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Race Differences in Cognitive Functioning Among Older Adults

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Objectives. Explaining race differences in cognitive functioning in later life continues to challenge researchers. This study was an attempt to incorporate the clinical literature, emphasizing biological correlates of cognitive functioning, and the social research literature, emphasizing social inequalities and consequent health outcomes, in the examination of sources of race differences in cognitive functioning in older adults.

Methods. With data from Wave 1 of the Assets and Health Dynamics of the Oldest Old survey, the authors used structural equation models (LISREL 8.30) to estimate the direct effects of race on cognitive functioning and indirect effects through social and biological risk factors for the total sample ($N = 5{,}955$).

Results. Race had a direct association with cognitive functioning. Race also had indirect effects on cognitive functioning through social risk factors—education and health insurance. There did not appear to be indirect effects of race through biological risk factors.

Discussion. The direct and indirect effects of race through social risk factors attest to the importance of examining different ways through which race can influence cognitive functioning of older adults. This research also emphasizes the need for researchers to investigate more closely race differences in dimensions of cognitive functioning and cognitive functioning over time.

PREVIOUS research has indicated consistent race differences in cognitive functioning (Inouye, Albert, Mohs, Sun, & Berkman, 1993; Leveille et al., 1998). Specifically, older African Americans appear to be more cognitively impaired than their White counterparts (Fillenbaum, Heyman, Prosnitz, & Burchett, 1990; Herzog & Wallace, 1997; Inouye et al., 1993; Leveille et al., 1998). Biological factors—such as age, vascular disease, and genetic markers—and education—a social correlate—are most commonly proposed as factors underlying observed race differences in cognitive functioning. In this research, we incorporated the clinical research, emphasizing biological correlates of cognitive functioning, and the social science research, emphasizing social inequalities and health outcomes, to examine sources of African Americans' disadvantaged cognitive functioning. We focused on the cohorts of African Americans and Whites born primarily between 1892 and 1922, who moved into young adulthood prior to and during the Great Depression.

Stratification and Cognitive Functioning Among Older Adults

The cumulative advantage and disadvantage theory provided our conceptual framework (Crystal & Shea, 1990; O'Rand, 1996; O'Rand & Henretta, 1999). Life course cumulative advantage and disadvantage theorists claim that initial inequalities establish intracohort differences in social phenomena such as educational achievement, employment history, and income inequality in later life (Crystal & Shea, 1990; O'Rand, 1996; O'Rand & Henretta, 1999). Race is a primary stratification system that yields initial social disadvantage to African Americans. Stratification was especially clear and rigid at the turn of the century, decades away from the post-World War II economic expansion and the liberation movements of the 1960s. Over time, initial racial inequalities may become divergent, thus accentuating the initial racial inequalities as people age and producing wider intracohort racial differences.

Socioeconomic inequality and health status are intertwined throughout the life course, yet there is clear evidence that earlier socioeconomic inequality yields health differences among older adults (Smith & Kington, 1997). Because education is strongly implicated in variation in cognitive functioning, even in clinical studies (Inouye et al., 1993; Katzman, 1993), initial racial inequalities in education may directly widen racial differences in cognitive functioning in later life and indirectly widen them through divergent employment experiences, the size and composition of adult income streams, and lifestyles and health behaviors. We argue that, in addition to a direct effect of race on cognitive functioning, race operates indirectly through biological and social risk factors, thus compounding racial disadvantage across the life course.

Cognitive Functioning

Biological correlates.—Poverty and racism limit African Americans' access to high-quality medical care and economic security, likely producing chronic stress over economic security and allowing health to deteriorate (Clark,

Anderson, Clark, & Williams, 1999; Cooper, Steinhauer, Schatzkin, & Miller, 1981; Harrell, 2000; Kessler, Mickelson, & Williams, 1999). The stressful consequences of economic insecurity and racism, combined with risky health behaviors, may increase African Americans' prevalence of biological risk factors for poor cognitive functioning: vascular disease, clinical depression, and physical comorbidity (Cooper et al., 1981; Dressler, 1991). Although all of these factors are important, and we will briefly discuss them, we are specifically interested in vascular diseases.

The most obvious cause of impaired cognitive functioning is the genetic predisposition to Alzheimer's disease (Stephenson, 1997). Vascular dementias, however, are much more prevalent than Alzheimer's disease. Indeed, some researchers have contended that vascular dementia is the leading cause of mental impairment among older adults (Hachinski, 1992). For example, high systolic blood pressure in middle age, a condition more common among African Americans than Whites, has been identified as a significant risk factor for cognitive impairment in old age (Friedl, Schmidt, Stronegger, & Reinhart, 1997; Launer, Masaki, Petrovich, Foley, & Havlik, 1995). Adult-onset diabetes mellitus in middle age is another risk factor for cognitive impairment in old age (Croxson & Jagger, 1995), primarily because of the vascular impact of the disease. But medical experts have yet to agree on a common definition of vascular dementia, because of disagreements as to its causes and clinical characteristics (Hachinski, 1992; Larson, 1993). Despite the controversy, physicians continue to recommend lifestyle changes and pharmacotherapy to prevent and reduce the impact of vascular disease. Treatment of hypertension and diabetes in middle age, especially among African Americans, may significantly improve cognitive functioning and reduce the economic and social costs of compromised cognitive functioning.

Other biological correlates of cognitive functioning include age, depression, illness, and functional limitations (Lindenberger & Reischies, 1999; Ofstedal, Zimmer, & Lin, 1999). A common belief among clinicians and lay people alike is that poor cognitive functioning is an inevitable result of aging. Yet prevalence rates may level off at the oldest ages, suggesting that the absolute size of the cognitively impaired older population may be lower than estimated (Ritchie & Kildea, 1995). Consequently, any mortality crossover between Whites and African Americans in this cohort may ameliorate somewhat the negative effects of cumulative disadvantage.

Late-life depression appears to operate as both a predictor and an outcome of poor cognitive functioning (Buntix, Kester, Bergers, & Knottnerus, 1996; Helmchen & Linden, 1993; Oxman, 1994). African Americans may be at greater risk of depression because of the stress of inequality, yet the condition may go untreated because it is less likely to be diagnosed (Baker, 1987). When African Americans do gain access to the formal medical care system, they likely present medical care providers with more severe symptoms and functional limitations, more advanced stages of chronic and acute conditions, and higher levels of physical comorbidity (Blendon, Aiken, Freeman, & Corey, 1994; Jaynes & Williams, 1989).

Social correlates.—We contend that, for this cohort of older adults, initial inequalities translated into differential access to education, especially higher quality education, which is the primary social correlate of cognitive functioning. African Americans who attended school were more likely than Whites to discontinue education earlier (Mare, 1995). In general, "poorer" children worked to sustain their households, whereas "richer" children attended school or were taught privately. African Americans were considerably less likely to attend school because of the confluence of poverty and racism (Jaynes & Williams, 1989). The education system was segregated, yielding schools with very limited resources for African Americans. Because of the need to staff African American schools with teachers, African American women in this cohort tended to have more schooling than men, a gender difference in direct contrast with that among Whites.

The consequence of educational differentials is the development of divergent pathways through the extant opportunity structure. Better education begets higher social class; more prestigious, safer, and higher paying occupations; better access to health care resources; and, ultimately, more economic security (House, Kessler, & Herzog, 1990). Job duties in more prestigious occupations may offer more cognitive stimulation, thus retarding age-related declines (Jorm, 1998). Relative economic security throughout the life course reduces the negative health effects of chronic stress and increases a person's ability to purchase access to health care, especially for preventive care and at an earlier point in the natural history of disease. The substantial racial inequalities of the educational system were mirrored in the occupational structure (Mare, 1995; Sandefur & Tienda, 1988), underlying African Americans' poorer health and higher mortality than Whites (Preston & Taubman, 1994).

Expected results.—We hypothesized that social and biological correlates would account fully for race differences in cognitive functioning. Thus, we expected to find that lower levels of education (and presumed poorer educational quality, though we could not measure this) among older African Americans would account for race differences in cognitive functioning. We expected to find that race differences in the quantity of education would also indirectly affect cognitive functioning through their influence on biological risk factors. Finally, we expected to find that African Americans' cumulative disadvantage would reflect their greater prevalence of biological risk factors, thus suggesting that race operates on cognitive functioning indirectly through increased biological risk.

Methods

Data and Sample

We used data from the first wave of the Assets and Health Dynamics of the Oldest Old (AHEAD), fielded in 1993–94, to estimate the potential direct effect of race on cognitive functioning and its indirect effects through social and biological risk factors. The AHEAD is a longitudinal panel study designed to be a nationally representative sample of community-dwelling adults in the continental United

States aged 70 and older. The oversampling of African Americans permits racial group comparisons as well as differential analyses within each group. Telephone or face-to-face interviews were completed with 8,222 of the 10,297 persons eligible (response rate = 80%). Proxy respondents accounted for 10.4% of the completed interviews. The study was designed to collect information on physical health and cognitive functioning, economic resources, and family structures and transfers. The primary advantages of these data include the singularly rich and diverse set of measures of health to describe the disablement process, a sufficient sample size of older African Americans, and a probabilistic sample of the oldest old necessary to generalize to the target population.

The sample (N = 5,955) for this analysis included older persons who identified themselves as African American (n = 833) or non-Hispanic White (n = 5,122). Latinos were excluded because their numbers in the AHEAD were too small for subgroup analyses, and the primary focus in this research was on race differences in cognitive functioning. The sample was further restricted to persons aged 70 and older and self-reporting respondents. There was approximately 4.5% missing data on cognitive functioning variables. In side analyses (data not shown), respondents who had missing data on the cognitive variables were significantly older, had lower education and income, and had greater numbers of physical health problems than respondents who provided complete information. Because the percentage of respondents who had missing data was relatively small, we deleted these respondents from the analyses. Consequently, we may have underestimated the extent of poor cognitive functioning in this sample, making it more difficult to observe indirect race effects.

Measures

Cognitive functioning.—The fields of cognitive psychology and cognitive neurobiology are developing rapidly, prohibiting a clear consensus on how to organize the age-based changes in basic cognitive functions. Current interest centers on prefrontal cortex executive function theory and medial temporal lobe declarative memory theory (Woodruff-Pak & Papka, 1999). Executive function, attention, and working memory are located in the frontal lobes of the cerebral cortex, the region of the brain most impaired by normal aging processes (Woodruff-Pak, 1997). Declarative memory, often measured by tests of immediate and delayed recall, is the brain memory system most affected by normal aging (Squire, Knowlton, & Musen, 1993; Woodruff-Pak & Papka, 1999).

In the late 1980s and early 1990s, however, cognition was often organized into fluid intelligence (e.g., information processing), crystallized intelligence (e.g., knowledge base), and memory (Christensen et al., 1994; Nesselroade, Pedersen, McClearn, Plomin, & Bergeman, 1988; Perlmutter, 1988; Perlmutter & Nyquist, 1990). The principal investigators of the AHEAD considered these dimensions when constructing the instrument (Herzog & Wallace, 1997). The AHEAD investigators selected these cognitive functioning measures to provide information on a comprehensive range of cognitive functions, to encompass several difficulty levels from competent cognitive functioning to impaired functioning, to be sensitive to change over time, to be able to be administered in a survey environment, and to be valid and reliable indicators (for a detailed discussion on these measures, see Herzog & Wallace, 1997). Many of these items were modeled after the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) or adapted from the Telephone Interview for Cognitive Functioning (TICS; Brandt, Spencer, & Folstein, 1988). Table 1 shows the cognitive functioning measures available in the AHEAD.

These cognitive measures are useful for addressing cognitive functioning among older adults in a population-based survey. Our intent in this research was to address potential race differences in cognitive functioning among a nationally representative sample of older adults, not to focus on measurement issues with respect to these scales. Thus, in this article we provide basic psychometric information, consistent with other researchers who have used these data (Ofstedal et al., 1999). An evaluation of the psychometric structure of these measures yielded two indicators of general cognitive functioning: a mental status index (TICS series and the serial 7s, eigenvalue = 3.07; Herzog & Wallace, 1997) and a memory index (immediate and delayed word recalls, eigenvalue = 1.07; Herzog & Wallace, 1997). Herzog and Wallace contended that the finding of two related factors (factors are correlated at r = .40) suggests that a composite measure is possible, which they created (α = .65) and we used in these analyses. To deal with potential race differences in each type of functioning, we addressed the mental status index and the memory index as separate concepts in two analyses, which we discuss further in the Results section.

We contend that the executive function theory is reflected in the mental status index and that the declarative memory theory is reflected in the memory index. Thus, the cognitive functioning composite measure contains components of both executive function and declarative memory theories. We expected to see indirect effects of race through social and biological correlates in the composite cognitive functioning measure.

Table 1. Cognitive Functioning Measures in the AHEAD Data

Measure	Components
Telephone interview for cognitive status	Respondent counts backward from 20 for 10 continuous numbers; names the day of the week and date; names the president and vice president; names the objects that "people usually use to cut paper," and the "kind of prickly plant that grows in the desert."
Serial 7s	Respondent starts at 100 and subtracts by increments of 7 for five trials.
Immediate word recall	Interviewer reads 10 words; respondent recalls as many as possible.
Delayed word recall	Later in the interview, respondent recalls as many of the 10 words as possible.
Combined cognitive score	Combination of the above scales; range 1–35; M = 19.72, SD = 5.84.

Note: AHEAD = Assets and Health Dynamics of the Oldest Old.

Social correlates.—Education shapes lifestyles and occupational opportunities, serving as the main social correlate through which race influences cognitive functioning. Education was measured as a self-report of the number of years of schooling, a measure consistent with much of the research on cognitive functioning (Fahlander et al., 2000; Herzog & Wallace, 1997; Ofstedal et al., 1999). We grouped the work experience of respondents into regular work in professional occupations (coded as 1) compared to work in nonprofessional occupations or nonparticipation in the labor force (coded as 0). Professional occupations included professional and technical workers, managers, officials, and proprietors. We measured income as a dichotomous variable with 1 reflecting above the median income. We included current income as an additional measure of lifetime inequality, an outcome that reflects earnings and savings histories. Higher income also confers the ability to purchase additional health and medical care insurance, which in turn reduces disability (Landerman et al., 1998). Accordingly we incorporated a measure of whether an individual had purchased health insurance (coded as 1).

Health correlates.—Vascular conditions were the primary biological or health correlates on which the study focused. Different types of vascular conditions have potential effects on cognitive functioning: hypertension or high blood pressure, diabetes, any heart condition, and stroke (Fahlander et al., 2000; Ofstedal et al., 1999). Because having a stroke is a relatively strong predictor of cognitive functioning (Ofstedal et al., 1999), stroke was examined separately. Respondents were asked if a doctor had ever told them that they had had a stroke (coded as 1). The other vascular conditions were combined into a dichotomous variable with 1 indicating presence of hypertension or high blood pressure, diabetes, any heart condition, any experience of chest pains, or heart attack. Functional limitations assess a higher order of functioning than do activities of daily living and instrumental activities of daily living limitations. In our study number of functional limitations was a continuous measure that addressed the number of difficulties that the respondents had walking several blocks, climbing one flight of stairs without resting, pushing or pulling large objects (like a chair), lifting or carrying weights of more than 10 lb (like a heavy sack of groceries), and picking up a dime from a table. The range was from 1 to 4 ($M = 1.24$, $SD = 1.44$).

Finally, to address the potential connection between depressive symptoms and cognitive functioning, we used the 10-item Center for Epidemiologic Studies-Depression (CES-D) scale in the AHEAD to measure level of depressive symptomatology. The original CES-D scale contains 20 items designed to address depressive symptoms in the general population (Radloff, 1977), but recent research has indicated that shorter versions of the scale do not influence the structure of the original scale (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993). The 10 items address mood and psychosomatic dimensions in the past week. The respondents were instructed to answer yes or not to questions including "Much of the time in the past week, I felt depressed," "Much of the time in the last week, I felt that everything I did was an effort," and "Much of the time in the last week, I felt lonely." The range of the CES-D scale is 0–10, with higher scores reflecting more depressive symptoms ($M = 2.11$, $SD = 2.26$).

Demographic correlates.—We controlled for several demographic variables that are associated with cognitive status. Our main demographic variable of interest was race, comparing African Americans (1) and Whites (0). We controlled for the confounding effects of gender; women were coded as 1. Marital status was organized into persons who were currently married (1) and unmarried (0). Age was a continuous variable with a range of 70 to 103 ($M = 77.25$, $SD = 5.58$).

Procedures

We first provide descriptive statistics on the total sample and the race-stratified sample, including mean and chi-square differences in the relevant model variables. Next, we provide a correlation matrix, which was the basis for building the structural equation models.

Structural equation models (SEM) are sets of techniques that permit path analysis and the estimation of direct and indirect effects (Alwin, 1988; Bollen, 1989; Kline, 1998). The measurement model aspect of SEM can be very important in the examination of latent or unobserved concepts, as well as the construct validity of a measurement instrument (Hoyle, 1995; Kline, 1998). The structural model aspect of SEM, on the other hand, focuses on the hypothesized relationships between the exogenous (independent) and endogenous (dependent) variables. All of the exogenous and endogenous variables in these analyses had only one indicator; therefore, the focus in these analyses was on the structural model and the estimation of direct and indirect effects of race, and of race through social and biological risk factors, on cognitive functioning (Kline, 1998; Peek, 2000).

We employed SEM using LISREL 8.30 (Joreskog & Sorbom, 1993) to estimate the direct and indirect effects of race in the full sample as well as the direct and indirect effects of social and biological risk factors by race-stratified samples. We stratified the analysis by race to understand better the effects of specific risk factors on cognitive functioning. We followed the procedure for estimating two models for Whites and African Americans simultaneously, as explained by Hayduk (1987). We estimated these "stacked" models by allowing some of the effect coefficients to be equal between both groups and then allowing the same coefficients to be "free." We then compared the two models with a chi-square difference test.

Estimation.—The coefficients for the LISREL models were weighted least squares based on matrices provided by PRELIS. Weighted least squares were used instead of maximum likelihood or generalized least squares because of the presence of both discrete and continuous measures in the data (Joreskog & Sorbom, 1993). We used PRELIS to generate a matrix of polychoric correlations and an accompanying matrix of asymptotic variances and covariances. With these matrices as input, we estimated the structural equation models using the weighted least squares fitting function in

LISREL 8.30, which is asymptotically distribution free (Joreskog & Sorbom, 1993).

Results

Table 2 indicates descriptive statistics for the total and race-stratified samples for the model variables. African Americans reported a lower cognitive functioning score (by about 6 points) than older Whites. As hypothesized, this cohort of older African Americans had greater social and biological risks than Whites for decreased cognitive functioning. A higher percentage of African Americans than Whites had a vascular condition other than a stroke. Also, African Americans reported slightly more functional limitations and depressive symptoms. A higher proportion of African Americans reported less than a high school education and less than the median income than older Whites. Finally, older African Americans were less likely than Whites to have additional insurance coverage.

Table 3 shows the correlation matrix for the model variables for the total sample only. All model variables except for gender were significantly associated with cognitive functioning. Two of the variables, having a professional occupational background and other vascular conditions, had relatively low correlations. Because there were other measures in the model that addressed similar health or biological and social correlates, these two variables were not used in the structural equation models.

The remainder of this section describes the results of the structural equation models estimating race differences in cognitive functioning. Table 4 presents the direct effects of race, social, and biological risk factors on cognitive functioning among the total sample. African Americans had significantly lower scores on the cognitive functioning index ($\beta = -.28$, $p < .001$), but race also had indirect effects on cognitive functioning through its effects on social risk factors. These indirect effects are not depicted in the table, so more detail is provided here. Being African American was associated with having less than a high school degree, which was, in turn, significantly associated with lower scores on cognitive functioning. In a similar fashion, being African American was associated with not having other insurance, which was significantly related to lower cognitive functioning scores. In contrast, race did not have significant indirect effects on cognitive functioning through biological risk factors. Also, biological risk factors did not have significant direct effects on cognitive functioning, with the exception of having had a stroke, and race was not associated with having had a stroke.

Table 4 also shows several indicators of model fit chosen from among the many indices that assess model fit (Chou & Bentler, 1995; Hu & Bentler, 1995; Kline, 1998; Mulligan, 1998). We report the chi-square goodness of fit, the adjusted goodness of fit index (AGFI), comparative fit index (CFI), nonnormed fit index (NNFI), and the standardized root mean squared residual (SRMR). The AGFI and CFI indices assess the relative amount of the variances and covariances that are predicted by the model. The CFI also adjusts for bias due to asymptotic sample size. The NNFI indicates the proportion of the improvement of the estimated model relative to the null model, with an adjustment for model complexity (Kline, 1998). All of these indices (except the SRMR) usually have values between 0 and 1. A value greater than .90 indicates the model fits the data reasonably well. Finally, the SRMR is a summary of covariance residuals. As the difference between observed and predicted covariances increases, the SRMR increases, so the fit of the model is improved as SRMR approaches 0; SRMR $< .10$ is deemed desirable (Kline, 1998). Table 4 shows that while the chi-square remained significant, the values of the other goodness-of-fit indices implied a relatively well-fitting model.

In summary, we hypothesized that race would have only indirect effects on cognitive functioning through social and biological risk factors and that once these indirect effects

Table 2. Characteristics of the AHEAD Total Sample and by Race

Model Variables	Total (N = 5,955)	African American (n = 833)	Non-Hispanic White (n = 5,122)
Demographic			
% African American	14.0	—	—
% Female	62.4	67.4	61.5
Age, *M* (*SD*)	77.1 (5.57)	77.4 (5.77)	77.2 (5.55)
% Married	49.1	30.4	52.1
Social			
% Low education	40.1	71.1	35.0
% Low income	50.6	76.5	46.4
% Professional background	20.8	8.5	23.4
% Having another source of health insurance	77.8	42.4	83.6
Biological			
% Stroke	7.5	8.6	7.3
% Other vascular conditions	66.3	74.7	64.9
Functional limitations, *M* (*SD*)	1.2 (1.44)	1.5 (1.51)	1.2 (1.43)
Depressive symptoms, *M* (*SD*)	2.1 (2.26)	2.5 (2.30)	2.1 (2.25)
Cognitive Functioning			
Total cognitive functioning, *M* (*SD*)	19.7 (5.84)	14.7 (6.07)	20.6 (5.38)

Notes: All model variables vary significantly ($p < .001$) between African Americans and Whites, except age and percentage of stroke. AHEAD = Assets and Health Dynamics of the Oldest Old.

Table 3. Correlation Matrix for Model Variables, Total Sample (N = 5,955)

Model Variables	1	2	3	4	5	6	7	8	9	10	11	12
1. African American												
2. Female	.04*											
3. Age	.01	.06*										
4. Married	−.16*	−.36*	−.23*									
5. Low education	.26*	.01	.15*	−.13*								
6. Low income	.21*	.21*	.19*	−.41*	.32*							
7. Professional background	−.13*	−.21*	−.03	.13*	−.30*	−.26*						
8. Insurance	−.34*	−.06*	−.08*	.19*	−.26*	−.26	.16*					
9. Stroke	.02	−.02	.06*	−.03	.06*	.05*	−.01	−.04				
10. Other vascular	.07*	.00	.02	−.04	.09*	.07*	−.04	−.02	.11*			
11. Functional limitations	.07*	.21*	.21*	−.17*	.16*	.20*	−.11*	−.10*	.17*	.18*		
12. Depressive symptoms	.06*	.10*	.12*	−.18*	.17*	.17*	−.12*	−.13*	.11*	.13*	.44*	
13. Cognitive functioning	−.34*	−.01	−.35*	.18*	−.43*	−.28*	.20*	.32*	−.11*	−.06*	−.22*	−.22*

*$p < .001$.

were accounted for, the direct effect of race would not be significant. However, this was not consistent with the results from these analyses. Furthermore, we conducted separate analyses in ordinary least squares regression (data not shown), and the models indicated that race continued to be a significant predictor of cognitive functioning when both social and biological factors were included. Race was a strong predictor for cognitive functioning in the bivariate model ($\beta = -.34$, t value = −28.21). When social factors were included, the effect of race still remained significant; however, it did decrease by approximately 38% ($\beta = -.21$, t value = −18.36). When biological factors were included in the model, the effect of race did not diminish at all ($\beta = -.21$, t value = −18.71). Thus, there are effects of race on cognitive functioning that go beyond the social and biological risk factors that we have addressed in this study.

Another possibility is that the influence of race differs across the components of the cognitive functioning variable.

Table 4. Structural Parameter Estimates[a] (Direct Effects) for Predicting the Influence of Race on Cognitive Functioning

Model Variable	Direct Effect
Demographic	
African American	−.28 (.05)/−5.83
Female	.00 (.02)/−0.16
Age	−.15 (.19)/−0.78
Married	−.03 (.06)/−0.55
Social	
Low Education	−.16 (.03)/−4.65
Low Income	−.03 (.03)/−1.11
Having another source of health insurance	.11 (.02)/4.65
Health	
Stroke	−.09 (.02)/−3.84
Functional Limitations	−.04 (.05)/−0.74
Depressive Symptoms	−.01 (.10)/−0.09
Model Fit	
Chi square/df	48.25/9
Adjusted goodness-of-fit index	1.00
Comparative fit index	1.00
Nonnormed fit index	.97
Standardized root-mean-squared residual	.04

[a]Unstandardized estimate (standard error)/t − value presented.

To address this, we analyzed two additional structural equation models (analysis not shown). First, we examined a model with race and the other model variables predicting two cognitive components (status = TICS series + serial 7s; memory = immediate word recall + delayed word recall). This model indicated that being African American had a significant direct effect on status ($\beta = -.58$, t value = −12.57), but race had no significant direct or indirect effect on memory, $\chi^2(29, N = 5{,}955) = 541.71$; AGFI = .98; CFI = .96; NNFI = .86; SRMR = .11. In the second model, we estimated a model with race and the model variables predicting four components of cognitive functioning (status1 = TICS series; status2 = serial 7s; memory1 = immediate word recall; memory2 = delayed word recall). Results from this model indicated significant direct effects of being African American on status1 ($\beta = -.39$, t value = −2.99), status2 ($\beta = -.55$, t value = −9.38), and memory1 ($\beta = -.28$, t value = −4.84), but not on delayed word recall, $\chi^2(16, N = 5{,}955) = 113.74$; AGFI = .99; CFI = .99; NNFI = .95; SRMR = .08. The presence of race differences appeared to vary depending on whether or not delayed word recall was included in the memory index construct.

To examine more closely the differential effects of social and biological risk factors across older African Americans and Whites, we conducted a race-stratified analysis in SEM. To conduct the stratified analyses, we first constrained the effects of social and biological risk factors to be equal across the two groups, and then we compared that model with one that allowed the effects of social and biological risk factors to vary between the two groups. Table 5 shows the direct effects of social and biological risk factors on cognitive functioning for the race-stratified sample from the model that allowed the effects to vary. When conducting a chi-square difference test, we found that the model that allowed the effects to vary fit significantly better than the model where the effects were constrained to be the same between the two groups; in the constrained model, $\chi^2(13, N = 5{,}955) = 82.52$; in the nonconstrained model; $\chi^2(2, N = 5{,}955) = 55.59$; difference $\chi^2(11, N = 5{,}955) = 26.93$, $p < .01$. In addition, we examined each of the social and biological risk factors paths in separate models. When the effects of having another source of insurance and of having low ed-

Table 5. Structural Parameter Estimates[a] (Direct Effects) by Race for Predicting the Influence of Social and Biological Risk Factors on Cognitive Functioning

	Direct Effect	
Model Variable	African American (n = 833)	White (n = 5,122)
Demographic		
Female	−.09 (.16)/−0.59	.04 (.08)/0.53
Age	−.50 (1.29)/−0.39	−.23 (.56)/−0.41
Married	−.08 (.17)/−0.47	.03 (.19)/0.15
Social		
Low education	−.52 (.17)/−3.17	−.35 (.07)/−4.91
Low income	.24 (.26)/0.94	.02 (.02)/1.00
Having another source of health insurance	.38 (.14)/2.67	.17 (.02)/8.96
Health		
Stroke	−.18 (.17)/−1.03	−.10 (.05)/−1.93
Functional limitations	.10 (.38)/0.26	−.06 (.15)/−0.40
Depressive symptoms	0.4 (.49)/0.08	.01 (.20)/0.04
Model Fit		
Chi square/df	55.59/2	
Goodness of fit index	1.00	
Comparative fit index	.99	
Nonnormed fit index	.70	
Standardized root mean squared residual	.05	

[a]Unstandardized estimate (standard error)/t − value presented.

ucation on cognitive functioning were not constrained to be equal between the two groups, the model fit improved considerably, $\chi^2(2, N = 5{,}955) = 6.67$, $p < .01$, and $\chi^2(1, N = 5{,}955) = 9.01$, $p < .001$, respectively.

However, Table 5 indicates that the effects of social and biological risk factors appeared to be similar across the two groups. A lower level of education was associated with lower cognitive functioning scores, and having another source of insurance was associated with higher cognitive functioning scores across both groups. Also, the indirect effects were similar across the two groups (coefficients not shown). For example, in both groups, being married was negatively associated with having less education, which was, in turn, associated with lower cognitive functioning scores. Age was associated with having a lower level of education, which was associated with poorer cognitive functioning. The stratified model did not fit as well as the model focusing on the total sample, however. Though the AGFI and other fit indices approached 1, the NNFI, which indicates the improvement of the estimated model relative to the null model, was only .70.

Discussion

In summary, race has a direct effect on late-life cognitive functioning and indirect effects through social correlates, primarily education and having another source of insurance. We expected there would not be direct effects of race, only indirect effects through social and biological correlates. These observed race differences raise issues about the measurement and conceptualization of cognitive functioning and the health impact of lifetime inequalities.

Several explanations of a persistent direct effect of race are possible. First, there is controversy about observed race differences in cognitive functioning. Are there unmeasured ethnic or cultural factors that influence cognitive functioning scores, or the measures that researchers use to assess cognitive functioning? Leveille and colleagues (1998) contended that the developers of the MMSE were quite aware that it performed differently for African Americans and Whites; however, after accounting for age and education, it was found to be an effective screening tool in both races. Furthermore, it has been used for clinical research purposes among various racial, ethnic, and cultural groups around the world. On the other hand, the extension of the use of the MMSE for nonclinical purposes, that is, measuring cognitive functioning, especially in nondemented persons, requires much more extensive and systematic research. It is therefore critical for future research to investigate both the cultural factors that may be influencing the lower scores on the cognitive measures for older African Americans as well as the specific ways in which education (and other social correlates) has its effects on cognitive functioning. Perhaps social theories such as cumulative advantage and disadvantage theory might prove to be useful organizing frameworks.

Second, a direct effect of race that remains after accounting for differences in social and biological risk factors may be attributed to unspecified race effects such as biological influences (e.g., genetics), racism, or unobserved heterogeneity. For example, perceived racism is associated with poor health outcomes, such as high blood pressure, poor well-being, and chronic conditions (Clark et al., 1999; Jackson et al., 1996), which may have effects on cognitive functioning over time. A third possibility for the remaining direct effects is misspecification of the model. We found potentially varying effects by race depending on whether or not delayed word recall was included in cognitive functioning. Essentially, persistent race effects are, at this point, uninterpretable and need further examination.

Cognitive functioning.—More immediately relevant are the measurement challenges posed by the various constructs and by the more global measures of cognitive performance used by clinicians. Fluid intelligence and aspects of executive function theory are difficult to measure in large population surveys, especially those using telephone interviews. Crystallized intelligence and memory are more flexible and easily incorporated into surveys. Yet there is no appreciable consensus on best measures, a consensus more observable in measurement of disability and functional health (i.e., activities of daily living). Finally, a distinction between laboratory and naturalistic tasks is warranted, providing clinicians with astute but parsimonious diagnostic tools for therapeutic regimens and assessments about an older adult's successful negotiation of his or her environment. The MMSE continues to serve the needs of both clinicians and survey researchers.

Race and life course inequalities.—It is difficult, of course, to fully evaluate life course theories of inequality without the most detailed longitudinal data. A full complement of

lifetime and temporally located biomarkers and socioeconomic transitions is ideal but virtually nonexistent in population surveys. Sample selectivity poses an additional challenge because persons in the lowest segments of socioeconomic status and health are least likely to survive to ages typically included in surveys of older adults. African Americans are disproportionately represented in these segments, making it difficult to observe any but the largest racial differences. Such a conservative bias deters full understanding of race differences in cognitive functioning, yet it suggests that the differences we observe in the AHEAD likely underestimate true race differences. We conclude that the cumulative advantage and disadvantage theory goes some distance, even with constrained data quality, in documenting that racial inequalities in education early in the life course yield African Americans' cognitive health disadvantage later in the life course.

More systematic research needs to address ethnoracial group and potential cultural differences in cognitive functioning, because some researchers have argued that, as far as culture is concerned, these effects on cognition may not necessarily magnify with age (Park, Nisbett, & Hedden, 1999). Because education is strongly implicated in variation in cognitive functioning, the initial inequalities of race and gender may be influenced by a cohort's educational experiences. To a certain extent, the results for the African American subsample in the AHEAD data with respect to education differences in cognitive functioning conform more to this hypothesis than for the Whites. In other words, the indirect effects of race on cognitive functioning through education suggest that, for this cohort of African Americans, having lower educational attainment can very much accumulate into a cognitive disadvantage over the life course.

The data reveal evidence of the cumulative disadvantage of early life inequalities on cognitive functioning, despite data and measurement limitations that make it difficult to do so. There are research tasks that may be accomplished with the same data. First, we focused only on Wave 1 of the AHEAD. Examining race differences in cognitive functioning using cross-sectional data may serve as a starting point to understanding potential differences, but to truly estimate race differences in cognitive levels and changes, researchers need longitudinal data. An important next step in this area of research is for investigators to estimate differences over time with all waves of the AHEAD and HRS between older African Americans and Whites in the changes in cognitive functioning and the correlates and consequences of these changes.

The AHEAD data do permit approximations of critical socioeconomic transitions across the life course, transitions that are relatively fixed characteristics among older adults. Education precedes lifetime occupational activity, both of which are usually completed by age 70. Locating in specific historical circumstances the average levels of economic resources (i.e., education, income, prestige) and the average age at transitions of social groups, say racial groups, allows insight into racial differences in outcomes observed in later life. Our results suggest the utility of considering individual biography and social structure, a life course theoretical framework, in understanding the profound racial differences in cognitive functioning, especially in efforts to synthesize clinical and social science research.

Acknowledgments

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