Associations Between Diabetes and Cognitive Function in Socioeconomically Diverse African American and White Men and Women

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ABSTRACT

Objectives: To examine whether race and poverty (income <125% of the federal poverty limit), modifies associations between diabetes and cognition in a biracial, urban-dwelling sample.

Methods: Cross-sectional data for 2066 participants (mean age = 47.6 years, 56.8% women, 56.2% African American, 38.6% below poverty) from the first wave of the Healthy Aging in Neighborhoods of Diversity across the Life Span study were used for analyses. Eleven tests measured cognitive function. Interactions among diabetes, race, and poverty status with cognition were assessed in multiple regression analyses.

Results: Significant interactions among diabetes, race, and poverty status were observed. Among African Americans below poverty, diabetic individuals performed lower than nondiabetic individuals on California Verbal Learning Test Free Recall Short Delay ($z = -0.444$ [0.123] versus $z = -0.137$ [0.045]) and Long Delay ($z = -0.299$ [0.123] versus $z = -0.130$ [0.045]), Digit Span Backward ($z = -0.347$ [0.109] versus $z = -0.072$ [0.041]), and the Brief Test of Attention ($z = -0.452$ [−0.099] versus $z = -0.099$ [0.047]), and higher on Category Fluency ($z = 0.114$ [0.117] versus $z = -0.118$ [0.044]). No consistent differences between diabetic and nondiabetic individuals were found for African American and white participants above poverty.

Conclusions: Diabetes was associated with poorer verbal memory, working memory, and attention among African Americans living in poverty. Diabetic African Americans below poverty may have increased risk of cognitive deficit at a younger age. Improving health literacy, doctor-patient communication, and multidisciplinary medical care for impoverished individuals may reduce differences. Additional research is needed to clarify mechanisms underlying these associations.

Key words: Type 2 diabetes, cognitive function, race, poverty status.

INTRODUCTION

Type 2 diabetes mellitus has been associated with dementia and cognitive decrements in cross-sectional and longitudinal studies (1). Possible mechanisms include increased microvascular pathology (2), vascular damage to white matter areas (3), inefficiency of glucose use during cognitive tasks (4), and diabetic comorbidities (1). Type 2 diabetes has been associated with decrements in performance on tests of attention, verbal and nonverbal memory, processing speed (1,5,6), executive function, and psychomotor speed and complex motor function (5).

African Americans have an increased risk of diabetes, and this risk is further increased with concomitant low socioeconomic status (7). Furthermore, African Americans are at an increased risk for diabetes-associated microvascular disease (8) and severe white matter lesions (9) compared

BMI = body mass index, **CES-D** = Centers for Epidemiologic Studies Depression Scale, **CRP** = C-reactive protein, **CVD** = cardiovascular disease, **CVLT** = California Verbal Learning Test, **HANDLS** = Healthy Aging in Neighborhoods of Diversity across the Life Span, **SBP** = systolic blood pressure, **WRAT** = Wide Range Achievement Test

SDC Supplemental Content

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with whites, suggesting that vascular-related white matter damage may be increased in diabetic African Americans compared with diabetic whites. Vascular pathology associated with diabetes (e.g., retinopathy) and white matter damage have both been shown to relate to cognitive decline (10–12).

Among nondiabetic individuals, African Americans are at higher risk for cognitive impairment (13) and dementia (14) than whites, and African Americans with diabetes are at increased risk for dementia compared with whites with diabetes (15). It has been estimated that eliminating racial disparities in Type 2 diabetes could reduce the relative difference in incidence of cognitive impairment and dementia between white and minority patients by 17% (16). Recent studies found that adjustment for socioeconomic status attenuated racial differences in risk of incident dementia (17) and racial differences in performance on neuropsychological tests (18,19), suggesting that the increased prevalence of dementia and cognitive decrements observed in African Americans may be due to lower socioeconomic status, rather than genetic variables. However, despite the importance of race and socioeconomic status as risk factors for the development of Type 2 diabetes, associated vascular complications, and dementia, we know of no studies that have examined the potential interactive relations among diabetes, race, and socioeconomic status to cognitive function among non-demented persons.

African Americans living in poverty may represent a population that is particularly vulnerable to the development of diabetes-associated vascular brain damage and correspondingly lower cognitive function. Therefore, for the current study, we examined the association between diabetes and cognitive function with race and poverty status as moderators of this association. We hypothesized that the magnitude of diabetes-associated decrement in cognitive function would be most pronounced in African Americans with household incomes below 125% of the poverty level.

METHODS

Sample

Data were taken from the first wave (July 2004–March 2009) of the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study (20). HANDLS is a population-based longitudinal study in which African Americans and whites aged 30 to 64 years were recruited as a fixed cohort of participants by household screenings from an area probability sample of 13 neighborhoods (contiguous census tracts) in Baltimore City. Approximately equal sample sizes were drawn for each cell, with cells defined by race African American or white), poverty status (above or below 125% of the federal poverty limit), age (in 5-year groups from 30 to 64 years), and sex. After sample selection, mobile research vehicles were driven to each neighborhood to collect physiological, behavioral, and cognitive data. More information on the study design and data collection procedures can be found elsewhere (20). The inclusion criteria for the study were as follows: a) ability to give informed consent, b) age 30 to 64 years, 3) ability to perform most study evaluations, and d) valid photograph identification. The exclusion criteria for enrollment in the study were as follows: a) currently pregnant; b) within 6 months of receiving chemotherapy, radiation, or biological treatments for cancer; c) previous diagnosis of AIDS; or d) uncontrolled high blood pressure (>160/100 mm Hg). Individuals with uncontrolled high blood pressure were excluded from further participation in the study because these participants were at increased risk for cardiovascular and cerebrovascular events during testing, and the mobile research vehicles were too far from emergency services to justify the risk.

Of the 2802 participants who completed the baseline medical examination at Wave 1, exclusionary criteria were applied in the following order: a) history of stroke (n = 60), b) probable dementia (n = 4), c) congestive heart failure (n = 63), d) diagnosis of HIV or AIDS (n = 66), e) dialysis (n = 1), f) Parkinson disease (n = 2), g) multiple sclerosis (n = 11), h) epilepsy (n = 84), i) history of transient ischemic attack (n = 51), j) missing all cognitive data (n = 19), k) not fasting before blood draw (n = 154), l) missing data for covariates in the basic model (n = 113), m) missing data for diabetes diagnosis (n = 55), or n) treated with insulin (n = 53) . Individuals treated with insulin were excluded because in some cases, it was difficult to discern the diagnosis of Type 2 diabetes from Type 1. The primary goal was to examine cognitive performance in individuals with Type 2 diabetes. A physician or nurse assessed all the exclusionary criteria by structured interview, with the exception of missing data and fasting status. Fasting status was determined by the physician or nurse at the time of blood draw. The final sample consisted of 2066 individuals. This study was approved by the MedStar institutional review boards, and informed consent for data collection was obtained from all participants.

Procedures

Data collection procedures are described in detail elsewhere (20). Briefly, participants were recruited via field interview, and demographic information was obtained through household interview. Individuals were then scheduled for a medical examination which took place on a Medical Research Vehicle. After an overnight fast, blood was drawn in the morning, followed by breakfast, cognitive testing, and medical examination. Blood samples were sent to Quest Diagnostics (Baltimore, MD; www.questdiagnostics.com) for analysis. Glucose, total cholesterol, high-density lipoprotein (HDL), and triglycerides were measured using a spectrophotometer (AU5400 Immuno Chemistry Analyser; Olympus, Center Valley, PA). High-sensitivity C-reactive protein (CRP) was assessed by the nephelometric method using latex particles coated with CRP monoclonal antibodies (Nephelometer II; Siemens/Behring, Minsk, Republic of Belarus). Glycated hemoglobin (HbA1c) was measured using the immunoturbidimetric method. The Healthy Eating Index-2010 (HEI-2010), an index of diet quality independent of quantity, is a measure of compliance with federal dietary guidelines for intake of the following foods: total fruit, whole fruit, total vegetables, greens and beans, whole grains, dairy, total protein foods, seafood and plant proteins, fatty acids, refined grains, sodium, and empty calories.

Diabetes was defined as glucose $\geq$ 7 mM, treatment, or self-reported history, and a glycemic control variable was computed as HbA1c < 7% (53 mmol/mol) (21). Height and weight were measured during the medical examination, and body mass index (BMI) was calculated as weight (in kilograms) divided by height (in meters) squared. Waist circumference (in meters) was measured using a nonextendable tape measure at the level of the iliac crest. Smoking status and alcohol consumption status (current/former/never) were determined by self-report. For these variables, current and former were combined into a single category. Poverty status was defined as an income below 125% of the federal poverty limit (22). Data on continuous income were not collected and therefore were not available for analysis. Drinking status measured by self-report. The Centers for Epidemiologic Studies Depression Scale (CES-D) (23) and the Wide Range

Achievement Test (WRAT) (24) were administered by trained examiners. Brachial systolic blood pressure (SBP) and diastolic blood pressure measurements were taken from the left and right arms in the seated position using an aneroid manometer and stethoscope after a 5-minute rest. The average of left and right arm pressures for each blood pressure measure was used for analyses.

Cognitive Variables

Cognitive variables, along with the specific abilities measured by each cognitive variable, are shown in Table 1. Details of test administration have been described previously (29). Note that some participants did not complete all cognitive tests, and therefore, sample size differed slightly for each regression analysis (described later). Both Trail Making A and Trail Making B were not normally distributed and were natural log transformed. All cognitive variables were *z* transformed based on their respective means and standard deviations. This linear transformation resulted in each cognitive variable having a mean of zero and a standard deviation of one, and facilitates comparison of results among cognitive measures. Scores for the Benton Visual Retention Test, Trail Making A, and Trail Making B were reversed, so that lower scores indicate lower performance.

Statistical Analyses

Multiple regression analyses (SAS 9.1 PROC GLM) were performed with diabetes status (yes/no) as the primary predictor and cognitive function variables as the outcome. First, we examined diabetes by race by poverty status interactions. We then tested diabetes by race and diabetes by poverty status, followed by examination of diabetes main effects. An α level of .05 was used for all analyses. A multivariate analysis of variance (MANOVA) was performed to control for the number of outcome variables. The MANOVA did not include Mini-Mental State Examination because this measure was included as a separate measure of cognitive status. Where interactions were significant, the pattern of adjusted means was examined. Covariates used in regression analyses included age (years), education (years), sex, WRAT total score, race, poverty status, SBP (mm Hg), current smoking (yes/no), triglycerides (mM), BMI (kg/m^2), drinker (yes/no), CES-D, CRP (nM), and the HEI-2010. These variables were used in the following covariate sets:

Demographic covariate set = age, education, sex, WRAT score, race, poverty status

Demographic + cardiovascular disease (CVD) risk covariate set = demographic covariates + SBP, smoking, triglycerides, BMI, drinker, CES-D, CRP, HEI-2010

To maintain sample size despite missing covariates, particularly CRP (n = 74 missing), and the HEI-2010 (n = 373 missing), values for covariates were imputed using PROC MI (10 imputations) and analyzed with PROC REG and PROC MIANALYZE.

RESULTS

Descriptive statistics for the overall sample, and by race and poverty status, are shown in Table 2. Within African Americans, participants above and below the poverty limit differed on age, education, WRAT score, BMI, waist circumference, low-density lipoprotein (LDL), HDL, triglycerides, CES-D, and percent smokers. Within whites, the pattern of results was similar, except that individuals above and below poverty did not differ on age, waist circumference, LDL, and triglycerides, and did differ on sex, proportion of diabetic individuals, and proportion of drinkers.

Unadjusted cognitive means (*z* scores) by poverty status for the overall sample and by diabetic status and poverty status for whites and African Americans are show in Table S1, Supplemental Digital Content 1, http://links.lww.com/PSYMED/A219. Individuals below poverty performed lower on all cognitive tests, with the exception of the California Verbal Learning Test (CVLT)–Total. This pattern was also observed among nondiabetic whites, but was less apparent for diabetic whites and African Americans.

A MANOVA for diabetes by race by poverty status interactions, with adjustment for the demographic covariate set, was significant ($F(10,1434) = 1.92$, $p = .038$, Wilks

TABLE 1. Description of Cognitive Tests Used in HANDLS

Cognitive Test Variable	*n*	Cognitive Ability Measured
Benton Visual Retention Test	2062	Visuomotor response, visuospatial perception, visual and verbal conceptualization, immediate memory span
CVLT—Total Trials A	2058	Verbal learning and memory
CVLT—Short Delay Free Recall	1653	Delayed verbal memory
CVLT—Long Delay Free Recall	1651	Delayed verbal memory
Digit Span Forward	2061	Attention
Digit Span Backward	2059	Attention and working memory
Category Fluency (Animals)	2045	Verbal fluency and executive function
Trail Making A	2043	Attention, psychomotor speed
Trail Making B	2036	Attention, psychomotor speed, executive function
Brief Test of Attention	1727	Auditory divided attention
Mini-Mental State Examination	2054	Mental status

CVLT = California Verbal Learning Test.

TABLE 2. Demographic Information and Health Characteristics for the Overall Sample and by Race and Poverty Status

Variable	Overall (N = 2066)	White			African American			p for Race by Poverty Interaction
		Above Poverty (n = 632)	Below Poverty (n = 270)	p	Above Poverty (n = 639)	Below Poverty (n = 525)	p	
Age, M (SD), y	47.5 (9.3)	48.1 (9.4)	47.2 (9.5)	.19	48.0 (9.3)	46.1 (9.0)	<.001	.25
Education, M (SD), y	12.6 (3.1)	13.4 (3.6)	11.4 (3.1)	<.001[a]	12.9 (2.7)	11.6 (2.3)	<.001[a]	.012
WRAT total score, M (SD)	42.4 (8.1)	46.1 (7.3)	42.1 (8.5)	<.001[a]	41.4 (7.1)	39.3 (8.3)	<.001[a]	.012
Women, n (%)	1173 (56.8)	344 (54.4)	173 (64.1)	.008	350 (54.7)	306 (58.3)	.24	.18
Glucose, M (SD), mg/dl	101.5 (34.1)	101.7 (33.7)	104.1 (36.1)	.34	101.8 (34.3)	99.6 (33.1)	.27	.15
HbA1c, M (SD), %	5.9 (1.1)	5.7 (1.0)	5.8 (1.1)	.26	6.0 (1.1)	6.0 (1.2)	.51[a]	.21
SBP, M (SD), mm Hg	120.2 (17.2)	118.0 (17.0)	119.3 (18.9)	.36[a]	121.4 (16.3)	122.0 (17.6)	.55	.69
DBP, M (SD), mm Hg	72.8 (10.7)	71.9 (10.2)	73.0 (11.0)	.15	73.1 (10.5)	73.6 (11.2)	.44	.55
Hypertension, n (%)	741 (35.8)	206 (32.6)	86 (31.9)	.88	253 (39.6)	196 (37.3)	.43	.76
Total cholesterol, M (SD), mg/dl	187.9 (43.0)	193.1 (41.6)	188.6 (44.6)	.14	186.6 (44.2)	182.7 (41.6)	.13	.87
LDL, M (SD), mg/dl	110.3 (36.4)	114.4 (35.2)	110.6 (36.2)	.15	110.3 (36.3)	105.5 (37.3)	.026	.76
HDL, M (SD), mg/dl	53.2 (17.4)	50.6 (14.8)	47.5 (13.9)	.003	54.7 (17.2)	57.4 (20.7)	.018[a]	<.001
Triglycerides, M (SD), mg/dl	124.3 (119.9)	143.2 (109.7)	161.7 (232.0)	.21[a]	109.3 (87.6)	100.5 (56.2)	.038[a]	.013
C-reactive protein, M (SD), mg/dl	4.7 (9.0)	4.2 (7.4)	5.0 (6.5)	.090[a]	4.6 (7.3)	5.3 (13.1)	.29[a]	.86
Diabetes (glucose), n (%)[b]	268 (13.0)	65 (10.3)	42 (15.6)	.032	98 (15.3)	63 (12.0)	.11	.006
Diabetes (HbA1c), n (%)[c]	337 (16.3)	63 (10.0)	42 (15.6)	.023	140 (21.9)	92 (17.5)	.066	.003
Duration of diabetes, M (SD)[d], y	6.1 (7.9)	7.8 (11.7)	5.0 (5.7)	.19[a]	4.9 (5.6)	7.0 (7.2)	.087	.052
Diabetes controlled, n (%)[e]	135 (50.4)	33 (50.8)	23 (54.8)	.70	50 (51.0)	29 (46.0)	.63	.48
Body mass index, M (SD), kg/m^2	30.0 (7.7)	29.8 (7.2)	30.8 (8.5)	.081[a]	30.6 (7.4)	29.1 (8.0)	<.001	<.001
Waist circumference, M (SD), cm	100.2 (26.6)	102.5 (40.0)	103.4 (18.4)	.62[a]	101.0 (16.4)	95.1 (17.8)	<.001[a]	.008
CES-D, M (SD)	14.7 (11.3)	13.4 (10.9)	18.0 (12.2)	<.001[a]	12.8 (10.4)	16.7 (11.5)	<.001[a]	.57
Current smoker, n (%)	895 (43.3)	226 (35.8)	145 (53.7)	<.001	242 (37.9)	282 (53.7)	<.001	.63
Drinker, n (%)	1133 (54.8)	374 (59.2)	270 (51.9)	.047	349 (54.6)	270 (51.4)	.29	.37

M = mean; SD = standard deviation; WRAT = Wide Range Achievement Test; HbA1c = glycated hemoglobin; SBP = systolic blood pressure; DBP = diastolic blood pressure; LDL = low-density lipoprotein; HDL = high-density lipoprotein; CES-D = Centers for Epidemiologic Studies Depression Scale.

t Tests were used to compare continuous measures, and Fisher exact test was used to compare proportions.

[a] t Test for unequal variances (Satterthwaite degrees of freedom).

[b] Diabetes defined as glucose ≥ 126 mg/dl, treatment, or self-reported history.

[c] Diabetes defined as A1c ≥ 6.5%, treatment or self-reported history.

[d] Includes only participants with diabetes.

[e] Percentage of diabetic individuals meeting the criteria for glycemic control, defined as A1c < 7% (16).

$\Lambda = 0.987$). With adjustment for the demographic covariate set, diabetes by race by poverty status interactions were observed for the CVLT Short Delay Free Recall ($p = .037$), CVLT Long Delay Free Recall ($p = .038$), Digit Span Backward ($p = .053$), Category Fluency ($p = .019$), and the Brief Test of Attention ($p = .044$). Comparisons between diabetic and nondiabetic participants within each of the four race/poverty status groups indicated that diabetic African Americans below poverty performed more poorly, compared with nondiabetic African Americans below poverty, on CVLT Short Delay Free Recall ($p = .019$), Digit Span Backward ($p = .018$), and the Brief Test of Attention

(p = .010). None of the comparisons between diabetic and nondiabetic individuals among the four groups differed for CVLT Free Recall Long Delay (all p > .20) and Category Fluency (all p > .062). All nonsignificant interactions with adjustment for the demographic covariate set are shown in Figure S1, Supplemental Digital Content 2, http://links.lww.com/PSYMED/A220.

With adjustment for the demographic + CVD risk model, significant diabetes by race by poverty status were observed for CVLT Short Delay Free Recall (p = .036), CVLT Long Delay Free Recall (p = .039), Digit Span Backward (p = .034), and the Category Fluency Test (p = .024). The diabetes by race by poverty status interaction was no longer observed for was marginal for the Brief Test of Attention (p = .056). For CVLT Free Recall Long Delay, no differences between diabetic and nondiabetic individuals in any of the race/poverty status groups were statistically significant (all p > .10). Adjusted means illustrating the nature of the interactions for CVLT Long Delay Free Recall, Digit Span Backward, Category Fluency, and the Brief Test of Attention are shown in Figure 1. Associations between diabetes and cognitive function were observed for only for African Americans below poverty status (p = .018, p = .018, p = .034, and p = .027, respectively). These associations were negative for all cognitive tests, with the exception of Category Fluency.

Results for regression analyses relating diabetes to cognitive variables for the Demographic model are shown in Table 3. Diabetes was related to Trail Making A and B, and the Brief Test of Attention with adjustment for the demographic model. With adjustment for the demographic + CVD risk model, a difference in performance

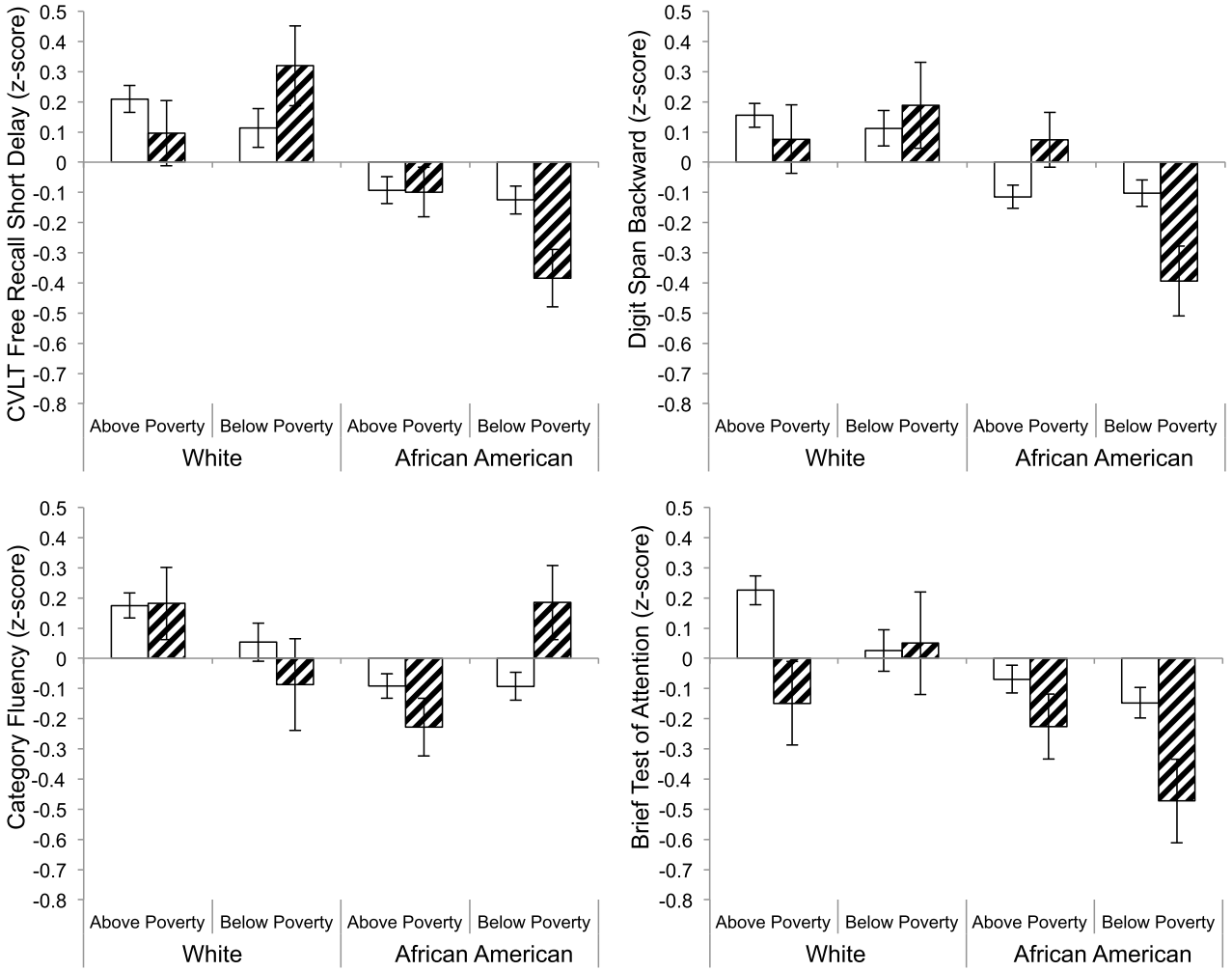

FIGURE 1. Adjusted means by diabetic status, race, and poverty status for CVLT Free Recall Short Delay (A), Digit Span Backward (B), Category Fluency (C), and the Brief Test of Attention (D). Means adjusted for age, education, sex, WRAT total score, race, poverty status, SBP, smoking (yes/no), triglycerides, BMI, drinker (yes/no), CES-D, and C-reactive protein. White bars, nondiabetic; cross-hatched bars, diabetic. Error bars indicate standard error of the mean. CVLT = California Verbal Learning Test; WRAT = Wide Range Achievement Test; SBP = systolic blood pressure; BMI = body mass index; CES-D = Centers for Epidemiologic Studies Depression Scale.

TABLE 3. Adjusted Means and SEM Illustrating the Association Between Diabetes and Cognitive Function

		Demographic Model[a]			Demographic + CVD Risk Model[b]		
Cognitive Outcome		Nondiabetic	Diabetic	*p*	Nondiabetic	Diabetic	*p*
BVRT errors[c]	Mean	0.012	−0.083	.11	0.014	−0.050	.32
	SEM	0.021	0.056		0.022	0.060	
CVLT Total A Trials	Mean	−0.003	0.022	.68	−0.018	0.010	.68
	SEM	0.022	0.058		0.023	0.063	
CVLT Short Delay Free Recall	Mean	0.009	−0.057	.31	0.010	−0.097	.12
	SEM	0.023	0.060		0.024	0.064	
CVLT Long Delay Free Recall	Mean	0.004	−0.023	.68	0.004	−0.095	.14
	SEM	0.023	0.060		0.024	0.063	
Digit Span Forward	Mean	0.012	−0.079	.13	−0.002	−0.040	.56
	SEM	0.021	0.056		−0.022	0.060	
Digit Span Backward	Mean	0.006	−0.039	.44	−0.0004	−0.006	.93
	SEM	0.020	0.053		0.021	0.057	
Category Fluency	Mean	−0.003	0.022	.68	0.008	−0.002	.88
	SEM	0.022	0.057		0.022	0.060	
Trail Making A[c]	Mean	0.017	−0.117	.028	0.017	−0.105	.064
	SEM	0.022	0.057		0.023	0.061	
Trail Making B[c]	Mean	0.015	−0.101	.038	0.012	−0.093	.079
	SEM	0.020	0.052		0.021	0.056	
Attention	Mean	0.032	−0.217	<.001	0.009	−0.215	.002
	SEM	0.024	0.063		0.025	0.068	
MMSE	Mean	0.008	−0.051	.25	0.003	−0.061	.25
	SEM	0.018	0.048		0.019	0.051	

SEM = standard error of the mean; CVD = cardiovascular disease; BVRT = Benton Visual Retention Test; CVLT = California Verbal Learning Test; MMSE = Mini-Mental State Examination; WRAT = Wide Range Achievement Test; SBP = systolic blood pressure; BMI = body mass index; CES-D = Centers for Epidemiologic Studies Depression Scale; CRP = C-reactive protein.

Cognitive variable means are in *z* score units.

[a] Demographic model = diabetes, age, education, sex, WRAT score, race, poverty status.

[b] Demographic + CVD risk model = demographic + SBP, smoking, triglycerides, BMI, alcohol consumption (yes/no), CES-D, and CRP.

[c] Outcome variable reversed, so that lower scores mean lower performance.

by diabetes status was only observed for the Brief Test of Attention. This association was qualified by a diabetes by race by poverty status interaction, as discussed earlier.

We also performed the earlier analyses with a) diastolic blood pressure substituted for SBP; b) HDL, LDL, or total cholesterol substituted for triglycerides; c) waist circumference substituted for BMI; and d) individuals treated with insulin included in the analyses. The pattern of results was the same, except that with individuals treated with insulin included, a diabetes by race interaction was observed for CVLT Short Delay Free Recall ($p = .04$). African Americans with diabetes performed the lowest on this test. We also performed analyses with HbA1c > 6.5% defined as diabetes, instead of glucose ≥ 126 mg/dl. Table 4 shows the proportion of individuals defined as diabetic according to the diagnostic criteria used by race and poverty status. Among white individuals, the proportion of diabetic individuals remained largely unchanged. However, when HbA1c was used as the criteria for diabetes, a higher proportion of African American participants were defined as diabetic, regardless of poverty status. Despite this difference in estimated prevalence of diabetes, the pattern of results was unchanged when diabetes was defined using HbA1c.

DISCUSSION

To our knowledge, this is the first study to examine associations between diabetes and cognitive performance as a function of both race and poverty status. Associations between diabetes and cognitive performance were observed only for African American individuals with household incomes below the poverty limit. With adjustment for demographic covariates, we found that these individuals performed lower on tests involving verbal memory, working memory, executive function, and attention. We found

TABLE 4. Proportion of Diabetic Individuals According to Diagnostic Criteria, by Race and Poverty Status

	Diagnostic Criteria	
Race/Poverty Status Group	Glucose[a]	HbA1c[b]
White, above poverty	10.3%	10.0%
White, below poverty	15.6%	15.6%
African American, above poverty	15.3%	21.9%
African American, below poverty	12.0%	17.5%

HbA1c = glycated hemoglobin.

[a] Diabetes defined as glucose ≥ 126 mg/dl, treatment, or self-reported history.

[b] Diabetes defined as HbA1c ≥ 6.5%, treatment, or self-reported history.

lower levels of performance in verbal memory, working memory, and attention even after adjusting for CVD risk factors. Findings for these particular domains of cognitive functions are largely consistent with previous studies examining associations between Type 2 diabetes and cognitive performance, which have similarly noted differences in tests of attention, verbal and nonverbal memory, and processing speed (1,5,6), as well as executive function (1,5).

Unexpectedly, we did find a positive association between diabetes and Category Fluency within African Americans below poverty. The reason why diabetic individuals perform higher than nondiabetic individuals in this group is unclear, and this may be a chance finding. The magnitudes of the cognitive differences observed in the current study were approximately 0.3 standard deviations. These are consistent with a small effect size but, at a population level, may represent meaningful effects. In that regard, the magnitude of the association between diabetes and cognitive function in African American participants living in poverty is approximately half of that that seen in older diabetic apolipoprotein E-epsilon 4 carriers (30,31).

Interestingly, we did not find consistent associations between diabetes and cognitive variables for any of the other race/poverty status groups. It should be noted, however, that the sample examined in this study was younger compared with previous studies relating diabetes to cognitive function (32). This lack of association between diabetes and cognitive function is consistent with the observation that diabetes-related cognitive deficits have been primarily observed in older (i.e., >65 years) rather than younger individuals (33). The current sample was relatively young (mean age = 47.5 years) compared with previous studies. This suggests that differences in cognitive performance associated with diabetes may develop slowly over time, largely occurring at older ages.

Differences in duration of diabetes may, at least in part, explain differences in associations between diabetes by race and poverty status group. African Americans below poverty had a higher self-reported duration of diabetes compared with African Americans living above poverty. Furthermore, whites above poverty, the only other group for which diabetes-associated cognitive decrement is observed, have the highest duration of diabetes. However, this association is seen most consistently in African Americans below poverty, suggesting that duration of diabetes may only partly explain diabetes-related decrements in cognitive function observed in diabetic African Americans below poverty.

That we do find evidence of lowered cognitive function in a sample of younger African Americans living in poverty seems to suggest that this group is the most vulnerable to diabetes-related cognitive deficits, and that these deficits may manifest at a younger age, compared with the other groups. The association between diabetes and cognitive function in African Americans below the poverty limit was observed after adjustment for other CVD risk factors, although diabetes severity and control were approximately the same as observed in the other groups (Table 2). In addition, diabetes-associated CVD risk factors (i.e., blood pressure, BMI, and cholesterol panel variables) did not differ significantly among the groups. CRP was slightly elevated and more variable in African Americans living in poverty, possibly suggesting the influence of inflammatory mechanisms in the association between diabetes and cognitive function in this group. However, CRP was included in covariate models, but statistical adjustment for CRP may not have accounted for all the effects of chronic inflammation.

A previous study (34) found that African American and white individuals with diabetes did not differ in terms of cognitive performance, with both groups performing lower on tests of semantic memory than nondiabetics. Similar results were noted in the current study; diabetes by race interactions were nonsignificant for all cognitive outcome variables. Therefore, it seems that African Americans may only be at decreased risk for diabetes-related cognitive deficit when these individuals are additionally burdened with poverty.

A higher prevalence of severe retinopathy has been observed in diabetic African Americans, and this increased prevalence is not related to an increase in risk factors such as age, HbA1c values, or duration of diabetes (8). Diabetic retinopathy has been shown to be associated with delayed word recall, processing speed, and executive function (35). In addition, African Americans have a higher prevalence of intracranial atherosclerotic stroke and lacunes compared with whites, and some of this increased vascular risk is associated with the increased prevalence of diabetes in this group (36). Indices of increased inflammation and diabetes comorbidities not assessed in this study (37) may also be related to the present findings.

Poverty has been shown to affect development of cognitive skills in childhood (38), and low socioeconomic status in childhood is an important risk factor for lowered cognitive function in middle age (39). Thus, impaired cognitive

development due to poverty and the increased burden of vascular damage as a result of diabetes may combine to result in lowered cognitive function. Furthermore, the cognitive decrements due to this doubly increased burden may manifest earlier than that associated with either poverty or diabetes alone, and this may be exacerbated in African Americans.

It has also been reported that a higher proportion of African Americans and those with lower incomes are less likely to have a reliable source of primary care and are more likely to be unable to obtain medical care or prescription medications, compared with whites and those with higher incomes (40). African Americans may be more likely to distrust the health care system than whites, perhaps due to increased experience of discrimination (41). In addition, African American adults and those below the poverty limit have a higher risk of having poor health literacy than the general population (42), and poor health literacy has been linked to higher HbA1c values (43). These factors may combine to exacerbate diabetes and related comorbidities, as well as resulting vascular pathology.

There may be other possibilities that explain our current findings. Low socioeconomic status is associated with impaired development of cognitive skills including attention, working memory, and literacy (38). Health literacy in particular has been shown to predict knowledge of diabetes (25,26), with low-literacy participants less likely to have knowledge regarding the signs of hypoglycemia and hyperglycemia, as well as corrective action required to address these issues (26,27). Therefore, poverty may lead to decreased ability to care for chronic diseases such as diabetes, which may lead to complications such as increased vascular damage to brain areas important to the ability to provide self-care, resulting in a vicious circle.

Limitations of this study should also be noted. First, the cross-sectional nature of this study did not allow us to examine change in cognitive function in association with diabetes. Second, we were unable to precisely determine Type 1 versus Type 2 diabetes, but we did exclude diabetic individuals treated with insulin to better isolate those with Type 2 diabetes. It is likely that some individuals with Type 2 diabetes on insulin therapy were excluded. However, the pattern of observed results was the same regardless of whether insulin-treated participants were included or excluded. Third, we did not collect data on continuous income, which would have allowed us to examine continuous and nonlinear associations between poverty and cognitive function. Fourth, we did not have continuous glucose monitoring, which would have allowed us to examine glucose control more closely.

This study has several strengths. This is the first study investigating the association between diabetes and cognitive function in the context of both poverty and race. Because of the design of HANDLS, a large number of individuals living in poverty (approximately 40% of the sample) and African Americans (approximately 56%) were recruited, thus allowing us to examine the influence of these important variables.

The findings of this study indicate that African Americans living in poverty may be at increased risk for poorer cognitive performance, and perhaps at a younger age, than other race/poverty status groups. Further study is needed to determine the directionality of these associations, as well as uncover possible treatment strategies to reduce any impact of diabetes on cognitive function within this group. Strategies aimed at improving health literacy, doctor-patient trust and communication, more inclusive medical care, and increased retinopathy screening in impoverished individuals may hold some promise. The full mobilization of the Affordable Care Act may offer promise for increasing access to care and disease prevention, but increased access to care may not fully attenuate disparities in control of cardiovascular risk factors (28). Ongoing efforts to understand the subjective experience of diabetes may provide more effective ways of encouraging self-care among diabetic patients with higher metabolic risk and increased risk for cognitive decrement.

Contribution Statement: G.A.D. performed statistical analyses, interpreted the results, and drafted the manuscript. S.R.W. contributed to statistical analyses, interpreted the results, and critically reviewed the manuscript for intellectual content. M.K.E. and A.B.Z. are the principal investigators for the HANDLS study, designed the study, interpreted the results, and critically reviewed the manuscript for intellectual content. All authors read and approved the final version.

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