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A Longitudinal Study of Black-White Disparities in Cognitive Aging

Author
Byrd, DeAnnah

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A Longitudinal Study of Black-White Disparities in Cognitive Aging

A dissertation submitted in partial satisfaction of the requirements for the Degree of Philosophy in Public Health

by

DeAnnah R. Byrd

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ABSTRACT OF THE DISSERTATION

A Longitudinal Study of Black-White Disparities in Cognitive Aging

by

DeAnnah R. Byrd

Doctor of Philosophy in Public Health

University of California, Los Angeles, 2017

Professor Gilbert Chee-Leung Gee, Chair

PURPOSE: As the U.S. population of older adults continues to grow, age-related cognitive impairment and dementia will become a greater concern for public health since both increase with age. Studies have found that blacks are disproportionately affected by these conditions (Zsembik and Peek 2001, Schwartz, Glass et al. 2004, Mehta, Stewart et al. 2009, Potter, Plassman et al. 2009, Masel, Raji et al. 2010). Education, stressful life events and experiences of discrimination may account for, or modify, some of these differences by race in cognitive impairment over time. Additionally, religion and spirituality may protect against cognitive impairment in old age. The current study seeks to better understand black and white differences in changes in cognitive impairment by examining education, stressful life events, discrimination, religion and spirituality as both direct and moderating factors.

METHODS: Analyses focused on black and white adults enrolled in all 5 waves (years 1986-2011) of the Americans' Changing Lives (ACL) study who were age 25 and older at baseline (N =3,617); additionally, a subsample of adults was analyzed in waves 4 and 5. The outcome of cognitive impairment (or number of cognitive errors) was assessed at each wave using a shortened version of the Short Portable Mental Status Questionnaire (SPMSQ). A 9-item Recent Life Events Index was used to measure stressful life events and a 5-item adaptation of the Williams Everyday Discrimination Scale was used to measure discrimination. Mixed-effects models were used to examine the longitudinal relationship between race and cognitive impairment changes. OLS regression was used to model the relationships between education, stressful life events, discrimination, religion, spirituality and cognitive impairment while controlling for demographic characteristics such as age, gender, income and marital status as well as for risk factors known to influence cognition, including depressive symptoms, chronic health conditions and smoking status.

RESULTS: The data showed that racial disparities in cognitive impairment existed at baseline such that blacks had higher cognitive impairment scores than whites. Second, these disparities widened over time (worsen with age) whereby blacks experience a more rapid cognitive decline than whites. Third, education alone, rather than the combined effect of education, recent stressful life events and discrimination, explained some of the race disparity. Fourth, religion and spirituality did not have a protective effect against cognitive impairment, such that those reporting high levels of religion and spirituality do not have lower levels of cognitive impairment versus those with low levels of religion and spirituality. Further, religion and spirituality also did not modify the race-cognition association.

CONCLUSIONS: Study findings demonstrated that blacks have more cognitive impairment than whites at baseline and these disparities worsened with age, even after adjusting for sociodemographic and other health-related factors. This finding highlights that racial disparities begin earlier than previously identified. This study suggests that interventions designed to address cognitive decline should be implemented at younger ages. Moreover, education rather than stressful life events and discrimination accounted for a significant amount of racial disparities but did not fully explain black-white differences. Thus, the types of interventions developed should consider the underlying educational differences between black and white adults.

The dissertation of DeAnnah R. Byrd is approved.

Steven P. Wallace

Chandra L. Ford

Megan McDonnell Sweeney

Gilbert Chee-Leung Gee, Committee Chair

University of California, Los Angeles

2017

DEDICATION PAGE

I dedicate this dissertation to my mother, Anita K. Byrd, who along with Almighty God guided and supported me every step of the way. I truly would not have been able to complete this journey without her unwavering support, love and encouragement. Most of all, I want to acknowledge the tremendous patience and guidance she provided me along this journey. At times, I wanted to give up but she would not allow me to do so and would often say, "quitting is not an option!" Thus, I stayed the course through the darkest of days and the loneliest and longest nights. With my family all the way across the country on the East Coast and none in Los Angeles, my mother always took my calls. What she did not realize was that her gentle, loving and kind voice allowed me to put one foot in front of the other and keep pushing. For a year, I had to write sitting on the side of my bed and she called nearly every single day to check on me. She knew I was frustrated, stressed out and most of all overly tired. And at one point she worked two jobs to support me and my other siblings, so it is with tremendous gratitude, humility and grace that I dedicate this work to Momma Byrd. I am also most proud to be the first in my family to earn a Doctor of Philosophy (PhD) and awarded the coveted title of Dr. DeAnnah Roshae Byrd.

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I would like to acknowledge my three siblings, Adrian, Corette, and Brandon Byrd for their loving support over the years. I remember our joyous times growing up together on a 3,500-acre cattle ranch in Whitmore, California and calling ourselves “the four ninja turtles.” It was those memories that help carry me through. I also want to acknowledge the amazing and wonderful Mr. Joe, who not only supported me financially but also made me laugh—a lot—along the way! In addition, I want to thank my surrogate Auntie, Mary P. Thomas, who spent hours editing my final dissertation with me as she literally went page by page and my Uncle Morgan who allowed me to steal her away. Moreover, I want to thank my mother’s work colleagues, Wanda Bryant and Tanya Keawe, for their assistance and support. Last, but surely not least I want to acknowledge my Aunt Eva Mae, who is the Matriarch of our family and a tireless prayer warrior. Had it not been for her and others praying for me, I certainly would not have come through this journey. Thus, I am truly blessed to have this level of support, love, and guidance.

VITA

2005-2007 Population Health Fellowship
School of Medicine and Public Health, University of Wisconsin, Madison, WI

2005 M.S. Population Health Sciences
Department of Population Health Sciences, School of Medicine and Public Health University of Wisconsin, Madison, WI

2002 B.S. Biology
Department of Biology, College of Agricultural and Life Sciences
University of Wisconsin, Madison, WI

PEER-REVIEWED PUBLICATIONS

Byrd, DR & McKinney, KJ. Individual, Interpersonal, and Institutional Level Factors Associated with the Mental Health of College Students. Journal of American College Health 2012; 60(3):185-193.

Byrd, DR. Race/Ethnicity and Self-Reported Levels of Discrimination and Psychological Distress, California, 2005. Preventing Chronic Disease 2012, 9:120042.

Byrd, DR, Katcher, ML, Peppard, P, Durkin, M, & Remington, PL. Infant Mortality: Explaining Black/White Disparities in Wisconsin. Maternal Child Health Journal 2007; 11(4):319-326.

ISSUE BRIEFS & OTHER PUBLICATIONS

Byrd, DR, Remington, PL, & Katcher, ML. Reducing Black Infant Mortality in Wisconsin: Best Practices and Model Programs. Issue Brief 2005; 6(3).

Byrd, DR, Friedsam, D, & Remington, PL. Healthiest Wisconsin 2010 and Health Disparities: How Do the State's Goals Relate to Wisconsin's Minority Populations? Issue Brief 2005; 6(4).

PROFESSIONAL PRESENTATIONS

Byrd, DR. 2017. "Examining Changes in Cognitive Impairment Symptoms among Older U.S. Black and White Adults" The IAGG World Congress of Gerontology & Geriatrics, Education, Race/Ethnicity, and Cognition Symposium, San Francisco, CA.

Byrd, DR. 2017. "Does Discrimination Play a Role in Racial Disparities in Cognitive Ability among Older U.S. Adults?" The IAGG World Congress of Gerontology & Geriatrics, Discrimination and Health Across the Life Course Symposium, San Francisco, CA.

Byrd, DR. 2015. “Black-White Changes in Cognitive Impairment among Older U.S. Adults” The Gerontological Society of America, Cognitive, Physical, and Psychological Health with Age: 25 years of the Americans’ Changing Lives Study Symposium, Orlando, FL.

Byrd, DR. 2015. “Examining Racial Disparities in Cognitive Ability over Time among Older U.S. Adults: Does Stress Play a Role?” American Public Health Association, Chicago, IL (poster).

Byrd, DR. 2010. “Psychological Distress and Racial Discrimination.” American Public Health Association, Denver, CO.

Byrd, DR. 2009. “Addressing Psychological Distress on College Campuses.” American Public Health Association, Philadelphia, PA (poster).

Byrd, DR. 2009. “Addressing Depression on College Campuses.” American Public Health Association, Philadelphia, PA (poster).

Byrd, DR. 2008. “Campus Life and Overall Wellness: An Online Survey.” American Public Health Association, San Diego, CA.

Byrd, DR. 2008. “Campus Life and Mental Health.” American Public Health Association, San Diego, CA (poster).

Byrd, DR. 2007. “Addressing Cancer Disparities via Navigation Outreach.” Bridging Health the Care Divide: Research and Programs to Eliminate Cancer Disparities, New Orleans, LA (poster).

Byrd, DR. 2006. “Addressing Cancer Disparities via Navigation Outreach.” Wisconsin Public Health Association, Wisconsin Dells, WI.

Byrd, DR. 2006. “Addressing Cancer Disparities via Navigation Outreach.” 5th Annual Cancer Survivorship Conference, Bethesda, MD (abstract).

Byrd, DR. 2005 “Infant Mortality: Black/White Disparities in Wisconsin.” American Public Health Association, Philadelphia, PA.

Byrd, DR. 2005. “Screening of Pregnant Women in the Emergency Department.” Medical Society of Milwaukee County, Milwaukee, WI.

Byrd, DR. 2005 “Infant Mortality: Black/White Disparities in Wisconsin.” 8th Annual Public Health Nursing (PHN) Conference. Understanding the Effect of Racism on Health: A Social Justice Issue for PHN Practice, Stevens Point, WI.

Byrd, DR. 2005 “Infant Mortality: Black/White Disparities in Wisconsin.” State Medical Society, Executive Committee of Public Health Council, Madison, WI.

CHAPTER 1. BACKGROUND

I. INTRODUCTION

Impaired cognitive functioning in older age is a serious public health concern, given the rapidly expanding population of older adults in the United States (U.S.). There are also clear racial differences in cognitive impairment (Zsembik and Peek 2001, Schwartz, Glass et al. 2004, Mehta, Stewart et al. 2009, Potter, Plassman et al. 2009, Masel, Raji et al. 2010, Rexroth, Tennstedt et al. 2013, Diaz-Venegas, Downer et al. 2016). Blacks have worse cognitive functioning than whites (Tang, Cross et al. 2001, Sloan and Wang 2005, Castora-Binkley, Peronto et al. 2013) and these disparities worsen over time (Masel and Peek 2009, Lee, Richardson et al. 2012). However, these studies have used either small sample sizes or been conducted in special populations, e.g., elders with mild cognitive impairment (Lee, Richardson et al. 2012); followed participants for a limited length of time, e.g., 3-12 years (Castora-Binkley, Peronto et al. 2013); or focused only on middle ages or older adults (51+ at baseline) (Tang, Cross et al. 2001, Sloan and Wang 2005, Alley, Suthers et al. 2007, Masel and Peek 2009, Lee, Richardson et al. 2012). The current study provides new information regarding racial differences in cognitive impairment scores across the full adult life course by examining a nationally representative sample of black and white adults over a 25-year period from 1986-2011. This study will also expand existing knowledge about education, stressful life events, discrimination, religion, spirituality and cognitive impairment among middle and older aged adults (25+ at baseline). I propose the following aims:

Aim 1: Determine whether black-white differences in cognitive impairment exist and evaluate whether these disparities persist over time.

Aim 2: Determine whether educational attainment, stressful life events and

discrimination are associated with worse cognitive impairment and determine the extent to which these three factors simultaneously account for racial differences in cognitive changes over time

Aim 3: Explore whether religion and spirituality buffer against cognitive decline and assess whether these factors attenuate black-white disparities.

II. LITERATURE REVIEW

A. Cognitive Impairment: Overview

In the past century, the older adult population in the United States (U.S.) has rapidly increased in number thanks to advances in health care and public health and longer life expectancies. As the "Baby Boomer" generation (those born between 1946 and 1964) continues to age, the number and proportion of older adults in the U.S. will further increase. The older population is not only expected to increase dramatically growing from 35 million to 72 million, but by 2030, it is projected that one in five people will be ages 65 or older (Federal Interagency Forum on Aging Related Statistics 2012). Moreover, the older population will become more racially and ethnically diverse than it is today. By 2030, the elderly minority population will have nearly doubled its current size to 20.2 million and Hispanics (projected to reach 8 million) will have surpassed blacks (projected to reach 7.5 million) as the largest minority group among older Americans (Pollard and Scommegna 2013). By 2050, it is projected that 58% of older adults will be non-Hispanic white, compared with 80% in 2010 (Federal Interagency Forum on Aging Related Statistics 2012). As the older U.S. population continues to grow and become more racially diverse, age-related cognitive impairment and dementia, including Alzheimer's disease and other neurodegenerative diseases, may burden our health care system and is therefore a growing public health concern (National Academies of Sciences 2017).

According to the fifth edition of the Diagnostic and Statistical Manual of Mental

Disorders (DSM-5), age-related cognitive impairment is categorized as a neurodegenerative disorder or as a cognitive disorder (Ganguli, Blacker et al. 2011). This category includes "disorders where the primary clinical deficit is in cognitive functioning and the cognitive decline is acquired rather than developmental, i.e., impaired cognition is not present at birth or in early life but appears later in life and represents a decline from a previously attained level of functioning" (Ganguli, Blacker et al. 2011)(p.2). More generally, cognitive impairment has been defined as the loss of ability to learn, process and remember information (Barnes, Lewis et al. 2012) because its symptoms can range from mild to severe, depending on the level or amount of one's impairment. For example, mild cognitive impairment (MCI) is typically the impairment that exists in the absence of a formal diagnosis of dementia; and it is often characterized as MCI or "cognitive impairment, no dementia" (Potter, Plassman et al. 2009). Mild cognitive impairment is further conceptualized as a transitional state between aging-related cognitive changes and dementia (Morris, Storandt et al. 2001, Petersen, Doody et al. 2001, Subramanyam and Singh 2016). Hence, individuals with mild cognitive impairment typically experience slight changes in cognitive functioning (e.g., problems with memory or changes in learning or attention) that are not serious enough to disrupt their daily lives and they are therefore able to continue functioning independently (National Academies of Sciences 2017). On the other hand, severe cognitive impairment is serious enough to reduce a person's ability to perform everyday activities and these individuals are seen as functioning non-independently and are usually diagnosed as having dementia (National Academies of Sciences 2017). As such, severe cognitive impairment is the basis or condition for diagnosing any form of dementia, including Alzheimer's (Manly and Mayeux 2004). Although cognitive impairment is commonly referred

to as “dementia”, this is not accurate since the clinical criteria for dementia encompasses cognitive impairment so severe it interferes with a person’s ability to perform everyday activities (Manly and Mayeux 2004). There is a high prevalence of mild cognitive impairment, with approximately 15 to 20% of people ages 65 or older classified as such (Roberts and Knopman 2013). Given these high estimates of mild cognitive impairment among older adults, this study focuses on mild forms of cognitive impairment rather than severe cases.

One of the hallmarks of cognitive impairment is memory loss. Cognitive impairment, especially those involving memory problems, can also be a risk factor for the development of Alzheimer’s and other dementias (Kantarci, Weigand et al. 2009, Mitchell and Shiri-Feshki 2009, Alzheimer's Association 2017). In fact, a systematic review of 32 studies report that, on average, 32% of individuals with MCI develop Alzheimer’s dementia within 5 years (Ward, Tardiff et al. 2013). Another meta-analysis of 41 studies puts these estimates at 38% for individuals who were also followed for 5 years or longer (Mitchell and Shiri-Feshki 2009). Estimates from each U.S. state and the District of Columbia (D.C.) reveal that between 2010 and 2025, all states, except DC, are expected to experience double-digit to triple-digit percentage increase in the prevalence of Alzheimer’s dementia among those 65 and older (range, 19% [Pennsylvania] to 116% [Alaska] (Weuve, Hebert et al. 2015). By 2025 older adults with Alzheimer’s will comprise a larger proportion of each state’s population than they did in 2010, with most of the population growth occurring in the oldest age groups (Weuve, Hebert et al. 2015). Although cognitive impairment and dementia, including Alzheimer’s, is not a normal part of aging, age is clearly the biggest risk factor. The percentage of people with Alzheimer’s dementia increases dramatically with age: 3% of people age 65-74, 17% of people age 75-84, and 32% of people age 85 or older have Alzheimer’s dementia (Hebert, Weuve et al. 2013).

Additionally, the number of people age 85 and over is projected to nearly quadruple to 19 million by 2050 (Federal Interagency Forum on Aging Related Statistics 2012). Given these growing estimates of Alzheimer's and the increase in the number of individuals 85 and older, these statistics have broad implications for cognitive impairment symptoms at earlier ages and for racial/ethnic minorities who tend to have a higher incidence and prevalence of both cognitive impairment and dementia.

Racial/ethnic and socioeconomic disparities in cognitive impairment and dementia have been widely documented and these disparities change with age (Mehta, Simonsick et al. 2004, Sloan and Wang 2005, Potter, Plassman et al. 2009, Sheffield and Peek 2011, Rexroth, Tennstedt et al. 2013, Diaz-Venegas, Downer et al. 2016). Overall evidence indicates that blacks have higher rates of cognitive impairment and dementia compared with whites (Potter, Plassman et al. 2009, Rexroth, Tennstedt et al. 2013, Diaz-Venegas, Downer et al. 2016). In particular, estimates from the 2006 Health and Retirement Study (HRS) show that blacks are approximately 2-4 times more likely than whites to have cognitive impairment, with greater differences among younger age groups (Lines and Wiener 2014). Blacks, for example, are four times more likely than whites to have cognitive impairment among people aged 55–64, but among people aged 85 and older, blacks are only about two times more likely than whites to have cognitive impairment (Alzheimer's Association 2011, Lines and Wiener 2014). Alzheimer's disease is also more prevalent among blacks than whites —with reported estimates ranging from 14% to 500% higher for blacks (Froehlich, Bogardus et al. 2001, Lines and Wiener 2014). Specifically, for Alzheimer's disease and other dementias, blacks ages 65-74 have a prevalence rate of 9.1% compared to only 2.9% for whites. Likewise, blacks ages 75-84 have a 19.9% prevalence rate

compared to 10.9% for whites and blacks 85 and older have a prevalence rate of 58.5% versus 30.2% for whites (Gurland, Wilder et al. 1999, Lines and Wiener 2014). Blacks 90 and older also have twice the incidence of Alzheimer's disease compared to whites (standardized incidence rate for blacks = 4.2% versus 1.9% for whites per person-year). Probable or possible Alzheimer's disease also occurs significantly more frequently among blacks (10.5%) than in whites (5.4%) (Tang, Cross et al. 2001). So, although there are more non-Hispanic whites living with Alzheimer's and other dementias in the U.S. compared to any other racial or ethnic group, older blacks are the hardest hit by dementia given that they are more likely than older whites to develop cognitive impairment, Alzheimer's disease and other dementias (Manly and Mayeux 2004).

Although there are clear racial differences in cognitive impairment and dementia and these disparities vary by age, the literature is inconsistent whether these disparities persist over time and whether blacks decline at a faster rate as they age. For instance, some studies have found that blacks experience a more rapid rate of cognitive decline (Sachs-Ericsson and Blazer 2005, Sawyer, Sachs-Ericsson et al. 2008), others have found that whites experience a more rapid rate of cognitive decline (Sloan and Wang 2005, Alley, Suthers et al. 2007, Karlamangla, Miller-Martinez et al. 2009, Early, Widaman et al. 2013, Wilson, Capuano et al. 2015) some have also found no difference (Atkinson, Cesari et al. 2005, Castora-Binkley, Peronto et al. 2013, Marsiske, Dzierzewski et al. 2013) and others present mixed results within the same study (Masel and Peek 2009, Wolinsky, Bentler et al. 2011).

Yet, it is unclear what factors contribute to these inconsistent findings. One issue is the measure of cognitive domain varies across studies (a global measure vs. specific domains). Previous studies have assessed cognition with brief global measures such as the Short Portable

Mental Status Questionnaire (SPMSQ) (Sachs-Ericsson and Blazer 2005, Sawyer, Sachs-Ericsson et al. 2008), Telephone Interview for Cognitive Status (TICS) (Sloan and Wang 2005, Alley, Suthers et al. 2007, Karlamangla, Miller-Martinez et al. 2009, Masel and Peek 2009, Wolinsky, Bentler et al. 2011), and Mini-Mental State Examination (MMSE) (Atkinson, Cesari et al. 2005), which vary in their measurement precision and ability to characterize specific domains of cognitive function since global measures tend to represent fewer cognitive abilities. Other studies have assessed specific domains of cognition such as Wilson and colleagues (2015) who evaluated five cognitive domains, including episodic memory, semantic memory, working memory, perceptual speed, and visuospatial ability and Early et al. (2013) who measured episodic memory, semantic memory, and executive functioning.

Second, several of these studies have used either small sample sizes (Early et al. 2013 – 116 black and 184 white; Wilson et al. 2015 – 647 black and 647 white) or been conducted in special populations, e.g., Medicare beneficiaries (Wolinsky, Bentler et al. 2011), elders with mild cognitive impairment (Lee, Richardson et al. 2012) or those clinically diagnosed with dementia as compared to those with normal cognition (Wilson, Aggarwal et al. 2010).

Third, these studies have followed participants for a limited length of time, e.g., 3-12 years (Castora-Binkley, Peronto et al. 2013) or have primarily focused on adults 51 and older at baseline (Masel and Peek 2009). Finally, some studies use different analyses within the same study and as a result report opposing findings, which further contributes to the inconsistencies. For example, Masel and Peek (2009) using data from the Health and Retirement Study included an entry age of 51 for participants and found strong racial differences in baseline scores of cognitive status. Cognitive status was assessed using two items from the TICS, one representing

"mental status" and the other representing "memory" using word recall items. However, they found mixed results when they examined rates of change in cognitive status as measured by these two separate indices of memory and mental status, such that black adults had significantly faster rates of memory decline than white adults, but there was no differences between blacks and whites and the rate of decline in mental status over time. When the authors examined the odds of a decline in memory and mental status score as greater than one standard deviation from the initial evaluation of cognition in 1996 to the last evaluation in 2004, they found no differences between blacks and whites for either measure. Yet, the authors do not discuss the implications of using two different methods (multilevel mixed models vs. multinomial logistic regression) to validate the findings. But do conclude "on the whole, the results may indicate that beginning in late middle age, disparities over time in cognitive scores are present, and change over time varies little by race/ethnicity (p.6)." Thus, depending on the analyses, race is not consistently associated with differences in rates of cognitive change. Their finding of a slightly faster rate of memory score decline in black respondents compared to white respondents, nonetheless, supports the idea that blacks have more rapid decline than whites and these disparities persist over time.

Likewise, Wolinsky et al. (2011) linked Medicare claims data to the baseline and final follow-up surveys from the Assets and Health Dynamics among the Oldest Old (AHEAD) study. They also found robust cross-sectional differences, such that blacks had lower cognitive-test scores at baseline (and final follow-up values) compared to whites. Yet, they report varying findings when using different analyses. In their first analyses of simple change scores (i.e., crude cognitive changes), whites showed greater declines than blacks on the immediate and delayed word recall tests, but in their second analyses in which the outcome was the last assessment

scores and the first assessment scores (taken in 1993-1994) were entered as a covariate in a residual change score multivariable linear regression model, blacks had greater cognitive decline than whites on all three cognitive outcomes (as measured by TICS-7, immediate and delayed word recall tests). Their finding that whites had better outcomes in the regression analyses further supports the idea that blacks decline more rapidly than whites and these differences remain over time.

Collectively, these studies show robust cross-sectional or baseline differences, such that blacks have lower cognitive-test scores than whites (Sloan and Wang 2005, Alley, Suthers et al. 2007, Karlamangla, Miller-Martinez et al. 2009, Masel and Peek 2009, Wolinsky, Bentler et al. 2011, Wilson, Capuano et al. 2015). On the other hand, these studies show inconsistent results in terms of which groups are at the greatest disadvantage when looking at longitudinal changes in cognition. Blacks declined more over time in some studies (and analyses), while whites declined more in others (see Appendix 1.1 for a table summarizing these studies, including the ages and measured used). Hence, cognitive decline during old age is not well understood, nor is it clear when racial inequalities in changes in cognitive functioning begin to appear in the life course (Early, Widaman et al. 2013). These inconsistencies may be due to the fact that most of these studies conducted secondary analyses of the same parent study, e.g., AHEAD data, but do not consistently report similar findings (Sloan and Wang 2005, Alley, Suthers et al. 2007, Karlamangla, Miller-Martinez et al. 2009, Wolinsky, Bentler et al. 2011). Additionally, none of these studies have included younger aged adults. Instead, most have focused on middle age and older adults at baseline (e.g., youngest age was 51 and above) (Masel and Peek 2009). Thus, studies clarifying the effects of race on cognitive trajectories among younger aged adults are

necessary to reconcile these inconsistencies because we see racial disparities change with age. Also, studies examining risk factors as potential explanations for racial differences in the incidence, prevalence, baseline levels and rate of change in cognitive functioning are also needed given the inconsistencies in the current literature. Although all of these studies control for years of education and various chronic conditions, additional work is necessary to understand the social factors contributing to these disparities.

Most studies that have documented racial differences in the incidence and prevalence of cognitive impairment and dementia have attributed these findings to well-established biological factors (such as age, gender, Apolipoprotein E (APOE) genotype and vascular conditions) as well as differences in social factors (education, SES and cultural differences) (Zsembik and Peek 2001, Mehta, Stewart et al. 2009, Potter, Plassman et al. 2009, Tschanz, Pfister et al. 2013). For example, a population-based study using data from the Cardiovascular Health and Cognition Study found that African Americans had a 4.4 times greater risk of mild cognitive impairment compared to whites, but the authors argued that most of this disparity could be explained by racial and ethnic differences in two biological factors, APOE genotype and cardiovascular health (Lopez, Jagust et al. 2003). Yet, these assertions about biological determinants were made without accounting for important social factors that differ between races, such as education.

Cultural differences have also been examined using data from the Aging, Demographics, and Memory Study. Potter and colleagues (2009) compared direct tests of cognitive functioning and found that blacks and whites differed in their perception and reporting of their older family member's cognitive changes. Whites' reported that their older family members were having cognitive difficulty in the past two years, and these reports were tied to higher odds of developing both mild cognitive impairment and dementia. However, blacks' reports of older

family members' cognitive difficulty was only linked to an increased odds of developing dementia, and not cognitive impairment. These results suggest that family members' reports of early cognitive changes may be influenced by cultural differences such that whites and blacks differ in their perception and reporting of mild cognitive impairment symptoms. These cultural differences in reporting may then lead to racial differences in the odds of developing cognitive impairment and subsequent dementia. However, more research is necessary to determine the influence of other social factors beyond cultural differences. Another potential social factor is the differences in education level.

B. Risk Factors and Cognitive Impairment

1. Education

Education may play a key role in explaining racial disparities in cognitive functioning. Education is a primary social correlate of cognitive ability (Zsembik and Peek 2001, Masel, Raji et al. 2010). Cognitive reserve refers to the efficient utilization of the brain network or "brain reserve" (i.e., structures that are related to learning and memory), which maximizes and optimizes normal performance (Stern 2002, Perneczky, Alexopoulos et al. 2011). Cognitive-reserve theorists argue that early-education has a direct effect on cognitive functioning by giving individuals the necessary cognitive resources or "cognitive reserve" to stave off later life cognitive decline (Mehta, Stewart et al. 2009). Brain reserve refers to an individual's brain structures, including the key neurons in the brain that are related to learning and memory (Mehta, Stewart et al. 2009, p.381). In contrast, cognitive reserve is developed early in life and can be shaped over time by education, life experiences and other social influences. For example, educational performance, mental ability or IQ, and linguistic ability are all related to or are

markers for cognitive reserve in early life. Thus, an individual's cognitive reserve can be influenced by education, including the quality, language skills and other early life experiences; such that, "those with a greater reserve may be able to function longer without reaching a threshold of cognitive impairment, compared to people with a lower reserve" (Mehta, Stewart et al. 2009, p.381). This may be why "a common metaphor used for brain reserve is that it is akin to computer hardware, while cognitive reserve is comparable to software, because it is developed over the life course" (Mehta, Stewart et al. 2009, p.381). As such, efforts to increase cognitive reserve early in life and throughout the life course should include increasing access to educational opportunities, as this may in fact stave off dementia in later life.

Indeed, research has shown that the quality of one's education has been linked to Alzheimer's and cognitive impairment. For example, (Mehta, Stewart et al. 2009) examined education quality as a correlate of Alzheimer's among multiple racial groups using data from the Aging, Demographics, and Memory Study (a complementary study of the Health and Retirement Study). They found that self-reported "below average" school performance was associated with a four fold increased odds of developing Alzheimer's disease among whites, blacks, and Hispanics, after accounting for the known effects of formal education and literacy. In additional analyses, the authors further found that self-reported "below average" school performance was associated with an increased odds of cognitive impairment; although they note that these analyses had limited power. The authors argue that as aging adults begin to suffer mental decline they may draw on their (educational) cognitive reserve to prevent the onset of Alzheimer's disease. "Not only is 'staying in school' important," they write, "but increasing school performance is equally important to stave off the potential long-reaching effects of low education quality" … and may offer the most promise as a "possible protective mechanism to

stave off the onset of Alzheimer's (p.386)." Thus, the quality of education may be a better measure of cognitive reserve than the level of education alone (Manly, Jacobs et al. 1999). Others scholars suggest that education may improve cognitive function in a variety of other ways as well. Alley and colleagues (2007) argue, "education may increase cultural competency, improve reading, math, and reasoning skills, as well as test-taking ability. At the same time, education may actually improve brain function by increasing the number of synapses" (p.74).

Although education may improve cognitive functioning via cognitive reserve and a variety of other ways, there are stark racial differences in reading ability and the quality of one's education. In grades 4, 8, and 12, blacks have considerably lower reading levels than whites (Aud, Fox et al. 2010). In 2007, more than half of black 4th (54%) and nearly half of black 8th (45%) graders scored below the basic reading achievement level compared with 22% of white 4th graders and 16% of white 8th graders (Aud, Fox et al. 2010) and these differences may be due to the quality of education/schooling. In comparison to whites, blacks have attended schools with very limited resources such as a shorter length of school terms, different quality of teachers, and poorer access to quality books and other resources, which result in a poorer quality education (Williams 1999). This poorer quality of education may then underlie racial differences in cognitive test performance among older black and white adults.

Indeed studies have looked at the quality of education as a means to account for the black-white differences in cognitive test performance. However, these studies first assessed the quantity of one's education when evaluating racial differences. Pedraza and colleagues (2012), for example, examined whether adjustments for age and years of education attenuated the differences in MMSE scores between older Caucasian and African American adults, ages 60 and

above. They found that African Americans had significantly lower unadjusted MMSE scores (23.0 ± 7.4) than Caucasians (25.3 ± 5.4). This disparity persisted despite adjustment of MMSE scores for age and years of education using established regression weights or newly derived weights. Given that the quantity of education did not eliminate the racial disparities in MMSE scores, the authors then adjusted the scores for age and quality of education, based on a measure of word reading from the Wide Range Achievement Test (WRAT-3). After controlling for dementia severity at baseline, age and the quality of education, the significant differences in MMSE scores between cognitively normal Caucasians and African Americans were attenuated but not eliminated. The authors emphasize that their findings "underscore the importance of the quality, and not just the quantity, of one's educational experience in shaping overall cognitive ability, particularly for African Americans" (p.331).

Similarly, Wood et al. (2006) compared standard MMSE scores to MMSE scores adjusted for age and level of educational attainment in a sample of 414 black (78% or 323) and white (22% or 91) elderly women, living independently in a community setting. Black participants scored significantly lower on the standard MMSE index than their white counterparts. However, even after scores were adjusted for age and years of education, this disparity remained, with blacks having lower mean scores than whites ($p = .003$), suggesting that racial differences may have an effect on MMSE performance independent of age and level of education and other factors such as the quality of one's education may play a role. Together, these studies suggest that the quantity of one's education must first be considered when evaluating the quality of education in explaining racial disparities, especially since disparities are further reduced but are still not completely eliminated when quality is taken into account above and beyond quantity.

Hence, additional research assessing the quantity of one's education is important given that higher educational attainment has consistently been linked to higher levels of cognitive performance in later life (Alley, Suthers et al. 2007, Masel and Peek 2009) and one's level of education also influences the scoring on existing measures of cognitive functioning, particularly for racial and ethnic minorities. For example, the Mini-Mental State Examination (MMSE) was derived from the Short Portable Mental Status Questionnaire (SPMSQ) and was originally designed to screen for cognitive status and dementia in a clinical setting (Folstein, Folstein et al. 1975). Currently, the MMSE is not only used as a sensitive indicator of dementia, but is also used extensively to screen for cognitive impairment and track changes in cognitive functioning over time in population and community-based research as well as neurological and even neurodegenerative research (Scuteri, Palmieri et al. 2005, Wood, Giuliano et al. 2006, Dahl, Berg et al. 2007). However, the MMSE's diagnostic utility tends to diminish when used to evaluate racial and ethnic minorities and individuals with varying levels of education. Moreover, the level of one's education has been shown to cause considerable bias in the MMSE's scores among older racial and ethnic minorities (Hawkins, Cromer et al. 2011, Matallana, de Santacruz et al. 2011, Pedraza, Clark et al. 2012).

For instance, scholars have looked at whether the quantity of education as well as age influences performance on the MMSE when assessing the difficulty of specific items and a standardized (common) method of administration among elderly blacks (Hawkins, Cromer et al. 2011). Hawkins and colleagues (2011), for example, examined the quantity of education on MMSE performance within an African American sample of older adults, ages 55-87 living independently in the community. MMSE scores were generated in two distinct ways for each

participant. In one, total MMSE score incorporates the serial 7's subtraction score. In the other, the total MMSE score incorporates "world" spelled backwards. They found that one's level of education was a primary source of variation on both MMSE scores, followed by age. These results indicate that the level of education and age should be taken into account when interpreting MMSE total scores (and perhaps somewhat more so when "world" rather than serial 7s is included), given that as age increased performance declined particularly for those with low levels of education.

Moreover, in an attempt to improve the accuracy of the MMSE as a screening tool for older blacks, studies have examined this issue of education quantity bias by assessing the difficulty level of specific items and adapting existing measures. Specifically, these studies have adapted existing measures by adjusting or reducing the cut-off scores, withdrawing items from the measure or using a different test when studying minority populations (Parker and Philp 2004). For example, Hawkins et al. (2011) compared the difficulty of two items used in the scoring of the MMSE: the serial 7's subtraction task vs. spelling the word "world" backwards. They found in a cohort of older African Americans that the MMSE with the serial 7's examination was a significantly harder test than when "world" spelled backwards was incorporated into the MMSE total score. Regardless of the level of education the difficulty differential between the two tests was consistent; such that across all levels of education, the total score with "world" spelled backwards was much higher (28.13, SD 1.97) than the total score with the serial 7's subtraction task (26.77, SD 2.50). These findings suggest that how a neuropsychological test is administered to minority populations (and ultimately scored) should be reported, particularly with regard to the use of serial 7's subtraction vs. "world" spelled backward.

In addition, neuropsychological tests are limited in their ability to detect subtle cognitive changes among racial minorities, especially blacks. For instance, after Woods and colleagues (2006) adjusted scores for age and years of education, 14 participants (all black) were moved from categories of mild cognitive impairment to unimpaired cognitive ability. The authors conclude that "as milder states of cognitive impairment become an increasing focus of neurodegenerative research, these differences have significant implications, since differences in scores that are solely dependent on administration method may approach or even exceed the standard deviations reported for particular clinical groups in studies comparing ostensibly cognitively normal, mildly impaired (mild cognitive impairment), and dementia groups. Significant proportions of a sample could fall on either side of a diagnostic threshold (e.g., a MMSE score of 28 for "normal") simply due to this one variation in MMSE administration (p.650)." Hence, how blacks perform based on their education level and the difficulty of the items included on the MMSE and other cognitive tests may contribute to the racial variation/differences in scores. Indeed research suggests that elderly blacks routinely score lower on the MMSE compared to their white counterparts with similar levels of cognitive impairment; and these differences have primarily been attributed to lower levels of education among blacks (Wood et al. 2006).

As such, racial disparities may be a reflection of the differential administration and assessment of cognitive ability as well as the differences in educational attainment between blacks and whites. Thus, a key determinant of racial disparities in cognitive impairment may be the substantial inequalities in education between blacks and whites (Barnes, Wilson et al. 2011). Blacks, on average, have lower levels of educational attainment (Williams 1999, Barnes, Wilson

et al. 2011). Although the gap in high school dropout and graduation rates for blacks and whites has narrowed, gaps in educational attainment, college enrollment and graduation remain (Mare 1995, Aud, Fox et al. 2010, Ryan and Siebens 2012). According to the 2009 American Community Survey (ACS), blacks are less likely than whites to have completed a college or graduate school degree, including a bachelor's, master's, doctorate or professional degree (Ryan and Siebens 2012).

In summary, considerable racial differences exist with regards to educational quality, attainment, and performance on cognitive measures. This highlights the complex influence of education quality and level of education on cognition and racial disparities as well as the limited ability of neuropsychological tests in detecting subtle cognitive decline among racial minorities. Although other scholars underscore the importance of education quality this study focuses on educational attainment while acknowledging that quality may still play a role in cognitive disparities. Thus, educational attainment may help to explain racial differences in cognitive impairment, but education may not be the only explanatory factor. Other important factors may play a role, such as stress.

2. Stress

Stress can have a profound impact on one's age-related physical and cognitive functioning via its ability to alter the structure of the brain itself, in particular the hippocampus, a region of the brain responsible for learning and the formation and storage of memories (Lupien, Maheu et al. 2007). Stress or "stressors" are defined as any environmental (entering school, starting a job), social (interpersonal interactions) or internal (emotional reactions) demand that requires an individual to change or readjust his or her routine pattern of behavior (Holmes and Rahe 1967). This 'readjusting' in one's usual life pattern, as a result of the changing demands of

the environment, induces a stress response and activation of the hypothalamic-pituitary-adrenal (HPA) axis. While activation of the HPA axis can be regarded as a basic adaptation in response to stress and major life changes, prolonged activation of this system is harmful to an individual's health (Lupien et al 2007, p.211). In particular, stress scholars argue that activation of the hypothalamic-pituitary-adrenal (HPA) axis signals the release of the major stress hormones, glucocorticoids (called corticosterone in animals, and cortisol in humans) and catecholamines (adrenaline and noradrenaline). HPA axis activation is the primary hormone-related physiological mechanism through which stress causes atrophy in the brain structures (Sapolsky 1986). Prolonged activation of the HPA axis further inhibits tissue growth and repair, suppresses immune functions, and causes cognitive impairment (see Lupien et al 2007, p.211; McEwen 1998).

For example, when an individual perceives a situation as stressful, a "stress reaction" occurs, the HPA axis is activated, and the body signals the release of the major stress hormones, glucocorticoids and catecholamines. In response to the release of these two stress hormones, the body activates a flight-or-fight response, which increases one's heart rate and blood pressure (Lupien et al 2007, p.211). However, prolonged stress exposure and highly elevated levels of glucocorticoid stress hormones cause the body to decrease glucose levels and increase inflammation, which then increases neuronal death in the hippocampus, a brain structure actively involved in learning, memory, and fear detection (McEwen 1999). The liposoluble nature of glucocorticoids allows them to cross the blood-brain barrier and bind to important brain receptors in regions, such as the hippocampus, amygdala, and frontal lobes. Thus, the

hippocampus is primarily changed by the glucocorticoids actions on the brain's receptors in this region of the mind (Lupien et al 2007, p.211).

Structural changes and disruptions in the hippocampus have been linked to age-related memory deficits (Yassa, Mattfeld et al. 2011). Early animal studies have shown that when middle-aged rats are exposed to high levels of glucocorticoids, they develop memory impairments and hippocampal atrophy similar to that seen in older-aged rats (Landfield, Waymire et al. 1978, Sapolsky 1986). Similar findings have been found in more recent work. For example, (Garrido, de Blas et al. 2012) found that rats exposed to high basal corticosterone also exhibited age-related damage in the hippocampus and prefrontal lobes of the brain. Chronic exposure to glucocorticoids such as corticosterone has also been shown to cause permanent damage to the hippocampus, by the loss of neurons (Sapolsky 1986). These findings suggest that prolonged exposure to the stress hormones not only have implications for age-related cognitive impairment, but that chronic stress exposure may also be another reason for racial differences in cognitive functioning. Thus, older adults and blacks may be particularly susceptible to the eroding effects of stress, especially stressful life events and discrimination as well as other social disadvantages such as poorer quality education.

For instance, Leng and his colleagues (2013) investigated the association between various measures of social stress and cognitive function in a middle- to older aged English sample using data from the European Prospective Investigation of Cancer (EPIC)-Norfolk prospective cohort study. Participants were followed prospectively for a 10.5-year period and cognition was measured using a shortened form of the Mini Mental State Examination (SF-MMSE). They found that subjective measures of social stress were significantly related to lower cognitive functioning, independent of socio-demographic factors, physical, and emotional health. This

effect of higher self-perceived (subjective) stress and lower cognitive functioning was restricted to those reporting the highest level of stress; whereby, participants who reported their life as being extremely stressful, were 1.9 times more likely to score worse on the SF-MMSE than those who reported no stress at all. However, this study used a single item to measure self-perceived stress and it focused primarily on older whites. Hence, multiple measures and/or items are needed to more comprehensively assess subjective experiences of stress and stressful life events, especially those that capture perceived stress level and experiences of discrimination among older blacks. The present study and the ACL used a multiple item index to assess the number of recent stressful life events and its impact on cognitive disparities.

3. Stressful Life Events

A growing body of research suggests that stressful life experiences linked to race can have adverse effects on the health of minority populations, particularly blacks (Barnes, Lewis et al. 2012). Yet, most studies of psychosocial stress and cognitive impairment have been conducted with older whites; thus, relatively little is known about the impact of psychosocial stressors on cognitive functioning in older blacks (Barnes, Lewis et al. 2012). This can be seen in several recent studies that have examined the impact of stressful life events on age-related cognition using the Mini Mental State Examination (MMSE) (a measure that overlaps greatly with the Short Portable Mental Status Questionnaire (SPMSQ) (Comijs, van den Kommer et al. 2011, Leng, Wainwright et al. 2013, Tschanz, Pfister et al. 2013) and other measures of cognitive performance, e.g., the Dementia Rating Scale (Rosnick, Small et al. 2007, Peavy, Salmon et al. 2009); whereby, most were conducted in predominately all white samples, focused on special populations (e.g., cognitively normal versus mildly impaired older adults) and/or failed to report race altogether. Although these findings can be seen in both U.S. based studies

as well as other countries such as Europe, Dutch and the Netherlands, their findings are conflicting in terms of the influence of stressful life events on cognitive aging (Comijs, van den Kommer et al. 2011, Leng, Wainwright et al. 2013).

For example, studies examining whether the association between the number of life events and the rate of cognitive decline is different for persons with normal cognition versus those with mildly impaired cognition yield inconsistent findings. Comijs and colleagues (2011) found that adults in the Netherlands (ages 55-85) with mild cognitive impairment improved on episodic memory tests (i.e., had higher retention scores) if they had experienced a greater number of aggregate life events over a 3-year time period. In contrast, Peavy et al. (2009) found that older U.S. adults (ages 65-97) with mild cognitive impairment and high stress ratings declined more rapidly over a 3-year time period than cognitively normal subjects on the memory subscale score and the total score from the Dementia Rating Scale.

Other longitudinal U.S. studies using cognitively normal participants and the MMSE have further found no link between aggregate stressful life events and cognitive decline; however, worse cognitive functioning and improved cognitive ability is also seen. For example, Tschanz (2013) found no overall association between indicators of stressful life events and cognitive decline in a population-based sample of non-demented older adults. On the other hand, another U.S. based cross-sectional study found that specific life events (e.g., having less money to live on over the past year and being a victim of a crime) were associated with worse cognitive function i.e., cognitive decline, while other life events (e.g., experiencing the injury or illness of a friend) were associated with improved cognitive ability in a cohort of older adults, ages 60–84 years. Surprisingly, having a friend get injured or become ill improved cognitive performance in this sample of older adults (Rosnick, Small et al. 2007).

Taken together, these studies indicate that stressful life events are associated with worse cognitive functioning; although no association or improved cognitive ability, as measured by the MMSE, the Dementia Rating Scale, three cognitive performance tasks (episodic memory, attention, and psychomotor speed tasks), and specific types of life events, is also seen (Rosnick, Small et al. 2007, Peavy, Salmon et al. 2009, Comijs, van den Kommer et al. 2011, Leng, Wainwright et al. 2013, Tschanz, Pfister et al. 2013). Although the research is not definitive, it is nonetheless plausible that stress contributes to cognitive impairment in older adults and may be even more salient for blacks. Blacks tend to experience a disproportionate burden of life stress compared to whites due, at least in part, to their relative disadvantaged position in U.S. society (Geronimus, Hicken et al. 2006, Paradies 2006); even though the above findings suggest that older whites are exposed to various life stressors; recent work has shown that there are clear racial differences in terms of the impact of stressful life events on cognition, particularly among blacks.

For instance, Sheffler (2013) examined the extent to which environmental factors, specifically stressful life events, influenced cognitive decline over a 3-year period and whether this relationship was stronger for blacks compared to whites. Using a community-dwelling sample of older adults from the Duke Established Populations for Epidemiologic Studies of the Elderly, she predicted that stressful life events would have a greater impact on the cognition of blacks versus whites; given that blacks are twice as likely as whites to develop dementia (Manly and Mayeux 2004) and stressful life events are more common among them (Barnes, Lewis et al. 2012). She found that blacks were more vulnerable to cognitive decline under low stress conditions compared to whites; however, at higher stress conditions blacks and whites had

similar rates of cognitive decline. Likewise, studies looking at the impact of chronic stress using allostatic load biomarkers have further found differences between blacks and whites. For example, Juster, McEwen, and Lupien (2010) examined health and cognition and found that in a low income neighborhood, the risk for having a higher allostatic load is 200% higher for blacks versus only 30% higher for whites. The current study will evaluate whether stressful life events have a more adverse affect on the cognition of blacks versus whites. Specifically, this study will explore whether stressful life events and discrimination modify the association between race and cognitive impairment among older black and white adults.

4. Discrimination

Scholars argue that discrimination is an important source of stress for racial and ethnic minorities compared to white Americans (Clark, Anderson et al. 1999, Williams 1999, Harrell, Burford et al. 2011). Specifically, discrimination and the stress that accompanies it has been conceptualized as a chronic, potent, and extremely negative experience that occurs across multiple contexts (i.e., individual, institutional, and cultural levels) and can be profoundly detrimental to racial minorities, particularly African Americans (Harrell 2000, Williams, Neighbors et al. 2003, Airhihenbuwa and Laveist 2006, Gee, Ro et al. 2009, Williams and Mohammed 2009).

Scholars further argue that blacks are disproportionately exposed to discrimination and other psychosocial stressors. For instance, (Geronimus 2000) emphasizes that African Americans are disproportionately exposed to harmful social stressors, such as highly segregated urban environments, intensified levels of poverty, and institutionalized discrimination. (James 1993) argues that "while it is certainly true that other U.S. racial and ethnic minorities have also suffered economic and social discrimination, few, if any, have faced these 'exposures' for as

long as have African Americans, nor have they faced them standing on an economic and cultural base that was systematically undermined by the larger society" (p.135). Profoundly, the impact and legacy of slavery and social and economic inequalities have not been erased from African American populations (Airhihenbuwa and Liburd 2006).

Furthermore, continued social disadvantage and discrimination may cause African Americans to be at greater risk for accumulated stress, which has serious implications for cognitive functioning in later life (Zsembik and Peek 2001, Geronimus, Hicken et al. 2006). Geronimus and her colleagues (2006) have proposed a "weathering hypothesis" for explaining why African Americans appear to be more vulnerable to health and cognitive deterioration in later life. They suggest that acute and chronic stressors are cumulative and lead to later life vulnerabilities and racial disparities in health and cognition. African Americans are thought to experience this "weathering" to a greater degree than other groups because of racial discrimination and other social disadvantages that have disproportionately affected blacks throughout history. In fact, blacks have also been found to have much higher mean scores on measures of allostatic load compared to whites (Geronimus, Hicken et al. 2006). Thus, older blacks may have experienced consistently higher stress throughout their lives, causing them to not only have higher allostatic loads, but also making them more vulnerable to discrimination and racial insults in later life. Hence, it is possible that because of the cumulative effects of life stressors and other social disadvantages, negative life events and experiences of discrimination may be associated with greater cognitive impairment in blacks.

There is evidence that experiences of discrimination negatively influence cognitive performance in both laboratory (or controlled) settings and in community-based cohorts of older

African Americans. For example, (Salvatore and Shelton 2007) presented fictional vignettes containing ambiguous and blatant cues of racial prejudice to black and white undergraduate students immediately before they were asked to complete the Stroop color naming task, a cognitive ability test. They found that subtle and blatant cues of racial prejudice were associated with poorer test performance for both groups. Their results suggest that there is an acute effect of perceived discrimination on cognitive functioning in a controlled setting, even when discrimination is not directed at the individual. This idea has been tested and termed stereotype threat by Steele and Aronson (1995) and is defined as an individual internalizing the negative stereotypes about one's group. This negative stereotype about one's racial group, in turn, causes a self-threat which then interferes with the intellectual functioning of the individual, particularly during standardized test taking. Indeed the authors found evidence supporting this hypothesis such that black college students underperformed in relation to whites in the presence of a racial stereotype regarding their intellectual ability (i.e., stereotype threat). Moreover, stereotype threat or self-threat has also been postulated as one possible mechanism by which self-reported school performance is associated with later dementia. For example, if one believes they are "not academic" enough they may subsequently avoid cognitively demanding tasks or at least those activities generally perceived to be demanding (Mehta, Stewart et al. 2009, p.381), which in turn limits their cognitive reserve and consequently their ability to stave off cognitive decline in old age.

Likewise, (Barnes, Lewis et al. 2012) found that higher levels of perceived discrimination were also related to poorer performance on cognitive tests among non-demented African Americans over 65. In particular, they found that participants who reported more instances of discrimination also had lower scores on both the global MMSE measure of cognitive functioning

as well as on specific measures of cognitive abilities, i.e., episodic memory (the hallmark of Alzheimer's disease) and perceptual speed (measured by four different tasks including a modified version of the Stroop Neuropsychological Screening test). Although, the magnitude of these associations remained unchanged after adjusting for demographic factors and vascular health conditions, they were no longer significant once depressive symptoms were taken into account. So it is key to evaluate whether discrimination is associated with worse cognitive outcomes after controlling for depressive symptoms.

Also, given that racism and discrimination makes the lives of blacks more stressful and thus leads to negative mental and physical health consequences (Krieger 1994, Clark, Anderson et al. 1999); it is equally important to examine the protective factors that buffer against the pernicious effects of stress and discrimination, which may lead to poorer cognitive outcomes among blacks. Religion and spirituality, for instance, maybe protective factors that blacks use to cope with stress and these factors may further buffer against later life cognitive decline; and ultimately account for racial disparities in cognitive impairment over time.

C. Protective Factors and Cognitive Impairment

1. Religion and Spirituality

Within a stress-coping framework, coping strategies are usually regarded as protective factors that buffer against the harmful effects of stress. Coping resources (such as a sense of control or mastery over life and self-esteem) and coping strategies (such as religious or spiritual coping) have been consistently shown to buffer the negative health effects of stress (Thoits 1995, Ryff, Friedman et al. 2012). In particular, when a situation is appraised as burdensome or exceeding one's ability to adapt, coping strategies are not only used to respond to this stressor,

but they are also used to manage the specific situational demands, including the negative emotions brought about because of it (Lazarus and Folkman 1984, Folkman 1997). Thus, coping strategies may either be directed at the situation or stressor itself (problem-focused response) or at the emotional reaction brought about because of the demand (emotion-focused response) (Lazarus and Folkman 1984). According to Allen et al. (2010) "the coping strategy chosen can lead to a favorable resolution, no resolution (e.g., the problem persists without change), or an unfavorable resolution. Favorable resolutions lead to positive emotions, while no resolution or unfavorable resolutions lead to emotional distress" (p.7). Positive emotions and their impact on the stress and coping process were not taken into account in Lazarus and Folkman's (1984) original stress model. However, Folkman's (1997) revised stress process model describes how an adverse event or ongoing stressor might actually lead to a positive emotional state. Specifically, Folkman's (1997) model postulates that people facing a seemingly unfavorable situation or one with no resolution (such as caring for a loved one with AIDS whose death is imminent and unpreventable) actually experience positive emotions and may also engage in meaning-based coping.

In Folkman's (1997) revised stress process model, meaning-based coping is seen as a resilient process that reduces the impact of negative life events (such as illness or death) on emotional outcomes and can lead to the experience of positive emotions. In other words, meaning-based coping facilitates positive emotions by buffering against the negative emotions that are formed when an adverse or stressful event is experienced. By moderating the relationship between stress and negative emotions, meaning-based coping (e.g., positive reappraisal, revised goals, positive events or activities and religious/spiritual beliefs) allows one to experience positive emotional outcomes. In other words, "the attainment of positive emotions

using meaning-based coping can be seen as a buffer between the stresses of an adverse event and the experience of negative emotions" (Allen, Haley et al. 2011) (p.8).

Recent empirical research has found evidence to support meaning-based coping as a distinct coping strategy with positive outcomes (Folkman 2008). Folkman's (1997) model further suggests that the uniqueness of meaning-based coping is in its ability to create positive emotional states rather than directly regulating the distress that is associated with unfavorable outcomes and thus sustains the coping process. Hence, spiritual and religious beliefs are one aspect of meaning-based coping that produces positive emotions, even in adverse situations.

In particular, a reliance on religion and spirituality is one form of meaning-based coping that helps an individual cope with and adapt to adversity. Although religion and spirituality are distinct concepts, there is quite a bit of overlap between them as they are closely linked and may function in similar ways. For example, Pargament (2000) views spirituality as a key function of religion. He emphasizes that spirituality, or the desire to connect with a force that goes beyond the individual, is the most basic function of religion. (Underwood 2006) also states that few people engage in religious activity without having an associated sense of spirituality, but further emphasizes that there are a small group of people who experience spirituality and are not connected to any formal religious belief or activity. Likewise, (Moreira-Almeida, Neto et al. 2006) argues that spirituality may or may not stem from the practice of religious customs that are formed when one joins a religious community. Thus, religion is the routine participation (via beliefs, customs or practices) in a specific organized religion (Agli, Bailly et al. 2014) and as such has been defined as the external, institutionalized, formal and doctrinal aspects of religious life (Hill and Pargament 2003).

Additionally, spirituality includes the concepts of faith and/or meaning but is still linked to one's religious beliefs via the faith component. Faith is the belief in a higher transcedent power that is both inside (internalized) and outside the human psyche (Agli, Bailly et al. 2014) and as such spirituality extends beyond the self and material world (Miller and Thoresen 2003) to include the concepts of God and the divine (Hill and Pargament 2003). Connectedness with this transcendent power, God or divine spirit, is an essential component of the spiritual experience, and as such spirituality is a personal and subjective experience that is linked to the concept of meaning (Agli, Bailly et al. 2014). "Meaning, or feeling that one's life has meaning, involves the deep conviction that we have both a role and a unique purpose in life, which is considered as a (fulfilling) gift. The faith component of spirituality is most often associated with religion and religious beliefs, while the meaning component seems to be a universal concept that can exist in people who are religious or not" (Agli, Bailly et al. 2014, p.2). In sum, spirituality is not only a faith-based relationship with the sacred or transcendent, but also an understanding of the meaning and purpose of one's life (Agli, Bailly et al. 2014).

Hence, a substantial proportion of people in stressful situations use both religion and spirituality to cope with stress and other hardships (Pargament, Koenig et al. 2000, Pargament 2001). In fact, early scholars report that religion is cited more frequently than any other form of coping among older adults and individuals of minority groups in the United States (Koenig 1998). Religion and spirituality may further play an important role for older adults and racial minorities, who are able to adapt positively to discrimination and other stressful life events, reinforcing the spiritual/religious resilience process. Spiritual/religious resilience "is a process in which a person uses spiritual and/or religious beliefs and behaviors as a means of coping in the face of adversity" (Allen, Haley et al. 2011) (p.8).

Research shows that religiosity and spirituality are especially important for older adults given the positive role it plays in maintaining health and recovering from illness. In fact, research shows that religion and spirituality are linked to better health and well-being outcomes among older adults, including coping strategies, quality of life and cognitive functioning (Agli, Bailly et al. 2014). In a systematic review, Agil and colleagues (2014) found positive effects of religion and spirituality on health outcomes in 10 out of the 11 studies examined.

Studies using the Mini-Mental State Exam (MMSE) have found an inverse link between religiosity and spirituality and cognitive decline. For example, (Coin, Perissinotto et al. 2010) found a higher level of religiosity was associated with a slower rate of cognitive and behavioral decline in patients with Alzheimer's dementia. Specifically, patients who reported no or low versus moderate or high religiosity on the Behavioral Religiosity Scale (BRS) also had markedly lower scores on both the total cognitive functioning and behavioral tests after one year. In this sample, lower levels of religiosity also coincided with a higher risk of cognitive impairment, considered as a 3-point decrease in the MMSE score. However, it is not clear whether the improvement in cognitive functioning in this sample was related to spirituality or taking a cholinesterase inhibitor medication, since this treatment was given to all of the participants at the outset of the study. A second study examining the effects of quality of life, spirituality, and religion on rate of progression of cognitive decline in Alzheimer's disease reported similar findings (Kaufman, Anaki et al. 2007). The authors found higher levels of spirituality and private religious practices were correlated with a slower rate of cognitive decline (as measured by the MMSE). They also report no association between the rate of cognitive decline and quality of life, independent of baseline level of cognition, age, sex, and education. Taken together these

studies suggest that various aspects of religion and spirituality may improve one's cognitive performance, enhance one's coping abilities and promote a better quality of life among older adults.

Older adults, particularly blacks, view religiosity and spirituality as very important and studies have found that in general, blacks are more likely than whites to use religious orientations and strategies as part of their coping repertoires when dealing with stress, difficult life situations, and a variety of health problems (Taylor, Chatters et al. 2004, Krause and Chatters 2005, Chatters, Taylor et al. 2008, Chatters, Taylor et al. 2009). For example, Krause and Chatters (2005) examined 17-diiferent prayer-related measures (e.g. social and substantive content of prayer, length of prayer, interpersonal aspects of prayer) in a national sample of older adults (66 years of age at baseline). They found that older blacks were more deeply engaged in prayer activities than their white counterparts. Taylor et al. (2004) examined data from the National Survey of Black Americans (a 15-year longitudinal study) and found that over the study period prayer was the most utilized form of coping for blacks. Roughly 9 out of 10 blacks reported relying on prayer or asking someone to pray for them when dealing with a serious personal problem. Other studies using data from the National Survey of American Life (NSAL), which includes a national sample of Blacks, Caribbean Blacks, and non-Hispanic Whites, report similar findings. Chatters and her colleagues (2008) examined black-white differences in attitudes regarding religious coping; they also found that close to 9 out of 10 blacks (and Caribbean blacks) versus nearly 6 out of 10 whites reported that prayer was an important source of coping when dealing with stress and that they looked to God for strength, support, and guidance. Further, for all 12 measures of religiosity, blacks reported higher levels of religious participation and spirituality compared with whites and these effects were independent of religious affiliation,

socioeconomic status, region, and other demographic factors (Chatters, Taylor et al. 2009).

Together, these findings underscore the overall importance of religious and spiritual coping in general among blacks, chiefly older blacks. Following this, blacks that report high levels of religious involvement and spirituality may then be protected against cognitive impairment in later life moreso compared with whites. Yet, few studies have considered whether religion or spirituality buffers against cognitive impairment in older adults or if these constructs are especially useful for blacks as a coping mechanism against declines in cognitive function. I hypothesis a similar protective influence of religion and spirituality on cognitive impairment, given that they are related concepts. I would also expect these two constructs to have equivalent buffering effects on stressful life events and discrimination, especially since the faith component of spirituality is most often associated with one's religion and religious beliefs. This study seeks to shed light on these relationships by exploring religion and spirituality as key protective factors against cognitive impairment that may be particularly valuable for blacks.

III. THEORETICAL FRAMEWORK

A. Stress and Coping Theory

The conceptual framework I used for this study (shown in Figure 1 and described below) incorporates the central idea from the stress and coping model of prolonged activation of the body's stress systems (including neural, neuroendocrine, and immune systems) as the primary pathogenic pathway by which stress leads to disease (McEwen 1998). In particular, repeated life stress is thought to cause cognitive impairment, by the chronic activation of the HPA axis, which leads to atrophy of the brain structures essential for learning and memory (i.e., hippocampus, amygdala, and frontal lobes). This illustration of the prolonged stress response and the subsequent health risk to an individual shows the negative adaptations to stress.

However, stress and coping theory further asserts that the occurrence of stress can evoke a positive response as well, allowing individuals to cope with the behavioral demands of a stressor and the associated physiological and emotional reactions brought about because of it (Lazarus and Folkman 1984). Thus, within this framework, coping resources and strategies are usually regarded as modifying or buffering the effects of stress on health, either by eliminating, avoiding or changing the meaning of the stressor (Brosschot, Gerin et al. 2006). Coping resources (such as a sense of control or mastery over life and self-esteem) and coping strategies (such as religious or spiritual coping) have been consistently shown to buffer the negative health effects of stress (Thoits 1995, Ryff, Friedman et al. 2012). Likewise, this study posits that stress leads to cognitive impairment and evaluates an individual's ability to cope or to readjust, despite prolonged activation of the body's stress response systems.

B. Conceptual Model

The constructs that will be measured and included in this study are in color and those that will not be measured are included in grey scale. The focal relationship is in blue, confounding factors are in pink, protective factors or coping strategies are in purple, and control variables are in green. Figure 1.1 shows that race/ethnicity is associated with (not a direct cause of) cognitive impairment. It also shows that education, stressful life events, and discrimination may confound the association between race/ethnicity and cognitive impairment and protective (religion/ spirituality) factors may buffer this association. Specifically, religion/spirituality may contribute to lower cognitive impairment, especially for blacks. Further, education, stressful life events, and experiences of discrimination can be influenced by demographic characteristics such as age, gender, income, and marital status, as well as by mental health status (i.e., depressive symptoms), chronic conditions (i.e., high blood pressure and diabetes) and health behaviors (i.e.,

smoking status). Similarly, spirituality, demographic characteristics, mental health status, chronic conditions and health behaviors can influence cognitive impairment.

Additional considerations not tested in this study are included in Figure 1 in grey scale. Although I will not be assessing the biological mechanisms, I do recognize the well-established genetic factors such as Apolipoprotein E (APOE) genotype that have been shown to influence cognitive functioning (Lopez, Jagust et al. 2003, Sheffler 2013).

C. Summary of Hypotheses

Based on the theoretical framework presented in Figure 1.1, the **first and second hypotheses** of this study is that race/ethnicity will influence (not cause) cognitive impairment and racial disparities in cognition will exist at baseline and over time, independent of risk factors known to influence cognition, including age, age-squared, gender, education, income, marital status, diabetes, stroke, smoking status and depressive symptoms (see pathways H1 and H2).

H1: Blacks will have higher cognitive impairment than whites at baseline.

H2: The black-white disparity in cognitive impairment will persist as individual's age.

The **third hypothesis** of this study is that education, stressful life events, and discrimination will be directly correlated with higher (worse) wave 5 cognitive impairment scores, independent of risk factors known to influence cognition (see pathway H3).

H3: Less education, more stressful life events and discrimination are independently associated with worse wave 5 cognitive impairment scores.

The **fourth hypothesis** of this study is that education, stressful life events and discrimination confounds the association between race/ethnicity and wave 5 cognitive impairment (see pathway H4).

H4: Education, stressful life events and discrimination will account for racial differences in wave 5 cognitive impairment scores.

The **fifth hypothesis** of this study is that education, stressful life events and discrimination will moderate the relationship between race/ethnicity and wave 5 cognitive impairment. Figure 1.2 depicts a graph of the anticipated interaction between education and race/ethnicity (see pathway H5).

H5: Education, stressful events and discrimination will moderate the race-cognitive impairment association, the greatest race disparity in cognitive impairment will occur at the lowest levels of education.

The **sixth hypothesis** of this study is that religion and spirituality will be directly correlated with better wave 5 cognitive impairment scores, independent of risk factors known to influence cognition and regardless of race (see pathway H6).

H6: Religion and spirituality will have a protective effect against declines in wave 5 cognitive impairment scores.

The **seventh hypothesis** of this study is that religion and spirituality will moderate the relationship between race/ethnicity and wave 5 cognitive impairment, such that at high levels of religion and spirituality blacks will have an advantage in terms of cognitive impairment compared to whites with similar levels of religion and spirituality (see pathway H7); religion and spirituality are expected to have a stronger protective effect on the cognition of blacks than whites based on previous studies examining racial differences in the impact of coping strategies on health and well-being (Williams 1994, Thompson 2006). Figure 1.3 depicts a graph of the anticipated interaction between religion, spirituality and race/ethnicity.

H7: Religion and spirituality will moderate the relationship between race/ethnicity and wave 5 cognitive impairment, such that at higher levels of religion and spirituality the black-white difference will be less.

The **eighth hypothesis** of this study is that religion and spirituality will buffer the relationships between stressful life events, discrimination and cognitive impairment, such that wave 5 cognitive impairment will be less among individuals reporting high levels of religion and spirituality (see pathway H8). Figures 1.4 and 1.5 depict graphs of the anticipated interactions between religion, spirituality, stressful life events and discrimination.

> H8: Religion and spirituality will buffer the relationship between stressful life events, discrimination and cognitive impairment.

Finally, the **ninth hypothesis** of this study is that there will be a 3-way interaction between race/ethnicity and stress (i.e., stressful life events and discrimination), religion and spirituality, such that blacks with low levels of religion and spirituality and low levels of stress will have worse wave 5 cognitive impairment scores compared with whites at these same levels. Additionally, blacks with high levels of religion and spirituality and high levels of stress will have better wave 5 cognitive impairment scores compared with whites at these same levels. Figure 1.6 depicts a graph of the anticipated 3-way interaction.

> H9: Blacks at lower levels of religion and spirituality and lower stress levels will have higher wave 5 cognitive impairment scores than whites, but at higher levels of religion and spirituality and even with higher stress blacks will do better than their white counterparts also reporting high levels of religion/spirituality and stress.

Figure 1.1 Conceptual Framework

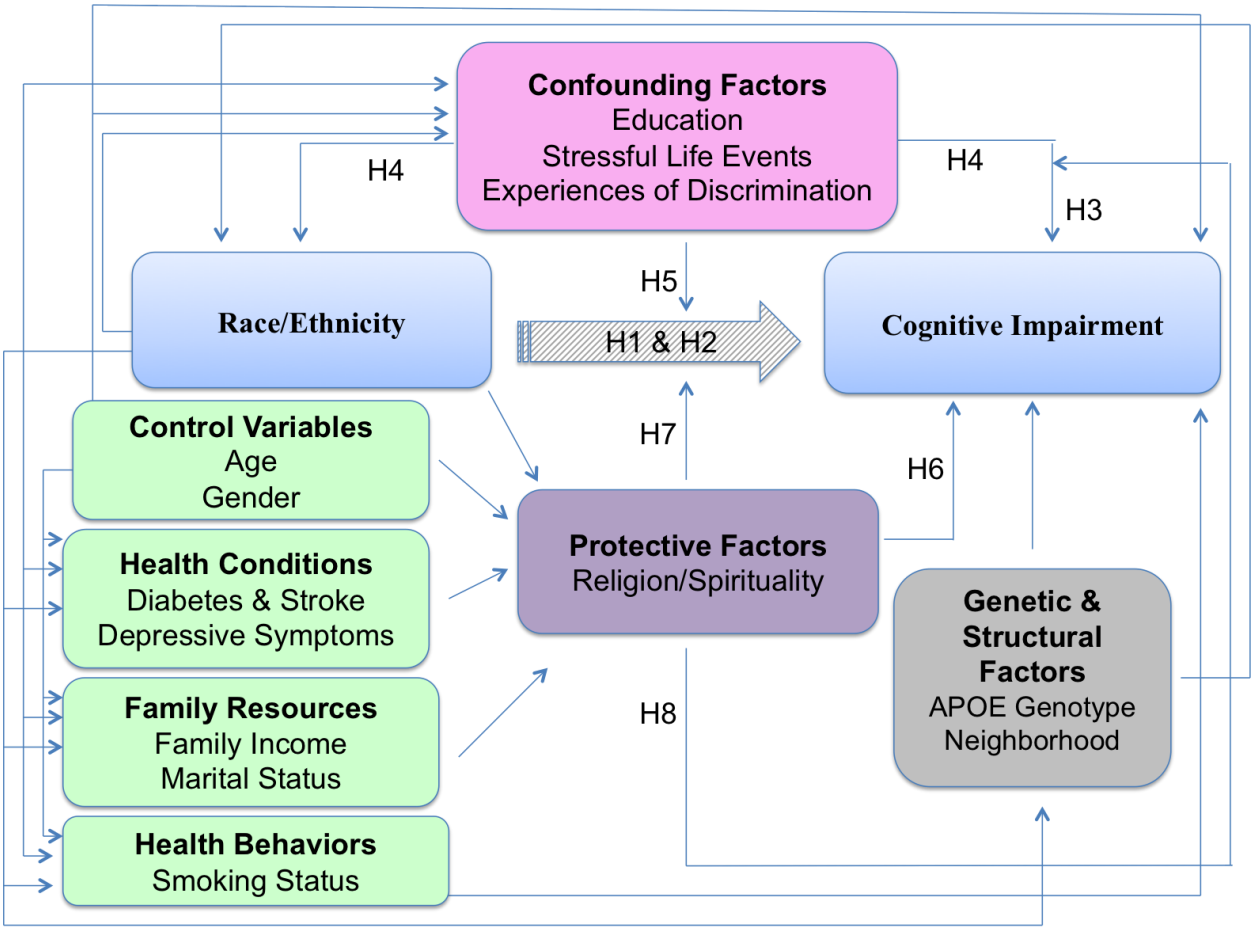


Figure 1.2 Anticipated Interaction Between Education and Race in Predicting Cogntive Impairment, ACL (waves 4 and 5)

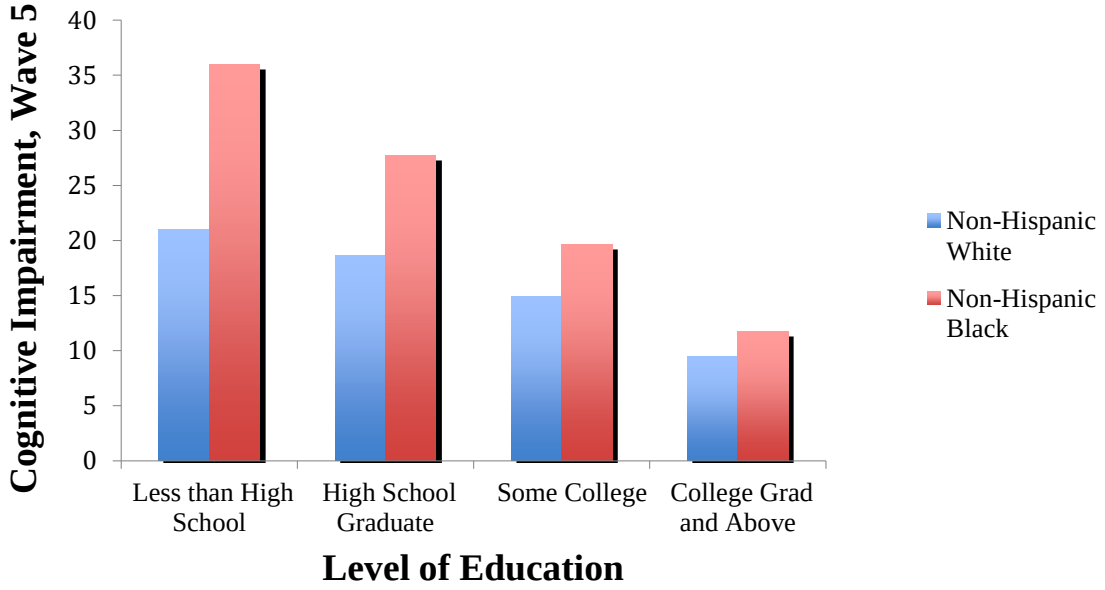


Figure 1.3 Anticipated Interaction Between Religion, Spirituality and Race in Predicting Cogntive Impairment, ACL (waves 4 and 5)

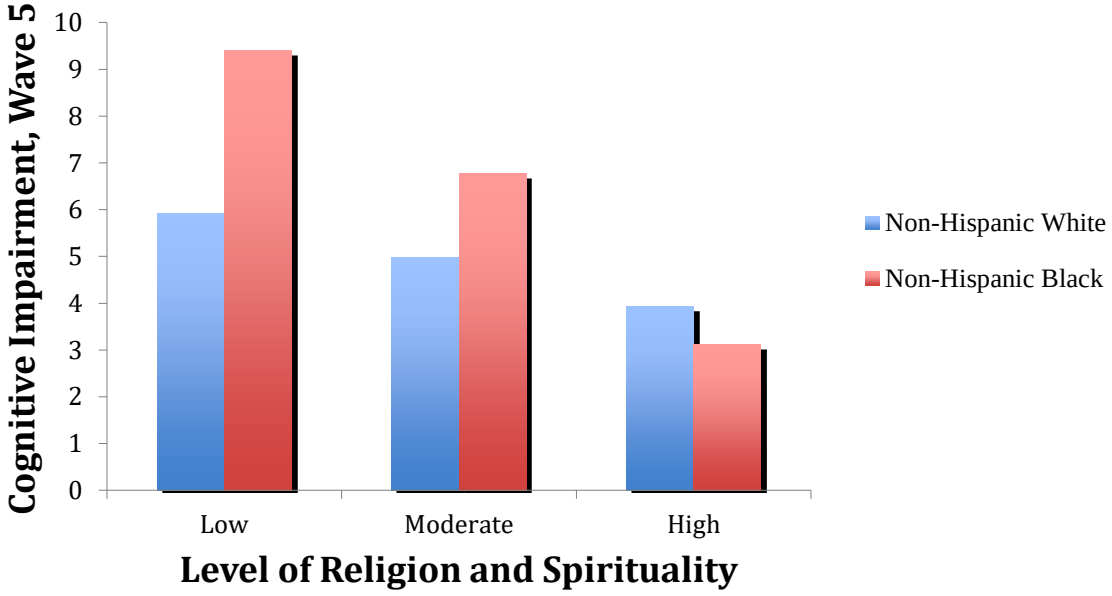


Figure 1.4 Anticipated Interaction Between Stressful Life Events, Religion and Spirituality in Predicting Cognitive Impairment, ACL (waves 4 and 5)

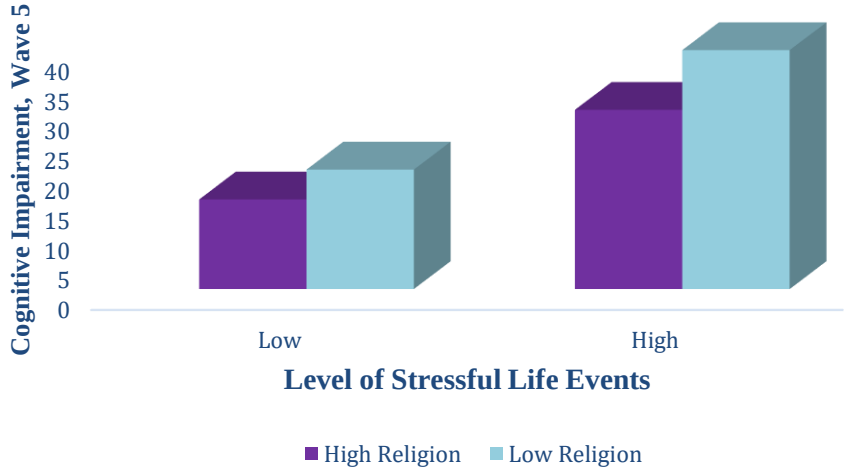


Figure 1.5 Anticipated Interaction Between Discrmination, Religion and Spirituality in Predicting Cognitive Impairment, ACL (waves 4 and 5)

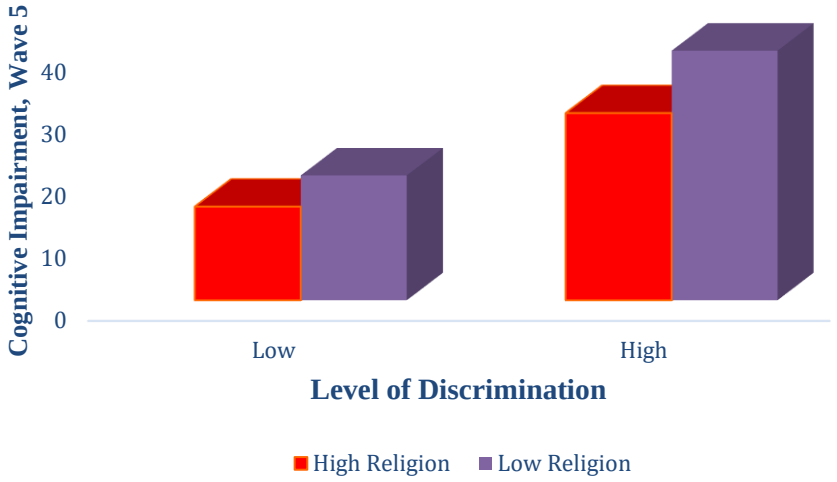
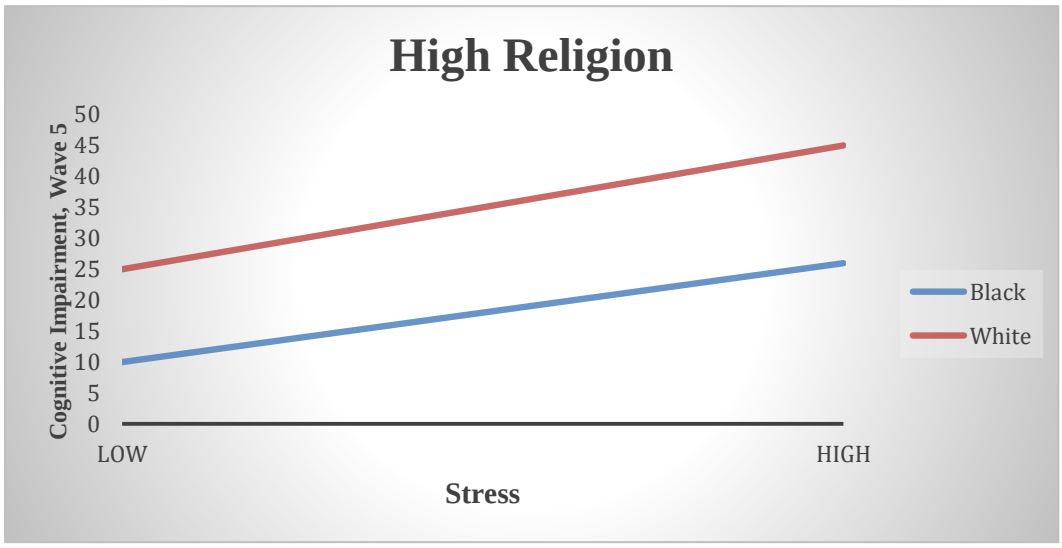


Figure 1.6 Anticipated 3-way Interactions Between Race, Stress (i.e., Stressful Life Events and Discrimination), Religion and Spirituality in Predicting Cognitive Impairment, ACL (waves 4 and 5)



CHAPTER 2: RESEARCH DESIGN AND METHODS

This dissertation is organized around three aims. The first aim will examine race differences in changes in cognitive impairment over time and determine whether disparities exist. The second aim will explore whether education, stressful life events, and experiences of discrimination account for racial disparities in cognitive impairment among a nationally representative sample of younger older U.S. black and white adults. Finally, my third aim will consider the protective influence of religion and spirituality on cognitive impairment and determine whether religion and spirituality buffers the relationship between race/ethnicity and cognitive impairment. In the following sections, I describe the data, measures, and analytic plan for each research objective. The key variables are summarized in Table 2.1 below.

Table 2.1 Summary of Concepts Assessed in the Americans' Changing Lives Study (ACL)

Construct	Wave 4 Variables	Wave 5 Variables
Cognitive Impairment (measured at all 5 waves)	Short Portable Mental Status Questionnaire (5-items)	Short Portable Mental Status Questionnaire (5-items)
Education	Self-Reported Level of Education	Self-Reported Level of Education
Stressful Life Events	Number of Recent Life Events Index (9-items)	Number of Recent Life Events Index (9-items)
Discrimination	Everyday Discrimination Scale (5-items)	Everyday Discrimination Scale (3-items)
Protective Factors	Religion and Spirituality (3-items) Spiritual Coping (2-items)	Religion and Spirituality (2-items)

The hypothesized relationships between individual characteristics, social and cultural risk factors, and cognitive function over time were modeled using the Americans' Changing Lives (ACL) survey. The ACL survey is a rich dataset to test the relationships proposed in this dissertation and examine changes in cognitive function over time. This chapter begins by

describing the ACL dataset used, including a description of the analytic sample, the measures and the analysis plan used to examine each of the specific aims detailed in Chapter 1.

I. Data Source: Americans' Changing Lives (ACL)

The Americans' Changing Lives (ACL) survey is an ongoing longitudinal study designed to explore social disparities in health and aging with a particular emphasis on examining differences between black and white adults who are in middle and late life (House, Lantz et al. 2005). The ACL cohort is comprised of a non-institutionalized sample of adults' ages 25 years and older living in the U.S. in 1986. It oversamples, at twice the rate of others, Black Americans and adults 60 and over (response rate = 68-70%) to increase the size of these groups and facilitate age and race comparisons. The individuals in the ACL cohort are interviewed and re-interviewed at five different time points (baseline survey conducted in 1986 and re-interviews in 1989, 1994, 2001/2002, and 2011), constituting waves one through five of the ACL survey. Wave 1 (baseline survey) of the ACL study began in 1986 with face-to-face interviews of 3,617 respondents. Wave 2, also involving face-to-face interviews, was done in 1989 with 2,867 respondents or 83% of the wave 1 survivors. Subsequent follow-up of wave 1 survivors was done in 1994 (83% of survivors), 2001/2002 (76%-80% of survivors), and 2011 (81% of survivors) making up waves 3, 4, and 5 of the data collection, respectively. At each wave of follow-up data collection, respondents reported on their stressful life events since the date of the last interview beginning in wave 4 and experiences of discrimination beginning in wave 3 (http://www.isr.umich.edu/acl/ accessed April 10, 2014).

II. Analytic Sample

The present analysis focused on black and white young to older aged adults enrolled in

all five waves of the Americans' Changing Lives study. Table 3.2 shows the race distribution of black and white respondents at all waves of the ACL. Black respondents at all waves include wave 1 (n=1,156), wave 2 (n=860), wave 3 (n=725), wave 4 (n=438) and wave 5 (n=401). White respondents at all waves include wave 1 (n=2,205), wave 2 (n=1,818), wave 3 (n=1,670), wave 4 (n=1,242) and wave 5 (n=916). Additionally, since recent stressful life events and everyday experiences of discrimination were only asked in later waves of the ACL study, a subsample of 1,105 adults was analyzed in waves 4 and 5. Respondents who were present in both waves 4 and 5 represent a total weighted analytic sample of n=1,105.

The ACL sample weights were provided with the data file and are designed to ensure that the respondents at each wave are representative of the 1986 non-institutionalized adult population in the continental U.S. (Burgard, Brand et al. 2007, p.372). This original sampling approach resulted in complex design effects (Herzog, Kahn, Moran, Jackson, & Antonucci, 1989) so the analyses in this chapter for waves 4 and 5 used weighted data to account for the wave 1 sampling variation in 1986. Analyses were weighted using the SVY command in Stata 13 to adjust for the oversampling of certain populations (i.e., Black Americans and people aged 60 and over) in the original 1986 ACL survey and the standard errors were adjusted to account for the complex sampling design (i.e., adjusting for the sampling error introduced at each level of sample selection).

These data are particularly useful for this study for a number of reasons, including (1) the collection of data at five time points, (2) the ability to examine differences between black and white adults who are in middle and late life, (3) the nationally representativeness of the data, (4) the breadth and depth of information collected on stressful life events, discrimination, religion

and spirituality, and cognition, and (5) the large sample sizes in subsequent waves of data collection.

III. Measures

A. Dependent Variable

Cognitive impairment was assessed at each wave using a shortened version of the Short Portable Mental Status Questionnaire (SPMSQ). The SPMSQ was developed for assessing the entire range of cognitive performance from intact functioning to severe impairment (Erkinjuntti, Sulkava et al. 1987). It has also been used in community samples to identify persons with cognitive impairment and clinically diagnosed Alzheimer's disease (Albert, Smith et al. 1991). This 5-item measure tests a participant's orientation (e.g., identification of today's date and day of the week), knowledge of current and past affairs (i.e., naming the current and former Presidents of the United States), and working memory (i.e., serial 3's subtraction test). The serial 3's subtraction test asked participants to subtract 3 from 20, report the number they get, and to continue subtracting in increments of 3 from each new number they get. Successful cognitive performance on the serial 3's test consists of the number of times a respondent is able to correctly count backwards by three until arriving at the number two. Thus, the entire series beginning with the first subtraction (i.e., 20-3=17) and the remaining subtractions (e.g., 17-3=14, 14-3=11, etc.) has to be performed accurately without missing any one of the subtractions in order to be scored as correct (0=correct). Missing any one of the subtractions (or any error in the series) or refusing to attempt the series is scored as incorrect (1=incorrect) (Pfeiffer, 1975, p.441). The total cognitive impairment score was calculated by adding the number of incorrect responses from each of the three domains (orientation, knowledge, and working memory) to

form a single summary measure, as done by other studies (Albert, Smith et al. 1991) with higher scores indicating poorer cognitive functioning. Sample range = 0 (less cognitive impairment) to 5 (more cognitive impairment).

Scholars should consider the potential education bias and the difficulty level of the items included when interpreting measures of cognitive function (Langley, 2000). For instance, having a lower score could be the result of either true cognitive impairment or lower educational attainment. In other words, one's ability to subtract 3's from 20 serially may not indicate cognitive dysfunction, rather may reflect a lower level of education. Thus, in discussing racial differences in cognitive performance, it is necessary to consider the inherent education bias, especially given that blacks have lower educational attainment than whites (Barnes, Wilson et al. 2011). Although, the original SPMSQ made adjustments for education level and race[1], the ACL survey did not make such adjustments instead it used unadjusted SPMSQ scores for ease of scoring and interpretation. The ACL survey also does not include a measure of education quality (e.g., reading level ability, self-assessed school performance) however, the SPMSQ measure included in the ACL survey does incorporate a serial 3's subtraction score in the total score, rather than spelling the word "world" backwards; thereby significantly increasing the difficulty of the SMPSQ measure regardless of one's education level (Hawkins, Cromer et al. 2011). Yet, this still does not address education bias or quality. Further, as noted previously when the MMSE (a measure that overlaps greatly with the SMPSQ) is used in minority populations (e.g., Hispanic, non-Hispanic Black, Africans or non European-American elders), its sensitivity and specificity are reduced (Wood, Giuliano et al. 2006, Matallana, de Santacruz et al. 2011,

[1] These adjustments allow one more error for a subject with only a grade school education; allow one less error for a subject with education beyond high school; and allow one more error for African American subjects, using identical educational criteria (see Pfeiffer, 1975).

Pedraza, Clark et al. 2012). Despite these potential biases racial differences may still have an effect on SPMSQ scores, independent of education level and quality. Table 2.2 below describes the overall scoring of the SPMSQ measure.

Additionally, I conducted sensitivity analyses of these items to test the claim that this measure may be biased against blacks because of educational level differences. Appendix 3.5 shows that the race effect is no longer significant once education level is controlled for in Model 2 for the question 'identifying today's date; but remains significant for the question that identifies what day of the week it is in Model 4. Appendix 3.6 shows for both items identifying the current and former President, the race effect remains significant but is reduced once level of education is controlled for in Models 2 and 4. Finally, the race effect remains significant and is reduced slightly for the serial 3's subtraction test in Model 2 of Appendix 3.2, after adjusting for one's education level. These supplemental analyses suggest that most of the items on the SPMSQ, except for the question 'identifying today's date' are influenced by education level. Therefore, the education (quantity) bias present in the SPMSQ should be taken into account when interpreting the results of the current study. In the ACL sample, blacks had significantly lower levels of education than whites and this may be why they scored lower on the SMPSQ. However, even after adjusting for years of education the black-white disparity is reduced but not eliminated, suggesting that other factors beyond education quantity may play a role.

Table 2.2 Dependent Variable. Composite Cognition Score. Americans' Changing Lives Study (ACL), 1986-2011 (waves 1-5).

Cognitive function was measured as the sum of the total number of errors for the following items on the SPMSQ*:

Domain	Variable	Description	Score
Orientation	Orientation to time	Identify today's date (month, day and year)	0-1
		Identify what day of the week it is	0-1
Knowledge	Current and past affairs	Identify current President	0-1
		Identify former President	0-1
Working Memory	Serial 3's subtraction test	Subtract 3 from 20 and continue subtracting each subsequent number for six trials	0-1
Total Score			0-5

*Short Portable Mental Status Questionnaire (SPMSQ)

B. Independent Variable

Race/ethnicity was characterized using two self-reported categories: Non-Hispanic white (referred to as whites; n=2,205), Non-Hispanic black (referred to as blacks; n=1,156). Individuals of other races, including non-Hispanic Native American (n=44), non-Hispanic Asian (n=30) and Hispanic (n=182), were excluded due to small sample sizes.

C. Confounding Variables

Level of education was self-reported and categorized as 0 = less than high school, 1 = high school graduate, 2 = some college, and 3 = college graduate and beyond.

Stressful life events were measured using a 10-item Number of Recent Life Events Index. Although this index does not assess lifetime occurrences of major/traumatic events, it has been used in other studies to measure recent negative life events (Lantz, House et al. 2005). Beginning with wave 2, ACL respondents were asked since the date of the last interview if they had experienced any of the following ten events (i.e., "Since we interviewed you in (e.g., month of interview, 2001/2002, 1994, 1989 or 1986), have you become…?"): (1) widowhood, (2) being robbed or having their home burglarized, (3) involuntarily job loss other than for retirement reasons, (4) being the victim of a serious physical attack or assault, (5) having a parent or step-parent die, (6) divorce, (7) having a child die, (8) having a close relative or friend, other than a spouse, parent or child die, (9) having any serious financial problems or difficulties, and (10) having anything else bad happened that upset the respondent a lot. The index was created by taking the sum of the number of "yes" responses to the ten different life events (scores range from 0-10). Additionally, a sensitivity analysis was done on the ninth event in the index relating to having any serious financial problems or difficulties because it was closely related to other

SES predictors of interest that were included in the regression analysis as controls (e.g., education, income, etc.). Thus, regression models were ran with and without this item and the findings are discussed in the results section (see Appendix 3.2). Finally, although information regarding stressors related to health was also available in the data; these two items were not included in the index. Two life events referring to health events (i.e., experiencing a life-threatening or serious illness or injury) were excluded because a serious illness or life-threatening injury such as a traumatic brain injury can lead to cognitive impairment, the dependent health outcome (see Appendix 2.1 for the complete measure).

Everyday discrimination was assessed using a shortened version of the Everyday Discrimination Scale (Williams, Yan et al. 1997). Wave 4 of the ACL used the 5-item shorten version of this scale (Cronbach's $\alpha = .71$), which was developed for the Chicago Community Adult Health Study (Sternthal, Slopen et al. 2011). ACL respondents in wave 4 were asked ''In your day-to-day life how often have any of the following things happened to you?'' The discriminatory events included being treated with less courtesy or respect than other people; receiving poorer service than other people at restaurants or stores; having people act as if the respondent was not smart; having people act as if they were afraid of the respondent and being threatened or harassed. For each event, respondents chose from five Likert response categories (1) at least once a week, (2) a few times a month, (3) a few times a year, (4) less than once a year and (5) never. The response categories were reverse-coded, summed and collapsed to (3) a few times a month or more, (2) a few times a year, (1) less than once a year and (0) never, so that higher scores reflect more discrimination and to match the four response categories in the wave 5 discrimination measure (see Appendices 2.2 and 2.3 for both measures). Sample range = 0 (never experiencing discrimination) to 15 (experiencing discrimination a few times a month or

more). Additionally, a sensitivity analysis was done on the discriminatory event "people act as if they think you are not smart" because it could be related to changes in someone's cognitive impairment (i.e., main outcome of interest). Thus, regression models were ran with and without this item (see Appendix 3.2).

To assess one's *religion and spirituality* several items were used, including two items measuring the frequency of religious attendance and participation in church activities, one item addressing the importance of religious or spiritual beliefs, and two items assessing one's use of spiritual coping (measured only in wave 4 of the ACL survey).

ACL respondents were read the following stem prompt, "Now I have a few questions about *religion*. Even people who have no religious affiliation may have some *religious feelings or beliefs or engage in some religious activities*, so we ask these questions of everyone." "How often do you usually attend religious services?" and "Besides religious services, how often do you take part in other activities at a church or place of worship?" For each question, respondents chose from six Likert response categories (1) more than once a week, (2) once a week, (3) 2-3 times a month, (4) about once a month, (5) less than once a month, and (6) never. Both items were summed and reverse-coded so that higher scores reflect greater frequency in religious attendance and participation in church activities (Cronbach's $\alpha = .79$). Sample range = 0 (never) to 5 (more than once a week). Other studies have used these questions to measure organizational religious participation, including frequency of attendance and participation in church activities (Chatters 2009).

ACL respondents were also asked a single question regarding the importance of their *religious or spiritual beliefs*. "In general, how important are religious or spiritual beliefs in your

day-to-day life? Would you say very important, fairly important, not too important, or not at all important? This item was reverse-coded such that higher scores reflect greater importance of religious or spiritual beliefs. Sample range = 0 (not at all important) to 3 (very important). This single-item question has been used by another study to measure subjective religiosity (see Chatters 2009).

Additionally, ACL wave 4 respondents were asked about their use of *spiritual coping*, using a 2-item spiritual coping index. Specifically, ACL wave 4 respondents were asked "Think about how you try to understand and deal with major problems in your life. To what extent is each of the following involved in the way you cope: (1) I work together with God as partners and (2) I look to God for strength, support, and guidance. Would you say a great deal, quite a bit, some, a little, or not at all?" The 2-item index was constructed such that items were reverse-coded and summed so that higher scores indicate greater use of spiritual coping (scores range from = 0-10). Another study has also used the second question in this scale to measure religious coping and one's overall orientation toward God as a resource (see Chatters 2008). For a review of other studies using similar measures see Ano and Vasconcelles (2005) and Agil et al. (2014).

D. Control Variables

All analyses account for major socio-demographic, chronic conditions, health behaviors and mental health variables known to influence cognitive functioning (Rexroth, Tennstedt et al. 2013). Therefore, I include participants' *age* and *age-squared* (measured in years), and *gender*. *Family income* is measured using both the respondent and spouse's income and treated as a continuous measure; family income was asked open-ended and respondents reported their income as actual dollar amounts. Some respondents refused to report or did not know the exact dollar amount of their income. In place of reporting a specific dollar amount, these respondents

were asked to select from a range of incomes. To create a single measure of family income across all participants, respondents selecting from the range of incomes were then assigned a "mid-point" value. This mid-point value was based on the average income values for those respondents who reported an exact dollar amount for that income range. The final family income variable is based on these assigned averaged "mid-point" values across all participants and is reported in dollars. Also, included *marital status* (married, separated, divorced or widowed, and never married), chronic health conditions (includes 2 conditions and respondents were asked "…if they have experienced any of the following health problems during the last 12 months": *diabetes* or high blood sugar, or taking medication for it, and *stroke*), and *smoking status* (current smoker, past smoker, never smoked). Given that these questions only asked respondents to report these health conditions during the last year, I created a variable for respondents who reported yes to having had these health conditions in a previous wave to identify them in subsequent waves. This was done to account for the shortened follow-up window relative to the time between waves, which is greater than a year.

I also control for depressive symptoms using an 11-item standardized version of the Center for Epidemiologic Studies Depression Scale (CES-D) (Radloff 1977). This scale measures the extent to which a person feels depressed and has become a standard in community mental health surveys over the past three decades (Eaton and Kessler 1981, Frerichs, Aneshensel et al. 1981, Ross, Mirowsky et al. 1983). Further, it is particularly useful when looking at depressive symptoms in old age groups, given that it includes both psychological and physiological components (Kim and Pai 2010). In ACL, respondents were asked to consider each of the following statements and choose the category that best described how often in the

past week they felt one of the following ways: “I felt depressed”; “I felt that everything I did was an effort”; “My sleep was restless”; “I was happy” (reverse); “I felt lonely”; People were unfriendly”; “I enjoyed life”(reverse); “I did not feel like eating”; “I felt sad”; “I felt that people disliked me” and “I could not get ‘going’.” Responses were (1) “never or hardly ever,” (2) “some of the time,” or (3) “most of the time.” The index was constructed such that items marked with (reverse) were reverse-coded so that higher scores indicated more depressive symptoms. Sample range = 0 (hardly ever) to 32 (most of the time). The Cronbach’s α = .82 for wave 4 and Cronbach’s α = .85 for wave 5. Depressive symptoms were measured at all fives waves.

III. Analysis Plan

A. Descriptive Analysis

Analyses began with descriptive statistics for the ACL sample, followed by bivariate associations between race/ethnicity and cognitive impairment across all 5 waves. Simple correlations between stressful life events, discrimination, religion, spirituality and cognitive impairment at waves 4 and 5 were conducted.

All descriptive and bivariate analyses were conducted using Stata13 (stataCorp 2013). Significance testing of the differences between blacks and whites was done using OLS regression, Pearson’s chi-square test statistics (using the CHI command in stata) and ordinal logit regression (using the OLOGIT command in stata).

B. Multilevel Analysis

To address aim 1 multilevel analysis was used to estimate a series of **individual growth models** using the MIXED procedure. Growth modeling is used when examining how individuals change over time and what factors contribute to that change. Growth modeling is appropriate when using nested data, which is longitudinal data with repeated measures within

individuals. Growth modeling techniques have the ability to examine individual growth trajectories by evaluating **within-individual** changes and **between-individual** differences in change over time.

The purpose of using the MIXED procedure to estimate a series of multilevel mixed-effects linear regression or growth models it two fold. First, the maximum likelihood estimate (mle) is an unbiased estimate in a mixed model that accommodates missing data on the dependent variable due to attrition over time. This estimation allows participants with missing data, or those lost in subsequent follow-up waves, to contribute to the model results. Secondly, MIXED allows one to examine the association or correlation between measures over time. In other words, the model allows for the observation of intra-individual variation within study participants; thereby addressing the non-independent nature of longitudinal data that has repeated measures over time within the same study subject (Masel and Peek 2009).

This dissertation aims to answer a series of questions within the multilevel growth modeling framework. Specifically, do individuals have different mean (baseline) levels of cognitive impairment? Are there individual changes in cognitive performance over time (i.e., test for random slopes across individuals)? If so, are there racial differences in these individual changes specifically, do these changes differ for blacks and whites? What are the predictors of the racial differences? (Singer and Willett pp.4, 8).

I hypothesize that racial disparities in cognitive impairment will exist at baseline and persist over time (see pathways H1 and H2). To address this hypothesis, I will model equation 2.0 below:

Cognitive Impairment Predicted (2.0)

$$Y_{ti} = \gamma_{00} + \gamma_{01}(Black_i) + \gamma_{10}(Time_{ti}) + \gamma_{11}(Black_i * Time_{ti}) + \gamma_{20}(Time_{ti})^2 + u_{0i} + \varepsilon_{ti}$$

Equation 2.0 illustrates a quadratic polynomial growth model for understanding black-white disparities in cognitive impairment over time. This equation is constructed from two components: (1) a level-1 sub model, known as an individual growth model, represents the within-individual change in cognitive impairment over time; and (2) a level-2 sub model representing between-individual differences in change.

This analysis permitted the estimation of individual differences in cognitive impairment as a function of age and to assess whether variability in cognitive changes could be predicted by race while controlling for sociodemographic variables. The influence of race on the growth of the number of cognitive errors (impairment), controlling for age and age-squared, as well as the interaction of age and race were also examined.

In the first component of a growth model, known as **level-1,** I evaluate **within-individual** changes in cognitive impairment over time. My goal in the level-1 analysis is to describe each person's pattern of change in cognitive impairment over time (i.e., each person's individual growth trajectory and the way his or her cognitive impairment values rise and fall over time) (Singer and Willett p.8). I will model level-1 using equation 2.1 below, which stipulates that each person's growth trajectory or slope is quadratic (non-linear) with time and includes three individual growth parameters β_{0i}, β_{1i} and β_{2i} that characterize the shape of each person's growth trajectory:

$$\text{Level-1: } Y_{ti} = \beta_{0i} + \beta_{1i}(Time_{ti}) + \beta_{2i}(Time_{ti})^2 + \varepsilon_{ti} \quad (2.1)$$

The notation *t* is used to index time (repeated measures of cognitive impairment) nested within individual *i*. The outcome Y_{ti} is the value of cognitive impairment at time *t* for individual *i*,

specified as a function of the growth process (Raudenbush and Bryk 2002) (p.162). β_{0i} (trajectory's intercept), is the expected value of cognitive impairment for individual *i* when all other predictors in the model are zero (Hox, p.52). β_{1i} (instantaneous growth rate at zero point) is individual *i*'s instantaneous rate of change in cognitive impairment at one specific moment, when time=0. β_{2i} (quadratic growth rate or curvature) is the quadratic or squared parameter associated with level-1 predictor time^2 and describes this changing rate of change (Singer and Willett, p.215). It is individual *i*'s quadratic growth rate or mean acceleration in cognitive impairment. Finally, ε_{ti} is the level-1 residual term. It represents the effect of random error, ε_{ti}, associated with the measurement of individual *i*'s cognitive impairment at time *t* and is assumed to be normally distributed with a mean of zero and homogenous variance σ_ε^2 across occasions and individuals—assumptions expressed in matrix notation as, $\varepsilon_{ti} \sim N(0, \sigma_\varepsilon^2)$. "The residual variance parameter σ_ε^2 captures the scatter of the level-1 residuals around each person's true change trajectory" (Singer and Willett, pp.54-55).

In the second component of the growth model, known as **level-2**, I examine **between-individual** differences in change. My goal in the level-2 analysis is to determine whether the individual pattern of change in the level-1 analysis is different for different racial groups. In other words, I will determine whether black and white adults experience different patterns of change with respect to cognitive impairment and what predicts these differences. Each level-1 parameter has its own level-2 sub model as such; the level-1 parameters become the outcomes in the level-2 equation (Singer and Willett, p.223). The level-2 sub model explores whether the variation in outcomes i.e., the level-1 individual growth parameters is related to race (a person-level covariate) (Singer and Willett, p.60). To examine the effect of race, I begin by postulating

a level-2 association with each level-1 parameter by modeling equations 2.2a-c below (Singer and Willett, p.223):

Level-2: $\beta_{0i} = \gamma_{00} + \gamma_{01}(\text{Black}_i) + u_{0i}$ (2.2a)

$\beta_{1i} = \gamma_{10} + \gamma_{11}(\text{Black}_i) + u_{1i}$ (2.2b)

$\beta_{2i} = \gamma_{20} + \gamma_{21}(\text{Black}_i) + u_{2i}$ (2.2c)

Where γ_{00} (level-2 intercept) is an overall initial status for whites. γ_{01} is the difference between blacks and whites in initial status in the level of cognitive impairment. γ_{10} (instantaneous rate of linear change) is the instantaneous growth rate specific to whites when time=0. γ_{11} is the difference between blacks and whites when time=0 and represents a point specific estimate of initial racial disparities in cognitive impairment (**Hypothesis 1**). γ_{20} is the quadratic growth rate or mean acceleration for whites and γ_{21} is the difference between blacks and whites in the quadratic growth rate. γ_{21} indicates a widening racial disparity in cognitive impairment over time corresponding to **Hypothesis 2**.

Similar to the level-1 residual term, the level-2 residuals (u_{0i}, u_{1i} and u_{2i}) are assumed to be normally distributed with mean zero, unknown variances and covariances τ_{00} through τ_{22}, respectively —represented using matrix notation by writing (Singer and Willett, pp.62-63),

$u_{0i} \sim N(0), \ \tau_{00}$ (2.2d)

$u_{1i} \sim N(0), \ \tau_{10} \ \tau_{11}$ (2.2e)

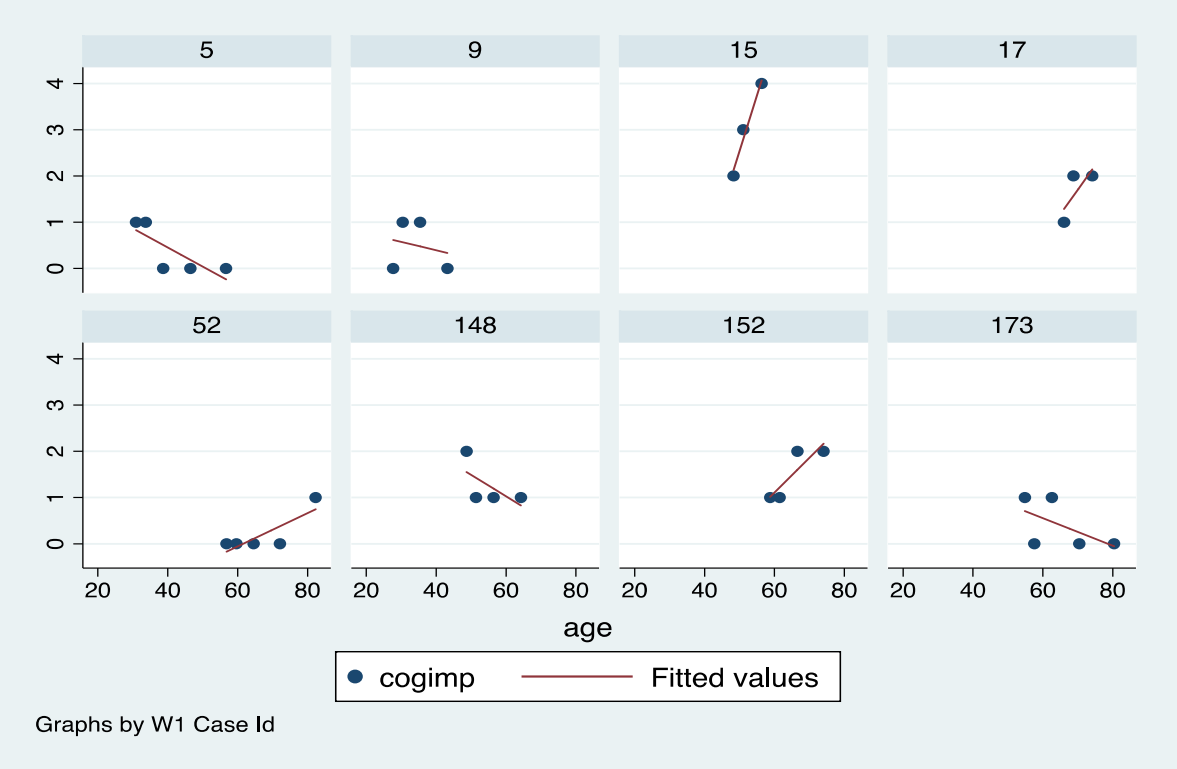
$u_{2i} \sim N(0), \ \tau_{20} \ \tau_{21} \ \tau_{22}$ (2.2f)

Conditional on the model's predictors, τ_{00} represents the variance across individuals in initial status or the value of cognitive impairment at age 0 (time point zero), τ_{11} represents the variance in the instantaneous growth rate at zero point, and τ_{22} represents the variance in the quadratic growth rate (Singer and Willett, pp.62-63). τ_{10} represents the covariance between initial

status and the instantaneous growth rate at zero point. τ_{20} represents the covariance between initial status and the quadratic growth rate. τ_{21} represents the covariance between the instantaneous growth rate at zero point and the quadratic growth rate.

To specify the quadratic polynomial growth model for the present study, I began by examining the individual growth trajectories of cognitive impairment to determine the initial form of growth (see Figure 2.1 below).

Figure 2.1 Identifying a Suitable Functional Form for the Level-1 Sub Model. Empirical Growth Plots for 8 Participants in the Americans' Changing Lives (ACL) Study.



NOTE: Plots of the underlying or crude relationship between cognitive impairment and age for participant ID numbers 5, 9, 15, 17, 52, 148, 152 and 173.

To inform model specification, Figure 2.1 presents empirical change plots with fitted values for participant ID numbers 5, 9, 15, 17, 52, 148, 152 and 173. These eight black and white adults were randomly selected from the larger ACL sample. For them, and for most of the

other participants not shown, the relationship between cognitive impairment (cogimp) and age appears nonlinear between ages 20 and 80. This suggest that I can posit a level-1 individual growth model that is non-linear (or quadratic) with age, $Y_{ti}\ \beta_{0i} + \beta_{1i}\,(Age_{ti}) + \beta_{2i}\,(Age_{ti})^2 + \varepsilon_{ti}$, where Y_{ti} is adult *i*'s value of cognitive impairment at time *t* and Age_{ti} is his or her age (in years) at that time. Thus, age will serve as the level-1 predictor in equation 2.1. I have centered age on 75 years to facilitate interpretation of the intercept. By using centered age (Age-75) as a level-1 predictor, instead of Time measured as continuous age or "real" ages, the intercept in equation 2.1 represents the expected value of cognitive impairment at age 75 for individual *i* when both predictors are zero (Hox, p.52). Had I simply used age as a level-1 predictor, with no centering the intercept in equation 2.1 would represent the expected value of cognitive impairment at age 0 or birth, an age that precedes the onset of data collection (Singer & Willet 2003 pp.52, 77). As such, this representation is less attractive because we do not know whether the growth trajectory extends back to birth non-linearly with age (Hox, p.52).

After determining the form of the growth, I then fit a quadratic polynomial to my data and then determined the appropriate random specification of the model. First, I estimated an intercept only model (model 1). Second, I estimated model 2, which includes the linear age term as a predictor variable. Third, I estimated model 3, which adds the quadratic (or squared) trend for the age term. Fourth, I estimated model 4, which models the linear age term as a random coefficient, as in equation (2.2b). Finally, I estimated model 5, which allows for the slopes for the quadratic age term to randomly vary as well, which leads to a model that does not converge given that it is beyond the ability of the data to do so, as in equation (2.2c).

Before going on and adding other explanatory variables, I conducted a likelihood ratio test comparing models 3 and 4 to determine the appropriate random specification of my model.

Specifically, I tested the fit of the model that has a random regression slope for the linear age term and the null hypothesis that the variance (variance of $u_{1i} = \tau_{11}$) and covariance (covariance of $u_{0i,}\ u_{1i} = \tau_{11}$) terms associated with the linear age term could be set to zero (see equations 2.2d and 2.2e). The chi-squared indicated, LR $\chi 2\ (1) = 0.00, p = 1.00$, I would reject the null hypothesis and prefer the model without a random effect for the linear age term. Overall, the best fitting model is one without the random effects of the linear and quadratic age terms.

I further tested an interaction between race and the quadratic age term. However, the interaction term was not significant as indicated by the chi-squared test ($\chi 2\ (1) = 2.29, p = 0.13$) and thus was not included in the final model. Therefore, the level-1/level-2 specification of the polynomial growth model for this study is represented below in equation 2.3:

Y_{ti} = Cognitive Impairment Predicted

Level-1: $Y_{ti} = \beta_{0i} + \beta_{1i}\,(Age_{ti}) + \beta_{2i}\,(Age_{ti})^2 + \varepsilon_{ti}$ (2.3)

Level-2: $\beta_{0i} = \gamma_{00} + \gamma_{01}\,(Black_i) + u_{0i}$ (2.3a)

$\beta_{1i} = \gamma_{10} + \gamma_{11}\,(Black_i)$ (2.3b)

$\beta_{2i} = \gamma_{20}$ (2.3c)

In summary, multilevel growth models were used (1) to estimate the longitudinal trajectories of cognitive impairment within individuals and (2) to evaluate the effect of race (i.e., racial differences) on these cognitive trajectories, independent of known covariates. Multilevel growth models for longitudinal or nested data provided estimates of both the predicted baseline level and rate of change in cognitive impairment scores across ages. These models further provided estimated effects (**fixed effects**) of the association between baseline level, rate of change (slope) in cognition and race/ethnicity that differs between-individuals (**level-2**) and

those that differs within-individuals such as one's score on the SPMSQ measure that may vary from wave to wave within an individual (**level-1**). Also, the random specification (**random effects**) of these models was tested to assess the between-individual variation in a person's initial value of cognitive impairment (random intercept) and his or her slope or change in cognition as predicted by their characteristics included in the model. A random intercept results from each person's tendency to be above or below the mean at baseline, while random slopes demonstrate how a person's trajectory changes more or less over time (Early et al. 2013, p.7).

C. OLS Analysis

For aims 2 and 3 I conducted ordinary least squares (OLS) analysis to model the relationships between education, stressful life events, discrimination, religion, spirituality and cognitive impairment. One of the main objectives of the dissertation is to use two waves of the ACL panel to assess whether social and cultural risk factors such as educational disadvantages, stressful life events and discrimination contribute to racial differences in cognitive performance over time for younger and older black and white adults (aim 2) and to determine whether religion and spirituality act as buffers (aim 3). I estimated a series of OLS regression models where the predictor variables are measured at wave 4 and cognitive impairment is measured at waves 4 and 5, using waves 4 and 5 of the ACL survey. Cognitive impairment at wave 5 (2011) was regressed on baseline cognitive impairment at wave 4 (2001/2002), controlling for main and interactive effects of race, level of education, stressful life events and discrimination to determine if these factors in combination account for the differences between blacks and whites. Including cognitive impairment at wave 4 controls for the effect of a prior level of cognitive performance on impairment at wave 5, and allows for the assessment of the effect of other variables on changes in cognition over time. This approach of measuring the main predictors of

education, stressful life events and discrimination prior to my cognitive impairment outcome clarifies temporal ambiguity, but does not prove causality. Significance of the interaction terms were tested based on the p-value as well as by using the CONTRAST command in Stata 13 and F-statistic to determine the overall significance.

Models testing the direct effects and whether education, stressful life events and discrimination confounds the association between race and cognitive impairment, such that this relationship will be attenuated when controlling for the simultaneous effects of these variables is presented below:

Hypothesis 3: direct effects of education, stressful life events and discrimination

W5 Cognitive Impairment Predicted = (2.4)

$\beta_0 + \beta_1$ (Black) - β_2 (Less Education) + β_3 (Stressful Life Events) + β_4 (Discrimination) + β_5 (W4 Cognitive Impairment) + β_6 (Risk Factors) + r_i

Hypothesis 4: confounding effects of education, stressful life events and discrimination

W5 Cognitive Impairment Predicted = $\beta_0 + \beta_1$ (Black) + r_i (2.5a)

W5 Cognitive Impairment Predicted = (2.5b)

$\beta_0 + \beta_1$ (Black) - β_2 (Less Education) + β_3 (Stressful Life Events) + β_4 (Discrimination) + β_5 (W4 Cognitive Impairment) + β_6 (Risk Factors) + r_i

Hypothesis 5: moderating effects of education, stressful life events and discrimination

W5 Cognitive Impairment Predicted = (2.6)

$\beta_0 + \beta_1$ (Black) - β_2 (Less Education) – β_3 (Black * Less Education) + β_4 (W4 Cognitive Impairment) + β_5 (Risk Factors) + r_i

W5 Cognitive Impairment Predicted = (2.7)

$\beta_0 + \beta_1$ (Black) + β_2 (Stressful Life Events) + β_3 (Black * Stressful Life Events) + β_4 (W4 Cognitive Impairment) + β_5 (Risk Factors) + r_i

W5 Cognitive Impairment Predicted = (2.8)

$\beta_0 + \beta_1$ (Black) + β_2 (Discrimination) + β_3 (Black * Discrimination) + β_4 (W4 Cognitive Impairment) + β_5 (Risk Factors) + r_i

Cognitive impairment at wave 5 (2011) was regressed on baseline cognitive impairment at wave 4 (2001/2002), controlling for main and interactive effects of race and religion/spirituality as well as education, stressful life events, discrimination and religion/spirituality to determine if these factors account for the differences between blacks and whites. Models illustrating whether religion and spirituality buffers the association between race and cognitive impairment, such that this relationship will be attenuated when controlling for the simultaneous effects of these variables is presented below:

Hypothesis 6: direct effects of religion and spirituality

W5 Cognitive Impairment Predicted = (2.9)

$\beta_0 + \beta_1$ (Black) – β_2 (Religion & Spirituality) + β_3 (W4 Cognitive Impairment) + β_3 (Risk Factors) + r_i

Hypothesis 7: moderating effects of religion and spirituality

W5 Cognitive Impairment Predicted = (3.0)

$\beta_0 + \beta_1$ (Black) – β_2 (Religion & Spirituality) + β_3 (W4 Cognitive Impairment) + β_4 (Risk Factors) + β_5 (Black) – β_6 (Black * Religion & Spirituality) + r_i

Hypothesis 8: buffering effects of religion and spirituality

W5 Cognitive Impairment Predicted = (3.1)

$\beta_0 + \beta_1$ (Black) – β_2 (Religion & Spirituality) + β_3 (W4 Cognitive Impairment) + β_4 (Risk

Factors) + β_5 (Stressful Life Events) – β_6 (Stressful Life Events * Religion & Spirituality) + r_i

W5 Cognitive Impairment Predicted = (3.2)

$\beta_0 + \beta_1$ (Black) – β_2 (Religion & Spirituality) + β_3 (W4 Cognitive Impairment) + β_4 (Risk Factors) + β_5 (Discrimination) – β_6 (Discrimination * Religion & Spirituality) + r_i

****Hypothesis 9: 3-way interaction between race, stress, religion and spirituality**

W5 Cognitive Impairment Predicted = $\beta_0 + \beta_1$ (Black) + β_2 (Stressful Life Events) + β_3 (Religion & Spirituality) + β_4 (Black * Stressful Life Events) + β_5 (Black * Religion & Spirituality) + β_6 (Stressful Life Events * Religion & Spirituality) + β_7 (Black * Stressful Life Events * Religion & Spirituality) + β_8 (W4 Cognitive Impairment) + β_9 (Risk Factors) + r_i

****Hypothesis 10: 3-way interaction between race, discrimination, religion and spirituality**

W5 Cognitive Impairment Predicted = $\beta_0 + \beta_1$ (Black) + β_2 (Discrimination) + β_3 (Religion & Spirituality) + β_4 (Black * Discrimination) + β_5 (Black * Religion & Spirituality) + β_6 (Discrimination * Religion & Spirituality) + β_7 (Black * Discrimination * Religion & Spirituality) + β_8 (W4 Cognitive Impairment) + β_9 (Risk Factors) + r_i

**The 3-way interactions will be tested if the prior 2-way interactions are significant or not.

D. Attrition Analysis

As is true for most longitudinal data, the ACL survey encountered sample attrition over time. The present study evaluated socio-demographic differences and other factors between those in my sample and those who dropped out between waves 4 and 5 (i.e., wave 4 only respondents). Additional information about the study design of ACL, including the survey and sampling

approach can be found elsewhere (House, Lantz et al. 2005). For my analysis, I will focus on black and white younger to older aged adults enrolled in all five waves of the Americans' Changing Lives (ACL) study. Additionally, since everyday experiences of discrimination and other life stressors were only asked in later waves of the ACL study, a subsample of 1,105 adults will be analyzed in waves 4 (unweighted n=1,787) and 5 (unweighted n=1,427). This subsample represents respondents who were present in both waves 4 and 5 for a total weighted analytic sample of n=1,105 (unweighted n=1,291).

Given that sample attrition occurs in the ACL panel, I compared survey respondents who were interviewed at both waves 4 and 5 (the study sample) with those respondents who dropped out after wave 4 along a number of dimensions (see Appendix 2.4). This analysis shows that respondents in my study sample were more likely to be younger, male, have more years of education, higher incomes, and be married than those who dropped out after wave 4. In addition, study sample respondents reported more depressive symptoms, stressful life events and experiences of discrimination than those who dropped out after wave 4 (non-responders). They also reported their religious beliefs as less important and had a lower mean spiritual coping index score than non-responders. Finally, those who remained in the panel reported lower wave 4 cognitive impairment scores than those who were loss to follow-up in subsequent waves.

Readers should use appropriate caution with respect to the generalizability of the results, given that there are significant differences between respondents who dropped out after wave 4 (non-responders) and those who remained in both waves 4 and 5. For example, the loss of blacks in both waves 4 and 5 could potentially affect my ability to detect racial differences in cognitive impairment scores and further bias my findings towards the null. Likewise, those remaining in the panel had lower cognitive impairment scores than non-responders, resulting in

less variation on my outcome measure. This may again bias my findings towards the null and influence my ability to detect differences in cognitive test performance, since those with higher cognitive impairment scores dropped out after wave 4.

E. Summary

Overall, this dissertation used a number of methods to evaluate changes in cognitive functioning with respect to black and white adults and address each of the study's specific aims:

1. Descriptive statistics and group comparisons were done to assess the racial variation in cognitive impairment scores at baseline and over time. Comparison of black and white adults in terms of cognitive functioning is determined by controlling for socio-demographic, chronic conditions, health behaviors and mental health characteristics.
2. A series of multilevel growth models were done to assess the presence of racial disparities in cognitive impairment at baseline and over time. Growth models assessing the differential effects of race, controlling for socio-demographic, chronic conditions, health behaviors and mental health characteristics are estimated.
3. The effects of education, stressful life events, discrimination, religion and spirituality on wave 5 cognitive functioning are assessed using ordinary least squares (OLS) regression models. OLS models assessing the differential effects of race, controlling for socio-demographic, chronic conditions, health behaviors and mental health characteristics measured at wave 4 are estimated. Sensitivity analyses were also performed on items that were related to either the main outcome or predictors of interest in the OLS models, e.g., wave 5 cognitive impairment and the ninth item in the stressful life events index on financial stress or strain.

CHAPTER 3: RESULTS

This chapter presents the findings for this dissertation. First, baseline descriptive statistics stratified by race are reported. Second, bivariate associations between key sociodemographic and health variables stratified by race are presented for each wave. Third, the growth modeling results assessing changes in cognitive functioning across all five waves of the Americans' Changing Lives (ACL) survey are presented. Finally, the OLS regression results are presented among a subsample of black and white adults from waves 4 and 5.

I. Descriptive Results

Baseline descriptive statistics for ACL respondents are presented in Table 3.1. Both white (60.9%) and black (66.1%) unweighted subsamples are predominantly female with mean ages of approximately 55 and 53, respectively. The ACL sample is diverse with respect to education and family income. As expected, blacks have significantly lower levels of education with 52% of blacks having less than a high school education compared to 28.5% of whites.

Similar to lower levels of education, blacks also had significantly lower incomes compared to whites. For example, 27.6% of blacks comprised the lowest income mid-point ($3,125) compared to 7.5% of whites. More than 60% of whites are married, and although nearly 40% of blacks are married, the majority is single either due to being separated, divorced, widowed or never married.

Further, the two samples differ significantly in terms of their risk factors, with the exception of stroke. A greater proportion of blacks (13.8%) versus whites (7.0%) reported having diabetes. More blacks (32.9%) are also current smokers compared to whites (27.5%). At baseline, blacks also report significantly more depressive symptoms than whites.

II. Bivariate Results

Table 3.2 displays the unweighted ranges, means and racial distribution among the key dependent and independent variables at each wave for blacks and whites. As shown in the table, there is wide variation in cognitive impairment scores, with a spike at wave 3 for both blacks and whites. As expected, at each wave of the ACL survey blacks have significantly higher unweighted cognitive impairment scores than whites, as shown in Figure 3.1. Contrary to expectation, blacks report significantly fewer stressful life events than whites at wave 4, but similar levels at wave 5. Blacks also report significantly more everyday discrimination at both waves 4 and 5 than whites. Similarly, blacks report significantly greater religious attendance and participation and importance of religious or spiritual beliefs than whites at waves 4 and 5. Blacks further report greater use of spiritual coping compared to their white counterparts at wave 4 only.

Simple correlations between education, stressful life events, discrimination, religion and spirituality and cognitive impairment, stratified by race/ethnicity are presented in Table 3.3. As shown in the table, there is a significant correlation between stressful life events and everyday discrimination at wave 4 for both whites ($r = 0.221$, $p < 0.001$) and blacks ($r = 0.316$, $p < 0.001$). There is also a significant negative correlation between stressful life events and wave 4 cognitive impairment for both whites ($r = -0.068$, $p < 0.05$) and blacks ($r = -0.117$, $p < 0.05$). Likewise, there are highly significant correlations between the religion and spirituality measures for both groups. For example, religious participation is significantly correlated with both religious importance and spiritual coping at wave 4, for both whites and blacks. However, there is a significant correlation between religious importance ($r = 0.102$, $p < 0.001$) and spiritual coping ($r = 0.108$, $p < 0.001$) and

wave 4 cognitive impairment for whites, but not blacks. It is interesting that religion and spirituality are related to more cognitive impairment at waves 4 and 5 for whites, but not correlated with impairment at either wave for blacks. For blacks there is a significant negative correlation between everyday discrimination and cognitive impairment at wave 4 ($r = -0.163$, $p<0.001$). There is also a highly significant correlation between wave 4 cognitive impairment and wave 5 cognitive impairment for both groups.

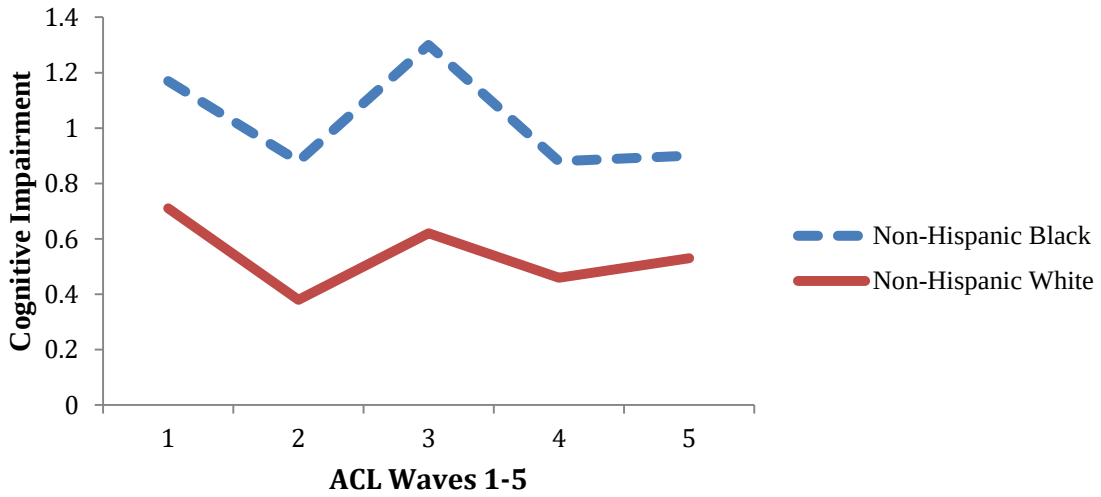
Figure 3.1 shows the racial distribution of mean cognitive impairment scores by wave (also found in Table 3.2), with a trend line added to show the overall association. Across all waves blacks have higher overall levels of cognitive impairment than whites and there is a spike at wave 3. Although the reason for this spike is unclear, it may be due to a period effect since the ACL survey did not increase the sample with new respondents or change the interview mode or items asked on the SMPSQ measure. Wave 3 of the ACL survey was conducted in 1994, the same year that then President Ronald Regan announced he had Alzheimer's disease. Some have suggested that regardless of his political views his legacy may be that he brought greater awareness and research funding to Alzheimer's and other neurodegenerative diseases (http://time.com/4473625/ronald-reagan-alzheimers-letter/ accessed August 23, 2017).

Figures 3.2 and 3.3 show the race and education distribution of cognitive errors for each item on the SPMSQ measure at baseline. Across all items blacks and those with less than a high school education make more cognitive errors than whites and those with greater levels of education, respectively. Similarly, Figure 3.4 shows that blacks, regardless of level education, make more cognitive errors on the serial 3's subtraction task than whites. Yet, for both of the date questions (i.e., today's date and day of the week) and the naming of the former president, blacks and whites across all levels of education report similar proportions of errors. However,

for the naming of the current president item, blacks at lower levels of education, including less than high school and high school graduate make more cognitive errors than whites with similar levels of education. But at higher levels of education, i.e., some college and college graduate and beyond blacks and whites report a similar proportion of errors.

Figure 3.5 shows the distribution of mean cognitive impairment scores by wave for those "surviving" to wave 5. Across all waves there is variation in the cognitive impairment scores for those who survived to wave 5. To look closer at this, I further developed a multiple imputation model and conducted several imputations for missing values on the wave 5 cognitive impairment scores using the prior waves scores, demographic variables, and health conditions (see Appendix 2.4). The results showed no significant differences between imputed values and those observed in my final regression models.

Figure 3.1 Racial Distribution of Mean Cognitive Impairment by Wave. Americans' Changing Lives Study (ACL), 1986-2011 (waves 1-5).



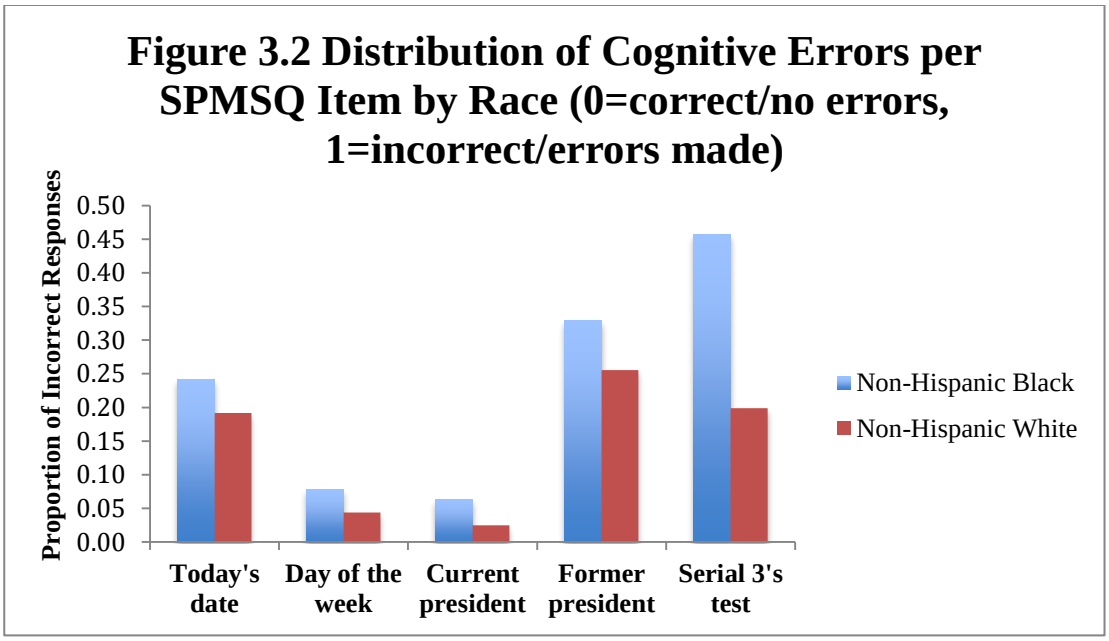


Figure 3.2 Distribution of Cognitive Errors per SPMSQ Item by Race (0=correct/no errors, 1=incorrect/errors made)
Proportion of Incorrect Responses
0.50
0.45
0.40
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
Today's date
Day of the week
Current president
Former president
Serial 3's test
Non-Hispanic Black
Non-Hispanic White

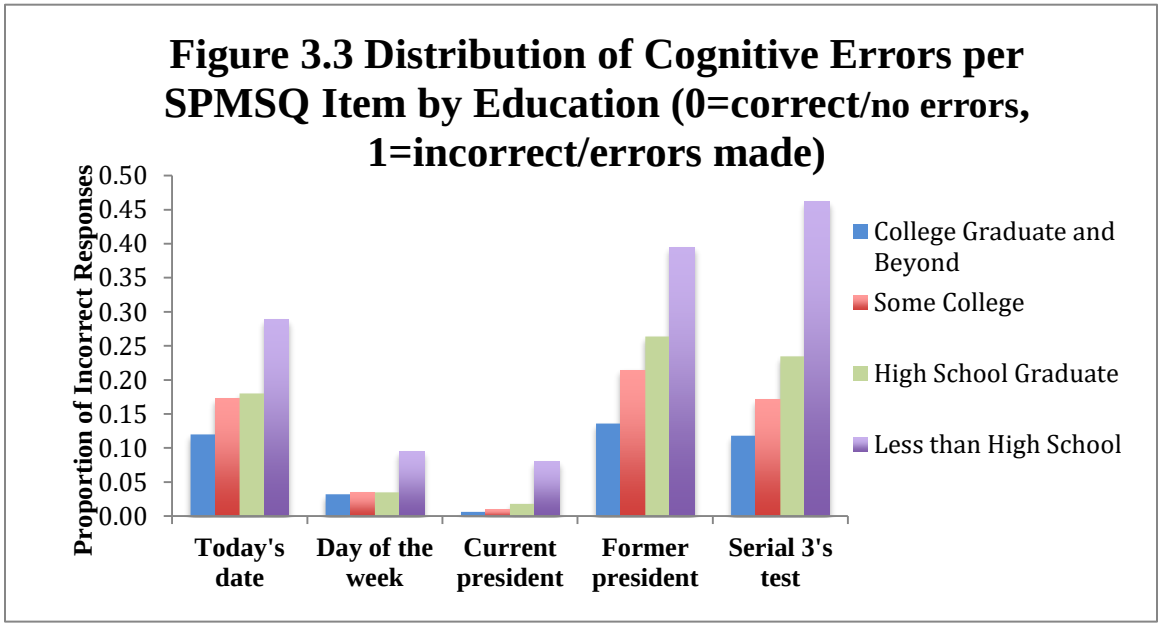


Figure 3.3 Distribution of Cognitive Errors per SPMSQ Item by Education (0=correct/no errors, 1=incorrect/errors made)
Proportion of Incorrect Responses
0.50
0.45
0.40
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
Today's date
Day of the week
Current president
Former president
Serial 3's test
College Graduate and Beyond
Some College
High School Graduate
Less than High School

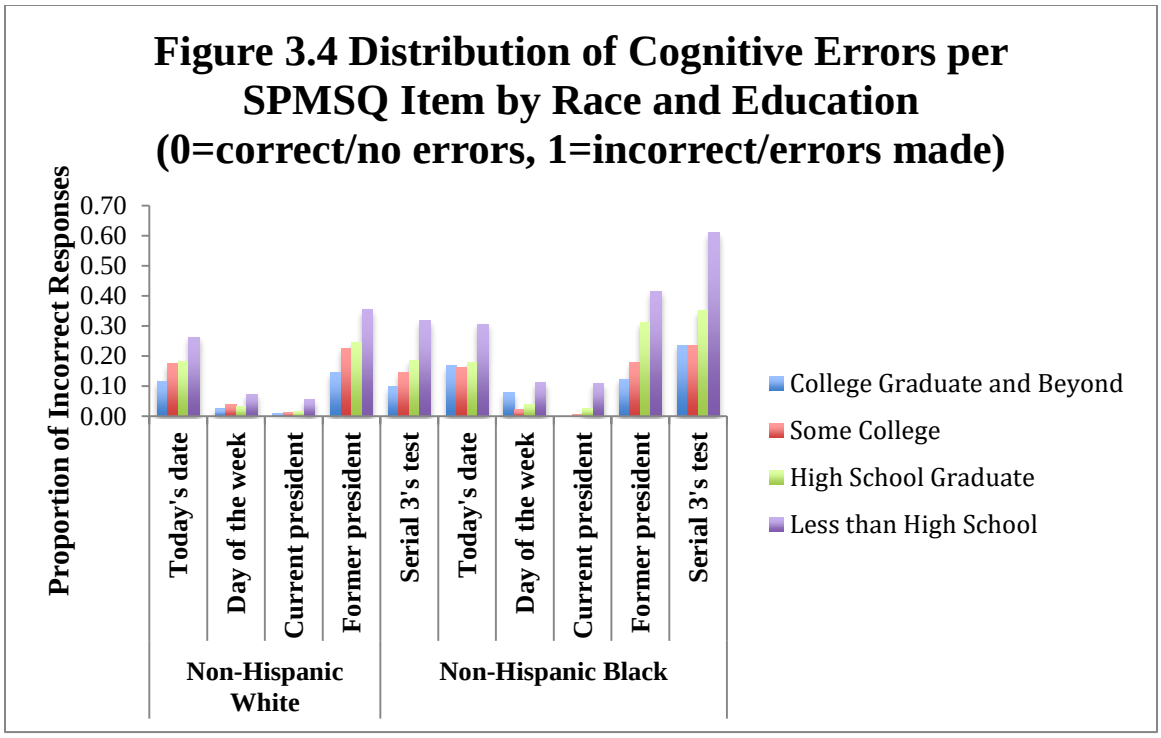


Figure 3.4 Distribution of Cognitive Errors per SPMSQ Item by Race and Education (0=correct/no errors, 1=incorrect/errors made)
Proportion of Incorrect Responses
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0.00
Today's date
Day of the week
Current president
Former president
Serial 3's test
Today's date
Day of the week
Current president
Former president
Serial 3's test
Non-Hispanic White
Non-Hispanic Black
College Graduate and Beyond
Some College
High School Graduate
Less than High School

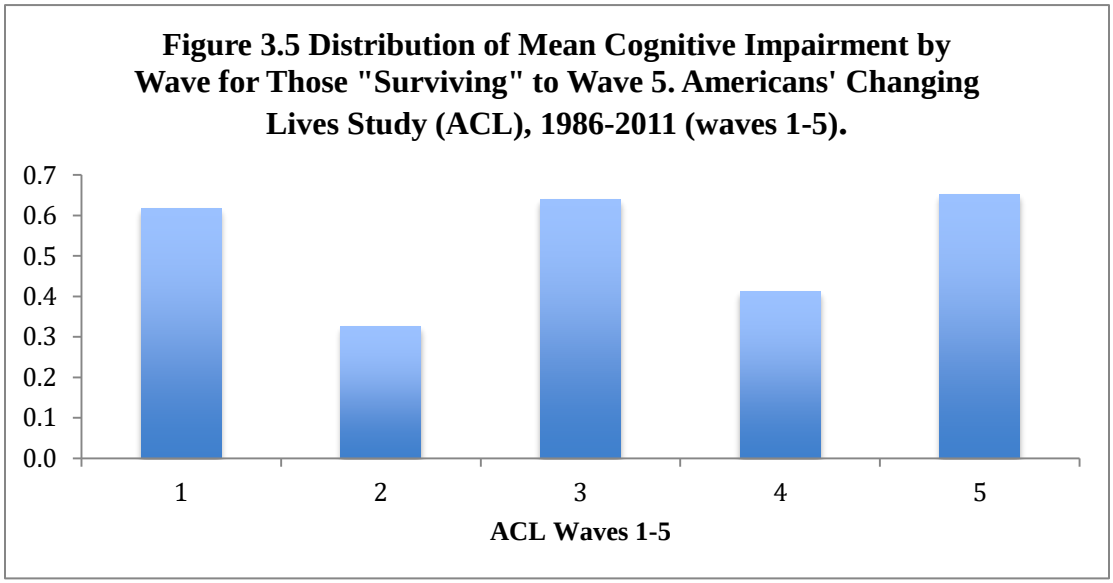


Figure 3.5 Distribution of Mean Cognitive Impairment by Wave for Those "Surviving" to Wave 5. Americans' Changing Lives Study (ACL), 1986-2011 (waves 1-5).
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
1
2
3
4
5
ACL Waves 1-5

Table 3.1 Baseline Characteristics for Adult Respondents, Stratified by Race/Ethnicity (unweighted). Americans' Changing Lives Study, 1986 (wave 1) (N=3,617).

Characteristic	Non-Hispanic Whites (percentage)	Non-Hispanic Blacks (percentage)	p-value for difference[a]
DEMOGRAPHICS			
Overall Proportion[b]	61.0	32.0	
Age, mean (SD)	54.9 (17.70)	52.8 (17.33)	
Female	60.9	66.1	= .003
Education			< .001
Less than High School	28.5	52.0	
High School Graduate	32.9	23.7	
Some College	21.6	16.6	
College Graduate and Beyond	17.0	7.7	
Baseline Income (adjusted mid-points)			< .001
$3,125	7.5	27.6	
$7,250	15.1	22.2	
$12,010	13.4	14.9	
$17,210	11.7	7.9	
$22,040	10.0	5.7	
$26,910	9.3	5.7	
$33,450	12.9	6.9	
$46,940	12.3	6.7	
$65,950	4.4	1.6	
$85,230	3.5	0.9	
Marital Status[c]			
Married	62.4	38.9	< .001
RISK FACTORS			
Diabetes			
Yes	7.0	13.8	< .001
Stroke			
Yes	0.8	1.0	= .424
Smoking Status			
Current Smoker	27.5	32.9	= .001
Depressive Symptoms (CESD-11), mean (SD)	15.16 (3.89)	16.51 (4.36)	< .001

[a]Tests of difference between races: OLS regression was used for age and depressive symptoms; Pearson's chi-square for gender, marital status, stroke, diabetes and smoking status; ordinal logit regression for education and income.

[b]Other races excluded Non-Hispanic Native American (n=44), Non-Hispanic Asian (n=30) and Hispanic (n=182).

[c]Not married includes separated, divorced, widowed and never married.

SD = standard deviation.

Table 3.2 Stratified Analysis By Non-Hispanic White and Black for Cognitive Impairment, Stressful Life Events, Discrimination, Religion and Spirituality (unweighted). Americans' Changing Lives Study (ACL), 1986-2011 (waves 1-5).

	Wave 1 (Baseline)	Wave 2	Wave 3	Wave 4	Wave 5
Study Measures	**N(%)**	**N(%)**	**N(%)**	**N(%)**	**N(%)**
Race Distribution					
White	2,205 (61.0%)[a]	1,818 (63.4%)	1,670 (65.3%)	1,242 (69.6%)	916 (64.2%)
Black	1,156 (32.0%)[a]	860 (30.0%)	725 (28.3%)	438 (24.5%)	401 (28.1%)
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**
Cognitive Impairment (range 0-5)					
White	0.71 (0.90)	0.38 (0.73)	0.62 (0.87)	0.46 (0.76)	0.53 (0.81)
Black	1.17 (1.18)***	0.88 (1.13)***	1.30 (1.22)***	0.88 (0.93)***	0.90 (0.86)***
Stressful Life Events (range 0-10)					
White				0.78 (1.08)	0.73 (1.20)
Black				0.65 (1.14)**	0.70 (1.28)
[b]Discrimination (wave 4 range 0-15; wave 5 range 0-9)					
White				2.38 (2.78)	2.00 (1.99)
Black				3.31 (3.18)***	2.66 (2.37)***
Religious Attendance & Participation (range 0-10)					
White				3.79 (3.02)	3.38 (2.96)
Black				5.47 (3.00)***	5.17 (3.03)***
Importance of Religious or Spiritual Beliefs (range 0-3)					
White				2.47 (0.84)	2.38 (0.92)
Black				2.87 (0.46)***	2.86 (0.48)***
[c]Spiritual Coping, wave 4 (range 0-10)					
White				5.62 (2.53)	
Black				7.21 (1.41)***	

[a]Percentages do not add to 100, other races excluded include Non-Hispanic Native American (n=44), Non-Hispanic Asian (n=30) and Hispanic (n=182).

[b]Everyday discrimination scale response categories: 3 = a few times a month or more, 2 = a few times a year, 1 = less than once a year and 0 = never.
[b]Wave 4 discrimination uses a 5-item scale. Wave 5 discrimination uses a 3-item scale.
[c]Use of spiritual coping was only asked in wave 4.
NOTE: OLS regression was used to tests for differences between races in cognitive impairment symptoms and other study measures.
SD = standard deviation.
***p < .05; **p < .01; ***p < .001**

Table 3.3 Correlations Between Stressful Life Events, Discrimination, Religion, Spirituality and Cognitive Impairment, Stratified by Race/Ethnicity (Non-Hispanic White estimates on top and Black estimates below) (unweighted). Americans' Changing Lives Study (ACL), 2001/2002 (wave 4) and 2011 (wave 5).

Variables	1. Stressful Life Events, wave 4	2. Everyday Discrimination, wave 4	3. Religious Participation, wave 4	4. Religious Importance, wave 4	5. Spiritual Coping, wave 4	6. Cognitive Impairment, wave 4	7. Cognitive Impairment, wave 5
1. Stressful Life Events, wave 4	Whites Blacks						
2. Everyday Discrimination, wave 4	0.221*** 0.316***	—					
3. Religious Participation, wave 4	-0.045 -0.026	-0.041 -0.043	—				
4. Religious Importance, wave 4	0.071* 0.027	-0.003 -0.055	0.462*** 0.286***	—			
5. Spiritual Coping, wave 4	0.050 -0.015	-0.044 -0.110*	0.545*** 0.285***	0.763*** 0.558***	—		
6. Cognitive Impairment, wave 4	-0.068* -0.117*	-0.035 -0.163***	0.004 -0.021	0.102*** -0.008	0.108*** -0.013	—	
7. Cognitive Impairment, wave 5	0.0001 0.0129	-0.059 0.002	0.040 0.063	0.092** 0.029	0.076* 0.078	0.341*** 0.322***	—

***p < .05; **p < .01; ***p < .001**

III. Growth Modeling Results

Aim 1 poses a series of questions within the multilevel growth modeling framework to evaluate changes in cognitive functioning with respect to black and white adults. Specifically, are there individual changes in cognitive performance over time? And is the change constant over time (versus is it accelerating or declining)? If so, are there differences in those individual changes by race? What are the predictors of the racial differences? Based on the nested structure of the data (i.e., repeated measures of cognitive impairment within individuals) it is appropriate to use growth modeling techniques to account for the multiple observations within each individual. Research on individual change identifies an explicit model of individual growth. One advantage of using growth models to study individual change is that the "number and spacing of measurement occasions may vary across persons" (Raudenbush and Bryk, 2002, pp.161-162). Aim 1 uses a growth curve model to test for racial differences in baseline levels of function and the rate of change in cognitive impairment symptoms over time. Thus, the analysis of Aim 1 provides the groundwork for Aims 2 and 3.

Table 3.4 shows the results for age centered at 75. Model 1 in Table 3.4, the null or empty model, tests if there are individual differences in mean levels of cognitive impairment. The null model contains no predictors, and is intended only to partition the variance in cognitive impairment into between-individuals (σ_ε^2) and within-individuals (τ_{00}) portions. The random effects (or level-2 covariance matrix), therefore, consists only of the intercept variance τ_{00}, whereas the level-1 residual variance is denoted by σ_ε^2. The variability in cognitive impairment (or number of errors) was nearly evenly split between levels, with an estimated between-person variability of $\sigma_\varepsilon^2 = 0.48$ and a within-person variability of $\tau_{00} = 0.52$ (intraclass correlation coefficient = 0.48, indicating that 48% of the variability was between-individuals and most of the

variability ~52% is within-individuals). The intercept in this model is significant (*b*=0.792, SE=0.014, *p*<0.0001) and indicates that the mean cognitive impairment score for the overall sample is 0.79 when age is 75. This is the overall initial status in cognitive impairment for blacks and whites. Model 2 (linear age term) shows that on average, individuals mean cognitive impairment scores change over time. In other words, it looks at whether people as a group change over time or on average, does the mean level of cognitive impairment change with age. It shows that the expected change in cognitive impairment for a 1-unit increase in age is 0.011. Stated differently, every year age increases cognitive impairment increases by 0.011. Model 3 (quadratic age term) determines whether this change is constant over time versus is the change accelerating or decelerating. The quadratic age terms shows that the rate of change in cognitive ability changes by 0.0004 across all fives waves of the ACL. The positive quadratic age slope indicates there was a significant convex relationship (i.e., the trajectory of cognitive functioning is convex to the time axis, with a single trough) between the quadratic age term and cognitive impairment, demonstrating that as age increases cognitive impairment is accelerating at a faster rate. A positive quadratic value also indicates the curvature is upwards and a quicker acceleration in the slope. Thus, the effect of age on cognition changes depending on your age. In other words, cognitive functioning gets worse with increasing age or as people get older. Model 4 (linear age and race interaction term) asks does the effect of age on cognitive impairment vary by race? In other words, do blacks have different linear age slopes than whites? This model shows that the intercept γ_{00} (*b*=0.740, SE=0.019, *p*<0.0001), the linear age slope γ_{10} (*b*=0.023, SE=0.001, *p*<0.0001), quadratic age slope γ_{20} (*b*=0.0004, SE=0.00003, *p*<0.0001) and race effect γ_{01} (*b*=0.747, SE=0.034, *p*<0.0001) were all significantly greater than zero. The intercept is

an overall initial status for whites and shows that the expected mean cognitive impairment score for whites is 0.740 when age is 75. The linear age term 0.023 is the instantaneous growth rate specific to whites when age is 75. The quadratic age term is the mean acceleration and shows how much the slope of cognitive functioning is changing by at each age. This term indicates that the instantaneous rate of change in cognitive ability changes by 0.0004 as age increases. The race effect is the difference between blacks and whites in initial status in the level of cognitive impairment and shows that blacks have a 0.747 higher mean level of cognitive impairment compared to whites at age 75. Blacks make approximately one more (0.75) cognitive error than whites on average at age 75. Further, there was a significant interaction between age and race on cognitive impairment; cognitive impairment levels increased at a greater rate over time for blacks compared to whites (b=0.011, SE=0.001, p<0.0001). The black-white gap in mean cognitive impairment scores widened by 0.011 per year of age, indicating that blacks on average made more errors and demonstrated a more rapid rate of cognitive decline than whites. Model 5 includes all covariates and shows the interaction term remains significant after adjusting for sociodemographic, chronic conditions, health behaviors and mental health characteristics (b=0.009, SE=0.001, p<0.0001). Likewise, the intercept (b=1.298, SE=0.046, p<0.0001), the linear age term (b=0.017, SE=0.001, p<0.0001) quadratic age slope (b=0.0004, SE=0.00003, p<0.0001) and race effect (b=0.483, SE=0.034, p<0.0001) also remain significant, although the race effect is reduced. Model 5 in Table 3.4 shows that controlling for sociodemographic, chronic conditions, health behaviors and mental health characteristics reduces the race effect by 20% [(0.747-0.483)/ 0.747] from the previous Model 4, which does not make adjustments for these covariates. Although, the race effect in Model 5 is reduced it remains significant indicating that blacks have worse cognitive functioning than whites at baseline over and above model

covariates.

Figure 3.6 demonstrates the effect of centered age on cognitive impairment varies over time by race. This figure presents the interaction between centered age (at 75) and race from Model 5 of Table 3.4 for ages 25 to 105. The effect of race on baseline scores and in the rate of change in scores is apparent in the separation of model-predicted scores at each age. There are clear racial differences in baseline scores and in the rate of change in scores such that the difference in predicted values of cognitive errors at each age differs for blacks and whites. Specifically, blacks have worse cognitive functioning and make more cognitive errors than whites at baseline or age 25 and these disparities widen over time, after adjusting for model covariates. Figure 3.7 shows the interaction between centered age (at 75) and race without education. As can be seen the relationship remains the same.

All of these models include a random intercept to test the variability in each person's starting point or their initial status in cognitive impairment symptoms. Further, random slopes were also tested and found not to be significant (model not shown) for the linear and quadratic age terms, suggesting that blacks and whites have the same degree of change (slopes) over time. A model with random slopes for the linear age term answers a central question in the multilevel analysis: are there individual changes in cognitive performance over time? This model looks at the overall variability in each person's trajectory or change in cognitive impairment over time and tests whether these slopes vary across individuals. The random slope for the linear age term was not significant after adding the quadratic (or squared) age term, suggesting similar cognitive impairment slopes across individuals and that the quadratic age term is accounting for the individual variability or differences in these slopes.

Appendix 3.1 shows the results of age not centered and the Figure 3.1a plots the interaction between uncentered or real ages and race from Model 5 for ages 25 to 95. This model was not used in the main analysis because it would be predicting cognitive impairment beyond the ACL data temporal limits. Using uncentered ages represents the expected value of cognitive impairment at age 0 or birth, an age that precedes the onset of data collection (Singer and Willett 2003, Hox 2010).

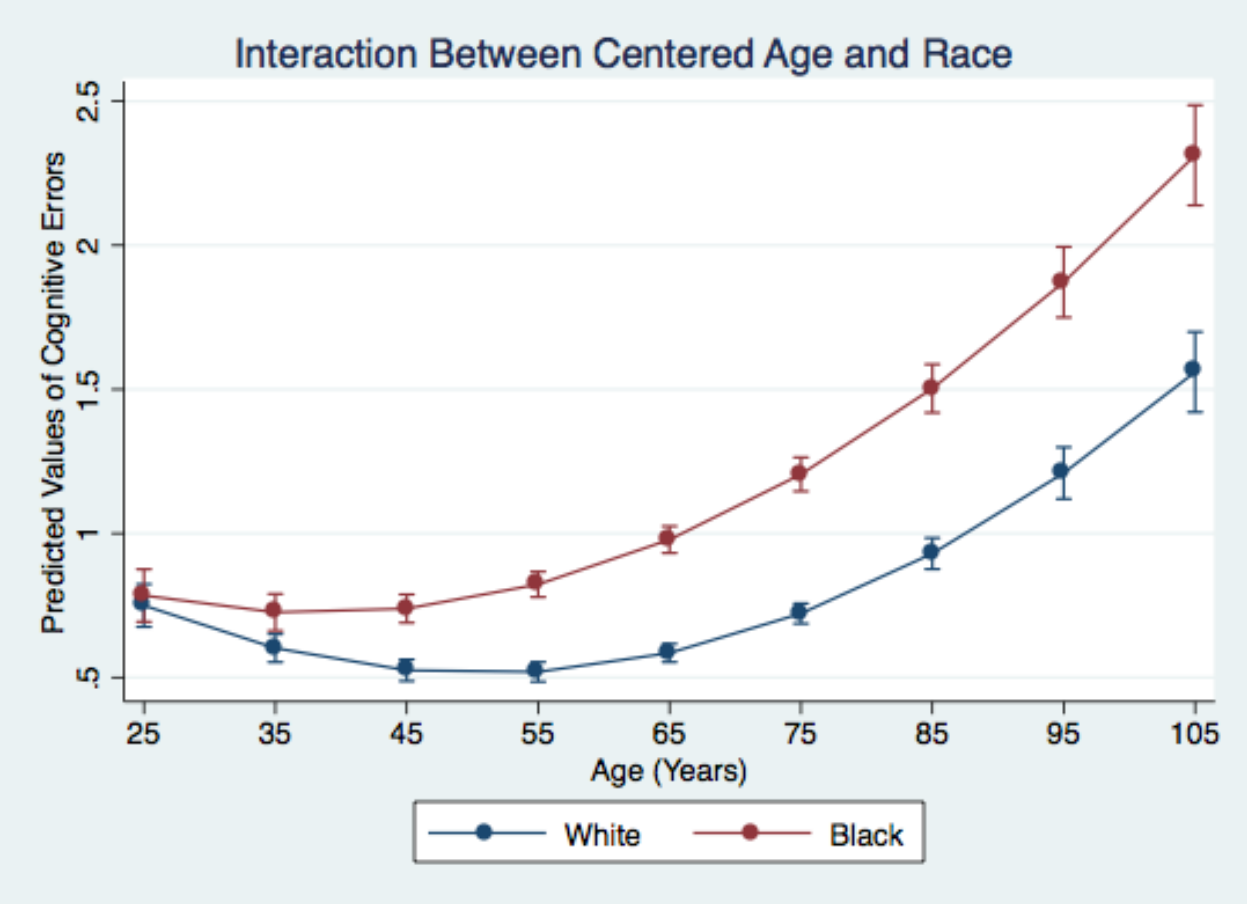
Table 3.4 Mixed Models of Cognitive Impairment on Sociodemographic, Chronic Conditions, Health Behaviors and Mental Health Characteristics for Non-Hispanic Black and White Respondents. Age Centered at 75. Americans' Changing Lives Study (ACL), 1986-2011 (waves 1-5)

	Model 1	Model 2	Model 3	Model 4	Model 5
Fixed Effects	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)
Age (centered at age 75)		0.011 (0.0006)***	0.024 (0.001)***	0.023 (0.001)***	0.017 (0.001)***
Age^2			0.0004 (0.00003)***	0.0004 (0.00003)***	0.0004 (0.00003)***
Black Race (ref=White)				0.747 (0.034)***	0.483 (0.034)***
Age*Black Race Interaction				0.011 (0.001)***	0.009 (0.001)***
Female					0.007 (0.025)
Education (ref=less than high school)					
High School Graduate					-0.327 (0.032)***
Some College					-0.442 (0.036)***
College Graduate and Beyond					-0.560 (0.042)***
Baseline Income (adjusted mid-points)[a]					
$7,250					-0.226 (0.043)***
$12,010					-0.336 (0.046)***
$17,210					-0.409 (0.051)***
$22,040					-0.395 (0.054)***
$26,910					-0.362 (0.055)***
$33,450					-0.407 (0.052)***
$46,940					-0.376 (0.054)***

$65,950					-0.390 (0.072)***
$85,230					-0.428 (0.080)***
Marital Status (ref=married)					
Separated, Divorced or Widowed					0.006 (0.022)
Never Married					0.011 (0.039)
Diabetes[b]					0.041 (0.029)
Stroke[b]					0.323 (0.067)***
Newly Diagnosed Stroke					0.149 (0.059)**
Current Smoker[b]					0.018 (0.026)
Depressive Symptoms					0.009 (0.006)
Intercept	0.792 (0.014)***	0.951 (0.017)***	0.967 (0.017)***	0.740 (0.019)***	1.298 (0.046)***
Random Effects (variance components)					
Random Intercept, τ_{00} (between-person variance)	0.48	0.44	0.44	0.35	0.26
Residual, σ_ε^2 (within-person variance)	0.52	0.52	0.51	0.51	0.52
X^2		271.64***	425.54***	982.68***	1777.33***
d.f.	0	1	2	4	24

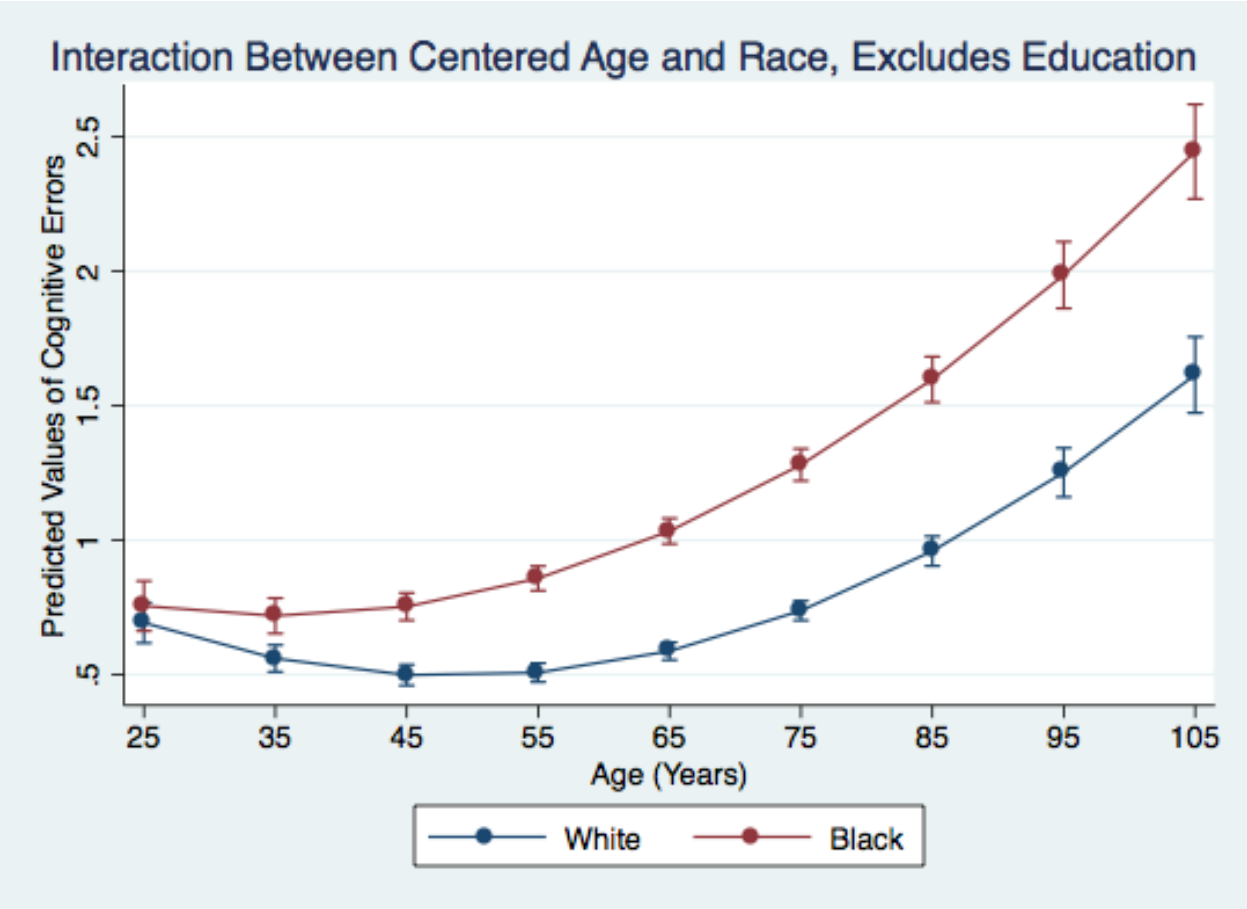
[a]$3,125 is the reference category.
[b]1=condition reported.
*p < .05; **p < .01; ***p < .001

Figure 3.6 The Relationship Between Centered Age at 75 and Cognitive Impairment as Moderated by Race, Americans' Changing Lives Study (Ages 25-105)



NOTE: Graph of Model 5 from Table 3.4 adjusting for sociodemographic, chronic conditions, health behaviors and mental health characteristics.

Figure 3.7 The Relationship Between Centered Age at 75 and Cognitive Impairment as Moderated by Race and Excluding Education, Americans' Changing Lives Study (Ages 25-105)



NOTE: Graph of Model 5 from Table 3.4 removing education and adjusting for sociodemographic, chronic conditions, health behaviors and mental health characteristics.

IV. Multivariate Results

A series of OLS regression models using the last two waves of the ACL survey -- where the predictor variables are measured at wave 4 and cognitive impairment is measured at waves 4 and 5 -- are estimated to examine the influence of specific covariates on black-white differences. The OLS regression Model 1 (Table 3.5) for aim 2 shows a black-white disparity; whereby,

blacks have significantly higher cognitive impairment score than whites (β = 0.379, $p < .001$). This disparity remains significant but is greatly reduced after adjusting for the simultaneous effects of education, stressful life events and discrimination and wave 4 (baseline) cognitive impairment in Model 2 (β = 0.153, $p < .05$). However, only the education coefficient is statistically significant, while stressful life events and discrimination are not. This finding indicates that only education rather than the combined effects of these variables confounds the association between race and cognitive impairment, such that this relationship is attenuated once education is controlled for. Although education level accounts for a significant amount of racial disparities it does not fully explain black-white differences. Thus, to further explore black-white differences in cognitive impairment at wave 5, I also conducted a sensitivity analysis examining the relative contribution of education in relationship to age, depressive symptoms, and baseline (wave 4) cognition (see Appendix 3.5 for results). The black-white disparity is no longer significant once the interactions between race/ethnicity, education, stressful life events and discrimination are controlled for in Models 3-5. Although I had anticipated the interactions for blacks and whites would be significant based on previous studies examining the impact of stressful life events and discrimination on mental health (Kessler 1979, Williams, Yan et al. 1997, Byrd 2012) and physical health (Schulz, Gravlee et al. 2006, Hicken, Lee et al. 2013, Hicken, Lee et al. 2014). None of the interaction terms are significant in these models, suggesting that education (F (3, 45) = 0.69, $p = .564$), stressful life events (F (1, 45) = 0.31, $p = .581$) and discrimination (F (1, 45) = 2.37, $p = .131$) do not moderate the race-cognitive impairment association.

Models 2-5 further shows that education has an independent effect on cognitive

impairment; whereby, individuals reporting less education have higher wave 5 cognitive impairment scores than those reporting more education, independent of their wave 4 cognitive impairment score and the risk factors known to influence cognition. Age, depressive symptoms and wave 4 cognitive impairment all remain significant in these models after adjusting for model covariates, demonstrating that these factors also have an independent effect on cognitive impairment such that older individuals, those with higher depressive symptoms and baseline cognitive impairment scores have higher levels of impairment than those who are younger and have lower levels of depressive symptoms and baseline cognitive impairment scores. Overall, these models explain approximately 25% (r^2 range = .246 to .247) of the variation in cognitive impairment scores.

Tables 3.6 and 3.7 for aim 3 tests the relationship between race/ethnicity and cognitive impairment and the protective influence of religion and spirituality. Model 1 shows that none of the religion and spirituality measures are significant. This finding indicates that individuals reporting higher levels of religion and spirituality do not have a lower wave 5 cognitive impairment score compared to those reporting lower levels of religion and spirituality, independent of their wave 4 cognitive impairment score and the risk factors known to influence cognition. Likewise, the interactions between race/ethnicity and religion/spirituality in Models 2 thru 4 are insignificant, suggesting that religion and spirituality do not moderate the relationship between race/ethnicity and cognitive impairment. As a result at higher levels of religion and spirituality there is no black-white difference in cognitive impairment scores, such that blacks reporting high levels of religion and spirituality do not have lower levels of wave 5 cognitive impairment when compared to whites with high levels of religion and spirituality. Table 3.7 shows the interactions separately for stressful life events, discrimination and religion and

spirituality. Models 2-4 show that none of the religion and spirituality measures buffers the relationship between stressful life events and cognitive impairment. The interactions between stressful life events and religion/spirituality including religious participation (F (1, 45) = 0.47, p = .496), religious importance (F (1, 45) = 0.19, p = .666), and spiritual coping (F (1, 45) = 1.36, p = .249) were all non-significant. Similarly, Models 5-7 shows that none of the interactions between discrimination and measures of religion/spirituality were significant including religious participation (F (1, 45) = 1.23, p = .274), religious importance (F (1, 45) = 0.63, p = .431), and spiritual coping (F (1, 45) = 0.04, p = .840). These findings suggest that religion and spirituality do not modify the association between discrimination and cognitive impairment. In all of these models (1-7) blacks have significantly higher cognitive impairment scores than whites, even after adjusting for model covariates. Further, the 3-way interactions between race/ethnicity, stressful life events, discrimination, religion and spirituality were also found to be non-significant (see Table 3.8).

I also conducted sensitivity analyses excluding those reporting financial stress or strain on the stressful life events scales to examine the possibility that this item might be closely related to other SES predictors of interest. Additionally, sensitivity analyses was done on the discriminatory event "people act as if they think you are not smart" because this could be related to changes in someone's cognitive impairment, the main outcome of interest. I found no evidence that excluding these items significantly affected the results; excluding these items from the analyses produced similar results as observed when including them (data shown in Appendices 3.2 and 3.3).

Table 3.5 Nested Ordinary Least Squares Models of the Effects of Education, Stressful Life Events, and Discrimination on Cognitive Impairment Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples from 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Variables	beta (SE)	beta (SE)	beta (SE)	beta (SE)	beta (SE)
Black Race (ref=White)	0.379*** (.063)	0.153* (.064)	0.010 (.171)	0.105 (.113)	0.038 (.088)
Age		0.015*** (.002)	0.015*** (.002)	0.015*** (.002)	0.014*** (.002)
Female		0.020 (.062)	0.027 (.059)	0.026 (.059)	0.026 (.061)
Education (ref=less than high school)					
High School Graduate		-0.259* (.100)	-0.300** (.110)	-0.262** (.100)	-0.266** (.100)
Some College		-0.333*** (.096)	-0.361*** (.102)	-0.336*** (.095)	-0.342*** (.094)
College Graduate and Beyond		-0.411*** (.082)	-0.444*** (.085)	-0.414*** (.081)	-0.419*** (.080)
Baseline Income (re-scaled per $10k)		-0.015 (.013)	-0.015 (.013)	-0.015 (.013)	-0.016 (.013)
Married (ref=not married)[a]		-0.017 (.091)	-0.020 (.089)	-0.021 (.089)	-0.014 (.091)
Diabetes		-0.064 (.121)	-0.080 (.128)	-0.073 (.126)	-0.064 (.124)
Current Smoker (ref=non-current smoker)		0.014 (.051)	0.012 (.049)	0.014 (.049)	0.018 (.050)
Depressive Symptoms		0.017*** (.005)	0.016*** (.005)	0.016*** (.005)	0.017*** (.005)
Stressful Life Events		0.017 (.017)		0.010 (.017)	
Everyday		-0.008 (.011)			-0.010 (.011)

Discrimination					
Cognitive Impairment, Baseline (wave 4)		0.405*** (.052)	0.400*** (.053)	0.404*** (.052)	0.403*** (.051)
Race*Education Interaction (ref=less than high school)					
Black*High School Graduate			0.301 (.224)		
Black*Some College			0.110 (.226)		
Black*College Graduate and Beyond			0.090 (.217)		
Black*Stressful Life Events				0.023 (.042)	
Black*Discrimination					0.030 (.020)
Intercept	0.474 ***(.026)	-0.133 (.174)	-0.108 (.173)	-0.150 (.175)	-0.100 (.166)
R^2	0.018	0.246	0.247	0.246	0.247

[a]Not married includes separated, divorced, widowed and never married.
NOTE: Estimates are weighted to account for the sampling design.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

Table 3.6 Nested Ordinary Least Squares Models of the Effects of Religion and Spirituality on Wave 5 Cognitive Impairment Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples from 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	Model 2	Model 3	Model 4
	beta (SE)	beta (SE)	beta (SE)	beta (SE)
Black Race (ref=White)	0.160** (.058)	0.155 (.100)	0.061 (.285)	0.032 (.279)
Age	0.015*** (.002)	0.015*** (.002)	0.015*** (.002)	0.015*** (.002)
Female	0.028 (.062)	0.028 (.059)	0.020 (.060)	0.033 (.061)
Education (ref=less than high school)				
High School Graduate	-0.261** (.097)	-0.263** (.099)	-0.262** (.097)	-0.267** (.098)
Some College	-0.334*** (.093)	-0.338*** (.094)	-0.336*** (.092)	-0.339*** (.093)
College Graduate and Beyond	-0.406*** (.078)	-0.415*** (.080)	-0.412*** (.092)	-0.413*** (.079)
Baseline Income (per $10k)	-0.015 (.013)	-0.016 (.013)	-0.015 (.013)	-0.016 (.013)
Married (ref=not married)[a]	-0.020 (.088)	-0.022 (.087)	-0.022 (.102)	-0.021 (.018)
Diabetes	-0.062 (.126)	-0.074 (.128)	-0.072 (.125)	-0.077 (.125)
Current Smoker (ref=non-current smoker)	0.016 (.052)	0.014 (.050)	0.017 (.054)	0.018 (.054)
Depressive Symptoms	0.017*** (.005)	0.016*** (.005)	0.017*** (.005)	0.017*** (.005)
Religious Participation[b]	0.0004 (.009)	-0.001 (.007)		0.014 (.015)
Religious Importance[b]	0.057 (.040)		-0.014 (.016)	
Spiritual Coping[b]	-0.017 (.014)			-0.003 (.008)
Cognitive Impairment, Baseline (wave 4)	0.402*** (.053)	0.401*** (.052)	0.402*** (.053)	0.403*** (.053)
Race*Religion & Spirituality Interactions				
Black*Religious Participation		-0.00003 (.017)		
Black*Religious Importance			0.047 (.043)	
Black*Spiritual Coping				0.018 (.040)
Intercept	-0.185 (.169)	-0.135 (.168)	-0.171 (.165)	-0.121 (.166)
R^2	0.245	0.244	0.245	0.245

[a]Not married includes separated, divorced, widowed and never married.
[b]Separate models were ran for each religion and spirituality measure to test for collinearity and found to be non-significant.
NOTE: Estimates are weighted to account for the sampling design.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

Table 3.7 Nested Ordinary Least Squares Models of the Effects of Religion and Spirituality on Stressful Life Events, Discrimination on Cognitive Impairment Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples from 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	beta (SE)	beta (SE)	beta (SE)				beta (SE)
Black Race (ref=White)	0.161** (.063)	0.150* (.063)	0.139* (.060)	0.142* (.057)	0.165** (.064)	0.156* (.062)	0.163** (.060)
Age	0.014*** (.002)	0.015*** (.003)	0.015*** (.002)	0.015*** (.002)	0.015*** (.002)	0.014*** (.002)	0.014*** (.002)
Female	0.021 (.065)	0.027 (.058)	0.017 (.061)	0.029 (.062)	0.022 (.059)	0.016 (.062)	0.031 (.063)
Education (ref=less than high school)							
High School Graduate	-0.255** (.102)	-0.258** (.102)	-0.262** (.097)	-0.272** (.097)	-0.259** (.102)	-0.260* (.100)	-0.266** (.100)
Some College	-0.330*** (.097)	-0.331*** (.098)	-0.335*** (.093)	-0.345*** (.094)	-0.336*** (.096)	-0.334*** (.094)	-0.340*** (.094)
College Graduate and Beyond	-0.401*** (.082)	-0.408*** (.083)	-0.410*** (.079)	-0.419*** (.080)	-0.413*** (.082)	-0.412*** (.080)	-0.415*** (.081)
Baseline Income (per $10k)	-0.015 (.013)	-0.015 (.013)	-0.015 (.013)	-0.016 (.013)	-0.016 (.013)	-0.015 (.013)	-0.017 (.013)
Married (ref=not married)[a]	-0.017 (.090)	-0.018 (.089)	-0.016 (.093)	-0.018 (.091)	-0.023 (.090)	-0.015 (.091)	-0.019 (.090)
Diabetes	-0.050 (.120)	-0.077 (.126)	-0.061 (.123)	-0.063 (.129)	-0.068 (.122)	-0.059 (.120)	-0.068 (.123)
Current Smoker (ref=non-current smoker)	0.016 (.053)	0.012 (.050)	0.014 (.050)	0.017 (.051)	0.016 (.050)	0.018 (.050)	0.018 (.051)
Depressive Symptoms	0.017*** (.005)	0.016*** (.005)	0.016*** (.005)	0.016*** (.005)	0.018*** (.005)	0.018*** (.005)	0.017*** (.005)
Religious Participation	0.001 (.009)	0.006 (.011)			0.007 (.009)		0.014 (.015)
Religious Importance	0.059 (.040)		-0.002 (.042)			0.034 (.030)	
Spiritual Coping	-0.017 (.014)			-0.015 (.013)			-0.004 (.012)
Cognitive Impairment, Baseline (wave 4)	0.405*** (.053)	0.403*** (.052)	0.402*** (.053)	0.403*** (.054)	0.402*** (.052)	0.401*** (.052)	0.402*** (.053)
Stressful Life Events (SLE)	0.016 (.016)	0.028 (.028)	-0.016 (.064)	-0.038 (.043)			

Everyday Discrimination	-0.009 (.011)				0.002 (.014)	0.008 (.021)	-0.010 (.019)
Stressful Life Events*Religion & Spirituality Interactions							
SLE*Religious Participation		-0.004 (.006)					
SLE*Religious Importance			0.012 (.027)				
SLE*Spiritual Coping				0.009 (.008)			
Discrimination*Religion & Spirituality Interactions							
Discrimination* Relig Participation					-0.003 (.002)		
Discrimination* Relig Importance						-0.006 (.008)	
Discrimination* Spiritual Coping							0.001 (.003)
Intercept	-0.182 (.173)	-0.186 (.182)	-0.145 (.187)	-0.062 (.177)	-0.147 (.170)	-0.198 (.173)	-0.090(.174)
R^2	0.246	0.244	0.246	0.246	0.246	0.247	0.245

[a]Not married includes separated, divorced, widowed and never married.
NOTE: Estimates are weighted to account for the sampling design.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

Table 3.8 Nested Ordinary Least Squares Models of the 3-way Interactions Between Race/Ethnicity, Stressful Life Events, Discrimination, Religion and Spirituality Predicting Cognitive Impairment Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples from 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	beta (SE)	beta (SE)	beta (SE)			
Black Race (ref=White)	0.247 (.193)	0.737 (.558)	0.429 (.456)	0.034 (.175)	0.374 (.659)	0.091 (.475)
Age	0.015*** (.003)	0.015*** (.002)	0.015*** (.002)	0.015*** (.002)	0.014*** (.002)	0.014*** (.002)
Female	0.027 (.058)	0.019 (.062)	0.030 (.063)	0.024 (.059)	0.019 (.061)	0.032 (.063)
Education (ref=less than high school)						
High School Graduate	-0.258** (.102)	-0.261** (.097)	-0.271** (.098)	-0.263** (.101)	-0.265** (.100)	-0.268* (.100)
Some College	-0.331*** (.099)	-0.337*** (.093)	-0.346*** (.094)	-0.341*** (.094)	-0.339*** (.094)	-0.343*** (.095)
College Graduate and Beyond	-0.406*** (.083)	-0.411*** (.079)	-0.420*** (.080)	-0.415*** (.081)	-0.415*** (.080)	-0.415*** (.081)
Baseline Income (per $10k)	-0.015 (.013)	-0.015 (.013)	-0.016 (.013)	-0.016 (.013)	-0.015 (.013)	-0.016 (.013)
Married (ref=not married)[a]	-0.021 (.088)	-0.018 (.089)	-0.021 (.091)	-0.019 (.091)	-0.010 (.092)	-0.015 (.091)
Diabetes	-0.079 (.126)	-0.077 (.126)	-0.065 (.128)	-0.069 (.122)	-0.061 (.120)	-0.068 (.123)
Current Smoker (ref=non-current smoker)	0.010 (.050)	0.012 (.050)	0.017 (.051)	0.018 (.051)	0.021 (.050)	0.022 (.052)
Depressive Symptoms	0.016*** (.005)	0.016*** (.005)	0.016*** (.005)	0.018*** (.005)	0.018*** (.005)	0.017*** (.005)
Religious Participation	0.009 (.012)					
Religious Importance		0.006 (.044)			0.040 (.030)	
Spiritual Coping			-0.013 (.014)			-0.002 (.012)
Cognitive Impairment, Baseline (wave 4)	0.402*** (.052)	0.401*** (.052)	0.403*** (.054)	0.405*** (.052)	0.403*** (.052)	0.405*** (.053)
Stressful Life Events (SLE)	0.032 (.031)	-0.004 (.065)	-0.030 (.045)			
Everyday Discrimination				0.002 (.015)	0.011 (.021)	-0.007 (.019)
Race*Stress Interactions						

Black*Stressful Life Events	-0.054 (.086)	-0.348 (.184)	-0.186 (.144)			
Black*Discrimination				0.024 (.033)	-0.067 (.091)	-0.014 (.058)
Race*Religion & Spirituality 2-way Interactions						
Black*Religious Participation	-0.035 (.030)			-0.0026 (.028)		
Black*Religious Importance		-0.228 (.199)			-0.122 (.225)	
Black*Spiritual Coping			-0.043 (.066)			-0.007 (.067)
Stressful Life Events*Religion & Spirituality 2-way Interactions						
SLE*Religious Participation	-0.006 (.007)					
SLE*Religious Importance		0.005 (.028)				
SLE*Spiritual Coping			0.007 (.009)			
Discrimination*Religion & Spirituality 2-way Interactions						
Discrimination*Relig Participation				-0.003 (.002)		
Discrimination*Relig Importance					-0.009 (.008)	
Discrimination*Spiritual Coping						-0.0004 (.003)
Race*SLE*Religion & Spirituality 3-way Interactions						
Race*SLE*Religious Participation	0.020 (.014)					
Race*SLE*Religious Importance		0.132 (.069)				
Race*SLE*Spiritual Coping			0.028 (.022)			
Race*Discrimination*Religion & Spirituality 3-way						

Interactions

Race*Discrimination*Relig Participation				0.002 (005)		
Race*Discrimination*Relig Importance					0.035 (.032)	
Race*Discrimination*Spiritual Coping						0.007 (.009)
Intercept	-0.194 (.182)	-0.164 (.185)	-0.074 (.175)	-0.142 (.168)	-0.204 (.174)	-0.094 (.173)
R^2	0.246	0.247	0.247	0.247	0.248	0.246

[a]Not married includes separated, divorced, widowed and never married.
NOTE: Estimates are weighted to account for the sampling design.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

CHAPTER 4: DISCUSSION

I. Introduction

The purpose of this longitudinal study was to explore and understand racial inequalities in cognitive impairment among black and white adults over time. The particular focus is on examining race differences and the possible risk and protective factors that underlie and or influence these disparities. Specifically, I examined whether education, stressful life events, and discrimination accounts for racial differences, changes in cognitive impairment, and if religion and spirituality act as buffers.

Thus, the main goals of this dissertation were to: (1) explore the relationship between race/ethnicity and changes in cognitive impairment over time; (2) examine the modifying and confounding influence of social and cultural risk factors—education, stressful life events, and discrimination; (3) and assess the protective effects of religion and spirituality on the focal relationship. This section discusses the major findings pertaining to these relationships.

My dissertation found that black-white differences in cognitive functioning exist at baseline (ages 25 and older) and these disparities widen and accelerate over time. In particular, my research shows that blacks have significantly higher levels of cognitive impairment at each wave and decline at a faster pace than whites do, even after adjusting for sociodemographic and other health-related factors. Education, rather than stressful life events and discrimination, accounted for a significant amount of racial disparities but did not fully explain black-white differences. Finally, the non-significant findings indicated that religion and spirituality are not related to cognitive impairment and do not buffer the relationship between race/ethnicity, stress and wave 5 cognitive impairment.

For ease of discussion this section first presents the multilevel findings, specifically, the significant linear and quadratic age effects and interaction between age and race. Comparison of the findings to previous studies follows the presentation of the data. Then the conditional relationship between race and education on cognitive impairment is discussed, including a review of the impact of education on this contingency. Next, the non-conditional relationship between race, stressful life events, discrimination and religion/spirituality is presented. This discussion also includes a review of the impact of religion and spirituality on cognitive impairment. The chapter ends with an overall review of the findings and their implications for future studies, practice, and theory.

II. Discussion of Major Findings

A. Black-White Differences in Cognitive Decline

The purpose of Aim 1 of this study was to determine whether black-white differences in cognitive impairment exist and evaluate whether these disparities persist over time. Two primary findings are notable. First, the finding of race being strongly associated with baseline cognitive impairment scores is consistent with other cross-sectional studies that have examined the effects of race/ethnicity on cognition in middle and older ages (Tang et al., 2001; Schwartz, Glass, et al. 2004 (ages 50-70 – Baltimore Memory Study); Rexroth et al. 2013 (mean age 73.6); Carlos Díaz-Venegas et al. 2016 (ages 51-80+). The second major finding was that race/ethnicity is associated with change in cognition over time: blacks experienced a faster rate of decline in their cognitive abilities than whites.

This finding is consistent with some studies; yet inconsistent with others. For instance, some studies have found that blacks experience a more rapid rate of cognitive decline (Sachs-

Ericsson and Blazer 2005, Sawyer, Sachs-Ericsson et al. 2008), while others have found that whites experience a more rapid rate of cognitive decline (Sloan and Wang 2005, Alley, Suthers et al. 2007, Karlamangla, Miller-Martinez et al. 2009, Early, Widaman et al. 2013, Wilson, Capuano et al. 2015), some have also found no difference (Atkinson, Cesari et al. 2005, Castora-Binkley, Peronto et al. 2013, Marsiske, Dzierzewski et al. 2013) and others present mixed results within the same study (Masel and Peek 2009, Wolinsky, Bentler et al. 2011).

Given the inconsistencies in the existing literature, the present study is valuable because it helps to reconcile some of these inconsistencies by clarifying the effects of race on a continuous measure of cognitive change. While previous studies have focused on only middle ages and/or older adults at baseline such as ages 51 and above (Masel and Peek 2009), my longitudinal study is innovative in that it examined racial changes in cognitive impairment scores across a larger part of the adult life course by studying a nationally representative sample of black and white adults who were ages 25 and older at baseline. Thus, to date my study is the first and only one to consider changes in cognition in younger ages and also whether there are black-white differences in these changes over time.

My study findings demonstrate that blacks have more cognitive impairment than whites at younger ages and these disparities widen over time, even after adjusting for sociodemographic and other health-related factors. These findings highlight that racial disparities begin earlier than previously identified, but are nonetheless consistent with other studies that have examined longitudinal changes in cognitive functioning in older black and white adults using the Short Portable Mental Status Questionnaire (SPMSQ) (Sachs-Ericsson and Blazer 2005, Sawyer, Sachs-Ericsson et al. 2008), which is the measure used in the current study.

For example, Sawyer and colleagues (2008) used a 10-item version of the SPMSQ and

created a continuous scale with higher scores indicating more cognitive difficulty (0-10 errors). They found a large effect size for the interaction of age and race (b=0.028, SE=0.008, p=0.0005). However, they did not estimate a non-linear association between age and cognitive impairment, as done by in the present study. Thus, the smaller effect size in the current study between age and race (b=0.009, SE=0.001, p<0.0001) may be due to the different approaches used. Although one can posit an individual growth model that is either linear or non-linear (quadratic) with age, this may influence the strength of the association between age, race and cognitive impairment. The smaller effect size may also be due to the fact that the ACL used a shorten version of the SPMSQ that included only 5-items. Yet, regardless of whether a linear or non-linear association is estimated or whether the full (10-items) or shorten (5-items) version is used the outcome is consistent in that blacks have a higher SPMSQ score at baseline (i.e., they make more errors) and on average demonstrate a more rapid cognitive decline than whites. Future work should be done to validate these findings using specific domains of cognition versus a brief global measure.

The present study further contributes to this literature by following participants for a longer period time given that the ACL tracked individuals for 25 years from 1986-2011. Other studies have followed participants on average for 3-12 years, less than half the time of the current study. Following participants over a longer period of time allows more information to be collected and leads to a more accurate analysis of the changes in cognitive functioning over time. Future studies should validate these findings with additional waves of follow-up to gather even more data points for a more robust analysis of these associations.

Additionally, several of these studies have used either small sample sizes (Early et al. 2013 – 116 black and 184 white; Wilson et al. 2015 – 647 black and 647 white) or have been

conducted in special populations, e.g., Medicare beneficiaries (Wolinsky, Bentler et al. 2011), elders with mild cognitive impairment (Lee, Richardson et al. 2012) or those clinically diagnosed with dementia as compared to those with normal cognition (Wilson, Aggarwal et al. 2010). The present study included a large sample of individuals with normal cognition and assessed changes in their cognitive abilities over time, which allows the generalizability of the results to apply to a broader segment of the population. As such the findings of this study extend beyond a specialized segment of the population and may be generalized to ordinary Americans.

B. Education Effect and Cognitive Decline

The purpose of Aim 2 of this study was to determine whether educational attainment, stressful life events and discrimination are associated with worse cognitive impairment and determine the extent to which these three factors simultaneously account for racial differences in cognitive changes over time, given that other studies have looked at these factors independently. My analysis of younger and older black and white adults yielded two major findings: (1) blacks have significantly higher wave 5 cognitive impairment scores compared to whites across the ages of 25 and 95 and (2) only education explains a significant portion of black-white disparities, while stressful life events and discrimination are not associated with racial differences.

The finding that whites have significantly lower wave 5 cognitive impairment scores compared to blacks is consistent with other studies that have found clear racial and ethnic differences in cognitive impairment among older adults, particularly for blacks and other ethnic minorities (Zsembik and Peek 2001, Schwartz, Glass et al. 2004, Mehta, Stewart et al. 2009, Potter, Plassman et al. 2009, Masel, Raji et al. 2010). However, this finding should be further validated with future waves, given the mixed and seemingly conflicting findings about racial differences in cognitive changes over time in the literature.

The finding that education alone, rather than stressful life events or discrimination, confounds or accounts for some of this disparity is contrary to my original hypothesis that collectively these three factors would confound the association between race and cognitive impairment (see hypothesis 4), but is consistent with the cognitive-reserve theory, which suggests that individuals with less education may not have the same brain reserve or cognitive resources as those with more education in staving off later life decline (Mehta, Stewart et al. 2009). While stress and discrimination are associated with a number of physical illnesses (Brondolo, Libby et al. 2008, Gee, Ro et al. 2009, Lewis, Barnes et al. 2009), this analysis failed to detect any association with cognitive problems.

The present study looked closer at the role of education level in race disparities over time at different ages by removing education from the multilevel growth model, as shown in Figure 3.7. This figure suggests that education level accounts for the race disparity at very early ages, but not at older ages. The cognitive reserve theory would explain this finding by stating that enriched educational experiences in early life produce an increased reserve that delays the onset of cognitive decline in later life, which allows individuals to draw upon the brain reserve in mid to later life rather than spending that capital as a young to middle age adult (Manly and Mayeux 2004). Indeed early-life markers of cognitive reserve such as, childhood low IQ scores (Whalley et al., 2000) and low linguistic abilities in early 20s have been associated with lower cognitive test scores and dementia later in life (Manly and Mayeux 2004).

Although variables such as years of education are often used as a proxy for an individual's degree of cognitive reserve, these variables may not necessarily represent innate ability or limited educational and occupational opportunities because of institutionalized racism

and poverty. Gee and colleagues (2012) infer that racism underlies the development of disparities over the life course. Specifically, his model emphasizes that there are disparities due to racism in both the amount of time and the quality of time spent in a particular life stage, which may ultimately shape health inequalities. This is important because the quantity (years of schooling) and the quality of time (educational quality) an individual spends in a life stage will either help build their capital, e.g., spending more time receiving a quality education, or take away from it, e.g., dealing with chronic unemployment or being incarcerated, and these disparities can then lead to lower life expectancies and greater cognitive decline (Gee, Walsemann et al. 2012).

Even though my study did not find an association between experiences of discrimination and cognitive decline, recent research has linked higher levels of perceived discrimination to poorer cognitive tests performance, particularly among non-demented African Americans over 65 (Barnes, Lewis et al. 2012). The divergent results may be contributed to the different study samples examined (only blacks over 65 vs. blacks and whites over 25), and the use of dissimilar scales and items (i.e., 18 performance-based tests assessing five specific domains of cognitive functioning that include episodic memory, semantic memory, working memory, perceptual speed and visuospatial ability vs. the SPMSQ global measure). Further, I found an educational quantity bias on most of the SPMSQ items; hence, my future work will incorporate additional comprehensive measures of educational experiences. This will enable me to better assess the impact of educational quality differences as a proxy of cognitive reserve. At the same time, consider the potential influence of racism and discrimination in shaping these disparities across the life course.

Additionally, the lack of a confounding effect for stressful life events may be due to the

fact that stressful life events were assessed using an aggregate global measure versus specific events or individual items. Studies have found that different life stressors affect cognition differently. For example, one study found no association between aggregate life events and cognitive decline among older adults aged 55-85 years in the Netherlands. This same study also found that specific life events (e.g., the illness of a partner or relative) looked at over a 3-year time period were associated with improved cognitive ability while other life events such as being relocated were associated with declines in cognitive functioning based on lower MMSE scores (Comijs, van den Kommer et al. 2011). Thus, future studies should consider the individual effects of life events rather than solely the aggregate.

Finally, accounting for depressive symptoms and other health conditions in combination with discrimination and other stressors in future work examining black and white differences in changes in cognitive impairment remains noteworthy; even though these stressors were not significant in this study. Depressive symptoms as well as age, education, and baseline scores of cognitive impairment all remained significant predictors of wave 5 cognitive impairment. This suggests that depressive symptoms may be a risk factor for cognitive impairment. Moreover, studies have linked discrimination and other stressors to several important risk factors for cognitive impairment in blacks, including high blood pressure/hypertension (Brondolo, Libby et al. 2008, Lewis, Barnes et al. 2009), depressive symptoms (Barnes, Mendes De Leon et al. 2004, Schulz, Gravlee et al. 2006) and psychological distress (Barnes, Mendes De Leon et al. 2004, Byrd 2012). Future work should consider more comprehensive measures of both stressful life events and discrimination and the role of depressive symptoms, age, education and other factors on these associations.

Currently, it is unclear the role that education (as well as race) plays in cognitive decline during middle and old age, given that a growing body of literature reports mixed findings. With some studies finding that these variables are *not* strongly associated with differences in rate of cognitive change (Karlamangla, Miller-Martinez et al. 2009, Masel and Peek 2009, Wilson, Hebert et al. 2009, Wilson, Aggarwal et al. 2010) and others showing there *are* significant differences by education (Wolinsky, Bentler et al. 2011). For example, Wolinsky et al. (2011) examined crude cognitive changes and found that those having grade school educations declined the least on the immediate and delayed word recall tests, while those with college educations declined the most. In a second analysis using a residual change score multivariable linear regression model, participants with grade school educations declined more than those going on to high school, while those attending college had the smallest declines in cognitive status across all three measures (TICS-7, immediate and delayed word recall tests). It may be that the assessment of cognition at only two time points, as done by the present study and Wolinsky et al. (2011), limits the ability to accurately detect consistent educational differences; thus, future work should validate the current study findings with additional follow-up waves.

C. Religion and Spirituality as Buffers

The purpose of Aim 3 of this study was to explore whether religion and spirituality protect against cognitive decline and assess whether these factors attenuate black-white disparities. Although, none of the findings were not significant they warrant further research and discussion. First, the finding that individuals reporting higher levels of religion and spirituality do not have a lower wave 5 cognitive impairment score compared to those reporting lower levels of religion and spirituality, after adjusting for model covariates is contrary to my original hypothesis. This finding is also inconsistent with other studies that have found an inverse link

between religion, spirituality and cognitive decline such that higher levels of religion, spirituality and private religious practices were correlated with a slower rate of both cognitive and behavioral decline (Kaufman, Anaki et al. 2007, Coin, Perissinotto et al. 2010). This may be due to the different religion and spirituality measures used (private vs. public religious practices) and the different populations examined (patients with Alzheimer's dementia vs. non-demented younger and older adults). Thus, the lack of a direct effect on religion and spirituality on cognition in this study may be due to these methodological issues. Future studies should consider these differences and other possible underlying mechanisms when examining the protective influence of religion and spirituality on changes in cognitive functioning.

Second, the finding that religion and spirituality do not modify the relationship between race and cognitive impairment is also inconsistent with my original hypothesis and existing literature. Studies have found that, in general, older blacks are more likely than whites to use religious orientations and strategies as part of their coping repertoires when dealing with stress, difficult life situations, and a variety of health problems (Taylor, Chatters et al. 2004, Krause and Chatters 2005, Chatters, Taylor et al. 2008, Chatters, Taylor et al. 2009). Moreover, some of these studies have longitudinally examined the use of religious and spiritual coping (e.g., over a 15-year time period) and report that prayer is the most utilized form of coping by blacks. Thus, we would anticipate that religion and spirituality would be an equalizer and function differently for blacks and whites in terms of coping with and/or protecting against cognitive decline, such that blacks reporting high levels of religious involvement and spirituality would have lower levels of cognitive impairment than whites reporting similar levels of religion and spirituality. This was not the case in the present study, rather at higher levels of religion and spirituality there

was no black-white differences in cognitive impairment scores. The lack of an effect modification for religion and spirituality on the race-cognition association suggests that these measures may not differ in terms of how they protect blacks and whites against cognitive decline. However, it may be that the experience and meaning of religion and spirituality differs between blacks and whites and this may be why blacks report a higher prevalence of using prayer and other religious strategies as a coping mechanism against discrimination and other life stressors. Prior studies have shown that the substantive meaning of religion and spirituality differs between older blacks and whites. For example, Krause and Chatters (2005) examined 17-different prayer-related measures (e.g., social and substantive content of prayer, length of prayer, interpersonal aspects of prayer, etc.) and found that blacks were more deeply engaged in prayer activities than their white counterparts. So it is important to not only look at the reported levels of use, but also the experience as well as the meaning of religion and spirituality as possible protective factors against cognitive decline. Although this study looked at various measures (i.e., religious attendance and participation, importance of religious or spiritual beliefs and the use of spiritual coping) it may be that these religion and spirituality measures were limited in their ability to influence changes in cognitive functioning over time as well as attenuate black-white disparities, given that they did not capture these areas. Future studies should consider broader measures such as those that look at the experience, meaning and/or content of religion and spirituality and how these measures function differently for blacks and whites and for younger as well as older adults in influencing racial differences in changes in cognition over time.

Finally, the finding that none of the religion and spirituality measures buffer the relationship between stressful life events, discrimination and cognitive impairment again conflicts with my original hypothesis and extant work, but was expected given that there was no

direct effect of stress on cognition and thus no stress effect to modify or buffer against. Further the 3-way interaction between race/ethnicity and stress (i.e., stressful life events and discrimination), religion and spirituality was also found to be non significant. So there was no difference in wave 5 cognitive impairment scores between blacks with low levels of religion and spirituality and low levels of stress compared with whites at these same levels. Additionally, blacks with high levels of religion and spirituality and high levels of stress had the same wave 5 cognitive impairment scores as their white counterparts also reporting high levels of religion/spirituality and stress.

Coping resources (such as a sense of control or mastery over life and self-esteem) and coping strategies (such as religious or spiritual coping) have been consistently shown to buffer the negative health effects of stress (Thoits 1995, Ryff, Friedman et al. 2012). Although, none of the religion and spirituality measures modified the associations between stressful life events, discrimination and cognitive impairment, this may be due to the different measures of stress examined (general stress vs. a global measure of stressful life events and discrimination). Moreover, none of the religion and spirituality measures differentially interact with stress to modify the influence of race on cognition and this may be due to the divergent measures examined (prevalence or reported level of use vs. the meaning and/or content of religion and spirituality). Future studies should consider other measures of stress, including the individual effects of life events, subtle or ambiguous discrimination (versus more blatant discriminatory treatment as in the everyday discrimination scale), and multiple dimensions of stress and inequality that simultaneously and interactively shape cognitive health over life the course (Brown, Richardson et al. 2016). Also, more comprehensive measures of religion and

spirituality should be explored. This may offer the most promise for understanding and addressing racial inequalities in cognitive trajectories.

III. Strengths and Limitations

Several limitations should be recognized. First, waves 4 and 5 of the ACL survey were carried out with a 10-year time interval between interviews. Although ACL is the oldest ongoing nationally representative longitudinal study, the lag time between waves varied; and different periods between interviews may influence the strength, statistical significance, and associations of variables over time. For example, the time span between adjacent waves keeps increasing (e.g., the time gap between waves 1 and 2 = 2.5 years, waves 2 and 3 = 5 years, waves 3 and 4 = 7.5 years and waves 4 and 5 = 10 years). So it is more likely that attrition occurred at later waves of the ACL survey. In this study, there were significant differences between those in my sample (waves 4 and 5) and those who dropped out after wave 4; whereby, those who had worse wave 4 cognitive impairment scores dropped out of the study sample (see Appendix 2.4). They may have dropped out simply due to the amount of time between data collection, rather than their higher cognitive impairment scores. Future work that follows individuals longitudinally should consider the amount of time between waves of data collection. This information will further contribute to our understanding of the long-term effects of education, stressful life events and discrimination on cognition. Second, the SPMSQ, the primary outcome measure, is not commonly used today to assess cognitive functioning, but when the ACL study began in 1986 it was one of the most prominent tools used. Moreover, given that the ACL study spans 25 years and has consistently used this tool, its continued use for the data analysis in this study is appropriate. Third, the measures of education, stressful life events, discrimination, and cognitive impairment used in this study are all self-reported and suffer the same challenges as all self-

report data. Specifically, these measures make it difficult to determine whether the associations found are valid or the extent to which they may reflect some other underlying factor such as early-stage dementia (Schulz, Gravlee et al. 2006). (Dohrenwend 2006) also notes that recall bias, unreliability of recall, and criterion and construct validity are all potential problems of self-reported measures. Fourth, these analyses did not test the specific biological mechanisms proposed in the conceptual framework linking genetic factors and cognitive impairment due to the limits of the data available in ACL. Yet, genetic factors still may only explain a small amount of racial disparities (Lopez, Jagust et al. 2003, Sheffler 2013). Finally, a key strength is that I do make use of all 5 waves of the ACL panel data, including a subsample taken from waves 4 and 5. This allows me to conduct a longitudinal analysis and helps me to disentangle the temporal relationships between race/ethnicity and changes in cognitive impairment when estimating my regression models. Likewise, in the subsample the predictor variables are measured at wave 4 and cognitive impairment is measured at waves 4 and 5. This strategy increases the likelihood that any relationships I find between stressful life events, discrimination, religion, spirituality and cognitive impairment in young and late life are not confounded by the simultaneous measurement of the main predictors in the model. Although this approach clarifies temporal ambiguity, it still does not prove causality so causal inferences remain limited.

IV. Summary and Conclusions

The overarching goal of this study was to better understand the social and cultural risk and protective factors of black and white declines in cognitive impairment symptoms in order to identify potentially modifiable risks for developing Alzheimer's disease or other dementias over time. Specifically, this study evaluated whether education, stressful life events, experiences of

discrimination, religion and spirituality are associated with changes in cognitive impairment by race, net of other known risk factors. Studies examining trajectories of cognitive changes in different racial/ethnic groups composed of individuals who represent a comprehensive view of the life course from young to old age are particularly relevant to understanding the factors that influence cognitive outcomes in older populations (Early, Widaman et al. 2013)

The Americans' Changing Lives Survey (ACL) is a rich dataset to test possible causal pathways for racial disparities given that it is the oldest ongoing nationally representative study designed to longitudinally explore social disparities in health and aging. More importantly, in its inception the ACL focused specifically on differences between black and white Americans in middle and late life in terms of understanding the ways health changes due to aging over the adult life course; therefore it included adults who were 25 and older at baseline.

My dissertation found that black-white differences in cognitive functioning exist at baseline and these disparities widen and accelerate over time. In particular, my research shows that blacks have significantly higher levels of cognitive impairment at each wave and decline at a faster pace than whites even after adjusting for sociodemographic and other health-related factors. Further, my findings reveal that educational differences underlie a substantial part of these racial inequalities and that religion and spirituality do not protect against cognitive decline as we age. These findings indicate that racial disparities begin earlier than previously identified, as existing studies have not included younger age groups at baseline. However, my findings are nonetheless consistent with other studies that have examined longitudinal changes in cognitive functioning in older black and white adults using the Short Portable Mental Status Questionnaire and other measures of cognition. Despite the fact that most cognitive changes occur at more advanced ages, my work captures a more comprehensive view of when cognitive changes begin

to take place and when racial differences emerge. By capturing early symptoms of cognitive impairment and decline, we may be able to develop interventions that will reduce the burden of dementia and Alzheimer's disease in our society and enable our elderly to have a better quality of life.

A. Implications for Future Studies

In summary, this research has important implications for future studies. First, future work should be done to validate these findings using different scales and tests, including specific domains of cognition versus a brief global measure. Second, future studies should validate these findings with additional waves of follow-up to more accurately detect race differences in trajectories of cognitive functioning and bring more consistency, given the mixed and seemingly conflicting findings that race differences exist in cognitive changes over time. Future work that follows individuals longitudinally should also consider the amount of time between waves of data collection. It is possible that using uniform or evenly spaced chunks of time a different pattern may emerge. This information will further contribute to our understanding of the long-term effects of education, stressful life events and discrimination on cognition.

Third, future work should consider more comprehensive measures of both stressful life events (such as the individual effects of life events rather than solely an aggregate measure) and discrimination and the role of depressive symptoms, age, education and other factors on these associations. Additionally, prospective studies are needed to further test the associations among discrimination, depressive symptoms and cognitive functioning in terms of testing the hypothesis that depression due to discrimination leads to poorer cognitive test performance. The results of the present study fit with this hypothesized scenario, but additional studies are needed to

replicate the findings. Future studies should also consider other measures of stress, including subtle or ambiguous discrimination (versus more blatant discriminatory treatment as in the Everyday Discrimination scale used in this study), and multiple dimensions of stress and inequality that simultaneously and interactively shape cognitive health over life the course (see Brown, Hargrove and Thomas 2016). This may offer the most promise for understanding and addressing racial inequalities in cognitive trajectories.

Finally, future studies should consider other possible underlying mechanisms when examining the potential protective influence of religion and spirituality on changes in cognitive decline. Specifically, studies should consider broader measures such as those that look at the experience, meaning and/or content of religion and spirituality and how these measures function differently for blacks and whites and for younger as well as older adults in influencing racial differences in changes in cognition over time. Studies should also consider if people get stressed out when they realize that their cognitive status is declining and explore whether they use religion and spirituality to cope with this "stress" as a way to reduce it. Although this may not change the rate of decline it may still be protective against it.

B. Implications for Public Health Practice

This research also has important policy implications for practice since it demonstrates that key social and cultural factors that differ by race (e.g., education) may be underlying racial inequalities in cognitive impairment among younger and older U.S. adults. Policies and interventions that impact these social and cultural risks and protective factors not only have intrinsic value, but they are also important with regard to successful aging and reducing health disparities. Interventions designed to address cognitive decline should be implemented at younger ages, given the implications of the study findings.

A current review of the effectiveness of 13 interventions designed for preventing or delaying the onset of age-related cognitive decline, mild cognitive impairment (MCI), or clinical Alzheimer's-type dementia (CATD) showed no evidence of a benefit to delay or prevent these diseases. The 13 classes of interventions examined included: cognitive training, physical activity, nutraceuticals, diet, multimodal interventions, hormone therapy, vitamins, antihypertensive treatment, lipid lowering treatment, nonsteroidal anti-inflammatory drugs (NSAIDs), anti-dementia drugs, diabetes treatment, and "other interventions. Specifically, the authors found moderate-strength evidence that cognitive training in adults with presumed normal cognition show improved performance in specific trained cognitive domains (i.e., memory, reasoning, or processing speed); however, this training did not transfer to improvement in other untrained cognitive areas nor was the much evidence that these benefits were experienced beyond two years. Although the authors reviewed several types of interventions they note that they "found no eligible studies for the following interventions: depression treatment, smoking cessation, and community-level interventions (p.1)." Thus, the benefits of these types of interventions, especially community-level interventions in preventing or delaying age-related cognitive decline, MCI, or CATD are unclear. The author's conclusions sum up the need for work in these areas "testing interventions that address modifiable risk factors can help to establish their causative role in MCI and CATD…. More work is needed to understand the relationship between intermediate outcomes such as cognitive test results and the onset of mild cognitive impairment and dementia…identifying interventions with the potential to prevent or delay the onset of dementia is an urgent public health priority (p.2)." (Kane, Butler et al. 2017)

Thus, efforts to address racial disparities in cognitive impairment should not only consider ways to prevent or slow the impairments in cognition that serve as a prelude to Alzheimer's disease and other dementias, these efforts should also design interventions that address social and cultural risk factors including educational level differences.

While there is evidence in the literature that stress contributes to cognitive decline and education quality (as a race-relevant variable) impacts cognition (Manly 2006), my research determined that education level is the primary contributor to cognitive impairment and underlies a significant part of racial disparities. Thus, the types of interventions developed should consider the underlying educational and occupational differences between black and white adults. Given that education quality and attainment both lead to more cognitively challenging occupations and higher incomes, employment discrimination also needs to be addressed. Higher education does not eliminate racial biases in the types of occupation obtained by those with less education, nor does it address the earning disparity between highly educated blacks and whites and the differential employment and hiring practices systematically applied to blacks. As a white friend once said, "it is easier for her husband as a convicted felon to get a job than it is for a higher educated black man."

Moreover, my findings suggest that interventions aimed at reducing poor cognitive outcomes among older African Americans should target and address depressive symptoms. Although it has been difficult to determine whether depression is a cause or a symptom of cognitive impairment, my study found that depressive symptoms are a risk factor for cognitive impairment and should be studied as such. As well, I found that discrimination is linked to depressive symptoms thus, there is a need for interventions addressing these risk factors collectively as well as individually. Other studies have found that depressive symptoms'

resulting from discriminatory treatment and/or practices reduces the association between discrimination and cognition (Barnes et al. 2012). Thus, interventions that consider how depressive symptoms affect cognitive status via discrimination is a worthy endeavor given that the depression effect via this pathway is not well understood.

C. Implications for Theory

Although the present study did not find that stress predicts decline nor does religion and spirituality protect against cognitive changes, identifying any protective influence of religion and spirituality against cognitive impairment and subsequent Alzheimer's dementia development will be an important mechanism for future interventions and theory. Studying the potentially protective influence of religion and spirituality reverses the bias in the current literature of only focusing on the deficits in the black community. The black church service is a functional community mental health resource for its participants and may provide specific cognitive resources that are useful for coping with various life stressors over one's lifetime. Although, this was not the case in the present study, interventions that promote supportive social networks in disadvantaged communities as well as theories that focus on the strengths of the black community may still mitigate cognitive disparities and reduce the overall burden of dementia.

In conclusion, the purpose of this study was to explore racial disparities in cognitive impairment trajectories and elucidate whether education, stressful life events, and discrimination accounts for these differences and determine if religion and spirituality act as buffers. This study found that racial disparities begin earlier in the life course than previously identified, such that blacks have more cognitive impairment than whites at baseline (ages 25 and older) and these disparities accelerate and worsen with age. Additionally, education level underlies a significant

portion of these disparities but does not fully explain black-white differences in cognitive aging. Lastly, religion and spirituality do not buffer against cognitive decline but the possible protective influence of these factors should still be considered. I hope my work sufficiently raises awareness of these issues and underscores the need to fuel further research addressing racial disparities and changes in cognitive performance over time.

APPENDIX 1.1 Table of Longitudinal Studies

Full Citation	**Dataset (follow-up waves and age groups)**	**Population or Sample**	**Main Objective Analyses Plan**	**Measure of Cognitive Function**	**Control Variables**	**Results**
Kathryn Sawyer, Natalie Sachs-Ericsson, Kristopher J. Preacher, Dan G. Blazer 2008 "Racial Differences in the Influence of the APOE Epsilon 4 Allele on Cognitive Decline in a Sample of Community-Dwelling	Duke EPESE study collected in **four waves over a 10-year period** (n = 2,076) Mean age=**71.6 years** The wave 1 study was conducted in 1986–1987 (n = 4,162, 54.3% Black), and follow-up interviews	A North Carolina sample of community residents selected from five contiguous counties (**community-dwelling older adults**). Participants were divided into two groups, those who had at least one APOE e4 allele and those who had no e4 allele.	We conducted multilevel growth curve analyses to examine the effect of race and APOE genotype on growth of cognitive errors with increasing age. We used multilevel models for repeated measures to examine racial differences in	10-item Short Portable Mental Status Questionnaire (SPMSQ) NOTE: items were summed to form a continuous scale (0–10 errors) with higher scores indicating more difficulty.	These variables include demographic variables (age, gender), socioeconomic variables (income, **education, literacy**), health variables [e.g., diabetes, high blood pressure (HBP), heart attack, stroke], and physical functioning (participants' ability to do heavy housework, walk	**Blacks experience a faster rate of decline than whites** There was a significant interaction of age and race, p = 0.0005, meaning that the increase in errors was significantly steeper for blacks than for whites. The gap in mean errors widened by 0.028 per year of age.

Older Adults" Blacks decline faster	were conducted in 1989–1990 (wave 2, n = 3,559, 54.6% AA), 1992–1993 (wave 3, n = 2,840, 55% AA), and 1996–1997 (wave 4, n = 1,767, 53.5% AA		participants' increase in errors on a continuous measure of cognitive functioning as they aged		up and down stairs, and walk one-half mile)	**Blacks made more errors on average and demonstrate more rapid Cognitive** **Decline**.
Sachs-Ericsson, Natalie Ph.D.; Blazer, Dan G. M.D., Ph.D. 2005 Racial Differences in Cognitive Decline in a Sample of Community-Dwelling Older Adults	Duke EPESE study collected in **four waves over a 10-year period** (n = 2,076) Mean age=**71.6 years**	A North Carolina sample of community residents selected from five contiguous counties (**community-dwelling older adults**).	We examined racial differences in cognitive decline (CD) and the role of education and literacy in mediating this relationship.	10-item Short Portable Mental Status Questionnaire (SPMSQ) NOTE: items were summed to form a continuous scale (0–10 errors) with higher scores indicating more difficulty.	These variables include demographic variables (age, gender), socioeconomic variables (income, **education, literacy**), health variables [e.g., diabetes, high blood pressure (HBP), heart attack, stroke], and physical	**Race predicted CD such that Blacks had higher rates than Whites.** When education and literacy were entered into the analysis, **the association between race and CD**, although remaining statistically significant, was

Blacks decline faster					functioning (participants' ability to do heavy housework, walk up and down stairs, and walk one-half mile)	reduced **and was of relatively weak magnitude**
Masel and Peek 2009 Black adults had significantly faster rates of memory decline than white adults, but there was no differences between blacks and whites and	Health and Retirement Study an entry age of 51	Health related variables included body mass index calculated from self-reported height and weight, participation in vigorous activity 3 or more timers per week or not (1=yes, 0=no), and self-report (1=yes, 0=no) of doctor diagnosis of heart disease,	Multinomial logistic regressions examined the odds of decline in memory and mental status score by one standard deviation between 1996 and 2004 and only those who were 63-66 in 1996 were evaluated longitudinally	Telephone Interview of Cognitive Status, representing "mental status," and word recall items, representing "memory."	At baseline, participants' age, sex (1=female, 0=male), and ethnicity were collected, and marital status in 1996 was categorized as married (1) or not married (0). **Education was measured as the number of years of school a participant reported**	The effect size for memory decline in African Americans (compared with Caucasians) was less than −0.01 (−.03/3.39), whereas the cross-sectional effect size for this comparison was .08 (−0.26/3.39). Rate of decline was generally larger, as would

the rate of decline in mental status over time		stroke, diabetes, or hypertension.	for mental status items.		**completing**. This measure was used as both a continuous and categorical (<8 years, 8-11 years, 12 years, ≥ 13 years) in the models. Household income in 1996	be expected, when younger individuals (51–70) were not included
Wolinsky et al. 2011 A prospective cohort study of long-term cognitive changes in older Medicare beneficiaries **Different results**	**Using only the first and last assessments in AHEAD data; Cognition was assessed at only 2 points:** we used the baseline (1993-1994) and biennial follow-up interviews through 2006 that were conducted as part of the	Used baseline and two year follow-up interviews from the **Assets and Health Dynamics among the Oldest Old** (AHEAD) study linked to Medicare claims from the same calendar years 1993-2007	Residual change score multiple linear regression analysis was used to predict cognitive function at the final follow-up using data from telephone interviews among 3,021 to 4,251 (sample size varied by cognitive outcome)	(3 assessments taken from the TICS) 7-item Telephone Interview for Cognitive Status (**TICS-7**) and 10-item **immediate** (taps episodic verbal memory i.e., working memory, fluid intelligence, or explicit memory) and **delayed word recall tests** (also taps episodic	Demographic and SES factors included age, sex, race, marital status, **years of education**, and income. Disease history was measured by a set of 10 binary indicators for whether the participant reported having been told by a physician that she	In analyses of simple change scores, **Caucasians showed declines that were (not equal to) greater than Blacks on the immediate and delayed word recall tests,** but in analyses in which scores from the last assessment were the outcome and

obtained from different analyses within the same publication: (1) Whites decline faster on immediate and delayed word recall using simple change scores; (2) Blacks decline faster on all 3 measures using residual change score linear regression (see p.8 results) so blacks decline more in some	AHEAD study; baseline (1993-1994) and a final two year post-baseline follow-up interview (1995-1996 thru 2006-2007) (up to13 years between first and last assessments, with an average of) conducted as part of the survey on **(AHEAD)** data linked to 1993-2007 Medicare claims Ages **≥ 70 years old**	8.7% Blacks	Two objectives: (1) examine long-term (an average of 7.2 years between assessments) changes in cognitive function in a nationally representative sample of older Medicare beneficiaries in the U.S.; (2) to identify the risk factors associated with those changes in cognitive function.	memory (i.e., acquired information, and processed and stored memory retrieval) because AHEAD data relies on phone interviews so unable to use the MMSE, but similar measures	had the particular diseases, and a binary indicator of ≥ 3 of the diseases to tap comorbidity. BMI, engaging in vigorous physical exercise, smoking, and alcohol consumption was used to measure health lifestyles. Functional status included measures of ADLs, IADLs, mobility, vision, hearing, and depressive symptoms	first assessment scores were entered as a covariate (residual change score multivariable linear regress), **whites had better outcomes or blacks had greater cognitive decline than whites on all three cognitive outcomes** Robust cross-sectional differences found, such that **African Americans had lower cognitive-test scores at baseline** than Caucasians (Early

analyses but not in others						et al. 2013, p. 3)
Sloan and Wang 2005 Disparities Among Older Adults in Measures of Cognitive Function by Race or Ethnicity Whites decline faster in total test score and word recall than blacks	Assets and Health Dynamics Among the Oldest Old (AHEAD), includes participants who, at baseline, were at least **70 years** **86% non-Hispanic White,** **10% Black, and 4% Hispanic** **7-year period between wave 1 through**	A national survey of US 7,443 Non-institutionalized persons aged 70 years. Follow-up interviews of the same sampled persons were conducted in 1995, 1998, and 2000 so individuals were tested 3 times between 1995 and 2000	Our main purpose in the longitudinal analysis was to assess trajectories in indicators of cognitive performance, as persons grew older.	TICS testing included **(1)** immediate and 5-minute **delayed word recall tests**; **(2) serial 7's** subtraction Test [as a test of working memory, attention, and calculation (scored 0–5)]; and **(3) other mental status items** (scored 0–10) that assess [orientation to time (date, month, year, day of the week); attention (counting backward from 20); language	Other demographic variables were age, gender, marital status, and **educational attainment (in years)**, and self-reported measures of an emotional, nervous, or psychiatric problem, depressive symptoms (20-item CESD), vision and hearing impairments, and overall health. We included measures of	Holding other factors constant, we found that, compared with Whites, at baseline (Wave 1), Blacks' total scores on the TICS were 3.5 points lower (p , .001) on average Total scores declined for Blacks, but at an annual rate of 0.06 less than that for Whites (p = .041). This lower rate of decline reflected a lower

	wave 4			(object naming), and knowledge of current affairs (president and vice president of the United States; 2 points)	household income, wealth, and a binary variable for negative net worth, set to 1 if the household's liabilities exceeded its assets.	rate of decline in word recall (p = .001), although scores declined for both groups blacks declined at a slower rate than whites did. The binary variables for self- or proxy-reported ability to perform personal tasks, several of which were limitations in instrumental activities of daily living (IADLs)
Alley, Suthers et al. 2007 Education and Cognitive Decline in Older Americans:	Assets and Health Dynamics Among the Oldest Old (AHEAD), includes participants	A national survey of US 7,443 Non-institutionalized persons aged 70 years. Follow-up	The authors performed growth curve modeling to examine the relationships between education, initial	Four tests that tap different cognitive abilities were used in this analysis: (1) delayed and (2) immediate verbal	Gender, **years of education** (no formal educ to 17 years or more), and several chronic health conditions	Blacks had lower baseline scores on each cognitive task relative to Whites. Racial and ethnic differences varied

Results From the AHEAD Sample	who, at baseline, were at least **70 years** **86% non-Hispanic White,** **10% Black, and 3.5% Hispanic (see Table 1)** **7-year period between wave 1 through wave 4**	interviews of the same sampled persons were conducted in 1995, 1998, and 2000 so individuals were tested 3 times between 1995 and 2000	cognitive score, and the rate of decline in cognitive function.	recall to 86assess verbal memory (fluid intelligence), (3) the Serial 7's to assess working memory (fluid intelligence), and the (4) Telephone Interview for Cognitive Status (TICS) to assess general mental status (a composite measure reflecting very basic crystallized intelligence).	have been identified as correlates of cognitive impairment, including high blood pressure, diabetes, heart disease, and stroke	by cognitive tasks, with Blacks experiencing a slower decline relative to Whites on tests of (1) delayed word recall and (2) immediate word recall. No racial differences were present in the rate of decline on the (3) Serial 7's test (p.86)
Karlamangla et al. 2009 Trajectories of Cognitive Function in Late Life in the United States: Demographic	Assets and Health Dynamics Among the Oldest Old (AHEAD), includes participants who, at	A national sample of 6,476 US adults born before 1924, who were tested 5 times between 1993 and 2002 on word recall, serial 7's, and	Applied mixed-effects modeling to characterize longitudinal change across the five assessment waves of the AHEAD study	Telephone Interview for Cognitive Status (a validated assessment tool comparable to the Mini-Mental State Examination	Demographic information collected included self-reported Sex, age, marital status. SES measures considered were	Non-Hispanic blacks (compared with non-Hispanic whites) declined slower African Americans showed slower decline on

and Socio-economic Predictors Whites decline faster in global cognition than blacks or cognitive decline was slower in Non-Hispanic black Americans	baseline, were **at least 70 years** **88.2% white, 7.6% black** **9-year period between wave 1 through wave 5**	other mental status items to determine demographic and socioeconomic predictors of trajectories of cognitive function in older Americans	7-item Telephone Interview for Cognitive Status (**TICS-7**) and 10-item **immediate** (taps episodic verbal memory i.e., working memory, fluid intelligence, or explicit memory) and **delayed word recall tests**	(MMSE) Testing included **(1)** immediate and 5-minute **delayed word recall tests**; **(2) serial 7's** subtraction Test [as a test of working memory, attention, and calculation (scored 0–5)]; and **(3) other mental status items** (scored 0–10) that assess [orientation to time (date, month, year, day of the week); attention (counting backward from 20); language (object naming), and knowledge of	**highest year of school/college completed,** household wealth (the sum of all components — e.g., primary residence, retirement accounts, savings —minus all debt), and annual household income	average in global cognition than Caucasians Compared with non-Hispanic whites, non-Hispanic blacks had lower baseline scores, had similar practice effects, and experienced slower declines (more positive slopes), so that the black-white difference diminished with aging

				current affairs (president and vice president of the United States)		
Early et al. 2013 Demographic Predictors of Cognitive Change in Ethnically Diverse Older Persons Whites decline faster in semantic memory and executive function than blacks	Participants were 404 (116 African Americans, 104 Hispanics, and 184 Caucasians) educationally, ethnically, and cognitively diverse older adults enrolled in an ongoing longitudinal study of cognition **Average age is 78.4 (includes ages greater than 60 years)** **Average follow-up time for all racial**	67% of participants were recruited through community-based recruitment protocols designed to enhance both the racial and ethnic diversity and the spectrum of cognitive dysfunction of the sample, **with an emphasis on normal cognition and mild cognitive impairment** (MCI) and 33% had a clinical diagnosis of	The purpose of the current study was to evaluate how race/ethnicity and educational attainment relate to cognitive trajectories in a diverse sample of older persons	This study used a subset of SENAS tests to measure three specific (or multiple) cognitive domains: **(1) episodic** memory, **(2) semantic** memory, **(3)** executive function SENAS= Spanish and English Neuropsychological Assessment Scales	Gender, **years of education** included an unusually broad range of education, from no formal schooling to doctoral degrees, age, language, clinical diagnosis (normal, MCI, dementia), follow-ups and # of evaluations Covariates included clinical diagnosis of normal, MCI and demented at baseline and recruitment	A more rapid decline in White persons relative to Black persons in semantic memory and executive function with no difference in episodic memory decline Indeed, Table 4 (Model 2 on p.23), shows that blacks showed significantly slower declines than Caucasians on two of the three cognitive outcomes, when fully adjusted for

	groups was approx. 4 years Continuous independent variables of age and education were centered at 70 and 12 years, respectively.	MCI at baseline			source (community vs. clinic)	model covariates
Wilson et al. 2015 Cognitive Aging in Older Black and White Persons Whites decline faster in semantic	**3 longitudinal cohort studies**: (1) The Minority Aging Research Study; (2) The Rush Memory and Aging Project (approx. 6% are Black; and (3) The Religious Orders Study (approx. 7% are	Religious Orders Study began in 1994 and involves annual clinical evaluations of Catholic nuns, priests, and monks from across the United States	We assessed different domains of cognitive function **at annual intervals for a mean of more than 5 years** in older Black and White persons matched for age, education, and number of cognitive	A battery of 17 cognitive tests from which composite measures of 5 abilities and a global measure of cognition were derived. Measures of 5 abilities incl. **(1) episodic** memory, **(2) semantic** memory, **(3) working memory, (4) perceptual speed,**	Age, years of education, and number of cognitive assessments, gender The present results suggest similar racial differences in cognitive aging, with less decline	Robust cross-sectional differences found, such that **African Americans had lower cognitive-test scores at baseline** than Caucasians OR Baseline level of MMSE score and in each cognitive

memory, perceptual speed, and visuospatial ability was than Black persons	Black) **Average # of follow-ups in terms of annual cognitive assessments, 6.3 (blacks) and 6.1 (whites)** Began the study at ages **55-90** years old (mean age at baseline 73.5 black and 73.6 white)		assessments	**and (5) visuospatial ability** On average, the annual rate of decline in semantic memory was 27% slower (0.021/0.078) in Black persons than White persons, decline in perceptual speed was 44% slower (0.040/0.090), and decline in visuospatial ability was 45% slower	in semantic memory, perceptual speed, and visuospatial ability in Black persons compared to White persons, and no differences in working or episodic memory. The basis of the observed differences in cognitive decline in Black persons compared to White persons is uncertain	domain was lower in the Black subgroup Rates of decline in semantic memory, perceptual speed, and visuospatial ability were slower in Black persons compared to White person There were no racial differences in rate of decline in episodic memory and working memory such that no diff in subgroups and rate of decline
Wilson et al. 2010 Cognitive	Chicago Health and Aging Project, had a **11-year follow-**	To measure the cognitive consequences of incident	We used mixed-effects models to examine change in cognitive	We used a composite index of **global cognition** based on all 4	MCI or AD differed by race, we conducted a second analysis	Over the course of 11 years, the annual rate of decline on a

decline in incident Alzheimer disease in a community population No difference between blacks and whites in cognitive decline among those diagnosed as normal, mildly cognitively impaired, or having dementia	**up period** **Mean age of 78.7 years**	Alzheimer disease (AD) in older African American and white subjects Persons without evidence of cognitive impairment (normal) were treated as a ref. group that was contrasted with MCI and AD subgroups. To test whether cognitive trajectories associated with	function following the diagnostic evaluation of mild cognitive impairment, dementia, and AD Analyses focused on cognitive change in persons after they underwent clinical evaluation by multiplying each variable by study time.	individual measures: **(1) immediate** and (2) **delayed word recall tests** (taps episodic memory), **(3) perceptual speed, and (4) MMSE** **A global measure** of cognition made up of multiple individual domains	with terms for the interaction of race with each diagnosis multiplied by study time.	factor-based composite measure of global cognition did not differ for African American and Caucasian individuals diagnosed as cognitively normal, mildly cognitively impaired, or having dementia

APPENDIX 2.1 ACL – Wave 4 Number of Recent Life Events Index

Next, I have some questions about experiences you may have had since we interviewed you last in FILL (MONTH OF IW, 1994, 1989 OR 1986). Since we interviewed you in FILL (MONTH OF IW, 1994, 1989 OR 1986)…?

	YES	NO
Have you become widowed?	1	5
Were you robbed or was your home burglarized?	1	5
Have you involuntarily lost a job for reasons other than retirement?	1	5
Have you been the victim of a serious physical attack or assault?	1	5
Has a parent or step-parent of yours died?	1	5
**Have you had a life-threatening illness or accidental injury?	1	5
**Have you had any serious, but not life threatening, illness or injury that occurred or got worse?	1	5
Have you gotten a divorce?	1	5
Has a child of yours died?	1	5
Other than a spouse, parent or child, has a close relative or one of your close friends died?	1	5
Have you had any serious financial problems or difficulties?	1	5
Has anything (else) bad happened to you that upset you a lot and that you haven't already told me about?	1	5

******Items excluded from analysis

APPENDIX 2.2 ACL – Wave 4 Everyday Discrimination Scale

In your day-to-day life how often have any of the following things happened to you? (Would you say at least once a week, a few times a month, a few times a year, less than once a year, or never?)

	AT LEAST ONCE A WEEK	A FEW TIMES A MONTH	A FEW TIMES A YEAR	LESS THAN ONCE A YEAR	NEVER
You are treated with less courtesy or respect than other people.	1	2	3	4	5
You receive poorer service than other people at restaurants or stores.	1	2	3	4	5
People act as if they think you are not smart.	1	2	3	4	5
People act as if they are afraid of you.	1	2	3	4	5
You are threatened or harassed.	1	2	3	4	5

APPENDIX 2.3 ACL – Wave 5 Everyday Discrimination Scale

In your day-to-day life how often have any of the following things happened to you? (Would you say at least once a week, a few times a month, a few times a year, less than once a year, or never?)

	A FEW TIMES A MONTH OR MORE	A FEW TIMES A YEAR	LESS THAN ONCE A YEAR	NEVER
You are treated with less courtesy or respect than other people.	1	2	3	4
You receive poorer service than other people at restaurants or stores.	1	2	3	4
People act as if they think you are not smart.	1	2	3	4

APPENDIX 2.4 Imputed Values for Wave 5 Cognitive Impairment Predicted by Demographic and Health Conditions. Americans' Changing Lives Study (ACL), Combined Samples for 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105) (N=1,680).

Characteristic	B	**(SE)**
Black Race (ref=White)	0.183***	0.063
Age	0.017***	0.002
Female	0.026	0.050
Education (ref=less than high school)		
College Graduate and Beyond	-0.189**	0.067
Some College	-0.290***	0.078
High School Graduate	-0.324***	0.089
Baseline Income (per $10k)	-0.026	0.014
Diabetes	-0.042	0.144
Current Smoker (ref=non-current smoker)	-0.011	0.055
Depressive Symptoms	0.062**	0.024
Cognitive Impairment, wave 4	0.316***	0.042
Intercept	1.303***	0.111

*** p < 0.05; **p < .01; ***p < .001**

APPENDIX 2.5 Comparison of Characteristics of Adult Respondents in Study Subsample (both waves 4 and 5, N=1,105) with Those Who Dropped Out After Wave 4 (non-responders, N=575) (unweighted). Americans' Changing Lives Study (ACL), Samples for 2001/2002 (wave 4) and 2011 (wave 5) (N=1,680).

	Mean		Percentage		
Characteristics	**Dropped out after wave 4 (N=575)**	**Study Subsample in waves 4 and 5 (N=1,105)**	**Dropped out after wave 4 (N=575)**	**Study Subsample in waves 4 and 5 (N=1,105)**	**p-value for difference[a]**
DEMOGRAPHICS					
Black Race (ref=White)			28.5	24.8	= 0.099
Age* **	58.2	42.4			< 0.001
Female*			66.6	61.8	= 0.053
Education* **					< 0.001
College Graduate and Beyond			11.8	22.8	
Some College			19.5	26.4	
High School Graduate			32.0	35.0	
Less than High School			36.7	15.8	
Baseline Income (adjusted mid-points)* **					< 0.001
$3,125			11.5	6.2	
$7,250			15.8	9.6	
$12,010			15.0	10.0	
$17,210			12.7	8.5	
$22,040			8.2	10.1	
$26,910			8.9	11.6	
$33,450			11.3	16.8	
$46,940			10.4	17.2	
$65,950			3.7	6.1	

$85,230			2.6	4.1	
Marital Status* **					< 0.001
Married			58.8	65.6	
Separated/Divorced/Widowed			35.3	20.1	
Never Married			5.9	14.3	
RISK FACTORS					
Diabetes, wave 4					= 0.132
No			20.6	29.5	
Yes			79.4	70.5	
Smoking Status, wave 4					= 0.084
Current Smoker			12.5	16.0	
Former Smoker			41.7	37.5	
Never Smoked			45.7	46.5	
Depressive Symptoms, wave 4* **	12.9	14.2			< 0.001
Cognitive Impairment, wave 4* **	0.9	0.4			< 0.001
Religious Participation, wave 4	4.2	4.3			= 0.488
Religious Importance, wave 4*	2.6	2.5			= 0.016
Spiritual Coping, wave 4*	6.2	5.9			= 0.018
Stressful Life Events, wave 4* **	1.3	1.6			< 0.001
Discrimination, wave 4* **	2.0	2.9			< 0.001

aTests of difference between groups: continuous variables comparisons evaluated with independent samples t-tests; Pearson's chi-square for race, gender, education, income, marital status, diabetes and smoking status.
*** p < 0.05; **p < .01; ***p < .001**

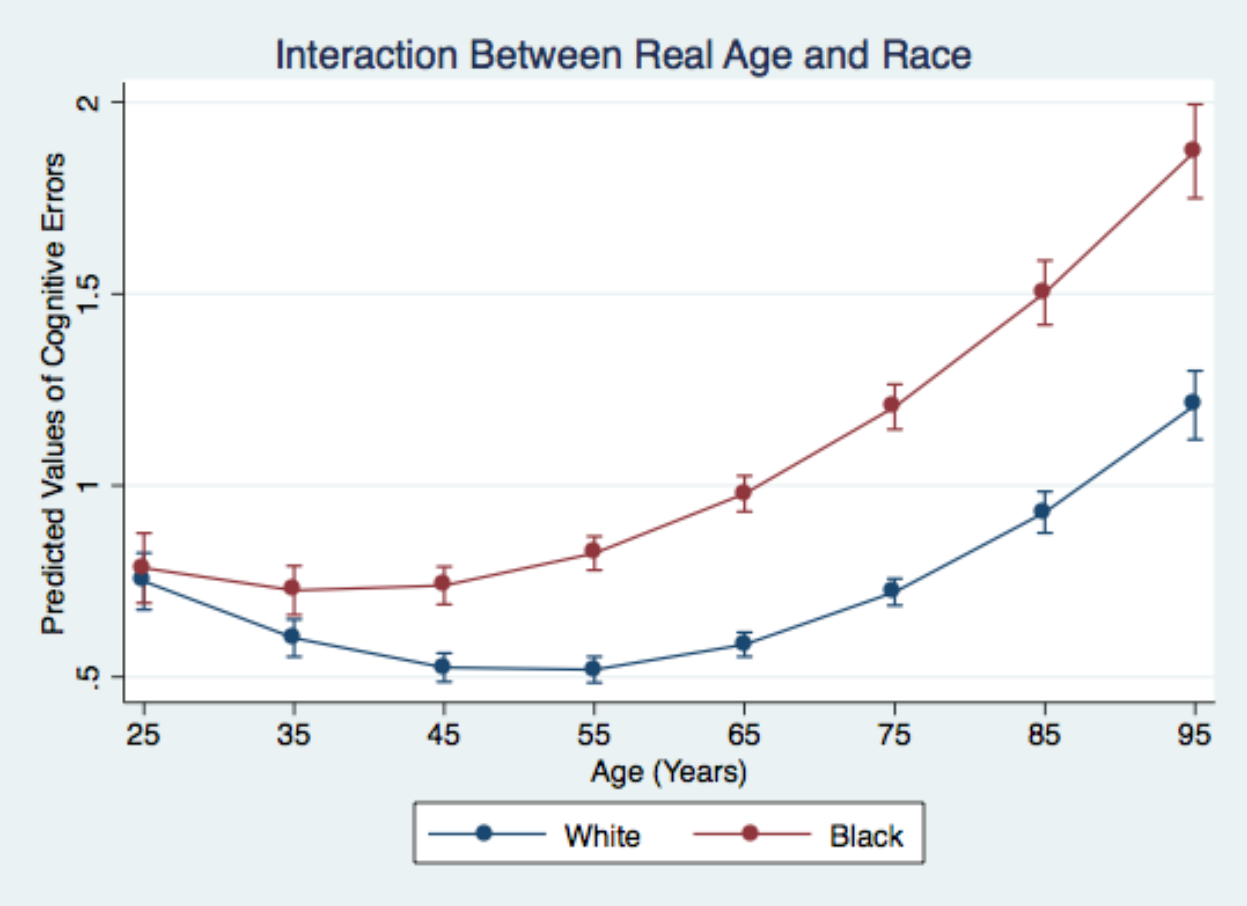
APPENDIX 3.1 Mixed Models of Cognitive Impairment on Sociodemographic, Chronic Conditions, Health Behaviors and Mental Health Characteristics for Non-Hispanic Black and White Respondents. Americans' Changing Lives Study (ACL), 1986-2011 (waves 1-5).

	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Fixed Effects	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)
Age		0.011 (0.0006)***	-0.032 (0.004)***	-0.035 (0.004)***	-0.036 (0.004)***
Age^2			0.0004 (0.00003)***	0.0004 (0.00004)***	0.0004 (0.00003)***
Black Race (ref=White)				0.200 (0.211)	-0.190 (0.079)*
Age*Black Race Interaction				-0.0002 (0.008)	0.009 (0.001)***
Age^2*Black Race Interaction				0.0001 (0.00007)	
Female					0.007 (0.025)
Education (ref=less than high school)					
High School Graduate					-0.327 (0.032)***
Some College					-0.442 (0.036)***
College Graduate and Beyond					-0.560 (0.042)***
Baseline Income (adjusted mid-points)[a]					
$7,250					-0.226 (0.043)***
$12,010					-0.336 (0.046)***
$17,210					-0.409 (0.051)***
$22,040					-0.395 (0.054)***
$26,910					-0.362 (0.055)***
$33,450					-0.407 (0.052)***

$46,940					-0.376 (0.054)***
$65,950					-0.390 (0.072)***
$85,230					-0.428 (0.080)***
Marital Status (ref=married)					
Separated, Divorced or Widowed					0.006 (0.022)
Never Married					0.011 (0.039)
Diabetes[b]					0.041 (0.029)
Stroke[b]					0.323 (0.067)***
Newly Diagnosed Stroke					0.149 (0.059)**
Current Smoker[b]					0.018 (0.026)
Depressive Symptoms					0.009 (0.006)
Intercept	0.792 (0.014)***	0.157 (0.041)***	1.267 (0.099)***	1.322 (0.102)***	2.008 (0.111)***
Random Effects (variance components)					
Random Intercept, τ_{00} (between-person variance)	0.48	0.44	0.44	0.35	0.26
Residual, σ_ε^2 (within-person variance)	0.52	0.52	0.51	0.51	0.52
X^2		271.64***	425.54***	982.68***	1777.33***
d.f.	0	1	2	4	24

[a]$3,125 is the reference category.
[b]1=condition reported.
*p < .05; **p < .01; ***p < .001

Figure 3.1a The Relationship Between Real Ages and Cognitive Impairment as Moderated by Race, Americans' Changing Lives Study (Ages 25-95)



NOTE: Graph of Model 5 from Appendix 3.1 above using uncentered (real) ages and adjusting for sociodemographic, chronic conditions, health behaviors and mental health characteristics**.**

APPENDIX 3.2 Sensitivity Analysis of the Effects of Education, Stressful Life Events, and Discrimination on Cognitive Impairment Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples for 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Variables	beta (SE)	beta (SE)			beta (SE)
Black Race (ref=White)	0.379*** (.063)	0.157* (.063)	0.010 (.171)	0.097 (.115)	0.069 (.082)
Age		0.015*** (.002)	0.015*** (.002)	0.015*** (.002)	0.015*** (.002)
Female		0.021 (.062)	0.027 (.059)	0.028 (.060)	0.024 (.061)
Education (ref=less than high school)					
High School Graduate		-0.261** (.101)	-0.300** (.110)	-0.265** (.099)	-0.265** (.100)
Some College		-0.335*** (.095)	-0.361*** (.102)	-0.340*** (.094)	-0.340*** (.095)
College Graduate and Beyond		-0.414*** (.081)	-0.444*** (.085)	-0.418*** (.080)	-0.418*** (.080)
Baseline Income (per $10k)		-0.016 (.013)	-0.015 (.013)	-0.016 (.013)	-0.016 (.013)
Married (ref=not married)[a]		-0.018 (.091)	-0.020 (.089)	-0.022 (.089)	-0.014 (.091)
Diabetes		-0.068 (.125)	-0.080 (.128)	-0.074 (.128)	-0.064 (.126)
Current Smoker (ref=non-current smoker)		0.015 (.050)	0.012 (.049)	0.015 (.049)	0.017 (.050)
Depressive Symptoms		0.017*** (.005)	0.016*** (.005)	0.016*** (.005)	0.017*** (.005)
Stressful Life		0.007 (.021)		-0.0004 (.021)	

Events (9-items)					
Everyday Discrimination (4-items)		-0.009 (.012)			-0.012 (.011)
Cognitive Impairment, Baseline (wave 4)		0.403*** (.051)	0.400*** (.053)	0.401*** (.051)	0.403*** (.051)
Race*Education Interaction (ref=less than high school)					
Black* High School Graduate			0.301 (.224)		
Black* Some College			0.110 (.226)		
Black* College Graduate and Beyond			0.090 (.217)		
Black* Stressful Life Events				0.037 (.054)	
Black* Everyday Discrimination					0.029 (.021)
Intercept	0.474 *** (.026)	-0.118 (.174)	-0.108 (.173)	-0.134 (.173)	-0.101 (.167)
R^2	0.018	0.246	0.247	0.246	0.246

[a]Not married includes separated, divorced, widowed and never married.

NOTE: Estimates are weighted to account for the sampling design.

beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

APPENDIX 3.3 Sensitivity Analysis of the Effects of Religion and Spirituality on Stressful Life Events, Discrimination and Cognitive Impairment Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples for 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**	**Model 7**
	beta (SE)	beta (SE)	beta (SE)				beta (SE)
Black Race (ref=White)	0.166** (.062)	0.154* (.062)	0.146* (.060)	0.151** (.056)	0.164** (.064)	0.155** (.062)	0.163** (.059)
Age	0.014*** (.002)	0.015*** (.002)	0.015*** (.002)	0.015*** (.002)	0.015*** (.002)	0.014*** (.002)	0.014*** (.002)
Female	0.022 (.065)	0.030 (.059)	0.019 (.062)	0.031 (.063)	0.022 (.059)	0.016 (.062)	0.030 (.063)
Education (ref=less than high school)							
High School Graduate	-0.256** (.100)	-0.259** (.101)	-0.263** (.095)	-0.273** (.096)	-0.259** (.102)	-0.259** (.100)	-0.265** (.100)
Some College	-0.331*** (.096)	-0.332*** (.0987)	-0.336*** (.091)	-0.346*** (.092)	-0.335*** (.096)	-0.331*** (.094)	-0.338*** (.095)
College Graduate and Beyond	-0.402*** (.081)	-0.410*** (.082)	-0.412*** (.077)	-0.420*** (.078)	-0.412*** (.082)	-0.410*** (.080)	-0.412*** (.081)
Baseline Income (per $10k)	-0.016 (.013)	-0.015 (.013)	-0.015 (.013)	-0.016 (.013)	-0.016 (.013)	-0.015 (.013)	-0.016 (.013)
Married (ref=not married)[a]	-0.018 (.090)	-0.019 (.088)	-0.017 (.092)	-0.019 (.090)	-0.022 (.090)	-0.016 (.091)	-0.019 (.090)
Diabetes	-0.054 (.124)	-0.078 (.128)	-0.067 (.124)	-0.069 (.128)	-0.071 (.124)	-0.064 (.122)	-0.070 (.124)
Current Smoker (ref=non-current smoker)	0.017 (.052)	0.011 (.050)	0.015 (.050)	0.019 (.051)	0.017 (.050)	0.018 (.050)	0.019 (.051)
Depressive	0.017***	0.016***	0.016***	0.017***	0.018***	0.018***	0.017***

Symptoms	(.005)	(.005)	(.005)	(.005)	(.005)	(.005)	(.005)
Religious Participation	0.0004 (.009)	0.010 (.012)			0.006 (.009)		
Religious Importance	0.060 (.040)		0.013 (.044)			0.036 (.031)	
Spiritual Coping	-0.017 (.014)			-0.011 (.013)			-0.002 (.012)
Cognitive Impairment, Baseline (wave 4)	0.404*** (.053)	0.401*** (.051)	0.401*** (.052)	0.402*** (.053)	0.403*** (.052)	0.402*** (.053)	0.403*** (.053)
Stressful Life Events (9-items)	0.016 (.020)	0.033 (.033)	-0.001 (.077)	-0.038 (.047)			
Everyday Discrimination (4-items)	-0.010 (.012)				0.002 (.016)	0.012 (.028)	-0.008 (.023)
Stressful Life Events*Religion & Spirituality Interactions							
Stressful Life Events*Religious Participation		-0.008 (.007)					
Stressful Life Events*Religious Importance			0.002 (.032)				
Stressful Life Events*Spiritual Coping				0.007 (.009)			
Discrimination* Religion & Spirituality Interactions							
Discrimination* Religious					-0.003 (.003)		

Participation							
Discrimination* Religious						-0.009 (.011)	
Importance							
Discrimination* Spiritual Coping							-0.0002(.004)
Intercept	-0.168 (.173)	-0.186 (.181)	-0.171 (.184)	-0.071 (.173)	-0.147 (.170)	-0.204 (.171)	-0.103 (.172)
R^2	0.246	0.245	0.245	0.245	0.246	0.247	0.245

[a]Not married includes separated, divorced, widowed and never married.
NOTE: Estimates are weighted to account for the sampling design.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

APPENDIX 3.4 Sensitivity Analysis of the Effects of Stressful Life Events, Discrimination, Religion and Spirituality on Depressive Symptoms Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples for 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	Model 2	Model 3	Model 4
	beta (SE)	beta (SE)	beta (SE)	beta (SE)
Black Race (ref=White)	0.560 (.318)	1.061*** (.335)	0.835 (.569)	0.503 (.484)
Age	-0.030** (.012)	-0.045*** (.011)	-0.043*** (.012)	-0.031** (.011)
Female	0.851*** (.260)	0.965*** (.269)	0.720 (.252)	0.916*** (.253)
Education (ref=less than high school)				
High School Graduate	-0.721 (.518)	-0.705 (.488)	-0.794 (.518)	-0.755 (.527)
Some College	-0.754 (.474)	-0.851 (.456)	-0.855 (.487)	-0.802 (.463)
College Graduate and Beyond	-0.958 (.492)	-1.110* (.505)	-1.022* (.515)	-1.012* (.502)
Baseline Income (per $10k)	-0.219*** (.060)	-0.270*** (.062)	-0.241 (.062)	-0.232*** (.058)
Married (ref=not married)[a]	0.734* (.355)	-0.763 (.371)	-0.828* (.356)	0.725* (.362)
Diabetes	1.103 (1.219)	1.336 (1.238)	1.311 (1.176)	1.100 (1.252)
Current Smoker (ref=non-current smoker)	0.247 (.299)	0.260 (.332)	0.272 (.297)	0.282 (.304)
Stressful Life Events	0.248* (.119)		0.367** (.140)	
Everyday Discrimination	0.211*** (.045)			0.229*** (.052)
Religious Participation		-0.010 (.056)		
Religious Importance		0.073 (.253)		
Spiritual Coping		-0.123 (.092)		
Race*Stressful Life Events Interaction			-0.065 (.279)	
Race*Discrimination Interaction				0.038 (.118)
Intercept	15.901*** (.795)	18.152*** (.764)	17.045*** (.789)	16.315*** (.710)

R^2	0.129	0.099	0.108	0.123

[a]Not married includes separated, divorced, widowed and never married.
NOTE: Estimates are weighted to account for the sampling design.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

In the above sensitivity analysis, I examined depressive symptoms as an outcome given that it is a possible risk factor for cognitive impairment and it attenuates the association between discrimination and poorer cognitive test performance among older African Americans. Thus, the goal of this sensitivity analysis (shown above in Appendix 3.4) was to disentangle the associations among discrimination, depressive symptoms, religion, spirituality, and cognition. The results show that both discrimination and stressful life events are significantly associated with depressive symptoms and these associations are not modified by race/ethnicity. Also, religion and spirituality do not buffer against depressive symptoms. These findings are consistent with the idea that the association of discrimination with poorer cognitive test performance is not independent of depressive symptoms. This mediated scenario is described by Barnes and her colleagues (2012) as "perceived discrimination might lead to increased depressive symptoms, which in turn might lead to poorer cognitive test performance (p.9)" Although, this study did not test the hypothesized mediated role of depression in the relationship between discrimination and cognitive impairment, future studies should given that discrimination is independently associated with depressive symptoms, depression is a significant predictor of cognitive impairment at wave 5, and the association between discrimination and cognition is reduced after adjusting for depression.

APPENDIX 3.5 Nested Ordinary Least Squares Models of the Effects of Age, Depressive Symptoms and Baseline Cognitive Impairment on Wave 5 Cognitive Impairment Among Non-Hispanic Black and White Adults (weighted). Americans' Changing Lives Study (ACL), Samples for 2001/2002 (wave 4) and 2011 (wave 5) (N=1,105).

	Model 1	**Model 2**	**Model 3**
Variables	beta (SE)	beta (SE)	beta (SE)
Black Race (ref=White)	0.379*** (.063)	0.187** (.061)	0.153* (.064)
Age		0.016*** (.002)	0.015*** (.002)
Depressive Symptoms		0.022*** (.005)	0.017*** (.005)
Cognitive Impairment, Baseline (wave 4)		0.463*** (.055)	0.405*** (.052)
Education (ref=less than high school)			
High School Graduate			-0.259* (.110)
Some College			-0.333*** (.096)
College Graduate and Beyond			-0.411*** (.082)
Stressful Life Events			0.017 (.017)
Everyday Discrimination			-0.008 (.011)
Female			0.020 (.062)
Baseline Income (re-scaled per $10k)			-0.015 (.013)
Married (ref=not married)[a]			-0.017 (.091)
Diabetes			-0.064 (.121)
Current Smoker (ref=non-current smoker)			0.014 (.051)
R^2	0.018	0.219	0.246

[a]Not married includes separated, divorced, widowed and never married.
NOTE: Estimates are weighted to account for the sampling design.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

In the above sensitivity analysis I examined whether the race parameter is confounded by age, depressive symptoms, and baseline (wave 4) cognition. The goal of this sensitivity analysis was to determine the relative contribution of education level in relationship to age, depression and baseline cognition. As shown in Model 2, the majority of the race effect is confounded by these three variables since they greatly reduce, but do not eliminate, the black-white disparity in

wave 5 cognitive impairment. In Model 3, education remains significant but has a modest effect in reducing racial disparities compared to age, depression and baseline cognition. Although the majority of the race effect is confounded by age, depression and baseline cognition, one's level of education continues to directly impact the rate of change in wave 5 cognitive impairment scores, over and above these covariates.

APPENDIX 3.6 Sensitivity Analysis of the Effects of Race and Education on Cognitive Impairment Orientation to Time Items Among Non-Hispanic Black and White Adults. Americans' Changing Lives Study (ACL), 1986 (wave 1) (N=3,617).

	Model 1[a]	**Model 2**	**Model 3[b]**	**Model 4**
	beta (SE)	beta (SE)	beta (SE)	
Black Race (ref=White)	0.293***(.087)	0.114 (.091)	0.607*** (.151)	0.371* (.156)
Education (ref=less than high school)				
High School Graduate		-0.588*** (.102)		-0.976*** (.194)
Some College		-0.646*** (.117)		-0.987*** (.226)
College Graduate and Beyond		-1.060*** (.152)		-1.048*** (.274)
Intercept	-1.438*** (.054)	-0.965*** (.076)	-3.079*** (.104)	-2.467*** (.130)
R^2	0.003	0.024	0.011	0.039

[a]Identify today's date (month, day and year) item.
[b]Identify what day of the week it is item.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

APPENDIX 3.7 Sensitivity Analysis of the Effects of Race and Education on Cognitive Impairment Knowledge of Current and Past Affairs Items Among Non-Hispanic Black and White Adults. Americans' Changing Lives Study (ACL), 1986 (wave 1) (N=3,617).

	Model 1[a]	Model 2	Model 3[b]	Model 4
	beta (SE)	beta (SE)	beta (SE)	
Black Race (ref=White)	0.960***(.182)	0.576*** (.188)	0.356*** (.079)	0.371* (.156)
Education (ref=less than high school)				
High School Graduate		-1.447*** (.256)		-0.976*** (.194)
Some College		-2.071*** (.395)		-0.987*** (.226)
College Graduate and Beyond		-2.506*** (.590)		-1.048*** (.274)
Intercept	-3.666*** (.137)	-2.735*** (.154)	-1.070*** (.049)	-2.467*** (.130)
R^2	0.024	0.101	0.006	0.039

[a]Identify current President item.
[b]Identify former President item.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

APPENDIX 3.8 Sensitivity Analysis of the Effects of Race and Education on Cognitive Impairment Serial 3's Subtraction Test Among Non-Hispanic Black and White Adults. Americans' Changing Lives Study (ACL), 1986 (wave 1) (N=3,617).

	Model 1[a]	**Model 2**
	beta (SE)	beta (SE)
Black Race (ref=White)	1.221***(.080)	1.007*** (.083)
Education (ref=less than high school)		
High School Graduate		-0.866*** (.094)
Some College		-1.292*** (.116)
College Graduate and Beyond		-1.634*** (.152)
Intercept	-1.395*** (.053)	-0.657*** (.071)
R^2	0.056	0.110

[a]Subtract 3 from 20 items.
beta = unstandardized coefficient; SE = standard error.
*p < .05 **p < .01 ***p < .001

REFERENCES

Agli O, Bailly N and Ferrand C (2014). "Spirituality and religion in older adults with dementia: A systematic review." Int Psychogeriatr: 1-11.

Airhihenbuwa CO and Laveist TA (2006). "Racial and ethnic approaches to community health (REACH) 2010." Health Promot Pract **7**(3 Suppl): 174S-175S.

Airhihenbuwa CO and Liburd L (2006). "Eliminating health disparities in the African American population: the interface of culture, gender, and power." Health Educ Behav **33**(4): 488-501.

Albert M, Smith LA, Scherr PA, Taylor JO, Evans DA and Funkenstein HH (1991). "Use of Brief Cognitive Tests to Identify Individuals in the Community with Clinically Diagnosed Alzheimer's Disease." International Journal of Neuroscience **57**(3-4): 167-178.

Allen RS, Haley PP, Harris GM and Fowler SN (2011). Resilience: Definitions, ambiguities and applications. Resilience in aging: Concepts, research and outcomes. B. Resnick, L. P. Gwyther and K. A. Roberto. New York, NY, Springer**:** 1-13.

Alley D, Suthers K and Crimmins E (2007). "Education and Cognitive Decline in Older Americans: Results From the AHEAD Sample." Res Aging **29**(1): 73-94.

Alzheimer's Association. (2011). "2010 Alzheimer's Disease Facts and Figures." Retrieved February 12, 2015, from https://www.alz.org/documents_custom/report_alzfactsfigures2010.pdf.

Alzheimer's Association (2017). "2017 Alzheimer's Disease Facts and Figures." Alzheimers Dementia **13**: 325-373.

Atkinson HH, Cesari M, Kritchevsky SB, Penninx BW, Fried LP, Guralnik JM and Williamson JD (2005). "Predictors of combined cognitive and physical decline." J Am Geriatr Soc **53**(7): 1197-1202.

Aud S, Fox MA and KewalRamani A (2010). Status and Trends in the Education of Racial and Ethnic Groups (NCES 2010-015). Washington, D.C.: U.S. Government Printing Office, U.S. Department of Education, National Center for Education Statistics.

Barnes LL, Lewis TT, Begeny CT, Yu L, Bennett DA and Wilson RS (2012). "Perceived discrimination and cognition in older African Americans." J Int Neuropsychol Soc **18**(5): 856-865.

Barnes LL, Mendes De Leon CF, Wilson RS, Bienias JL, Bennett DA and Evans DA (2004). "Racial differences in perceived discrimination in a community population of older blacks and whites." J Aging Health **16**(3): 315-337.

Barnes LL, Wilson RS, Hebert LE, Scherr PA, Evans DA and Mendes de Leon CF (2011). "Racial differences in the association of education with physical and cognitive function in older blacks and whites." J Gerontol B Psychol Sci Soc Sci **66**(3): 354-363.

Brondolo E, Libby DJ, Denton EG, Thompson S, Beatty DL, Schwartz J, Sweeney M, Tobin JN, Cassells A, Pickering TG and Gerin W (2008). "Racism and ambulatory blood pressure in a community sample." Psychosom Med **70**(1): 49-56.

Brosschot JF, Gerin W and Thayer JF (2006). "The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health." J Psychosom Res **60**(2): 113-124.

Brown TH, Richardson LJ, Hargrove TW and Thomas CS (2016). "Using Multiple-hierarchy Stratification and Life Course Approaches to Understand Health Inequalities: The Intersecting Consequences of Race, Gender, SES, and Age." J Health Soc Behav **57**(2): 200-222.

Byrd DR (2012). "Race/Ethnicity and self-reported levels of discrimination and psychological distress, California, 2005." Prev Chronic Dis **9**: E156.

Castora-Binkley M, Peronto CL, Edwards JD and Small BJ (2013). "A longitudinal analysis of the influence of race on cognitive performance." J Gerontol B Psychol Sci Soc Sci.

Chatters LM, Taylor RJ, Bullard KM and Jackson JS (2009). "Race and Ethnic Differences in Religious Involvement: African Americans, Caribbean Blacks and Non-Hispanic Whites." Ethn Racial Stud **32**(7): 1143-1163.

Chatters LM, Taylor RJ, Jackson JS and Lincoln KD (2008). "Religious Coping Among African Americans, Caribbean Blacks and Non-Hispanic Whites." J Community Psychol **36**(3): 371-386.

Clark R, Anderson NB, Clark VR and Williams DR (1999). "Racism as a stressor for African Americans. A biopsychosocial model." Am Psychol **54**(10): 805-816.

Coin A, Perissinotto E, Najjar M, Girardi A, Inelmen EM, Enzi G, Manzato E and Sergi G (2010). "Does religiosity protect against cognitive and behavioral decline in Alzheimer's dementia?" Curr Alzheimer Res **7**(5): 445-452.

Comijs HC, van den Kommer TN, Minnaar RW, Penninx BW and Deeg DJ (2011). "Accumulated and differential effects of life events on cognitive decline in older persons: Depending on depression, baseline cognition, or ApoE epsilon4 status?" J Gerontol B Psychol Sci Soc Sci **66 Suppl 1**: i111-120.

Dahl A, Berg S and Nilsson SE (2007). "Identification of dementia in epidemiological research: a study on the usefulness of various data sources." Aging Clin Exp Res **19**(5): 381-389.

Diaz-Venegas C, Downer B, Langa KM and Wong R (2016). "Racial and ethnic differences in cognitive function among older adults in the USA." Int J Geriatr Psychiatry **31**(9): 1004-1012.

Dohrenwend BP (2006). "Inventorying stressful life events as risk factors for psychopathology: Toward resolution of the problem of intracategory variability." Psychol Bull **132**(3): 477-495.

Early DR, Widaman KF, Harvey D, Beckett L, Park LQ, Farias ST, Reed BR, Decarli C and Mungas D (2013). "Demographic predictors of cognitive change in ethnically diverse older persons." Psychol Aging **28**(3): 633-645.

Eaton WW and Kessler LG (1981). "Rates of symptoms of depression in a national sample." Am J Epidemiol **114**(4): 528-538.

Erkinjuntti T, Sulkava R, Wikstrom J and Autio L (1987). "Short Portable Mental Status Questionnaire as a Screening Test for Dementia and Delirium Among the Elderly." Journal for the American Geriatrics Society **35**: 412-416.

Federal Interagency Forum on Aging Related Statistics (2012). Federal Interagency Forum on Aging Related Statistics. Older Americans 2012: Key Indicators of Well-Being. Washington, DC, U.S. Government Printing Office.

Folkman S (1997). "Positive psychological states and coping with severe stress." Soc Sci Med **45**(8): 1207-1221.

Folkman S (2008). "The case for positive emotions in the stress process." Anxiety Stress Coping **21**(1): 3-14.

Folstein MF, Folstein SE and McHugh PR (1975). ""Mini-mental state". A practical method for grading the cognitive state of patients for the clinician." J Psychiatr Res **12**(3): 189-198.

Frerichs RR, Aneshensel CS and Clark VA (1981). "Prevalence of depression in Los Angeles County." Am J Epidemiol **113**(6): 691-699.

Froehlich TE, Bogardus ST, Jr. and Inouye SK (2001). "Dementia and race: Are there differences between African Americans and Caucasians?" J Am Geriatr Soc **49**(4): 477-484.

Ganguli M, Blacker D, Blazer DG, Grant I, Jeste DV, Paulsen JS, Petersen RC, Sachdev PS and The Neurocognitive Disorders Work Group of the American Psychiatric Association's DSMTF (2011). "Classification of Neurocognitive Disorders in DSM-5: A Work in Progress." The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry **19**(3): 205-210.

Garrido P, de Blas M, Del Arco A, Segovia G and Mora F (2012). "Aging increases basal but not stress-induced levels of corticosterone in the brain of the awake rat." Neurobiol Aging **33**(2): 375-382.

Gee GC, Ro A, Shariff-Marco S and Chae D (2009). "Racial discrimination and health among Asian Americans: Evidence, assessment, and directions for future research." Epidemiologic Reviews **31**(1): 130-151.

Gee GC, Walsemann KM and Brondolo E (2012). "A Life Course Perspective on How Racism May Be Related to Health Inequities." American Journal of Public Health **102**(5): 967-974.

Geronimus AT (2000). "To mitigate, resist, or undo: Addressing structural influences on the health of urban populations." Am J Public Health **90**(6): 867-872.

Geronimus AT, Hicken M, Keene D and Bound J (2006). ""Weathering" and age patterns of allostatic load scores among blacks and whites in the United States." Am J Public Health **96**(5): 826-833.

Gurland BJ, Wilder DE, Lantigua R, Stern Y, Chen J, Killeffer EH and Mayeux R (1999). "Rates of dementia in three ethnoracial groups." Int J Geriatr Psychiatry **14**(6): 481-493.

Harrell CJ, Burford TI, Cage BN, Nelson TM, Shearon S, Thompson A and Green S (2011). "Multiple pathways linking racism to health outcomes." Du Bois Rev **8**(1): 143-157.

Harrell SP (2000). "A multidimensional conceptualization of racism-related stress: Implications for the well-being of people of color." Am J Orthopsychiatry **70**(1): 42-57.

Hawkins KA, Cromer JR, Piotrowski AS and Pearlson GD (2011). "Mini-Mental State Exam performance of older African Americans: effect of age, gender, education, hypertension, diabetes, and the inclusion of serial 7s subtraction versus "world" backward on score." Arch Clin Neuropsychol **26**(7): 645-652.

Hebert LE, Weuve J, Scherr PA and Evans DA (2013). "Alzheimer disease in the United States (2010-2050) estimated using the 2010 census." Neurology **80**(19): 1778-1783.

Hicken MT, Lee H, Ailshire J, Burgard SA and Williams DR (2013). ""Every shut eye, ain't sleep": The role of racism-related vigilance in racial/ethnic disparities in sleep difficulty." Race Soc Probl **5**(2): 100-112.

Hicken MT, Lee H, Morenoff J, House JS and Williams DR (2014). "Racial/ethnic disparities in hypertension prevalence: Reconsidering the role of chronic stress." Am J Public Health **104**(1): 117-123.

Hill PC and Pargament KI (2003). "Advances in the conceptualization and measurement of religion and spirituality. Implications for physical and mental health research." Am Psychol **58**(1): 64-74.

Holmes TH and Rahe RH (1967). "The social readjustment rating scale." Journal of Psychosomatic Research **11**(2): 213-218.

House JS, Lantz PM and Herd P (2005). "Continuity and change in the social stratification of aging and health over the life course: Evidence from a nationally representative longitudinal study from 1986 to 2001/2002 (Americans' Changing Lives Study)." J Gerontol B Psychol Sci Soc Sci **60 Spec No 2**: 15-26.

Hox JJ (2010). Multilevel Analysis: Techniques and Applications, Second Edition (Quantitative Methodology Series). New York, NY, Routledge.

James SA (1993). "Racial and ethnic differences in infant mortality and low birth weight. A psychosocial critique." Ann Epidemiol **3**(2): 130-136.

Kane R, Butler M, Fink H, Brasure M, Davila H, Desai P, Jutkowitz E, McCreedy E, Nelson V, McCarten J, Calvert C, Ratner E, Hemmy L and Barclay T (2017). Interventions To Prevent Age-Related Cognitive Decline, Mild Cognitive Impairment, and Clinical Alzheimer's-Type Dementia. Comparative Effectiveness Review No. 18. Rockville, MD.

Kantarci K, Weigand SD, Przybelski SA, Shiung MM, Whitwell JL, Negash S, Knopman DS, Boeve BF, O'Brien PC, Petersen RC and Jack CR, Jr. (2009). "Risk of dementia in MCI: combined effect of cerebrovascular disease, volumetric MRI, and 1H MRS." Neurology **72**(17): 1519-1525.

Karlamangla AS, Miller-Martinez D, Aneshensel CS, Seeman TE, Wight RG and Chodosh J (2009). "Trajectories of cognitive function in late life in the United States: Demographic and socioeconomic predictors." Am J Epidemiol **170**(3): 331-342.

Kaufman Y, Anaki D, Binns M and Freedman M (2007). "Cognitive decline in Alzheimer disease: Impact of spirituality, religiosity, and QOL." Neurology **68**(18): 1509-1514.

Kessler RC (1979). "Stress, social status, and psychological distress." J Health Soc Behav **20**(3): 259-272.

Kim J and Pai M (2010). "Volunteering and trajectories of depression." J Aging Health **22**(1): 84-105.

Koenig HG (1998). "Religious attitudes and practices of hospitalized medically ill older adults." Int J Geriatr Psychiatry **13**(4): 213-224.

Krause N and Chatters LM (2005). "Exploring race differences in a multidimensional battery of prayer measures among older adults." Sociology of Religion **66**(1): 23-43.

Krieger N (1994). "Epidemiology and the web of causation: has anyone seen the spider?" Soc Sci Med **39**(7): 887-903.

Landfield PW, Waymire JC and Lynch G (1978). "Hippocampal aging and adrenocorticoids: quantitative correlations." Science **202**(4372): 1098-1102.

Lantz PM, House JS, Mero RP and Williams DR (2005). "Stress, life events, and socioeconomic disparities in health: results from the Americans' Changing Lives Study." J Health Soc Behav **46**(3): 274-288.

Lazarus RS and Folkman S (1984). Stress, appraisal, and coping. New York, Springer.

Lee HB, Richardson AK, Black BS, Shore AD, Kasper JD and Rabins PV (2012). "Race and cognitive decline among community-dwelling elders with mild cognitive impairment: Findings from the Memory and Medical Care Study." Aging Ment Health **16**(3): 372-377.

Leng Y, Wainwright NWJ, Hayat S, Stephan BCM, Matthews FE, Luben R, Surtees PG, Khaw K-T and Brayne C (2013). "The association between social stress and global cognitive function in a population-based study: The European Prospective Investigation into Cancer (EPIC)-Norfolk study." Psychological Medicine **43**(03): 655-666.

Lewis TT, Barnes LL, Bienias JL, Lackland DT, Evans DA and Mendes de Leon CF (2009). "Perceived discrimination and blood pressure in older African American and white adults." J Gerontol A Biol Sci Med Sci **64**(9): 1002-1008.

Lines LM and Wiener JM (2014). Racial and ethnic disparities in Alzheimer's Disease: A literature review. U.S. Department of Health and Human Services, Assistant Secretary for Planning and Evaluation and A. Office of Disability, and Long-Term Care Policy. Washington, DC, Research Triangle Institute (RTI) International.

Lopez OL, Jagust WJ, Dulberg C, Becker JT, DeKosky ST, Fitzpatrick A, Breitner J, Lyketsos C, Jones B, Kawas C, Carlson M and Kuller LH (2003). "Risk factors for mild cognitive impairment in the Cardiovascular Health Study Cognition Study: Part 2." Arch Neurol **60**(10): 1394-1399.

Lupien SJ, Maheu F, Tu M, Fiocco A and Schramek TE (2007). "The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition." Brain Cogn **65**(3): 209-237.

Manly JJ (2006). "Deconstructing race and ethnicity: implications for measurement of health outcomes." Med Care **44**(11 Suppl 3): S10-16.

Manly JJ, Jacobs DM, Sano M, Bell K, Merchant CA, Small SA and Stern Y (1999). "Effect of literacy on neuropsychological test performance in nondemented, education-matched elders." J Int Neuropsychol Soc **5**(3): 191-202.

Manly JJ and Mayeux R (2004). Ethnic differences in dementia and Alzheimer's disease. Critical Perspectives on Racial and Ethnic Differences in Health in Late Life. N. Anderson, R. Bulatao and B. Cohen. Washington DC, National Academies Press.

Mare RD (1995). Changes in educational attainment and school enrollment. State of the union: America in the 1990s. R. Farley. New York, NY, Russell Sage**:** 155-213.

Marsiske M, Dzierzewski JM, Thomas KR, Kasten L, Jones RN, Johnson KE, Willis SL, Whitfield KE, Ball KK and Rebok GW (2013). "Race-related disparities in 5-year cognitive level and change in untrained ACTIVE participants." J Aging Health **25**(8 Suppl): 103S-127S.

Masel MC and Peek MK (2009). "Ethnic differences in cognitive function over time." Ann Epidemiol **19**(11): 778-783.

Masel MC, Raji M and Peek MK (2010). "Education and physical activity mediate the relationship between ethnicity and cognitive function in late middle-aged adults." Ethn Health **15**(3): 283-302.

Matallana D, de Santacruz C, Cano C, Reyes P, Samper-Ternent R, Markides KS, Ottenbacher KJ and Reyes-Ortiz CA (2011). "The relationship between education level and mini-mental state examination domains among older Mexican Americans." J Geriatr Psychiatry Neurol **24**(1): 9-18.

McEwen BS (1998). "Stress, adaptation, and disease. Allostasis and allostatic load." Ann N Y Acad Sci **840**: 33-44.

McEwen BS (1999). "Stress and hippocampal plasticity." Annu Rev Neurosci **22**: 105-122.

Mehta KM, Simonsick EM, Rooks R, Newman AB, Pope SK, Rubin SM and Yaffe K (2004). "Black and white differences in cognitive function test scores: What explains the difference?" J Am Geriatr Soc **52**(12): 2120-2127.

Mehta KM, Stewart AL, Langa KM, Yaffe K, Moody-Ayers S, Williams BA and Covinsky KE (2009). ""Below average" self-assessed school performance and Alzheimer's disease in the Aging, Demographics, and Memory Study." Alzheimers Dement **5**(5): 380-387.

Miller WR and Thoresen CE (2003). "Spirituality, religion, and health. An emerging research field." Am Psychol **58**(1): 24-35.

Mitchell AJ and Shiri-Feshki M (2009). "Rate of progression of mild cognitive impairment to dementia--meta-analysis of 41 robust inception cohort studies." Acta Psychiatr Scand **119**(4): 252-265.

Moreira-Almeida A, Neto FL and Koenig HG (2006). "Religiousness and mental health: A review." Rev Bras Psiquiatr **28**(3): 242-250.

Morris JC, Storandt M, Miller JP, McKeel DW, Price JL, Rubin EH and Berg L (2001). "Mild cognitive impairment represents early-stage Alzheimer disease." Arch Neurol **58**(3): 397-405.

National Academies of Sciences E, and Medicine (2017). Preventing Cognitive Decline and Dementia: A Way Forward. Washington, DC.

Paradies Y (2006). "A systematic review of empirical research on self-reported racism and health." Int J Epidemiol **35**(4): 888-901.

Pargament KI (2001). The psychology of religion and coping: Theory, research, practice. New York, NY, Guilford Press.

Pargament KI, Koenig HG and Perez LM (2000). "The many methods of religious coping: Development and initial validation of the RCOPE." J Clin Psychol **56**(4): 519-543.

Parker C and Philp I (2004). "Screening for cognitive impairment among older people in black and minority ethnic groups." Age Ageing **33**(5): 447-452.

Peavy GM, Salmon DP, Jacobson MW, Hervey A, Gamst AC, Wolfson T, Patterson TL, Goldman S, Mills PJ, Khandrika S and Galasko D (2009). "Effects of chronic stress on memory decline in cognitively normal and mildly impaired older adults." Am J Psychiatry **166**(12): 1384-1391.

Pedraza O, Clark JH, O'Bryant SE, Smith GE, Ivnik RJ, Graff-Radford NR, Willis FB, Petersen RC and Lucas JA (2012). "Diagnostic validity of age and education corrections for the Mini-Mental State Examination in older African Americans." J Am Geriatr Soc **60**(2): 328-331.

Perneczky R, Alexopoulos P, Schmid G, Sorg C, Forstl H, Diehl-Schmid J and Kurz A (2011). "[Cognitive reserve and its relevance for the prevention and diagnosis of dementia]." Nervenarzt **82**(3): 325-330, 332-335.

Petersen RC, Doody R, Kurz A, Mohs RC, Morris JC, Rabins PV, Ritchie K, Rossor M, Thal L and Winblad B (2001). "Current concepts in mild cognitive impairment." Arch Neurol **58**(12): 1985-1992.

Pollard K and Scommegna P (2013). Today's research on aging: The health and life expectancy of older Blacks and Hispanics in the United States. Washington, DC, Population Reference Bureau.

Potter GG, Plassman BL, Burke JR, Kabeto MU, Langa KM, Llewellyn DJ, Rogers MA and Steffens DC (2009). "Cognitive performance and informant reports in the diagnosis of cognitive impairment and dementia in African Americans and whites." Alzheimers Dement **5**(6): 445-453.

Radloff L (1977). "The CES-D scale: A self-reprt depression scale for research in the general population." Applied Psychological Measurement **1**(385).

Raudenbush SW and Bryk AS (2002). Hierarchical Linear Models Applications and Data Analysis Methods. Thousand Oaks, CA, Sage Publications.

Rexroth DF, Tennstedt SL, Jones RN, Guey LT, Rebok GW, Marsiske MM, Xu Y and Unverzagt FW (2013). "Relationship of demographic and health factors to cognition in older adults in the ACTIVE study." J Aging Health **25**(8 Suppl): 128S-146S.

Roberts R and Knopman DS (2013). "Classification and epidemiology of MCI." Clin Geriatr Med **29**(4): 753-772.

Rosnick CB, Small BJ, McEvoy CL, Borenstein AR and Mortimer JA (2007). "Negative life events and cognitive performance in a population of older adults." J Aging Health **19**(4): 612-629.

Ross CE, Mirowsky J and Huber J (1983). "Dividing work, sharing work, and in-between: Marriage patterns and depression." Am Sociol Rev **48**(6): 809-823.

Ryan CL and Siebens J (2012). Educational Attainment in the United States: 2009. **Washington, DC: U.S. Census Bureau**, U.S. Department of Commerce, Economics and Statistics Administration.

Ryff C, Friedman E, Fuller-Rowell T, Love G, Miyamoto Y, Morozink J, Radler B and Tsenkova V (2012). "Varieties of Resilience in MIDUS." Soc Personal Psychol Compass **6**(11): 792-806.

Sachs-Ericsson N and Blazer DG (2005). "Racial differences in cognitive decline in a sample of community-dwelling older adults: The mediating role of education and literacy." The American Journal of Geriatric Psychiatry **13**(11): 968-975.

Salvatore J and Shelton JN (2007). "Cognitive costs of exposure to racial prejudice." Psychol Sci **18**(9): 810-815.

Sapolsky RM (1986). "Glucocorticoid toxicity in the hippocampus: Reversal by supplementation with brain fuels." J Neurosci **6**(8): 2240-2244.

Sawyer K, Sachs-Ericsson N, Preacher KJ and Blazer DG (2008). "Racial Differences in the Influence of the APOE Epsilon 4 Allele on Cognitive Decline in a Sample of Community-Dwelling Older Adults." Gerontology **55**(1): 32-40.

Schulz AJ, Gravlee CC, Williams DR, Israel BA, Mentz G and Rowe Z (2006). "Discrimination, symptoms of depression, and self-rated health among african american women in detroit: results from a longitudinal analysis." Am J Public Health **96**(7): 1265-1270.

Schwartz BS, Glass TA, Bolla KI, Stewart WF, Glass G, Rasmussen M, Bressler J, Shi W and Bandeen-Roche K (2004). "Disparities in cognitive functioning by race/ethnicity in the Baltimore Memory Study." Environ Health Perspect **112**(3): 314-320.

Scuteri A, Palmieri L, Lo Noce C and Giampaoli S (2005). "Age-related changes in cognitive domains. A population-based study." Aging Clin Exp Res **17**(5): 367-373.

Sheffield KM and Peek MK (2011). "Changes in the prevalence of cognitive impairment among older Americans, 1993-2004: Overall trends and differences by race/ethnicity." Am J Epidemiol **174**(3): 274-283.

Sheffler JL (2013). Stress, race, and APOE: Understanding the risk factors of cognitive decline Electronic Theses, Treatises and Dissertations, Florida State University.

Singer JD and Willett JB (2003). Doing data analysis with the multilevel model for change. Applied Longitudinal Data Analysis: Modeling Change and Event. New York, NY, Oxford University Press**:** 75-137.

Sloan FA and Wang J (2005). "Disparities among older adults in measures of cognitive function by race or ethnicity." J Gerontol B Psychol Sci Soc Sci **60**(5): P242-250.

Stern Y (2002). "What is cognitive reserve? Theory and research application of the reserve concept." J Int Neuropsychol Soc **8**(3): 448-460.

Subramanyam A and Singh S (2016). "Mild cognitive decline: Concept, types, presentation, and management." Journal of Geriatric Mental Health **3**(1): 10-20.

Tang MX, Cross P, Andrews H, Jacobs DM, Small S, Bell K, Merchant C, Lantigua R, Costa R, Stern Y and Mayeux R (2001). "Incidence of AD in African-Americans, Caribbean Hispanics, and Caucasians in northern Manhattan." Neurology **56**(1): 49-56.

Taylor RJ, Chatters LM and Levin JS (2004). Religion in the Lives of African Americans: Social, Psychological and Health Perspectives. Thousand Oaks, Sage Press.

Thoits PA (1995). "Stress, coping, and social support processes: Where are we? What next?" J Health Soc Behav **Spec No**: 53-79.

Thompson VLS (2006). "Coping responses and the experience of discrimination." Journal of Applied Social Psychology **36**(5): 1198-1214.

Tschanz JT, Pfister R, Wanzek J, Corcoran C, Smith K, Tschanz BT, Steffens DC, Ostbye T, Welsh-Bohmer KA and Norton MC (2013). "Stressful life events and cognitive decline in late life: Moderation by education and age. The Cache County Study." Int J Geriatr Psychiatry **28**(8): 821-830.

Underwood LG (2006). "Ordinary spiritual experience: Qualitative research, interpretive guidelines, and population distribution for the daily spiritual experience scale." Archive for the Psychology of Religion **28**(1): 181–218.

Ward A, Tardiff S, Dye C and Arrighi HM (2013). "Rate of conversion from prodromal Alzheimer's disease to Alzheimer's dementia: a systematic review of the literature." Dement Geriatr Cogn Dis Extra **3**(1): 320-332.

Weuve J, Hebert LE, Scherr PA and Evans DA (2015). "Prevalence of Alzheimer disease in US states." Epidemiology **26**(1): e4-6.

Williams DR (1994). The measurement of religion in epidemiologic studies: Problems and prospects. Religious factors in aging and health: Theoretical foundations and methodological frontiers. J. S. Levin, Sage**:** 125-148.

Williams DR (1999). "Race, socioeconomic status, and health. The added effects of racism and discrimination." Ann N Y Acad Sci **896**: 173-188.

Williams DR and Mohammed SA (2009). "Discrimination and racial disparities in health: Evidence and needed research." J Behav Med **32**(1): 20-47.

Williams DR, Neighbors HW and Jackson JS (2003). "Racial/ethnic discrimination and health: Findings from community studies." Am J Public Health **93**(2): 200-208.

Williams DR, Yan Y, Jackson JS and Anderson NB (1997). "Racial differences in physical and mental health: Socio-economic status, stress and discrimination." J Health Psychol **2**(3): 335-351.

Wilson RS, Aggarwal NT, Barnes LL, Mendes de Leon CF, Hebert LE and Evans DA (2010). "Cognitive decline in incident Alzheimer disease in a community population." Neurology **74**(12): 951-955.

Wilson RS, Capuano AW, Sytsma J, Bennett DA and Barnes LL (2015). "Cognitive Aging in Older Black and White Persons." Psychology and aging **30**(2): 279-285.

Wilson RS, Hebert LE, Scherr PA, Barnes LL, Mendes de Leon CF and Evans DA (2009). "Educational attainment and cognitive decline in old age." Neurology **72**(5): 460-465.

Wolinsky FD, Bentler SE, Hockenberry J, Jones MP, Weigel PA, Kaskie B and Wallace RB (2011). "A prospective cohort study of long-term cognitive changes in older Medicare beneficiaries." BMC Public Health **11**: 710.

Wood RY, Giuliano KK, Bignell CU and Pritham WW (2006). "Assessing cognitive ability in research: use of MMSE with minority populations and elderly adults with low education levels." J Gerontol Nurs **32**(4): 45-54.

Yassa MA, Mattfeld AT, Stark SM and Stark CE (2011). "Age-related memory deficits linked to circuit-specific disruptions in the hippocampus." Proc Natl Acad Sci U S A **108**(21): 8873-8878.

Zsembik BA and Peek MK (2001). "Race differences in cognitive functioning among older adults." J Gerontol B Psychol Sci Soc Sci **56**(5): S266-274.