

Disparities in Cognitive Functioning by Race/Ethnicity in the Baltimore Memory Study

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The Baltimore Memory Study is a cohort study of the multilevel determinants of cognitive decline in 50–70-year-old randomly selected residents of specific city neighborhoods. Prior studies have demonstrated that cognitive function differs by race/ethnicity, with lower scores in minorities than in whites, but the underlying basis for these differences is not understood. Studies have differed in the rigor with which they evaluated and controlled for such important confounding variables as socioeconomic status (SES), health-related behaviors, comorbid illnesses, and factors in the physical environment. The goal of this study was to describe differences in neurobehavioral test scores by race/ethnicity, before and after control for a four-dimensional measure of SES and health-related behaviors and health conditions, in a cross-sectional analysis of first visit data. Random samples of households in the study area were selected until enrollment goals were reached. Among the 2,351 persons on whom eligibility was determined, 60.8% were scheduled for an enrollment visit; of these, 1,140 (81.3%) were enrolled and tested. These study participants were 34.3% male and 65.7% female and were from 65 Baltimore, Maryland, neighborhoods. After adjustment for age, sex, and testing technician, there were large and statistically significant differences in neurobehavioral test scores by race/ethnicity, with African-American scores lower than those for whites, for both men and women. After adjustment for individual SES (educational status, household income, household assets, and occupational status), the average difference declined by 25.8%. After additional adjustment for SES, health-related behaviors and health conditions, and blood lead, the average difference declined another 10%, but large differences persisted; African Americans had test scores that averaged 0.43 standard deviation lower than those for whites across all neurobehavioral tests. These differences were present in all cognitive domains, including tests that would not be characterized as susceptible to differential item functioning by race/ethnicity, suggesting that the results are not due to race/ethnicity-associated measurement error. *Key words:* cognitive function, health disparities, race/ethnicity, socioeconomic status. *Environ Health Perspect* 112:314–320 (2004). doi:10.1289/ehp.6727 available via <http://dx.doi.org/> [Online 24 November 2003]

The impending retirement of the baby-boom cohort, along with geometric growth in the relative size of the older population, will dramatically alter the public health challenges of the 21st century. Demographics ensure that the numbers of persons with dementia and cognitive decline will increase in the coming decades (Brookmeyer et al. 1998). The determinants of cognitive dysfunction with increasing age are complex, multifactorial, and synergistic, involving features of the physical and social environments, as well as endogenous biologic (e.g., genetic) and behavioral factors. Although results are not entirely consistent (e.g., Munoz et al. 2000), there is substantial evidence that neurobehavioral test scores, cognitive decline over time, and dementia risk vary substantially by race/ethnicity (Fillenbaum et al. 1998; Graham et al. 1998; Gurland and Katz 1997; Gurland et al. 1999; Hall et al. 2000; Launer et al. 1999; Perkins et al. 1997; Shadlen et al. 1999; Stern et al. 1994; Wiederholt et al. 1993). The underlying basis for these differences

has not been clearly delineated. Potential explanations include uncontrolled confounding by socioeconomic status (SES), comorbid illnesses that could influence cognitive function (e.g., cardiovascular disease), and chronic stress associated with race/ethnicity that is not fully captured by traditional measures of race/ethnicity, SES, or other indicators of the social environment. Previous studies have a number of limitations, including populations that are too old, samples that are not representative of underlying target populations, and incomplete control for important confounding variables, especially SES.

In considering the role of the social environment, neighborhood-level (or contextual) factors must be distinguished from individual-level (or compositional) factors, and these have in fact been separate foci of interest in earlier studies (Diez Roux 2001; Glass and Balfour 2003; Macintyre et al. 2002). Individual-level social variables that have been considered generally include those subsumed under the category of SES, which consists of such

attributes as education, occupation, income, and wealth, but no prior studies have rigorously controlled for this set of measures. Although the evidence is compelling that individual SES is associated with cognitive function in late life, the pathways through which this association operates have yet to be elucidated.

Population-specific differences in the presence of disease, health outcomes, or access to health care have been termed health disparities, and understanding the causes of these disparities and eliminating them is a primary goal of the Health Resources and Services Administration, the National Institutes of Health, and other American public health and research agencies (e.g., U.S. Department of Health and Human Services 2000). The National Institute of Environmental Health Sciences [National Institutes of Health (NIH), Department of Health and Human Services] has an active research program designed to disentangle the roles that the natural, built, and social environments play in disease causation. Here we report on the Baltimore Memory Study, which is funded under the trans-NIH research program. We present the detailed methods of the study, describe the disparities in neurobehavioral test scores in a large community sample of 50–70-year-old individuals from selected neighborhoods in Baltimore, Maryland, and evaluate selected individual-level social, physical environmental, and behavioral factors that account partially for these racial/ethnic differences in test scores. This work represents a case study in multilevel, multidisciplinary research, aimed at integrating knowledge within and across biologic, environmental, social, behavioral, and mathematical sciences.

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Materials and Methods

Study design. The Baltimore Memory Study, one of the studies funded by the NIH's Initiative to Eliminate Racial and Ethnic Disparities in Health is a cohort study of the multilevel determinants of cognitive decline in Baltimore city residents. The first of three study visits was completed between 30 May 2001 and 20 September 2002. We present analyses of cross-sectional data from the first study visit to describe the extent and magnitude of the disparities across race/ethnic groups on a battery of neurobehavioral tests designed to assess a full range of cognitive abilities in adults.

Study population. The target population consisted of 50–70-year-old residents of 65 contiguous neighborhoods in central and north Baltimore city who had lived within the greater Baltimore area for at least the previous 5 years. Neighborhoods were selected to ensure wide variability on characteristics of interest, including availability of services, socioeconomic deprivation, and racial composition, within and across race/ethnic groups.

Baltimore city consists of 200 census tracts and 264 neighborhoods named by the city (termed "neighborhood statistical areas"). Baltimore city's named neighborhood boundaries were defined by the Baltimore City Department of Planning in collaboration with the Johns Hopkins Center for Metropolitan Planning and Research in the late 1970s (Taylor 1979) and revised after the 1990 and 2000 censuses. In this study, we use the named neighborhoods, not census tracts, to define the geography of neighborhoods. The study area consisted of 81 contiguous neighborhood statistical areas using the 2002 definitions (these neighborhoods overlay approximately 60 census tracts; Baltimore City Department of Planning, Division of Urban Design, Baltimore, MD). A total of 1,140 study participants from 65 neighborhoods were enrolled in the study; most of the neighborhoods that did not provide study subjects had no residential population (e.g., Johns Hopkins University, areas of downtown). A total of 37 neighborhoods provided 10 or more study subjects, with a mean \pm SD of 27 ± 19 study subjects per neighborhood (ranging from 10 to 86). The study was approved by the Committee for Human Research of the Johns Hopkins Bloomberg School of Public Health; all participants provided written, informed consent before testing, and all study participants were paid \$50 for participating.

Sampling households and neighborhoods. The Maryland Department of Planning MdProperty View 2000 (MPV) database (Maryland Office of Planning, Baltimore, MD) was used to randomly select households for recruitment. Making use of Department

of Assessments and Taxation data, the MPV database contains all property addresses within Baltimore city in a geocoded format (i.e., all properties occupy an x,y-coordinate position on a map). Each record represents a household and includes fields for parcel identification number, owner's name, address, and ZIP code. The Stewart Directory (Stewart Directories, Inc., Cockeysville, MD) was used to supplement the MPV database with address-linked telephone numbers. Systematic differences in the recording of addresses were corrected before linking on the address field. The MPV database consisted of 233,267 properties, of which 207,309 records were residential addresses and 54,290 were in our study area. After accounting for multiunit properties ($n = 599$), the total number of residences in the study area was 64,037. Of these, there were 24,511 records with telephone numbers.

Subject selection and recruitment. A series of six random samples of households with telephone numbers (because it was not financially feasible to do home visits) was selected for recruitment until enrollment goals were reached. A total of 18,826 households with unique telephone numbers were selected and contacted to determine whether an eligible person resided there. Each household was called until a disposition could be established (Table 1) or until the household had been telephoned 10 times. Among the 2,351 subjects on whom eligibility was determined, 60.8% were scheduled for an enrollment visit. Of the 1,403 subjects scheduled for a clinic appointment, 1,140 (48.5% of subjects on whom eligibility was determined) were enrolled and tested. Overall, approximately 10% of selected residential units and 3% of apartments provided a scheduled subject.

Data collection. All data collection was performed by trained research assistants at the Baltimore Memory Study Clinic, located in north-central Baltimore city. Study participants first provided written, informed consent. Data were then collected in the following order: neurobehavioral testing, blood pressure, height, weight, spot urine collection, structured interview, and venipuncture; before departing, participants completed a satisfaction survey about the visit.

Neurobehavioral battery. Specific neurobehavioral tests were selected with four considerations in mind: *a*) variation by age, *b*) variation by race/ethnicity and SES, *c*) validity and reliability across the SES spectrum and in different race/ethnicity groups, and *d*) documented association of scores with at least one of our primary physical environmental exposures of interest (e.g., lead, mercury). We tried to minimize measurement error by race/ethnicity in the testing in three ways. First, technicians read each participant a standard statement that was designed to

orient all participants to the purpose and format of the questions in a way that maximized comfort level and avoided any reference to testing as a means to evaluate individual ability. Second, testing was performed by both female and male technicians of white and African-American race/ethnicity with random assignment. Third, to avoid differential information bias by SES or race/ethnicity groups, we specifically selected tests that do not require complex verbal responses (e.g., Welsh et al. 1995).

The 90-min test battery consisted of the following tests in this order (for details, see Table 2): Boston Naming Test (Kaplan et al. 2001; every second item of the 60 items was administered, to shorten the test), Raven's Coloured Progressive Matrices (Psychological Corporation, San Antonio, TX; Raven 1965; Raven et al. 1995), Rey Complex Figure copy, Rey Auditory Verbal Learning Test (RAVLT) immediate recall (Schmidt 1996), Purdue Pegboard (dominant hand, nondominant hand, both hands, and assembly) (model 32020; Lafayette Instrument Corporation, Lafayette, IN; LIC 1999), Stroop Test (A, B, and C forms), Trail-Making Tests A and B, Symbol Digit Paired Associate Learning, Rey Complex Figure delayed recall, finger tapping (dominant, nondominant), RAVLT delayed recall, RAVLT recognition, Simple Reaction Time (with the Standard Reaction Time Tester; Software Science, Cincinnati, OH; Wilkinson and Houghton 1982), Letter Fluency, and Category Fluency (Table 2). The complete test session was recorded on audiotape, and a random sample was regularly reviewed to evaluate quality. Neurobehavioral tests were scored by two technicians, and discrepancies were corrected by review and agreement.

Structured interview. The structured interview obtained information on self-report of

Table 1. Final dispositions of households with telephone numbers selected for recruitment, Baltimore Memory Study, 2001–2002.

Disposition	No. (%) ^a
Eligible	2,351 (12.5)
Eligible and scheduled	1,430 (7.6)
Eligible but not interested	921 (4.9)
Not eligible	7,526 (40.0)
Incorrect address	1,892 (10.0)
Age ineligible	5,220 (27.7)
Residency duration ineligible	27 (0.001)
Vacant or business	387 (2.1)
Unable to determine eligibility	7,702 (40.9)
Hangup after reached person	3,133 (16.6)
Disconnected, not in service	2,879 (15.3)
Repeated no contact	1,690 (9.0)
No final disposition	1,247 (6.6)
Number retired after enrollment goal reached	1,247 (6.6)
Total	18,826 (100.0)

^aPercent of total of 18,826 households with telephones selected from target neighborhoods in six random samples.

race/ethnicity [using the Census 2000 method distribution summarized in Table 3 (Grieco and Cassidy 2001)], housing and residential history, instrumental activities of daily living, medications (including detailed questions about current and historical use of nonsteroidal anti-inflammatory medications, estrogens, and oral birth control), childhood lead poisoning history, medical history, vascular risk factors (the Rose Questionnaire; Rose 1962), and chronic conditions. We also gathered data on a range of psychosocial and behavioral factors, including depressive symptoms [using the Center for Epidemiologic Studies depression scale (Ratloff 1977)], self-efficacy (adapted from Pearlin and Schooler 1978), history of alcohol and tobacco consumption, social networks (Glass et al. 1997), anxiety symptoms (Symptom Checklist-90 revised, anxiety scale only; Derogatis et al. 1973, 1976), social and

productive activities from the Enacted Function Profile (Glass 1998), received social support (Barrera 1980), and self-rated health and quality of life (both measured as single-item global measures).

Individual-level SES. Recognizing the weaknesses in existing approaches to individual SES, we developed and tested a new instrument that assesses individual and household SES along three dimensions: educational status, occupational status, and household wealth (Figure 1). The entire SES assessment tool consists of 110 questions and takes approximately 17 min to administer. Educational status includes measures of self-reported years of education completed (attainment) as well as credentials acquired (e.g., degrees, certificates, trade school). We used information from both attainment and credentialing to create a nine-level ordinal index of educational status.

Occupational status was based on a self-report of the degree of supervision and decision latitude of job duties (i.e., who had control in deciding what work was done and how it was completed) in four categories for the longest held job.

It is especially important when assessing individual-level SES in older adults to include measures of assets (Liberatos et al. 1988). Failure to measure assets can lead to drastic misclassification in the socioeconomic position of African Americans because race/ethnic disparities in assets are substantially greater than for income [based on data from the Panel Study of Income Dynamics as reported by Juster et al. (1999)]. Household wealth was assessed along two subdimensions: income and transfers, and assets. Our instrument asks about several sources of income (e.g., salaries, bonuses, extra income) and transfers (e.g., social security, welfare, Supplemental Security Income), with the respondent and the spouse/partner each asked separately. The instrument is equipped with bracketed value ranges to reduce missing data on study participants who do not report exact dollar amounts for the various items. Information from these items was combined to form total measures of household income and assets, which together are a measure of wealth. Household wealth was used as the main measure in the analyses, with separate terms for household income and household assets.

Laboratory methods. A 10-mL blood specimen was obtained by venipuncture by a trained phlebotomist and initially stored at -20°C as whole blood, buffy coat, and plasma. A spot urine specimen was obtained and frozen. Samples were transferred to Johns Hopkins Bloomberg School of Public Health and stored at -70°C. Blood lead was measured from whole blood by anodic stripping voltammetry in the laboratories of the Kennedy Krieger Institute in Baltimore (Bannon and Chisolm 2001). The limit of detection was 1 µg/dL, and the intra- and interday coefficients of variation (for 5.9 µg/dL) were 11 and 7%, respectively.

Statistical analysis. The main objectives of the analysis were to a) describe the associations of neurobehavioral test scores and blood pressure with race/ethnicity, after adjustment for age and sex; and b) describe the associations after further adjustment for physical environmental variables (blood lead levels), socioeconomic variables (household income, household assets, educational attainment, occupational status), and specific medical conditions (blood pressure, diabetes, vascular disease).

Trail-Making Tests A and B and Simple Reaction Time were natural-log(ln)-transformed, because of departures from normality, and negated to standardize the signs of the β coefficients. For all neurobehavioral

Table 2. Description of neurobehavioral testing battery, Baltimore Memory Study, 2001–2002.

Test name	Units of outcome	Range ^a	Cognitive domain
Boston Naming	Number correct	7–30	Language
Category Fluency	Number correct	17–106	Language
Coloured Progressive Matrices	Number correct	3–36	Nonverbal reasoning/ general intelligence
Finger tapping (mean of three trials)			
Dominant hand	Number of taps	7.8–67.4	Simple motor speed
Nondominant hand	Number of taps	6.8–61.8	Simple motor speed
Letter Fluency	Number correct	7–92	Language
Purdue Pegboard (mean of three trials)			
Dominant hand	Number of pegs	2.7–18.3	Eye–hand coordination/ manual dexterity
Nondominant hand	Number of pegs	1–18	Eye–hand coordination/ manual dexterity
Both hands	Number of pegs	1–19.3	Eye–hand coordination/ manual dexterity
Assembly	Number of pieces	2.7–47.3	Executive abilities
RAVLT			
Trials 1–5	Number correct	15–73	Verbal learning
Recognition ^b	Number	–16–15	Verbal memory
Delayed recall	Number correct	0–15	Verbal memory
Rey Complex Figure			
Copy	Score	1–36	Visuo-construction/ visuoperception
Delayed recall	Score	0–27	Visual memory
Simple Reaction Time (mean of 64 trials)	Seconds	0.19–0.95	Psychomotor speed
Stroop Test			
C form minus A form	Seconds	–31–276	Executive abilities
Symbol Digit Paired Associate Learning	Number correct	1–21	Visual memory
Trail-Making Tests			
Part A	Seconds	12–256	Executive abilities
Part B	Seconds	17–408	Executive abilities

^aIn study subjects. ^bNumber correct minus number incorrect.

Table 3. Distribution of study subjects by race/ethnicity, ^a Baltimore Memory Study, 2001–2002.

Race/ethnicity ^b	No. (%)	For analysis
White	598 (52.5)	Reference group
White/Native American	14 (1.2)	Reference group
Black/African American	474 (41.7)	Black
African American/mixed	30 (2.6)	Black–mixed race/ethnicity
Asian or Hawaiian	9 (0.8)	Other
Native American	11 (1.0)	Other
Missing or refused	4 (0.4)	Other
Total	1,140 (100.0)	

^aStudy participants could self-report as many of these race/ethnicity categories as they desired. ^bA total of 11 study participants reported they were Hispanic or Latino; of these, seven reported white race/ethnicity and four reported black or African-American race/ethnicity.

tests, a negative coefficient indicates that test performance declines with increasing values of the predictor variable. Educational attainment was modeled as a categorical variable in nine categories, using high school graduate with trade school or other credential as the reference group (because this was a large category in the middle of the range; Table 4).

Linear regression was used to evaluate differences in neurobehavioral test scores in three race/ethnicity groups, with whites as the reference group: Subject reported black only, black and another race/ethnicity, or other race/ethnicity. In model 1, the base model, differences by race/ethnicity were adjusted for age, sex, and testing technician. Next, in model 2, these differences were additionally adjusted for educational attainment (nine categories), occupational status (four categories), household income (ln-transformed), and household assets (ln-transformed). Finally, in model 3, these differences were adjusted for the covariates in model 2 as well as blood lead, time of day of testing (afternoon or evening vs. morning), taking medications for anxiety (yes vs. no), history of diabetes (yes vs. no), taking medications for hypertension (yes vs. no), tobacco use (current vs. never and previous vs. never), history of stroke (yes vs. no), alcohol consumption (reported at least one drink in past month vs. none), and body mass index (kilograms per square meter).

Results

Description of study subjects. The 1,140 study subjects consisted of 391 (34.3%) men and 749 (65.7%) women. Study subjects were mainly of white or African-American race/ethnicity (Table 3) and were living in 19 ZIP codes, but six ZIP codes provided 880 (77.2%) study participants. There were prominent differences in the prevalence of health conditions, medication use, and other covariates by sex and race/ethnicity (Table 5).

Models of neurobehavioral test scores by race/ethnicity. We compared the results of the three models of neurobehavioral test scores. In the base model (model 1; Table 6), controlling for age, sex, and testing technician, African Americans performed significantly worse than whites on each of the 20 neurobehavioral tests. The differences were large and appeared in all cognitive domains, including those assessed by tests with and without potential for differential measurement properties by race/ethnicity. In the base model, examination of standardized coefficients (after Z-transformation of the neurobehavioral test scores) revealed that, on average across all neurobehavioral tests, African Americans performed 0.64 standard deviation (SD) unit worse than whites (SD of these differences across all neurobehavioral tests = 0.16; the differences ranged from 0.39 for Purdue Pegboard dominant to 1.02 for Boston

Naming). For a normally distributed outcome, a span of 4 SDs encompasses approximately 95% of the distribution, so on average, 0.64 SD units is approximately 16% worse performance ($0.64 \div 4$).

In model 2, adjusting for the variables in model 1 and household income, household assets, educational attainment, and occupational status, the average difference in test performance of African Americans compared with

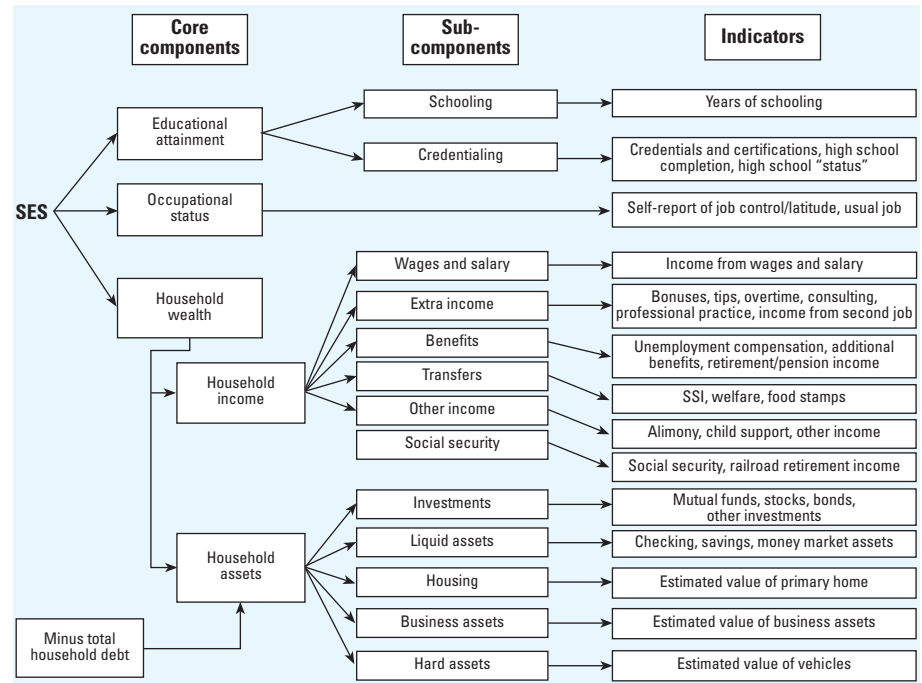


Figure 1. Conceptual model of individual SES used in the Baltimore Memory Study, 2001–2002. SSI, Supplemental Security Income.

Table 4. Distribution of study subjects by educational status,^a Baltimore Memory Study, 2001–2002.

Education	No. (%)	Percent	
		Blacks	Whites
< High school education	154 (13.5)		
< 10th grade	48 (4.2)	5.3	3.1
≥ 10th grade	76 (6.7)	8.2	5.6
Completed trade school ^b	30 (2.6)	4.6	1.0
High school graduate (or equivalency)	438 (38.5)		
Without trade school	194 (17.0)	21.7	12.8
With trade school ^c	244 (21.4)	31.9	13.4
Some college or associate degree	66 (5.8)	7.8	4.3
Baccalaureate degree	136 (11.9)	7.4	15.7
Some postbaccalaureate education	110 (9.7)	4.9	13.3
Postbaccalaureate degree	235 (20.6)	8.2	30.9
Total	1,139 ^d (100.0)		

^aBased on self-reported years of education plus additional questions on high school equivalency (i.e., GED), trade school and other credentials, and highest degree obtained. ^bTrade, nursing, or other similar credential. ^cThe reference group in the analysis. ^dOne person was missing information on education.

Table 5. Selected covariates by race/ethnicity (African American and white^a) and sex, Baltimore Memory Study, 2001–2002.

Variable	African American		White ^b	
	Women	Men	Women	Men
No.	339	135	368	244
Body mass index (kg/m ² , mean ± SD)	32.3 ± 7.4	28.7 ± 5.7	28.6 ± 7.1	28.4 ± 5.1
Blood lead level (µg/dL, mean ± SD)	3.2 ± 2.0	4.5 ± 2.8	3.0 ± 1.9	4.4 ± 2.8
History of diabetes [n (%)]	90 (26.5)	27 (20.0)	42 (11.4)	26 (10.7)
Taking anxiolytic medications [n (%)]	11 (3.2)	3 (2.2)	31 (8.4)	10 (4.1)
Taking antidepressant medications [n (%)]	10 (2.9)	3 (2.2)	58 (15.8)	14 (5.7)
Never used tobacco products [n (%)]	151 (44.5)	29 (21.5)	141 (38.3)	83 (34.0)
History of stroke [n (%)]	15 (4.4)	11 (8.1)	10 (2.7)	1 (0.004)
Consumed alcoholic beverage in past month [n (%)]	115 (33.9)	78 (57.8)	260 (70.7)	190 (77.9)

^aOther race/ethnicity groups were not tabulated because of small sample sizes. ^bIncludes 14 subjects who reported both white and Native American race/ethnicity.

whites declined by 25.8% compared with the base model (Table 6). Each of the four dimensions of SES was an independent and consistent predictor of neurobehavioral test scores. Educational attainment, occupational status,

household income, and household assets were associated ($p < 0.05$) with 20, 9, 5, and 13 of the 20 neurobehavioral tests, respectively. Lower levels of education (the lowest two categories, Table 4) were associated with worse

performance on all or almost all tests, whereas the highest two categories were associated with better performance on generally all tests except those with manual dexterity components (e.g., Purdue Pegboard, finger tapping, Simple Reaction Time). Occupational status was associated with several verbal tests (e.g., Boston Naming, RAVLT, Letter Fluency). There was no apparent prominent domain in which income associations were observed, whereas assets were associated with better performance on virtually all tests except Rey Complex Figure and RAVLT.

Finally, in model 3, also adjusting for blood lead, time of day of testing, taking medications for anxiety, history of diabetes, taking medications for hypertension, tobacco use, history of stroke, alcohol consumption, and body mass index, the average difference in test performance of African Americans compared with whites declined by 35.1% in relation to the base models (Table 7). However, significant differences persisted between African Americans and whites in these models. Examination of standardized coefficients revealed that after this adjustment, on average across all neurobehavioral tests, African Americans still had scores that were 0.43 SD units lower than those of whites (ranging from 0.12 for Purdue Pegboard dominant to 0.84 for Boston Naming).

Table 6. Associations of neurobehavioral test scores with race/ethnicity in the base model (model 1) and base model plus SES (model 2).

Test name	Model 1 β coefficients ^a			Model 2 β coefficients ^b		
	Black	Black-mix	Other	Black	Black-mix	Other
Boston Naming	-3.579 [#]	-2.646 [#]	-3.353 [#]	-2.983 [#]	-2.003 [#]	-2.915 [#]
Category fluency	-13.64 [#]	-9.721 [#]	-14.41 [#]	-9.396 [#]	-5.774 [*]	-11.72 [#]
Colored Progressive Matrices	-4.172 [#]	-3.129 [#]	-2.408 ^{**}	-2.766 [#]	-1.920 [*]	-1.492
Finger tapping, dominant	-6.100 [#]	-4.909 ^{**}	-2.615	-4.647 [#]	-3.569 [*]	-1.857
Finger tapping, nondominant	-4.759 [#]	-3.611 ^{**}	-2.509	-3.516 [#]	-2.482	-1.796
Letter fluency	-9.088 [#]	-7.460 ^{**}	-7.020 ^{**}	-5.634 [#]	-4.601 [*]	-4.701 [*]
Purdue Pegboard						
Dominant hand	-0.878 [#]	-1.313 ^{**}	-0.275	-0.482 [#]	-0.886 [*]	-0.020
Nondominant hand	-1.029 [#]	-1.033 ^{**}	-0.470	-0.671 [#]	-0.619	-0.239
Both hands	-1.077 [#]	-1.206 ^{**}	0.251	-0.700 [#]	-0.814 [*]	-0.411
Assembly	-5.501 [#]	-4.697 [#]	-1.613	-4.222 [#]	-3.496 ^{**}	-1.023
RAVLT						
Trials 1-5	-6.779 [#]	-4.143 [*]	-4.664 ^{**}	-4.872 [#]	-2.257	-3.396 [*]
Recognition	-1.118 [#]	-0.010	-0.842	-0.796 [#]	0.244	-0.528
Delayed recall	-1.906 [#]	-0.072	-1.330 [*]	-1.500 [#]	0.297	-1.037
Rey Complex Figure copy	-4.717 [#]	-5.238 [#]	-1.701	-3.542 [#]	-4.241 [#]	-0.843
Rey Complex Figure delayed	-2.673 [#]	-2.952 [#]	-2.394 [*]	-2.041 [#]	-2.352 ^{**}	-1.965 [*]
Simple Reaction Time ^c	-0.114 [#]	-0.103 ^{**}	-0.165 [#]	-0.079 [#]	-0.063	-0.145 [#]
Stroop Test	-16.33 [#]	-16.95 ^{**}	-10.08	-12.14 [#]	-13.25 [*]	-7.422
Symbol Digit	-2.610 [#]	-2.144 [#]	-2.413 [*]	-1.829 [#]	-1.422	-1.861 [*]
Trails A ^d	-0.302 [#]	-0.272 [#]	-0.280 [#]	-0.234 [#]	-0.193 ^{**}	-0.227 ^{**}
Trails B ^d	-0.396 [#]	-0.392 [#]	-0.311 [#]	-0.288 [#]	-0.275 [#]	-0.232 ^{**}
Systolic blood pressure	-9.585 [#]	-9.739 ^{**}	-2.100	-7.955 [#]	-8.652 [*]	-0.773
Diastolic blood pressure	-3.138 [#]	-2.437	-2.953	-3.050 [#]	-2.823	-2.576

^aAll coefficients have been standardized, so negative always indicates worse function; adjusted for age, sex, and technician; white race/ethnicity is the reference group. ^bAlso adjusted for educational attainment (nine categories), occupational status, household income (ln-transformed), and household assets (ln-transformed). ^cTrails A and B and reaction time were ln-transformed. ^d* $p < 0.05$; ** $p < 0.01$; # $p < 0.001$.

Table 7. Associations of neurobehavioral test scores with race/ethnicity in the base model plus SES plus health (model 3).

Test name	Model 3 β coefficients ^a		
	Black	Black-mix	Other
Boston Naming	-2.928 [#]	-1.982 [#]	-2.797 [#]
Category Fluency	-9.082 [#]	-5.520 [#]	-11.43 [#]
Coloured Progressive Matrices	-2.514 [#]	-1.758 [*]	-1.375
Finger tapping, dominant	-4.407 [#]	-3.385 [*]	-1.774
Finger tapping, nondominant	-3.492 [#]	-2.257	-1.587
Letter Fluency	-5.117 [#]	-4.543 [*]	-4.297
Purdue Pegboard			
Dominant hand	-0.246	-0.548	0.015
Nondominant hand	-0.442 ^{**}	-0.165	-0.170
Both hands	-0.502 [#]	-0.400	0.377
Assembly	-3.255 [#]	-2.336	-1.008
RAVLT			
Trials 1-5	-4.576 [#]	-2.256	-2.970
Recognition	-0.848 [#]	0.282	-0.476
Delayed recall	-1.409 [#]	0.295	-0.895
Rey Complex Figure copy	-3.361 [#]	-3.667 [#]	-0.631
Rey Complex Figure delayed	-1.932 [#]	-2.077 [#]	-1.742
Simple Reaction Time ^b	-0.084 [#]	-0.053	-0.131 ^{**}
Stroop Test	-12.28 [#]	-10.37 [*]	-7.201
Symbol Digit	-1.660 [#]	-1.206	-1.629
Trails A ^d	-0.213 [#]	-0.183 ^{**}	-0.207 [*]
Trails B ^d	-0.266 [#]	-0.244 ^{**}	-0.219 ^{**}
Systolic blood pressure	-5.961 [#]	-7.085 [*]	-3.066
Diastolic blood pressure	-2.650 [#]	-2.160	-3.516

^aAll coefficients have been standardized, so negative always indicates worse function; adjusted for age, sex, technician, education (nine categories), occupational status, household income (ln-transformed), household assets (ln-transformed), blood lead, time of day of testing (afternoon or evening vs. morning), taking medications for anxiety (yes vs. no), history of diabetes (yes vs. no), taking medications for hypertension (yes vs. no), tobacco use (current vs. never and previous vs. never), history of stroke (yes vs. no), alcohol consumption (reported at least one drink in past month vs. none), and body mass index (kg/m²). ^bTrails A and B and reaction time were ln-transformed. * $p < 0.05$; ** $p < 0.01$; # $p < 0.001$.

Discussion

In a large, community-based population sample of adults 50–70 years of age, randomly selected from the general population in Baltimore, there were large differences in neurobehavioral test scores by race/ethnicity in all assessed cognitive domains; these differences declined by approximately 25% after adjusting for individual SES and another 10% after adjusting for additional individual factors mainly relating to health and health-related behaviors. However, after potential confounders were included in the model, large differences in the cognitive test scores were still observed across three race/ethnicity groups. Each of the four dimensions of SES was an independent predictor of neurobehavioral test scores, suggesting that studies may not be able to rely on simple surrogates of SES (e.g., years of education) when examining race/ethnic differences.

Studies have demonstrated that the determinants of cognitive dysfunction, cognitive decline, and neurodegenerative disease are complex and multifactorial, and encompass biologic, environmental, behavioral, and social pathways. For example, a higher risk of Alzheimer's disease is consistently observed among those with low education levels (Evans et al. 1993, 1997a, 1997b; Geerlings et al. 1999; Graham et al. 1998; Gurland et al. 1999; Hall et al. 2000; Letenneur et al. 1999;

Prencipe et al. 1996; Wiederholt et al. 1993). Low education is also a predictor of cognitive decline, even in subjects < 60 years of age (Farmer et al. 1995; Lyketsos et al. 1999). Growing evidence indicates that various environmental influences, including traumatic injury, oxidative stress, neurotoxins, and medications, interact with endogenous biologic factors to influence cognitive function and risk of Alzheimer's disease (Small 1998). It is likely that other measures of SES, including income and occupational prestige, also predict risk of Alzheimer's disease (Evans et al. 1997b; Stern et al. 1994). Household income was associated with neurobehavioral test scores among our study subjects, as was, to a lesser degree, occupational status. Notably, few studies have evaluated household assets, which was a stronger predictor of neurobehavioral test scores than was household income.

Studies that have attempted to account for multiple domains of risk factors have reported that vascular changes in magnetic resonance imaging of the brain, measures of brain atrophy, apolipoprotein E (*ApoE*) genotype, age, education, and race are all associated with lower cognitive function scores in older individuals (Kuller et al. 1998). Approximately 50% of the variance in cognitive function in the elderly may be explained by genetic factors and educational achievement alone (Brandt et al. 1993), with heritability accounting for 30% of the total and shared environment accounting for an additional 18% of the variance. Notably, heritability of Alzheimer's disease in African Americans appears to account for less variance than it does in whites, suggesting a larger role for environmental factors in African Americans (Devi et al. 2000), an issue of particular note with regard to the *ApoE-ε4* allele and risk of Alzheimer's disease.

We examined the representativeness of our study sample in two ways. First, using 2000 Census data (U.S. Census Bureau 2002), we estimate that in our study area there reside 31,195 persons between the ages of 50 and 69 years who reported only a single race as African American or white. Of these, 19.1% were white men, 27.8% were African-American men, 18.4% were white women, and 34.6% were African-American women. Among our study subjects, the corresponding proportions were 22.0, 12.5, 33.8, and 31.7%, respectively. These data suggest that African-American men were underrepresented and white women were overrepresented in our sample. Two main factors could have influenced the representativeness of our study sample: differential phone ownership by race/ethnicity and differential participation rates by race/ethnicity. Second, we examined enrollment success in our study neighborhoods by race/ethnicity using 2000 Census data (U.S. Census Bureau 2002), and found no apparent trend in enrollment success

by neighborhood by the proportion of African Americans in the study neighborhoods. Although this is an imperfect way to evaluate differential recruitment or participation by race/ethnicity, the lack of a trend is reassuring and supports the notion that the study subjects represent the source population with telephones.

In summary, we observed large differences in neurobehavioral test scores by race/ethnicity. The differences became smaller but did not disappear after adjusting for SES, selected health measures, and health-related behaviors. Future analysis will continue to disentangle the complex web of determinants of cognitive dysfunction, with a broad set of determinants under investigation.

REFERENCES

- Bannon DI, Chisolm JJ. 2001. Anodic stripping voltammetry compared with graphite furnace atomic absorption spectrophotometry for blood lead analysis. *Clin Chem* 47:1703–1704.
- Barrera M. 1980. A method for the assessment of social support networks in community survey research. *Connections* 3:8–13.
- Brandt J, Welsh KA, Breitner JC, Folstein MF, Helms M, Christian JC. 1993. Hereditary influences on cognitive functioning in older men. A study of 4000 twin pairs. *Arch Neurol* 50:599–603.
- Brookmeyer R, Gray S, Kawas C. 1998. Projections of Alzheimer's disease in the United States and the public health impact of delaying disease onset. *Am J Public Health* 88:1337–1342.
- Derogatis LR, Lipman RS, Covi L. 1973. SCL-90: an outpatient psychiatric rating scale—preliminary report. *Psychopharmacol Bull* 9:13–28.
- Derogatis LR, Rickels K, Rock AF. 1976. The SCL-90 and the MMPI: a step in the validation of a new self-report scale. *Br J Psychiatry* 128:280–289.
- Devi G, Ottman R, Tang MX, Marder K, Stern Y, Mayeux R. 2000. Familial aggregation of Alzheimer disease among whites, African Americans, and Caribbean Hispanics in northern Manhattan. *Arch Neurol* 57:72–77.
- Diez Roux AV. 2001. Investigating neighborhood and area effects on health. *Am J Public Health* 91:1783–1789.
- Evans DA, Beckett LA, Albert MS, Hebert LE, Scherr PA, Funkenstein HH, et al. 1993. Level of education and change in cognitive function in a community population of older persons. *Ann Epidemiol* 3:71–77.
- Evans DA, Beckett LA, Field TS, Feng L, Albert MS, Bennett DA, et al. 1997a. Apolipoprotein E epsilon4 and incidence of Alzheimer disease in a community population of older persons. *JAMA* 277:822–824.
- Evans DA, Hebert LE, Beckett LA, Scherr PA, Albert MS, Chown MJ, et al. 1997b. Education and other measures of socioeconomic status and risk of incident Alzheimer disease in a defined population of older persons. *Arch Neurol* 54:1399–1405.
- Farmer ME, Kittner SJ, Rae DS, Bartko JJ, Regier DA. 1995. Education and change in cognitive function. The Epidemiologic Catchment Area Study. *Ann Epidemiol* 5:1–7.
- Fillenbaum GG, Peterson B, Welsh-Bohmer KA, Kukull WA, Heyman A. 1998. Progression of Alzheimer's disease in black and white patients: the CERAD experience, part XVI. Consortium to Establish a Registry for Alzheimer's Disease. *Neurology* 51:154–158.
- Geerlings MI, Schmand B, Jonker C, Lindeboom J, Bouter LM. 1999. Education and incident Alzheimer's disease: a biased association due to selective attrition and use of a two-step diagnostic procedure? *Int J Epidemiol* 28:492–497.
- Glass TA. 1998. Conjugating the “tenses” of function: discordance among hypothetical, experimental, and enacted function in older adults. *Gerontologist* 38:101–112.
- Glass TA, Balfour JL. 2003. Neighborhoods, aging and functional limitations. In: *Neighborhoods and Health* (Berkman LF, ed). New York:Oxford University Press, 303–334.
- Glass TA, Mendes de Leon CF, Seeman TE, Berkman LF. 1997. Beyond single indicators of social networks: a LISREL analysis of social ties among the elderly. *Soc Sci Med* 44:1503–1518.
- Graham C, Howard R, Ha Y. 1998. Dementia and ethnicity. *Int Psychogeriatr* 10:183–191.
- Grieco EM, Cassidy RC. 2001. Overview of race and Hispanic origin. Census 2000 Brief. Washington, DC:U.S. Census Bureau.
- Gurland BJ, Katz S. 1997. The subjective burden of depression. *Am J Geriatr Psychiatry* 5:188–191.
- Gurland BJ, Wilder DE, Lantigua R, Stern Y, Chen J, Killeffer EH, et al. 1999. Rates of dementia in three ethnoracial groups. *Int J Geriatr Psychiatry* 14:481–493.
- Hall KS, Gao S, Unverzagt FW, Hendrie HC. 2000. Low education and childhood rural residence: risk for Alzheimer's disease in African Americans. *Neurology* 54:95–99.
- Juster FT, Smith JP, Stafford F. 1999. The measurement and structure of household wealth. *Labour Econ* 6:253–273.
- Kaplan E, Goodglass H, Weintraub S. 2001. Boston Naming Test. 2nd ed. Baltimore, MD:Lippincott Williams & Wilkins.
- Kuller LH, Shemanski L, Manolio T, Haan M, Fried L, Bryan N, et al. 1998. Relationship between ApoE, MRI findings, and cognitive function in the Cardiovascular Health Study. *Stroke* 29:388–398.
- Launer LJ, Andersen K, Dewey ME, Letenneur L, Ott A, Amaducci LA, et al. 1999. Rates and risk factors for dementia and Alzheimer's disease: results from EURODEM pooled analyses. EURODEM Incidence Research Group and Work Groups. *European Studies of Dementia. Neurology* 52:78–84.
- Letenneur L, Gilleron V, Commenges D, Helmer C, Orgogozo JM, Dartigues JF. 1999. Are sex and educational level independent predictors of dementia and Alzheimer's disease? Incidence data from the PAQUID project. *J Neurol Neurosurg Psychiatry* 66:177–183.
- Liberatos P, Link BG, Kelsey JL. 1988. The measurement of social class in epidemiology. *Epidemiol Rev* 10:87–121.
- LIC. 1999. Quick Reference Guide for the Purdue Pegboard #32020—Test Administrator's Manual. Rev ed. Lafayette, IN:Lafayette Instrument Company.
- Lyketsos CG, Chen LS, Anthony JC. 1999. Cognitive decline in adulthood: an 11.5-year follow-up of the Baltimore Epidemiologic Catchment Area study. *Am J Psychiatry* 156:58–65.
- Macintyre S, Ellaway A, Cummins S. 2002. Place effects on health: how can we conceptualise, operationalise and measure them? *Soc Sci Med* 55:125–139.
- Munoz DG, Ganapathy GR, Eliasziw M, Hachinski V. 2000. Educational attainment and socioeconomic status of patients with autopsy-confirmed Alzheimer disease. *Arch Neurol* 57:85–89.
- Pearlin LI, Schooler C. 1978. The structure of coping. *J Health Soc Behav* 18:2–21.
- Perkins P, Annegers JF, Doody RS, Cooke N, Aday L, Vernon SW. 1997. Incidence and prevalence of dementia in a multiethnic cohort of municipal retirees. *Neurology* 49:44–50.
- Prencipe M, Casini AR, Ferretti C, Lattanzio MT, Fiorelli M, Culasso F. 1996. Prevalence of dementia in an elderly rural population: effects of age, sex, and education. *J Neurol Neurosurg Psychiatry* 60:628–633.
- Ratloff LS. 1977. The CES-D scale: a self report depression scale for research in the general population. *Appl Psychol Meas* 1:385–401.
- Raven J. 1965. The Coloured Progressive Matrices Test. London:Lewis Publishers.
- Raven JC, Court JH, Raven J. 1995. Manual for Raven's Coloured Progressive Matrices and Vocabulary Scales. Oxford, UK:Oxford Psychologists Press.
- Rose G. 1962. The diagnosis of ischaemic heart pain and intermittent claudication in field surveys. *Bull WHO* 27:645–658.
- Schmidt M. 1996. Rey Auditory Verbal Learning Test—A Handbook. Los Angeles, CA:Western Psychological Services.
- Shadlen MF, Larson EB, Gibbons L, McCormick WC, Teri L. 1999. Alzheimer's disease symptom severity in blacks and whites. *J Am Geriatr Soc* 47:482–486.
- Small GW. 1998. The pathogenesis of Alzheimer's disease. *J Clin Psychiatry* 59(suppl 9):7–14.
- Stern Y, Gurland B, Tatemichi TK, Tang MX, Wilder D, Mayeux R. 1994. Influence of education and occupation on the incidence of Alzheimer's disease. *JAMA* 271:1004–1010.
- Taylor RB. 1979. Toward a Neighborhood-Based Data File. Baltimore, MD:Johns Hopkins University Center for Metropolitan Planning and Research.

- U.S. Census Bureau. 2002. Census 2000 Summary File 3—Maryland. Washington, DC:U.S. Census Bureau.
- U.S. Department of Health and Human Services. 2000. Eliminating Racial and Ethnic Disparities in Health. Available: <http://raceandhealth.hhs.gov/sidebars/sbinitOver.htm> [accessed 26 January 2004].
- Welsh KA, Fillenbaum G, Wilkinson W, Heyman A, Mohs RC, Stern Y, et al. 1995. Neuropsychological test performance in African-American and white patients with Alzheimer's disease. *Neurology* 45:2207–2211.
- Wiederholt WC, Cahn D, Butters NM, Salmon DP, Kritzer-Silverstein D, Barrett-Connor E. 1993. Effects of age, gender and education on selected neuropsychological tests in an elderly community cohort. *J Am Geriatr Soc* 41:639–647.
- Wilkinson RT, Houghton D. 1982. Field test of arousal: a portable reaction timer with data storage. *Hum Factors* 24:487–493.
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