

# Racial Disparities in Cognitive Performance in Mid- and Late Adulthood: Analyses of Two Cohort Studies

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**OBJECTIVES:** To examine whether the attenuation of racial disparities observed in physical health outcomes at older ages can be extended to cognitive outcomes in mid- and late-life samples.

**DESIGN:** Cross-sectional associations between race and cognitive functioning were examined as a function of age.

**SETTING:** The National Survey of Midlife Development in the United States (MIDUS) and the Washington Heights-Inwood Columbia Aging Project (WHICAP).

**PARTICIPANTS:** Non-Hispanic African American or white individuals aged 40 and older (MIDUS; n = 3875, 10.5% African American) and non-Hispanic African American or white individuals aged 65 and older without a diagnosis of dementia (WHICAP; n = 2,729, 53.8% African American).

**MEASUREMENTS:** Composite scores of executive functioning and episodic memory.

**RESULTS:** Independent of main effects of age, birth cohort, sex, education, and chronic health conditions, significant interactions between age and race indicated that racial disparities in episodic memory and executive functioning were larger at younger than older age in both samples.

**CONCLUSION:** Attenuation of racial inequalities in older age can be extended to cognitive outcomes, which probably reflects selective survival. Research on cognitive disparities or on race-specific causes of cognitive outcomes in old age must incorporate corrections for selective survival if the goal is to identify causal predictors of cognitive outcomes rather than merely statistical predictors. *J Am Geriatr Soc* 64:959–964, 2016.

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Racial disparities exist for many health outcomes and multiple domains of cognition. Cognitive disparities are relevant to the study of dementia risk, which has been estimated to be twice as high in African Americans as in non-Hispanic whites.<sup>1</sup> Previous work has described how the magnitude of health disparities appears to depend on the age of the population under study. Health disparities for many outcomes appear to be less prominent in late life, which probably reflects selective survival.<sup>2,3</sup> However, health disparities for other outcomes appear to be more prominent in late life or stable across the life course.<sup>4</sup> The current study sought to extend previous research on physical health outcomes to cognition. Comparing the magnitude of cognitive disparities in younger people with those of older people will advance understanding of specific biases in research on dementia risk. Evidence of selective survival with respect to cognition would imply that conventional risk factor patterns for dementia may be distorted in older African Americans, and clinical predictions based on less-selected white peers will be inaccurate.

Older African Americans obtain lower scores than whites on a wide range of cognitive assessments.<sup>5–7</sup> Educational factors that differ across racial groups may account for these differences. More and better education leads to greater learning opportunities throughout life, as well as higher literacy levels, income, and occupational status, all of which may affect performance on neuropsychological testing.<sup>8,9</sup> The theory of cumulative advantage and disadvantage (CAD) posits that stress and resource limitations throughout different stages of life that accumulate over time lead to worse health outcomes.<sup>10–12</sup> Thus, CAD theory would suggest that racial disparities in cognitive performance would increase or widen with age.

Racial disparities in physical health have been well documented. For example, older African Americans have a higher prevalence of diabetes mellitus, hypertension,<sup>13,14</sup>

and cardiovascular disease<sup>15</sup> than whites, although there is evidence that the magnitude of these health disparities is smaller at older ages, which has been described as the age-as-leveler hypothesis.<sup>2,3</sup> Although the age-as-leveler hypothesis is often contrasted with CAD, it has been demonstrated that, for educational inequalities, selective survival processes could attenuate population-level disparities in old age, even as individual-level inequalities worsened.<sup>16</sup> With respect to racial disparities, higher mortality and shorter life expectancy for African Americans in early and midlife<sup>4</sup> results in a smaller proportion of African Americans than of whites surviving to late life. By definition, survivors have greater hardiness (physical and mental resilience), which would lead to the observed narrowing of racial disparities at later ages.<sup>17,18</sup> If the course of cognitive aging mirrors that of other health conditions, the age-as-leveler hypothesis would suggest that racial inequalities in cognitive performance would also appear smaller in the oldest individuals.

The current study tested whether cognitive disparities were larger or smaller at later ages in two large cohort studies in the United States: the National Survey of Midlife Development in the United States (MIDUS) and the Washington Heights-Inwood Columbia Aging Project (WHICAP). Larger disparities at later ages would be consistent with CAD theory, whereas smaller disparities at later ages would be consistent with the age-as-leveler hypothesis, although as discussed above, the age-as-leveler hypothesis and CAD theory are not mutually exclusive. The implications of this research question extend to any study that uses between-person contrasts to evaluate racial disparities or to seek the determinants of healthy cognitive aging across racial groups. Smaller cognitive disparities in older participants would suggest that selective survival is a major source of bias in studies determining racial patterns in cognitive aging.

## METHODS

### Participants and Procedures

Data for this cross-sectional study were drawn from the second wave (2004–06) of MIDUS (MIDUS-II), including the Milwaukee oversample, and all three waves (1992, 1999, 2009) of WHICAP.

MIDUS is a national sample of noninstitutionalized, English-speaking adults selected using random digit dialing.<sup>19</sup> MIDUS-II included a cognitive battery, as described below. Inclusion criteria for the current study were aged 40 and older (range 40–83, median 57), self-reported ethnicity of non-Hispanic, and self-reported race of white or black/African American at the time of MIDUS-II. If data on racial and ethnic identity were not available for the MIDUS-II visit, participant responses were imputed from the first MIDUS visit. The final sample included 3,453 whites and 404 African Americans.

WHICAP is a community-based longitudinal study of aging and dementia in northern Manhattan.<sup>1,20</sup> Participants were recruited from Medicare lists in three waves beginning in 1992, 1999, or 2009. The current study included data only from participants' baseline neuropsychological evaluations. Inclusion criteria for the current

study were aged 65 and older (range 65–102, median 75), self-reported ethnicity of non-Hispanic, self-reported race of white or black/African American, and no consensus diagnosis of dementia. The final sample included 1,260 whites and 1,469 African Americans.

The appropriate institutional review boards approved MIDUS and WHICAP, including informed consent. The institutional review board of Columbia University approved the current secondary data analysis.

### Cognitive Outcomes

Cognitive testing in MIDUS-II was conducted over the telephone using the Brief Test of Adult Cognition by Telephone (BTACT).<sup>21,22</sup> Previous factor analysis of the BTACT in MIDUS-II revealed that it comprises two factors, reflecting episodic memory and executive functioning.<sup>23</sup> Cognitive testing in WHICAP was conducted in person using a comprehensive neuropsychological battery.<sup>24</sup> Because MIDUS-II had a more-limited neuropsychological battery than WHICAP, cognitive composites were derived from the WHICAP battery to best match those available from MIDUS-II. A complete list of tests included in the episodic memory and executive functioning composites used in this study is shown in Table 1. Composite scores were computed separately in MIDUS-II and WHICAP as mean *z*-scores within each domain.

### Covariates

All analyses controlled for main effects of age, sex, race, education, and health (number of chronic conditions). In both studies, age was self-reported at the time of the cognitive assessment included in the current study. In MIDUS-II, self-reported education was quantified as a 12-category variable ranging from no school or some grade school to PhD, EdD, MD, DDS, LLB, LLD, JD, or other professional degree. In WHICAP, education was quantified as self-reported years of school (0–20). In MIDUS-II, health was quantified as the number of self-reported chronic conditions out of a list of 30 potential conditions. In WHICAP, health was quantified as the number of self-reported chronic conditions out of a list of 10 potential conditions.

**Table 1. Corresponding Cognitive Domains Assessed in the National Survey of Midlife Development in the United States (MIDUS) and Washington Heights-Inwood Columbia Aging Project (WHICAP) Samples**

Domain	MIDUS	WHICAP
Episodic memory		
Word list immediate	+	+
Word list delayed	+	+
Executive functioning		
Category fluency	+	+
Digits backward	+	–
Number series	+	–
Backward counting	+	–
Stop and go switch task	+	–
Letter fluency	–	+
Verbal abstraction	–	+

Because WHICAP participants included in this study were drawn from three recruitment waves, birth cohort was used as an additional covariate in WHICAP analyses. Birth cohort was a five-category variable reflecting birth year: (0 = lowest–1909, 1 = 1910–1919, 2 = 1920–1929, 3 = 1930–1939, 4 = 1940–highest).

**Statistical Analysis**

Racial differences were evaluated separately in each sample using *t*-tests for continuous variables and chi-square tests for categorical variables. Separate linear regressions were used to estimate main effects of age, race, and their interaction (product term), to predict episodic memory and executive functioning composites in MIDUS-II and WHICAP. As defined above, all analyses controlled for sex, education, and health. WHICAP analyses also controlled for birth cohort. Main-effects models stratified at the sample-specific medians ( $\geq 57$  for MIDUS,  $\geq 75$  for WHICAP) were used to interpret the linear interaction terms.

**RESULTS**

**Racial Differences**

Racial differences in MIDUS and WHICAP are shown in Table 2. African Americans in MIDUS were older, were more likely to be female, were less likely to have attended college, reported more chronic health conditions, and scored lower on episodic memory and executive functioning composites than whites. African Americans in WHICAP were more likely to be female, attended fewer years of school, reported more chronic health conditions, and scored lower on episodic memory and executive functioning composites than whites.

**Interactions Between Race and Age**

Results from linear regressions in MIDUS and WHICAP are shown in Table 3. In both samples, older age and African American race were each independently associated with poorer episodic memory and executive functioning. There were also significant interactions between age and race for episodic memory and executive functioning in both samples, indicating that racial differences were attenuated in older individuals.

Next, linear regressions stratified according to age group (split at sample-specific medians of  $\geq 57$  for MIDUS and  $\geq 75$  for WHICAP) were estimated. In MIDUS, the independent effect of race on episodic memory performance was stronger in younger ( $B = -0.420$ , 95% confidence interval (CI) =  $-0.552$  to  $-0.288$ ;  $P < .001$ ) than in older ( $B = -0.333$ , 95% CI =  $-0.474$  to  $-0.191$ ;  $P < .001$ ) participants. Similarly, the independent effect of race on executive functioning performance was stronger in younger ( $B = -0.855$ , 95% CI =  $-0.972$  to  $-0.739$ ;  $P < .001$ ) than in older ( $B = -0.673$ , 95% CI =  $-0.799$  to  $-0.547$ ;  $P < .001$ ) participants in MIDUS. In WHICAP, the independent effect of race on episodic memory performance was stronger in younger ( $B = -0.398$ , 95% CI =  $-0.495$  to  $-0.301$ ;  $P < .001$ ) than older

**Table 2. Characteristics of African-American and White Sample Members in the National Survey of Mid-life Development in the United States (MIDUS) and Washington Heights-Inwood Columbia Aging Project (WHICAP)**

Characteristic	African American	White	P-Value
<b>MIDUS</b>			
Age, mean $\pm$ SD	56.7 $\pm$ 10.6	58.0 $\pm$ 11.3	.01
Female, %	64.9	53.8	<.001
Any college, %	55.0	66.8	<.001
Number of chronic conditions, mean $\pm$ SD	3.53 $\pm$ 3.63	2.50 $\pm$ 2.47	<.001
Episodic memory, mean $\pm$ SD	-0.36 $\pm$ 0.98	0.00 $\pm$ 0.99	<.001
Executive functioning, mean $\pm$ SD	-0.82 $\pm$ 0.97	0.05 $\pm$ 0.96	<.001
<b>WHICAP</b>			
Age, mean $\pm$ SD	76.1 $\pm$ 6.5	76.2 $\pm$ 6.8	.57
Birth cohort, %			.07
Lowest–1909	5.3	5.4	
1910–1919	24.5	21.9	
1920–1929	37.6	38.3	
1930–1939	20.2	18.7	
1940–highest	12.3	15.8	
Female, %	70.6	61.5	<.001
Education, years, mean $\pm$ SD	11.5 $\pm$ 3.7	13.8 $\pm$ 3.7	<.001
Chronic conditions, mean $\pm$ SD	2.30 $\pm$ 1.66	2.03 $\pm$ 1.56	<.001
Episodic memory, mean $\pm$ SD	-0.20 $\pm$ 0.88	0.23 $\pm$ 0.95	<.001
Executive functioning, mean $\pm$ SD	-0.28 $\pm$ 0.75	0.34 $\pm$ 0.84	<.001

SD = standard deviation.

( $B = -0.264$ , 95% CI =  $-0.355$  to  $-0.174$ ;  $P < .001$ ) participants. Similarly, the independent effect of race on executive functioning performance was stronger in younger ( $B = -0.463$ , 95% CI =  $-0.540$  to  $-0.387$ ;  $P < .001$ ) than in older ( $B = -0.324$ , 95% CI =  $-0.95$  to  $-0.252$ ;  $P < .001$ ) participants in WHICAP. Interactions between race and age are displayed visually in Figure 1.

**DISCUSSION**

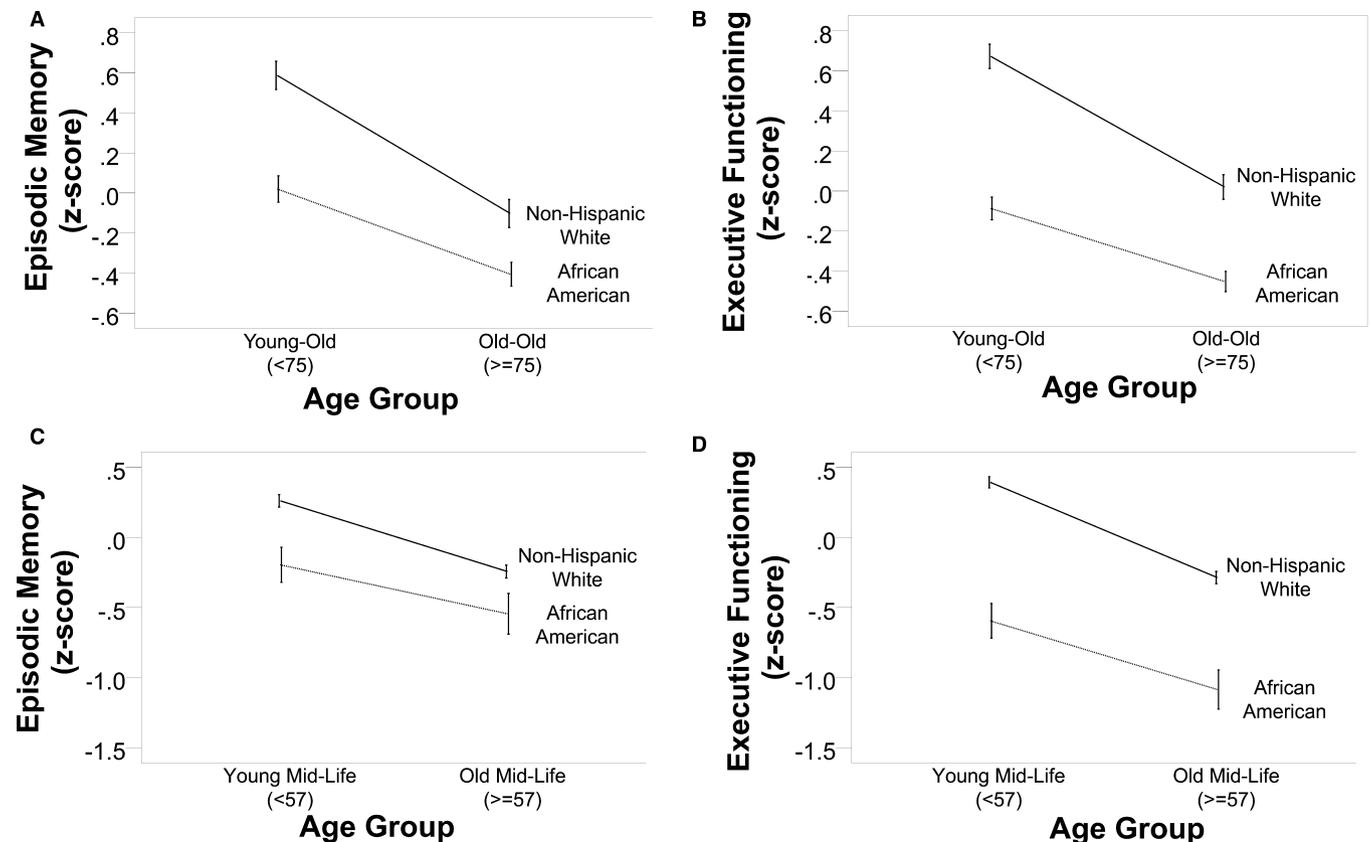
The results of this study indicate that the attenuation of racial inequalities at older ages that is observed for many physical health outcomes can be extended to cognitive outcomes. Specifically, racial differences in episodic memory and executive functioning were present across age groups but were smaller in older people in two large cohort studies conducted in middle-aged and older individuals. These findings were identified in the context of numerous racial differences in cognitive performance and demographic characteristics and were independent of birth cohort, sex, education, and burden of chronic health conditions. Results are consistent with the age-as-leveler hypothesis,<sup>3</sup> which may hold true if a smaller, more-resilient subset of African Americans than of whites survive to late life.

The present finding of attenuated racial disparities at later ages differs from that of a cross-sectional study of

**Table 3. Predictors of Cognition in Linear Models**

Predictor	Episodic Memory		Executive Functioning	
	Estimate (SE)	95% CI	Estimate (SE)	95% CI
<b>National Survey of Midlife Development in the United States</b>				
Age	-0.037 (0.005)	-0.047 to -0.027	-0.040 (0.005)	-0.049 to -0.031
Female	0.508 (0.031)	0.447-0.568	-0.088 (0.027)	-0.141 to -0.034
African American	-0.998 (0.263)	-1.515 to -0.481	-1.283 (0.233)	-1.739 to -0.827
Education	0.073 (0.006)	0.061-0.085	0.129 (0.005)	0.119-0.140
Chronic conditions	-0.022 (0.006)	-0.033 to -0.010	-0.035 (0.005)	-0.045 to -0.025
Age by African American	0.011 (0.005)	0.002-0.020	0.009 (0.004)	0.001-0.017
<b>Washington Heights-Inwood Columbia Aging Project</b>				
Age	-0.060 (0.008)	-0.077 to -0.044	-0.030 (0.007)	-0.042 to -0.017
Female	0.303 (0.034)	0.237-0.370	-0.043 (0.027)	-0.095-0.009
African American	-1.323 (0.368)	-2.045 to -0.602	-1.453 (0.285)	-1.982 to -0.857
Education	0.059 (0.005)	0.050-0.068	0.090 (0.004)	0.082-0.097
Chronic conditions	-0.014 (0.011)	-0.035-0.007	-0.007 (0.009)	-0.024-0.010
Birth cohort	0.052 (0.024)	0.004-0.099	0.193 (0.019)	0.156-0.231
Age by African American	0.013 (0.005)	0.004-0.022	0.013 (0.004)	0.006-0.021

SE = standard error; CI = confidence interval.



**Figure 1.** Cognitive composite scores plotted separately according to cognitive domain and study: (A) episodic memory in Washington Heights-Inwood Columbia Aging Project (WHICAP), (B) executive functioning in WHICAP, (C) episodic memory in the National Survey of Midlife Development in the United States (MIDUS), (D) executive functioning in MIDUS. Errors bars reflect 95% confidence intervals.

task-switching and processing speed that found significant interactions between age and race.<sup>25</sup> Specifically, larger racial disparities were found in older members of a sample recruited from Veterans Affairs medical centers, although that study was conducted in a highly specific population of veterans diagnosed with diabetes mellitus and included only 25 African Americans. The age-as-leveler hypothesis

would not necessarily be expected to apply when the healthiest older African Americans were likely to have been excluded from the study sample.

The present cross-sectional study is consistent with longitudinal studies showing lower rates of cognitive decline in older African Americans than in older whites.<sup>26,27</sup> That is, if whites start out at a higher cognitive

level but experience greater cognitive decline with age, then racial differences in cognitive performance in a longitudinal study would be expected to be smaller at each follow-up. In an early longitudinal study in the WHICAP cohort, racial disparities in the incidence of Alzheimer's disease appeared to be largest in the youngest group of older adults.<sup>1</sup> In a cross-sectional study, lower rates of cognitive decline in older African Americans would manifest as smaller racial differences between participants recruited at older ages. Lower rates of cognitive decline in older African Americans may also reflect selective survival, although other studies have reported faster rates of cognitive decline in older African Americans than in older whites.<sup>28–30</sup> Thus, additional longitudinal research is needed to identify the sources of disparate findings across cohorts (e.g., statistical bias due to baseline adjustment).<sup>31</sup> A major limitation of this study was that it was cross-sectional, although a longitudinal study comparing rates of cognitive decline between comparable samples of African Americans and whites (before the emergence of selective survival) would require extremely long follow-up.

Other limitations of this study include the disparate measures included in the executive functioning composites in MIDUS-II and WHICAP, because only one measure (category fluency) was common to both studies, although the use of composites rather than individual tests lessens the effect of these differences, and the consistency of results across studies despite measurement differences increases confidence in the finding of attenuated racial disparities at older ages. Because unequal interval scaling of the cognitive measures could obscure interpretation of the results, future studies should endeavor to include cognitive outcome measures calibrated across the entire range of ability using item response theory. It is possible that age-related racial differences in older adults' decisions to participate in research studies could have confounded the results. Specifically, it is possible that African Americans with cognitive difficulties are more likely than whites to decline participation as they get older. Such an explanation would reflect underlying inequalities, consistent with the finding of greater functional impairment in African Americans than in whites with similar chronic illness burden.<sup>4,32</sup> The current study attempted to address this by coordinating analyses from two large cohort studies that differed in many ways, including age, recruitment methods, racial composition, and geographical distribution.

Attenuation of social disparities at older ages is widely, although not universally, interpreted as indicating selective survival, such that the surviving members of the disadvantaged group are people with exceptional biological, environmental, psychological, or social profiles that provide resilience. Such selective survival has a challenging implication for research on the determinants of healthy cognitive aging; it is likely that any study that is based on between-person contrasts is biased. This would include, for example, studies based on prevalent or incident dementia or analyses that include between-person differences in estimating longitudinal changes. Many studies of differences in genetic causes of dementia between African Americans and whites would be vulnerable to such a bias. For example, if a gene is related to mortality and dementia, and its association with mortality is stronger in African

Americans, then its estimated effects on dementia in African Americans would be weaker. Multiple data sets have revealed weaker effects of the apolipoprotein E  $\epsilon$ 4 allele on dementia in African Americans than in whites.<sup>33,34</sup> The current study results strongly suggest that selective survival is a major determinant of racial patterns in cognitive aging. Research on disparities or on race-specific causes of cognitive outcomes in old age must incorporate corrections for such selective survival if the goal is to identify causal predictors of cognitive outcomes (which can be intervention targets), rather than merely statistical predictors.

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**Conflict of Interest:** The editor in chief has reviewed the conflict of interest checklist provided by the authors and has determined that the authors have no financial or any other kind of personal conflicts with this paper.

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## REFERENCES

1. Tang M-X, Cross P, Andrews H et al. Incidence of AD in African-Americans, Caribbean Hispanics, and Caucasians in northern Manhattan. *Neurology* 2001;56:49–56.
2. Crimmins EM, Hayward MD, Seeman TE. Race/ethnicity, socioeconomic status, and health. In: Anderson NB, Bulatao RA, Cohen B, eds. *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*. Washington, DC: National Academies Press, 2004.
3. Kim J, Miech R. The black-white difference in age trajectories of functional health over the life course. *Soc Sci Med* 2009;68:717–725.
4. Mendes de Leon CF, Barnes LL, Bienias JL et al. Racial disparities in disability: Recent evidence from self-reported and performance-based disability measures in a population-based study of older adults. *J Gerontol B Psychol Sci Soc Sci* 2005;60B:263–271.
5. Sisco S, Gross AL, Shih RA et al. The role of early-life educational quality and literacy in explaining racial disparities in cognition in late life. *J Gerontol B Psychol Sci Soc Sci* 2015;70B:557–567.
6. Brewster PWH, Marquine MJ, MacKay-Brandt A et al. Life experience and demographic influences on cognitive function in older adults. *Neuropsychology* 2014;28:846–858.
7. Schwartz BS, Glass TA, Bolla KI et al. Disparities in cognitive functioning by race/ethnicity in the Baltimore Memory Study. *Environ Health Perspect* 2004;112:314–320.
8. Stern Y, Gurland B, Tatemichi TK et al. Influence of education and occupation on the incidence of Alzheimer's disease. *JAMA* 1994;271:1004–1010.
9. Manly JJ, Jacobs DM, Touradji P et al. Reading level attenuates differences in neuropsychological test performance between African American and white elders. *J Int Neuropsychol Soc* 2004;8:341–348.

10. Byrd DA, Miller SW, Reilly J et al. Early environmental factors, ethnicity, and adult cognitive test performance. *Clin Neuropsychol* 2006;20:243–260.
11. Whystal S, Shea D. Cumulative advantage, cumulative disadvantage, and inequality among elderly people. *Gerontologist* 1990;30:437–443.
12. O’Rand AM, Henretta JC. *Age and Inequality: Diverse Pathways through Later Life*. Boulder, CO: Westview Press, 1999.
13. Pieterse AL, Carter RT. An exploratory investigation of the relationship between racism, racial identity, perceptions of health, and health locus of control among black American women. *J Health Care Poor Underserved* 2010;21:334–348.
14. Whitson HE, Hastings SN, Landerman LR et al. Black-white disparity in disability: The role of medical conditions. *J Am Geriatr Soc* 2011;59:844–850.
15. Margellos H, Silva A, Whitman S. Comparison of health status indicators in Chicago: Are black-white disparities worsening? *Am J Public Health* 2004;94:1612–1617.
16. Dupre ME. Educational differences in age-related patterns of disease: Reconsidering the cumulative disadvantage and age-as-leveler hypotheses. *J Health Soc Behav* 2007;48:1–15.
17. Glymour MM, Weuve J, Chen JT. Methodological challenges in causal research on racial and ethnic patterns of cognitive trajectories: Measurement, selection, and bias. *Neuropsychol Rev* 2008;18:194–213.
18. Johnson NE. The racial crossover in comorbidity, disability, and mortality. *Demography* 2000;37:267–283.
19. Brim O, Ryff C, Kessler R. *How Healthy Are We? A National Study of Well-Being at Midlife*. Chicago: University of Chicago Press, 2004.
20. Manly JJ, Tang M-X, Schupf N et al. Frequency and course of mild cognitive impairment in a multiethnic community. *Ann Neurol* 2008;63:494–506.
21. Lachman ME, Tun PA. Cognitive testing in large-scale surveys. Assessment by telephone. In: Hofer SM, Alwin DF, eds. *Handbook of Cognitive Aging: Interdisciplinary Perspectives*. Thousand Oaks, CA: Sage Publications, 2008, pp 506–523.
22. Tun PA, Lachman ME. Age differences in reaction time and attention in a national sample of adults: Education, sex, and task complexity matter. *Dev Psychol* 2008;44:1421–1429.
23. Lachman ME, Agrigoroaei S, Murphy C et al. Frequent cognitive activity compensates for education differences in episodic memory. *Am J Geriatr Psychiatry* 2010;18:4–10.
24. Stern Y, Andrews H, Pittman J et al. Diagnosis of dementia in a heterogeneous population. Development of a neuropsychological paradigm-based diagnosis of dementia and quantified correction for the effects of education. *Arch Neurol* 1992;49:453–460.
25. Obidi CS, Pugged JP, Fan X et al. Race moderates age-related cognitive decline in type 2 diabetes. *Exp Aging Res* 2008;34:114–125.
26. Early DR, Widaman KF, Harvey D et al. Demographic predictors of cognitive change in ethnically diverse older persons. *Psychol Aging* 2013;28:633–645.
27. Barnes LL, Wilson RS, Li Y et al. Change in cognitive function in Alzheimer’s disease in African American and white persons. *Neuroepidemiology* 2006;26:16–22.
28. Lee HB, Richardson AK, Black BS et al. Race and cognitive decline among community-dwelling elders with mild cognitive impairment: Findings from the memory and medical care study. *Aging Ment Health* 2012;16:372–377.
29. Sachs-Ericsson N, Blazer DG. Racial differences in cognitive decline in a sample of community-dwelling older adults: The mediating role of education and literacy. *Am J Geriatr Psychiatry* 2005;13:968–975.
30. Sawyer K, Sachs-Ericsson N, Preacher KJ et al. Racial differences in the influence of the APOE epsilon 4 allele on cognitive decline in a sample of community-dwelling older adults. *Gerontology* 2009;55:32–40.
31. Glymour MM, Weuve J, Berkman LF et al. When is baseline adjustment useful in analyses of change? An example with education and cognitive change. *Am J Epidemiol* 2005;162:267–278.
32. Ostchega Y, Harris TB, Hirsch R et al. The prevalence of functional limitations and disability in older persons in the U.S.: Data from the National Health and Nutrition Examination Survey III. *J Am Geriatr Soc* 2000;48:1132–1135.
33. Marden JR, Walter S, Tchetgen Tchetgen EJ et al. Validation of a polygenic risk score for dementia in black and white individuals. *Brain Behav* 2014;4:687–697.
34. Tan M-X, Stern Y, Marder K et al. The APOE-epsilon4 allele and the risk of Alzheimer disease among African Americans, whites, and Hispanics. *JAMA* 1998;279:751–755.