Lifecourse Pathways to Racial Disparities in Cognitive Impairment among Elderly Americans

Zhenmei Zhang, Michigan State University
Mark D. Hayward, University of Texas at Austin
Yan-Liang Jerry Yu, Michigan State University

Extended Abstract for PAA 2013

Cognitive impairment and dementia are major health problems confronting older persons. In 2002, approximately 13.9% of people in the United States aged 71 and older had dementia, and 22.2% had cognitive impairment without dementia, with the prevalence rate rising sharply with age. Blacks are especially hard hit by cognitive impairment and dementia. It was estimated that among those aged 71 years and older Blacks were approximately two times more likely to have dementia than Whites (Plassman et al., 2007; Plassman et al., 2008). Despite developments in understanding risk factors associated with cognitive impairment and dementia in recent years, very few population-level studies have investigated the origins and mechanisms through which the racial gap in cognitive impairment is produced.

Theoretical focus

The Long Arm of Childhood. Increasingly, the life course approach has been used to evaluate the determinants of cognitive functioning in late-life. Cumulative disadvantage theory posits that insults throughout the life course (e.g., childhood illness and poverty, family disruption, unemployment, illness and unhealthy behavior in adulthood) can accumulate and negatively affect people's health in late-life. The theory also suggests that early risk factors can be compounded across the life course by setting people onto different life trajectories such that earlier disadvantages lead to later disadvantages, which result in increasing health inequalities in later life (Crystal & Shea, 1990; O'Rand & Hamil-Luker, 2005). Although the life course perspective has been used to examine racial disparities in mortality and physical diseases, few studies have used it to examine the origins of racial disparities in cognitive health in later life. Recent research in developed and developing countries consistently reveals that dementia may have its origins in childhood (Deary et al., 2004; Zhang, Gu, &Hayward, 2008). Although the exact mechanisms linking childhood conditions and late-life cognitive impairment are still not clear, two conceptual models are used to explain the links.

The critical period model suggests that early life adversity can have ***direct*** effects on late-life cognition. The brain grows most during the prenatal period and the first 3 years of life and continues to grow in childhood and adolescence (Kim et al., 2003). Early life deprivation can lead to impaired brain development. The negative effects of the impaired brain development may be small until aggravated by the aging process (Moceri et al., 2000). A growing body of research has reported that childhood socioeconomic disadvantages are directly associated with cognitive impairment and dementia in later life (Huang et al., 2008; Petot et al., 2007).

The pathway model suggests that that early life circumstances may ***indirectly*** influence cognitive impairment via education attainment, occupational exposures, health behaviors, and chronic disease. Education in early-life promotes brain growth in the formative years and enables the brain network to operate more efficiently, thus providing protection against cognitive decline in later life (Fritsch et al., 2007; Kaplan et al., 2001). Higher levels of education often lead to occupations that involve cognitive challenges and practice, which could further enhance or maintain cognitive functioning in adulthood (Schooler, 1987).In addition, poor early life conditions can increase the risk of hypertension, heart disease, and stroke in adulthood, which in turn affects late-life cognitive functioning (O'Rand & Hamil-Luker, 2005;Turrell et al., 2002). Given the substantial socioeconomic disadvantages the African Americans are exposed to during childhood and early adulthood (Fillenbaum et al., 1998), it is important to understand how these early disadvantages may shape into higher adult risks in cognitive impairment and dementia.

In this study, using data from 7 waves of the Health and Retirement Study (1998-2010), we analyzed how racial differences in cognitive impairment are tied to the racial stratification of childhood resources and health, adult health, health behaviors, and socioeconomic achievement in adulthood among 9,044 non-Hispanic Whites and Blacks aged 65 and older in 1998.

Data and Methods

Data

We used data from the 1998, 2000, 2002, 2004, 2006, 2008 and 2010 waves of HRS to analyze the relationship between early life conditions and late-life cognitive impairment, because childhood conditions were first asked of all HRS respondents in 1998. The 1998 wave of HRS is a nationally representative sample of noninstitutionalized adults older than 50 and includes information from 21,384 respondents and oversamples of Blacks and Hispanics. HRS collects detailed information on cognitive, physical, economic, health behavior, work, and family conditions every 2 years, either by telephone or in-person. About 10% of the interviews are done with proxies (spouses or child) for sample members who cannot complete the survey (Langa et al, 2005). At baseline (1998 HRS), there are 1,226 Blacks and 7,818 Whites aged 65 and over.

Measures

Our dependent variable is cognitive impairment. The measurement of cognitive status in the HRS differs for self- and proxy- respondents. The cognitive tests were administered in all 7 waves of HRS for self-respondents and can be used to track the cognitive transitions among respondents over time. Following previous research (Alley, Suthers, & Crimmins, 2007; Herzog & Wallace, 1997), we used the summary measure of cognitive function, which is based on the modified version of TICS, to classify respondent's cognitive status. The modified version of TICS includes date identification, object naming, naming of the President and Vice President, a serial 7s subtraction, and the tests of immediate and delayed verbal of a list of 10 words. The summary score

ranges from 0 (severely impaired) to 35 (highly functioning). A small percentage of respondents (0.8%-3.1%) refused to participate in tests of immediate and delayed recall and Serial 7s, and because these data were not missing on random, the HRS has developed a multiple imputation strategy that imputed cognitive variables for all waves (Ofstedal, Fisher & Herzog, 2005). We used the imputed cognitive variables released by HRS in the analysis. Based on our previous validation study, we used different cut-points for cognitive impairments for Blacks and Whites. Among Blacks, those who scored between 0 and 12 were classified as cognitively impaired, and 13 to 35 as normal. For Whites, those who scored between 0 and 14 were classified as cognitively impaired, and 15 to 35 as normal. For persons who were unable to participate in the cognitive tests due to health issues, proxies were asked to report on a list of symptoms of cognitive impairment. Five symptoms were consistently asked from 1998 to 2010: got lost in a familiar environment; wandered off and not returned 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 highly associated with diagnosis of dementia. We thus constructed a summary score of the respondent's cognition ranging from 0 (no symptoms of cognitive impairment) to 7 (severely impaired) based on proxy reports of five symptoms of cognitive impairment and difficulties with two daily activities of living. Respondents who had two or more symptoms were classified as "cognitively impaired".

Our independent variables include race (non-Hispanic Blacks=1, non-Hispanic Whites=0), childhood health and socioeconomic status (SES) as well as adulthood health, lifestyles, and SES. *Childhood SES* includes four items: father's education, mother's education, father's occupation, and subjective assessment of family's financial situation from birth to age 16 (i.e., whether the family was financially pretty well-off, about average, or poor). *Childhood health* is based on the respondent's rating of his or her health as a child on a 5-point scale ranging from poor to excellent. Previous research has shown that the retrospective measure of overall childhood health was valid and had good reliability over time (Haas, 2007). Four blocks of variables are created to evaluate the direct and indirect effects of early-life environment on racial gaps in late-life cognitive impairment. *Adult SES* is measured using education and the respondent's longest-held occupation in 1998. *Adult health conditions and health behaviors* include body mass index, the number of chronic diseases (heart disease, hypertension, diabetes, stroke, and high cholesterol), smoking history, and exercise. *Adult social support* includes marital status and number of children. These are time-varying measures. We also include demographic variables such as age and gender.

Methods

We begin by looking at racial differences in the prevalence of cognitive impairment at baseline (i.e., 1998). This reveals racial differences in the burden of cognitive impairment among persons who survived to age 65 and above. We then examine the relationship between early life conditions and racial gaps in cognitive impairment. Five logistic regression models will be estimated. The baseline model will include age, gender and race. Model 2 will add early life conditions and estimate whether the coefficient for Blacks is reduced by adding these variables. Model 3 will add

adulthood SES; Model 4 will additionally control for adulthood health and health behaviors, and Model 5 will control for social support. We hypothesize that early life conditions have both direct and indirect effects on the racial gaps in cognitive impairment and that adulthood conditions mediate much of the effects of early life conditions on racial gaps in cognitive impairment at baseline.

Next, we will estimate a series of nested multistate models in discrete-time to examine whether and by what mechanisms early life conditions contribute to racial differences in the onset of cognitive impairment across the twelve year period. Specifically, person-interval (i.e., two-year interval) record files are created from 1998 to 2010 and multinomial logit model is used for the discrete-time event history analysis.

We will first estimate a model that includes age, gender and race only. Then the full set of early-life characteristics and adult SES, health, and health behaviors are added *sequentially*. 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 racial gaps in cognitive impairment (Warner & Hayward, 2006). We estimate all models using SAS. All models are based on weighted data using 1998 HRS sampling weights.

Preliminary Results

Our preliminary results showed that older Blacks were about three times more likely to suffer from cognitive impairment than Whites in 1998, controlling for age and gender. Childhood health, mother's education, father's education, and father's occupation were strongly associated with odds of cognitive impairment, and they played an important role in accounting for racial disparities in cognitive impairment in late life. We expect that racial differences in adulthood SES and health will further explain racial differences in cognitive impairment.

We have just started to examine racial differences in the onset of cognitive impairment from 1998 to 2010. Preliminary analysis showed that Blacks are more likely to experience the onset of cognitive impairment than Whites, controlling for age and gender, and that childhood conditions continued to play a role in explaining Blacks' higher risk of cognitive impairment during the 12-year follow-up.