quality (Bulanda and Brown 2007; Sarkisian and Gerstel 2004) may be another important pathway to racial differences in cognitive impairment. Last, due to racism and residential segregation, blacks are more likely to live in neighborhoods characterized by poverty, social disorder, and exposure to environmental toxins (Aneshensel et al. 2011; Williams and Sternthal 2010). Future research needs to examine how different quality of living environment of blacks and whites contributes to racial differences in cognitive impairment, as recent research suggested that neighborhood disadvantage may influence cognitive health in later life through their effects on individual's psychological stress, health, health behaviors, and access to resources (Aneshensel et al. 2011; Wu, Prina, and Brayne 2015).

Despite these limitations, our study makes contributions to the current discussion on racial disparities in cognitive impairment by investigating the issue with a nationally representative longitudinal data set. Although our models do not fully explain blacks' higher risk of cognitive impairment compared to whites, our results suggest that early-life disadvantages experienced by blacks contribute to their lower socioeconomic status in adulthood, which significantly increases their risk of cognitive impairment in later life. Reducing racial disparities in health is a national policy priority, and a great deal of effort has been devoted to improving health care access, advocating positive health behaviors, and finding treatment for diseases. While these efforts are important, our results suggest that policymakers need to put more emphasis on reducing racial gaps in socioeconomic resources over the entire life course. There are multiple windows of opportunity for intervention, and the most promising ones are early life and young adulthood because these are periods that shape individuals' socioeconomic trajectories into their later life. Social policies such as improving educational resources and capabilities in disadvantaged communities, providing economic support to poor students and their families, improving graduation rates in high schools and colleges, and eliminating discrimination against blacks in job markets may significantly reduce racial disparities in cognitive impairment in later life.

Biographies

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Mark D. Hayward is Professor of Sociology and Centennial Commission Professor in the Liberal Arts at the University of Texas at Austin. Currently, he is co-PI of an NIA supported research network on health dynamics and disparities over the life course. His recent work examines the role of educational attainment in the context of the life course in influencing adult health and mortality, and historical changes in these processes.

Yan-Liang Yu is a doctoral student in the Department of Sociology at Michigan State University. His research focuses on health disparities, and particularly how marital experiences influence health over the life course. His recent work examines the effects of interracial marriage on health. His dissertation explores how marital biography influences the progression of chronic diseases among older Americans.

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Table 1

Weighted Descriptive Statistics by Race, Health and Retirement Study, 1998.

Variable	Whites (n=7,734)	Blacks (n= 1,212)
Cognitive impairment (%)	3.09	10.58[*]
Childhood conditions		
Fair/poor childhood health (%)	6.05	7.58
Cumulative childhood adversity (0–4)	2.06 (.02)	2.84[*] (.03)
Southern birth (%)	26.65	87.54[*]
Adulthood SES		
Education (%)		
0–7 years	4.69	28.45[*]
8–11 years	23.44	34.42[*]
12 years	36.24	20.24[*]
13 years and above	35.63	16.89[*]
Household income ($)	36,335 (875)	18,389[*] (822)
Net household wealth ($)	318,965 (13,449)	63,990[*] (3,736)
Adult health and health behaviors		
Chronic disease (%)	64.67	75.38[*]
Smoking (%)		
Current smokers	10.48	15.08[*]
Former smokers	46.69	42.59
Never smoked	42.82	42.33
Drinking (%)		
Never drank	53.27	73.74[*]
Former drinkers	18.14	13.36[*]
Current light drinkers	20.72	9.02[*]
Current heavy drinkers (>7 drinks/week)	7.88	3.87[*]
No vigorous exercise(%)	59.97	72.37[*]
Controls		
Age (years)	74.44 (.10)	73.93 (.29)
Women (%)	57.80	62.18[*]
Marital status (%)		
Married/cohabiting	58.56	38.96[*]
Divorced/separated	6.32	14.67[*]
Widowed	32.41	41.92[*]
Never married	2.71	4.44
Proxy (%)	7.17	12.54[*]

Note: Values in parentheses are standard errors of means.

*
Statistically significant difference between whites and blacks at the .05 level.

Table 2

Prevalence of Cognitive Impairment by Race and Gender Groups, Health and Retirement Study, 1998.

Age	White Men	White Women	Black Men	Black Women
65	.7	.8	2.8	3.1
70	1.3	1.4	5.0	5.4
75	2.3	2.5	8.7	9.4
80	4.0	4.4	14.8	15.8
85	7.1	7.6	23.9	25.4
90	12.2	13.0	36.3	38.1

Note: All values are weighted. $N = 8,946$. The prevalence figures are estimates derived from logistic regression model by regressing the log odds of cognitive impairment on age, race, and gender. Interaction between race and gender was tested and was statistically insignificant. Parameter estimates are used to calculate predicted rates.

Table 3

Logistic Regressions of the Odds of Cognitive Impairment among Older Americans, Health and Retirement Study, 1998.

	Model 1	Model 2	Model 3
Blacks	3.69[**]	2.77[**]	2.36[**]
Childhood conditions			
Fair/poor childhood health		1.07	.99
Cumulative childhood adversity		1.11	1.02
Southern birth		1.47[**]	1.25
Education (reference=12 years)			
0–7 years			2.76[**]
8–11 years			1.40
13 years and above			1.07
Controls			
Age	1.10[**]	1.10[**]	1.09[**]
Women	1.57[**]	1.62[**]	1.79[**]
Marital status			
(reference= married/cohabiting)			
Divorced/separated	.93	.91	.82
Widowed	1.06	1.01	.95
Never married	1.99	2.00	1.89
proxy	21.38[**]	20.25[**]	19.01[**]

[*]
$p < .05$;

[**]
$p < .01$.

N=8,946.

Table 4

Multinomial Logistic Regressions of the Odds of Cognitive Impairment, Death, and Loss to Follow-Up among Cognitively Unimpaired Older Americans, Health and Retirement Study, 1998–2010.

	Cognitively Impaired vs. Cognitively Unimpaired						Dead vs. Cognitively Unimpaired	Missing vs. Cognitively Unimpaired
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 6	Model 6
Blacks	2.52 **	1.86 **	1.62 **	1.45 **	1.43 **	1.43 **	.80 **	1.07
Childhood conditions								
Fair/poor childhood health		1.23	1.14	1.12	1.11	1.09	1.12	.77
Cumulative childhood adversity		1.11 **	1.01	1.00	1.00	.99	.97	1.09 **
Southern birth		1.44 **	1.32 **	1.31 **	1.31 **	1.23 **	1.00	.99
Adulthood SES								
Education (reference=12 years)								
0–7 years			2.41 **	2.15 **	2.16 **	2.09 **	1.03	1.20
8–11 years			1.66 **	1.54 **	1.54 **	1.50 **	1.04	1.12
13 years and above			.92	1.02	1.03	1.09	1.02	.89
Household income[a]				.78 **	.78 **	.80 **	.85 **	.97
Net household wealth[a]				.91	.92	.95	.90 **	1.12 *
Adult health and health behaviors								
Chronic disease					1.24 **	1.21 **	1.94 **	1.20 **
Smoking (reference=never smoked)								
Current smoker						1.39 *	2.84 **	1.16
Former smoker						1.07	1.56 **	1.01
Drinking (reference=current light drinkers)								
Never drank						1.86 **	1.79 **	1.11
Former drinker						1.50 **	1.32 **	.96
Heavy drinker (>7 drinks/week)						1.07	.97	1.06
No vigorous exercise						1.22 *	1.67 **	.99
Controls								
Age	1.13 **	1.13 **	1.13 **	1.13 **	1.12 **	1.12 **	1.11 **	1.00

	Cognitively Impaired vs. Cognitively Unimpaired						Dead vs. Cognitively Unimpaired	Missing vs. Cognitively Unimpaired
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 6	Model 6
Women	1.20 *	1.19 *	1.26 **	1.19 *	1.19 *	1.11	.60 **	1.10
Marital status (reference= married/cohabiting)								
Divorced/separated	.98	.96	.94	.83	.84	.81	1.31 **	.97
Widowed	1.10	1.07	1.01	.92	.93	.90	1.14 *	.87
Never married	1.01	1.00	1.00	.88	.88	.88	1.63 **	.85
Proxy	3.68 **	3.45 **	3.01 **	2.96 **	2.97 **	2.84 **	2.03 **	2.77 **

*
$p < .05$;

**
$p < .01$