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Differential Associations of Socioeconomic Status with Global Brain Volumes and White Matter Lesions in African American and White Adults: the HANDLS SCAN study

Shari R. Waldstein, Ph.D.^{1,2,3}, Gregory A. Dore, Ph.D.⁴, Christos Davatzikos, Ph.D.⁵, Leslie I. Katzel, M.D., Ph.D.^{2,3}, Rao Gullapalli, Ph.D.⁶, Stephen L. Seliger, M.D.⁷, Theresa Kouo, M.D.⁶, William F. Rosenberger, Ph.D.⁸, Guray Erus, Ph.D.⁵, Michele K. Evans, M.D.⁴, and Alan B. Zonderman, Ph.D.⁴

¹Department of Psychology, University of Maryland, Baltimore County

²Division of Gerontology & Geriatric Medicine, University of Maryland School of Medicine

³Geriatric Research Education and Clinical Center, Baltimore VA Medical Center

⁴Laboratory of Epidemiology and Population Sciences, National Institute on Aging Intramural Research Program

⁵Section of Biomedical Image Analysis, Department of Radiology, University of Pennsylvania

⁶Department of Diagnostic Radiology, University of Maryland School of Medicine

⁷Division of Nephrology, Department of Medicine, University of Maryland School of Medicine

⁸Department of Statistics, George Mason University

Abstract

Objective—Examine interactive relations of race and socioeconomic status (SES) to magnetic resonance imaging (MRI) - assessed global brain outcomes with previously demonstrated prognostic significance for stroke, dementia, and mortality

Methods—Participants were 147 African Americans (AAs) and Whites (ages 33 to 71 years; 43% AA; 56% female; 26 % below poverty) in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) SCAN sub-study. Cranial MRI was conducted using a 3.0 Tesla unit. White matter (WM) lesion volumes and total brain, gray matter (GM) and WM volumes were computed. An SES composite was derived from education and poverty status.

Results—Significant interactions of race and SES were observed for WM lesion volume ($b = 1.38$; $\eta^2 = 0.036$; $p = .028$), total brain ($b = 86.72$; $\eta^2 = 0.042$; $p = <.001$), GM ($b = 40.16$; $\eta^2 = 0.032$; $p = .003$), and WM ($b = 46.56$; $\eta^2 = 0.050$; $p < .001$). AA participants with low SES exhibited significantly greater WM lesion volumes than white participants with low SES. White

Corresponding Author: Shari R. Waldstein, Ph.D., Department of Psychology, University of Maryland, Baltimore County, 1000 Hilltop Circle, Baltimore, MD 21250, waldstei@umbc.edu, Phone: 410-455-2374, Fax: 410-455-1055.

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DISCLOSURES

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participants with higher SES had greater brain volumes than all other groups (albeit within normal range).

Conclusions—Low SES was associated with greater WM pathology – a marker for increased stroke risk – in AAs. Higher SES was associated with greater total brain volume – a putative global indicator of brain health and predictor of mortality – in Whites. Findings may reflect environmental and interpersonal stressors encountered by AAs and those of lower SES and could relate to disproportionate rates of stroke, dementia, and mortality.

Keywords

subclinical brain pathology; brain volume; MRI; socioeconomic status; race

INTRODUCTION

Although disparities between African Americans (AAs) and Whites are apparent for all leading causes of death in the United States, the differential mortality associated with stroke is most pronounced (1). Indeed, AAs have twice the risk of incident stroke than Whites. AA-White disparities extend to multiple additional neurological endpoints including post-stroke dementia, vascular dementia, and Alzheimer's disease (2–4). Self-identified AA race further confers greater risk for cognitive decline, decreased physical function, disability, frailty, and magnetic resonance imaging (MRI)-assessed subclinical brain pathology (5–12).

Three of the most commonly derived MRI-based indices of subclinical brain pathology include white matter (WM) lesions, subclinical or “silent” brain infarcts, and brain atrophy. WM lesions reflect cerebral small vessel disease commonly associated with vascular risk factors (and/or cerebral amyloid angiopathy) and include diffuse areas of nonspecific injury (13). Subclinical or “silent” infarcts are characterized by focal areas of infarction (e.g., lacunes) without clinical signs of stroke (14). These types of pathology can be characterized by clinical ratings or reflected in a quantitative WM lesion volume score. Brain (or cerebral) atrophy refers to glial, synaptic, dendritic, and/or neuronal loss with age and/or pathology that is reflected in lesser brain volume assessed quantitatively or by clinical rating. However, it's important to note that there is much variability in “normal” brain volumes with no clear cut scores as to what constitutes pathology. Nonetheless, each of these MRI-based measures of brain health are of notable public health concern as they have demonstrated prognostic significance for future cognitive decline, progression to dementia, stroke, and mortality, even among initially healthy individuals (14–20). Furthermore, total brain volume has been suggested to be a global indicator of brain health that has predictive utility with respect to all-cause mortality risk (21).

AAs are more likely than Whites to have lower socioeconomic status (SES), another potent risk factor for stroke (22), dementia (23), cognitive and functional decline and disability (6, 24), and subclinical brain pathology (25, 26). Relations of AA race to clinical brain health endpoints (e.g., stroke) remain significant following statistical adjustment for SES (27). However, lower SES may *potentiate* poor brain health outcomes among AAs. In that regard, a more pronounced relation of SES to stroke has indeed been reported in Black versus White men (23). Furthermore, both AAs and persons of lower SES have a greater prevalence

and/or more severe cardiovascular and metabolic risk factors such as smoking, hypertension, and diabetes (28) that are known to confer risk for stroke, dementia, and subclinical brain pathology (13,14). However, to our knowledge, it is unknown whether lower SES is associated with greater subclinical brain pathology among AAs than Whites.

Noble and colleagues have demonstrated elegantly that the relation of lower family SES to lesser MRI-assessed brain volumes in children is independent of genetic markers of African ancestry (29). However, race is a social construct, and it is possible that self-identified AAs are more vulnerable to SES influences on brain health, in part, due to exposure to multiple environmental and interpersonal stressors in the United States (28).

Individual level SES is also a social construct with multiple indicators such as income, education, occupation, and wealth. However, no single SES indicator fully captures all complexities of social disadvantage (30). Further, it's been argued that examination of single SES indicators oversimplifies the construct and can overestimate the effects attributable to that particular variable. Therefore, the utility of combining SES indicators has been suggested and may better capture dimensions of cumulative risk. Here we created a composite index of SES predicated on both education and poverty status.

It has more generally been recommended that research in health disparities give dual consideration to race and SES (28). Indeed, these variables are often conflated, and it may not be possible to fully disentangle their respective influences on health outcomes. Williams and colleagues emphasize the need to explicitly assess potential interactions of race and SES as sole statistical adjustment of one for the other can yield highly misleading findings (28).

Here, we examined potential interactions of self-identified race and SES to global measures of MRI-assessed WM lesion volumes, and total brain, gray matter (GM), and WM volumes. We posited disparities in these early indicators of brain health such that synergistic associations of race and SES would reveal the greatest vulnerability for lower SES AAs with respect to subclinical brain pathology.

METHODS

Participants

Participants were 147 AA and White adults (aged 33 – 71 years; 43% AA; 56% female; 26 % living in poverty) who had recently participated in Wave 3 of the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) parent study between the years 2009 and 2013. HANDLS is an ongoing longitudinal investigation of race- and SES-related health disparities among AA and White adults residing in 13 Baltimore neighborhoods pre-selected based on their likelihood to yield representative distributions of race and sex across a range of socioeconomic circumstances (31). The HANDLS study was approved by the Institutional Review Board of the National Institute of Environmental Health Sciences, NIH. All participants provided written informed consent. HANDLS study exclusions were 1) outside of the age range 30– 64 years; 2) currently pregnant; 3) within six months of receiving chemotherapy, radiation, or biological treatments for cancer; 4) AIDS diagnosis; 5) unable to provide informed consent due to mental incapacity resulting from drug or alcohol

intoxication, severe developmental disability, or dementia; 6) unable to provide at least five data measures on the mobile medical research vehicle; 7) without a verifiable address or valid government issued identification at time of consent.

HANDLS SCAN participants were recruited from the HANDLS parent study with the following additional exclusions: history of dementia, stroke, transient ischemic attack, carotid endarterectomy; MRI contraindications (e.g., indwelling ferromagnetic material); terminal illness (e.g., metastatic cancer, end-stage liver or pulmonary diseases); HIV positive status; or other neurological disorder (e.g., multiple sclerosis). Two hundred twenty-five participants were recruited for the present protocol. Of those, 41 did not complete neuroimaging for the following reasons (n = 12 deemed medically ineligible; n = 19 claustrophobia; n = 4 excessive girth; n = 6 equipment or scheduling issues). Of the 184 participants with neuroimaging, 9 were excluded for partial scans (due to motion artifact); 15 for incidental clinical findings, 8 because of a history of cocaine or opiate use, and 5 had missing data for key study variables. Complete data were available for 147 participants in this ongoing investigation.

All participants provided written informed consent and HIPAA consent approved by the University of Maryland, Baltimore's Institutional Review Board (and also approved by the University of Maryland, Baltimore County) for the HANDLS SCAN sub-study. They were then seen by a physician at the University of Maryland General Clinical Research Center for a brief medical evaluation to identify acute medical problems since their last HANDLS visit, review current medications, administer the MRI eligibility checklist, assess potential contraindications to the performance of HANDLS SCAN testing, and complete brief physical function assessment. They next underwent MRI acquisition in the Department of Diagnostic Radiology at the University of Maryland School of Medicine. Individuals received \$50 and coverage of transportation costs for their participation.

Measures

Demographic variables—Degree of SES risk was indexed as below median education (0 = 12 years or above; 1 = < 12 years) or income below 125% of the 2004 poverty line (\$18,850 per year for a family of 4) relative to family size and household income (0 = above poverty; 1 = below poverty). A composite index of SES was computed as a dichotomous variable, with low SES defined as below median education (< 12 years), and/or income below 125% of the federal poverty line. Participants were classified as high SES if they met neither of these criteria. Also considered were continuous and trichotomous education (0 = > 12 year; 1 = 12 years; 2 = < 12 years); and a trichotomy derived from poverty status and self-reported categories of annual income level (1=below poverty line; 2= above poverty and < \$50,000 income; 3 = above poverty and >= \$50,000 income). Self-identified race (0 = White; 1 =AA), sex (0 = female; 1 = male) and age (in years) were also assessed.

Clinical variables—Systolic and diastolic blood pressure were obtained by brachial artery auscultation following a 5 min rest in a seated position. One measure was obtained in each arm; those measures were averaged. Hypertension was defined by self-reported history, use

of antihypertensives, and/or resting systolic pressure of ≥ 140 mm Hg or diastolic pressure ≥ 90 mm Hg.

Blood samples were obtained from an antecubital vein following an overnight fast. Levels of total serum cholesterol, and fasting glucose were assessed by standard laboratory methods at Quest Diagnostics (Chantilly, VA; www.questdiagnostics.com). Diabetes was defined as fasting blood glucose ≥ 126 mg/dl, self-reported history, and/or use of relevant medications.

Height and weight were obtained using calibrated equipment, and body mass index was computed as weight divided by height-squared (kg/m^2). Waist circumference was measured to the nearest 0.1 cm with a flexible tape measure placed at the midpoint between the lower rib margin and the iliac crest at the end of exhalation during normal breathing.

Cardiovascular disease co-morbidity was coded as present = 1 or absent = 0 based on the presence of any of the following conditions: coronary artery disease, myocardial infarction, peripheral artery disease, or coronary artery bypass surgery. Conditions were documented by a HANDLS physician or nurse practitioner following a comprehensive physical examination and medical history.

Smoking status and alcohol use were dichotomized as 0 = never used and 1 = ever used (i.e., former and current users).

Magnetic Resonance Imaging—Cranial magnetic resonance images were obtained using a Siemens Tim-Trio 3.0 Tesla unit. Volumetric T1-weighted magnetization prepared rapid gradient echo imaging (MP-RAGE) images were obtained covering the entire brain in the sagittal plane at 1.2 mm thickness for a total of 160 slices (TR/TE/TI=2300/2.9/900 ms; FOV 25.6cm). These images were reformatted into axial sections to match the orientation of other anatomical images. Axial FLAIR images were obtained at a slice thickness of 3.0 mm with no gap in two concatenated groups of 24 slices each for a total of 48 slices (TR/TE/TI = 8000/71/2500ms, FOV=23cm). T2-weighted axial images were also obtained at 3mm thickness with no gap using turbo spin-echo acquisition from the same location as the FLAIR images (TR/TE = 6600/93ms, FOV=23cm; turbo factor 7).

Structural MRI scans were preprocessed by applying newly developed in-house techniques in the Section of Biomedical Image Analysis, Department of Radiology at the University of Pennsylvania. The processing steps first include removal of extra-cranial material on T1-weighted image (skull-stripping) using a multi-atlas registration based method that requires minimal manual correction (32), followed by joint bias correction and tissue segmentation into GM, WM, and cerebrospinal fluid using multiplicative intrinsic component optimization (33).

Preprocessed images were co-registered to a common brain atlas (template) using deformable registration via attribute matching and mutual-saliency weighting (34). Regional analysis of volumes examined in normalized space (RAVENS) maps (35) were generated to enable comparative analysis of tissue volumes on the common template space. The RAVENS approach has been extensively validated and applied in various studies (34–36). In

this investigation, GM, WM and ventricular RAVENS maps were generated, each quantifying the amount of respective tissue present in each brain region. The RAVENS maps were normalized by individual intracranial volume to adjust for global differences in intracranial size, down-sampled to $2 \times 2 \times 2 \text{ mm}$, and smoothed for incorporation of neighborhood information using an 8mm-diameter Gaussian filter.

A supervised learning based multimodal lesion segmentation technique, which uses a model trained on manually segmented lesions, was applied to segment ischemic lesions (37). The method involved co-registration of T1, T2, FLAIR and PD scans, histogram normalization to a template image, feature extraction, voxel wise label assignment and false-positive elimination. A new multi-atlas label fusion method was applied for segmenting the brain into a set of anatomical regions of interest (ROI; 38). Volumetric measurements for normal and abnormal (with lesion) tissue were calculated within each ROI, as well as in larger anatomical regions obtained by grouping single ROIs within a hierarchical representation.

Statistical Methods

A series of multiple regression analyses (SAS 9.4 PROC GLM) were conducted to assess potential interactive relations of race and the SES composite score to WM lesion volumes, total GM and WM volumes, and total brain volumes. Models included the interactions of race $\times$ SES in addition to the first order terms of race and SES. All analyses were adjusted for age and sex. Significant interactions were decomposed by contrasting all groups by t-test using the least squares means procedure in PROC GLM.

Subsequent sensitivity analyses were computed to assess respective influences of hypertension, diabetes, smoking, alcohol, total cholesterol, body mass index, and waist circumference as covariates. Analyses were also repeated with those having CVD diagnoses excluded. Lastly, a series of exploratory analyses examined interactive relations of race with several individual SES indicators including continuous education, dichotomous education, trichotomous education, poverty dichotomy, income trichotomy for each of the outcome measures.

RESULTS

Table 1 shows demographic data for the overall sample, and by SES. Study non-completers had significantly lower levels of education than study completers ($p = .006$), but were otherwise similar sociodemographically (see Table S1, Supplemental Digital Content 1). Scatterplots of age associations with each MRI outcome are displayed in Supplemental Digital Content Figures S1–S4.

As described in Table 2 and illustrated in Figure 1, significant interactions between race and the SES composite were observed for WM lesion volume ($p = .028$)¹, in addition to total brain volume ($p < .001$), GM volume ($p = .003$), and WM volume ($p < .001$). Significant group contrasts showed that AA participants with low SES exhibited significantly greater WM lesion volumes compared with white participants with low SES ($p < .05$). In addition, higher SES Whites had greater total brain, WM, and GM volumes than low SES Whites, low SES AAs, and higher SES AAs (p 's $< .05$).

Results of sensitivity analyses (see Table 3) indicated that the interactions of race and SES remained significant following adjustment for concurrent hypertension, diabetes, body mass index, waist circumference, total cholesterol, smoking, and alcohol use. Findings also remained significant following exclusion of those with CVD comorbidity.

As displayed in Table 4, findings from the exploratory multiple regression analyses in which several individual SES indicators were substituted for the SES composite score yielded significant race by SES indicator interactions for total brain volume, GM, and WM volumes across most measures. However, findings for WM lesion volume were not significant for any individual SES indicator.

DISCUSSION

Both self-identified AA race and lower SES have known associations with a plethora of poor clinical brain health outcomes such as stroke and dementia. However, race and SES are commonly conflated due to the substantially greater prevalence of lower SES among racial/ethnic minorities. To our knowledge, this is the first investigation to explicitly address potential differences in several global indices of MRI-assessed brain health outcomes according to race. Similar findings were noted for the ratio of WM lesion volume to total white matter volume. and SES. Partially consistent with our hypothesis, low SES AAs displayed greater WM pathology than low SES Whites, although no other groups differed significantly. Contrary to expectations, our findings also showed that higher SES Whites had greater mean GM, WM, and total brain volumes than low SES Whites, low SES AAs, and higher SES AAs. The above findings are notable in that greater WM lesions and lesser global brain volumes have demonstrated prognostic significance for poor clinical outcomes such as stroke, dementia, cognitive decline, disability, and/or mortality (14–21).

The present results suggest that the combination of low SES (indexed by either low education, living in poverty, or both) and self-identified AA race may indeed confer the greatest vulnerability for WM lesion burden. These findings remained significant following a series of adjustments for concurrent vascular risk factors commonly associated with WM pathology. WM lesions are typically acquired across the middle to later adult lifespan and are a form of subclinical brain pathology that may have particularly important predictive utility for future stroke (15, 19). WM lesions have also shown robust associations with dementia, cognitive performance and decline, and physical function (15–19), and thus may be a key risk marker for poor, clinically significant brain health outcomes.

Interpretation of findings related to global brain volumes is more complicated. From a clinical perspective, it has been suggested that brain volume may be a global indicator of brain health that is pertinent to risk for mortality and dementia (21). Thus, a combination of higher SES and self-identified White race may confer the greatest advantage for brain health. That higher SES did not similarly advantage AAs may, in part, reflect unique stressors encountered by such persons in their work and neighborhood contexts. For instance, higher SES AAs report greater levels of perceived discrimination than lower SES AAs (39). It has further been suggested that AAs disproportionately experience multiple additional dimensions of social disadvantage compared to Whites (30).

It is critical to note that there are no clear cut-offs regarding ranges of brain volume that are considered “normal” versus pathological. The largely overlapping distributions of brain volumes among all of our subgroups of participants suggest that our findings reflect variability within a normal range. Furthermore, MRI-assessed brain size is only one of multiple morphological and functional characteristics that are associated with functional status. Such global macrostructural measures do not capture key microstructural variables (e.g., synaptic density), and anatomical or functional connectivity (40). Thus, one must refrain from interpretation of these findings with respect to individual level functioning. Rather, just as incrementally higher levels of blood pressure, even within the normal range, confer greater relative risk for stroke, dementia, and other poor brain health outcomes, it is possible that a similar association applies to the normal spectrum of brain volume. If brain volume is indeed a robust indicator of overall brain health, this may translate into population level mortality significance.

Importantly, it is impossible to tease apart the degree to which the present findings regarding brain volumes are attributable to neurodevelopmental factors versus atrophic changes acquired over the life span. In that regard, a growing body of literature reveals that (a) AAs are more likely than Whites to have been exposed to early life disadvantage including low SES (41); (b) that children of lower SES show lesser brain volumes in multiple regions (e.g., prefrontal, hippocampal, amygdala) (42, 43); and (c) that lower childhood SES predicts lesser adult brain volumes (44–45). Thus, the lesser brain volumes in AAs and lower SES Whites may, in part, have roots in lower childhood SES. However, the presence of greater WM lesion volumes in low SES AA than low SES Whites, which were unlikely to be present in childhood, suggests possible cumulative impact of various life-long exposures that are more pronounced for AAs and those of lower SES. These may include multi-level variables such as income inequality, neighborhood stress, toxic environmental exposures, racial/ethnic discrimination, lesser access to healthy food and health care, poorer health habits, greater cardiovascular and metabolic risk factors, and greater overall burden of systemic disease. The “brain battering hypothesis” has previously proposed that these factors may explain race-related disparities in cognitive aging (5).

More generally, the present findings related to brain GM, WM, and total brain volumes are consistent with results of prior investigations showing that higher levels of education or income confer greater health benefits for Whites than AAs with respect to various systemic health outcomes such as coronary heart disease, subclinical atherosclerosis, and inflammatory markers (46, 47). However, an alternative explanation is that SES indicators differ systematically for AAs and Whites (28). For example, the average quality of education is better for Whites than for AAs. Furthermore, for each level of income, Whites have more wealth than AAs.

The present investigation has several notable strengths. First, the HANDLS parent study, from which the present participants were recruited, was explicitly designed to disentangle respective influences of self-identified AA and White race from poverty status. Thus, there was a diverse spectrum of SES within our AA and White samples. Second, our SES indicator combined two variables - poverty status and education - that have potent influences on multiple health endpoints. The current work also has several limitations. Specifically, the

findings do not generalize to ethnic minority populations other than AAs. In addition, results may be unique to the urban environment of Baltimore city. We did not have a full spectrum of SES indicators such as occupational status or wealth, nor did we have detailed characterization of annual income. It will be important to address both singular and cumulative influences of multiple SES indicators and determine whether they are linked to different mechanistic pathways to brain health. Future studies should also consider whether relations of race and SES to brain health outcomes are further moderated by age or sex, issues that we were underpowered to address herein. Relatedly, our sample size conferred limited statistical power to detect group differences that are small in magnitude, or to explore multi-level predictors of brain volumes within our study subgroups. We expect to examine the latter issue as our sample size increases. Lastly, we did not correct our group contrasts for multiple comparisons due to concerns about potential Type II error in this novel and largely exploratory study. Thus, the chance of Type I error remains a concern.

CONCLUSIONS

In sum, associations of self-identified race and SES with MRI-assessed outcomes were not uniform across all measures but rather subject to effect modification. Among persons of lower SES, AAs displayed a greater burden of WM lesions than Whites. It is possible that these findings may translate into a more pronounced risk for stroke and other poor clinical brain health outcomes in lower SES AAs, at least in part, reflecting the pernicious impact of poverty for AAs. With respect to global brain volumes, higher SES was associated with greater total brain, GM, and WM volumes in Whites but not AAs. These findings may, in part, reflect contextual stressors encountered by higher SES AAs, and may have important prognostic significance for future clinical brain health outcomes and mortality. Although MRI-based indices of brain health are only one in a complex and interrelated set of risk indicators relevant to clinical outcomes, further identification and reduction of such risk factors may assist in the elimination of race and SES related brain health disparities. It will be critical understand the multi-level mechanisms underlying these disparities, and to determine whether early interventions can alter trajectories toward poorer clinical brain health outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Acronyms

AA	African Americans
AIDS	acquired immunodeficiency syndrome

FLAIR	fluid attenuated inversion recovery
FOV	field of view
GM	gray matter
HANDLS	Healthy Aging in Neighborhoods of Diversity across the Lifespan
HIV	human immunodeficiency virus
MP-RAGE	magnetization prepared rapid gradient echo imaging
MRI	magnetic resonance imaging
NIA	National Institute on Aging
NIH	National Institutes of Health
PD	proton density
RAVENS	regional analysis of volumes examined in normalized space
ROI	region of interest
TE	echo time
TI	inversion time
TR	repetition time
WM	white matter

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Figure 1. Least-squares (adjusted) brain volume means according to race and SES. Error bars represent standard error of the mean. Means adjusted for age and sex.

Table 1

Demographic and health variables for the overall sample, and by SES

	Overall N=147	Low SES N = 63	High SES N = 84	
Variable	M (SD) or N (%)	M (SD) or N (%)	M (SD) or N (%)	p
Age (years)	52.1 (9.5)	49.9 (9.3)	53.7 (9.4)	0.016
Education (years)	12.9 (3.2)	11.0 (2.7)	14.2 (2.8)	<.001
Low Education (%)	37 (25.1)	37 (58.7)	0.0	
Below Poverty (%)	43 (29.3)	43 (68.3)	0.0	
Female (%)	82 (55.8)	42 (66.7)	41 (48.8)	0.042
African American (%)	85 (57.8)	32 (50.8)	30 (35.7)	0.13
Ever Smoker (%)	104 (70.7)	48 (76.2)	56 (66.7)	0.34
Ever Alcohol Use (%)	132 (89.8)	55 (87.3)	76 (90.5)	0.57
Hypertension (%)	67 (45.6)	32 (49.2)	31 (36.9)	0.12
Diabetes (%)	24 (16.3)	10 (15.9)	14 (16.7)	>.99
CVD comorbidities (%)	3 (2.0)	3 (4.8)	0.0	0.17
Body mass index (kg/m ²)	30.2 (6.7)	29.9 (7.2)	30.5 (36.4)	0.60
Waist circumference (cm)	103.5 (16.2)	101.8 (17.5)	104.7 (15.2)	0.30
Total cholesterol (mg/dl)	188.2 (43.4)	182.8 (38.5)	191.7 (46.1)	0.25
Total brain volume (cc)	975.6 (102.4)	948.1 (87.9)	996.1 (108.0)	0.005
Gray matter volume (cc)	517.6 (54.7)	504.3 (47.8)	527.5 (57.6)	0.011
White matter volume (cc)	458.0 (50.6)	443.8 (43.6)	468.6 (53.1)	0.003
White matter lesion volume (cc)	2.1 (1.8)	2.0 (2.0)	2.2 (1.6)	0.67

CVD comorbidities variable includes participants with any of the following: coronary artery disease, myocardial infarction, peripheral artery disease, and coronary artery bypass surgery.

t-tests were used to compare continuous variables; Fisher's exact test was used to compare categorical variables.

Table 2

Sociodemographic variable and brain volume measures (N = 147) *

Predictor	Outcome											
	Total Brain Volume			Gray Matter Volume			White Matter Volume			White Matter Lesion Volume		
	b	part η^2	p	b	part η^2	p	b	part η^2	p	b	part η^2	p
Age	-1.81	0.026	0.007	-1.34	0.050	<.001	-0.47	0.007	0.17	0.031	0.023	0.075
Sex	122.64	0.340	<.001	63.91	0.324	<.001	58.72	0.319	<.001	-0.052	0.0002	0.87
Race	-83.95	0.089	<.001	-47.47	0.100	<.001	-36.48	0.069	<.001	-0.417	0.007	0.31
SES	-64.73	0.053	<.001	-30.35	0.041	<.001	-34.38	0.061	<.001	-0.645	0.017	0.13
SES $\times$ Race	86.72	0.042	<.001	40.16	0.032	0.003	46.56	0.050	<.001	1.384	0.036	0.028

* Data reflect unstandardized regression coefficients (b), semipartial η^2 , and p-values

Sex coded as: 0=female, 1=male

Race coded as: 0=White, 1=African American

Table 3

The associations of the interaction between SES and race with brain volume measures adjusted for additional covariates; base model includes age, sex, race, SES, and race*SES interaction (N = 147) *

Covariate	Outcome											
	Total Brain Volume				Gray Matter Volume				White Matter Volume			
	b	part η^2	p	b	part η^2	p	b	part η^2	p	b	part η^2	p
Base Model	86.72	0.042	<.001	40.16	0.032	0.003	46.56	0.050	<.001	1.38	0.036	0.028
Alcohol Use	81.29	0.036	0.003	36.84	0.026	0.009	44.46	0.044	0.002	1.49	0.039	0.027
Cigarette Use	78.70	0.035	0.003	34.92	0.024	0.010	43.78	0.044	0.002	1.53	0.042	0.020
Hypertension	81.05	0.037	0.002	36.19	0.026	0.008	44.86	0.046	0.001	1.44	0.037	0.027
Diabetes	81.20	0.037	0.002	36.26	0.026	0.008	44.94	0.046	0.001	1.52	0.041	0.021
Waist circumference	81.36	0.036	0.002	35.84	0.025	0.010	45.52	0.046	0.001	1.42	0.036	0.030
Body Mass Index	80.09	0.036	0.002	35.84	0.026	0.009	44.25	0.045	0.001	1.48	0.040	0.023
Total cholesterol	80.20	0.034	0.003	35.63	0.024	0.012	44.57	0.043	0.002	1.59	0.044	0.018
CVD excluded	89.33	0.044	<.001	41.46	0.034	0.002	47.88	0.052	<.001	1.27	0.030	0.045

* Data reflect unstandardized regression coefficients (b), semipartial η^2 , and p-values illustrating associations of the SES $\times$ Race interaction with brain volume measures with adjustment for additional individual covariates, each in separate analyses.

CVD comorbidities variable includes participants with any of the following: coronary artery disease, myocardial infarction, peripheral artery disease, and coronary artery bypass surgery.

Table 4

Multivariate analyses of the interactions between Education and Income with Race as related to brain volume measures (N = 147)

Interaction Term	Outcome											
	Total Brain Volume			Gray Matter Volume			White Matter Volume			White Matter Lesion Volume		
	b	part η^2	p	b	part η^2	p	b	part η^2	p	b	part η^2	p
Continuous Education $\times$ Race	-9.42	0.016	0.041	-5.26	0.017	0.028	-4.16	0.013	0.085	-0.065	0.003	0.56
Trichotomous Education $\times$ Race												
Education 2 nd Tertile $\times$ Race	-62.56	0.014	0.051	-31.82	0.013	0.058	-30.74	0.014	0.068	-0.834	0.009	0.28
Education 3 rd Tertile $\times$ Race	-87.01	0.025	0.010	-43.30	0.022	0.014	-43.71	0.026	0.013	-0.884	0.009	0.28
Dichotomous Education $\times$ Race	73.54	0.024	0.012	37.00	0.021	0.015	35.53	0.024	0.017	0.949	0.013	0.18
Poverty Status $\times$ Race	58.33	0.016	0.038	29.00	0.014	0.049	29.33	0.017	0.046	0.689	0.007	0.32
Trichotomous Income $\times$ Race												
Income 2 nd Tertile $\times$ Race	-49.74	0.011	0.090	-23.64	0.009	0.12	-26.10	0.012	0.090	-0.497	0.004	0.49
Income 3 rd Tertile $\times$ Race	-94.71	0.017	0.034	-47.88	0.015	0.040	-46.84	0.017	0.045	0.758	0.018	0.12

Data reflect unstandardized regression coefficients (b), semipartial η^2 , and p-values illustrating associations between Education $\times$ Race and Income $\times$ Race interactions with brain volume measures for various education and income variables