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Ethnoracial Differences in the Clinical Characteristics of Alzheimer Disease at Initial Presentation at an Urban Alzheimer's Disease Center

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Abstract

Objective—To compare presentation of Alzheimer disease (AD) at the time of initial evaluation at a university specialty clinic across three ethnoracial groups in order to understand similarities and differences in the demographic, clinical, cognitive, psychiatric, and biologic features.

Design—Cross-sectional study.

Participants—A total of 1,341 self-identified African American, Latino (primarily of Caribbean origin), and white non-Hispanic ("WNH") subjects were recruited from primary care sites or by referral by primary care physicians.

Measurements—Demographic variables and age of onset of AD, as well as cognitive, functional, and mood impairments at the time of initial presentation and frequencies of apolipoprotein E genotypes, were compared across groups.

Results—Differences among ethnoracial groups were found for nearly all variables of interest. In particular, the largely immigrant Puerto Rican Latino group had an earlier age of onset of AD, more cognitive impairment, and greater severity of cognitive impairment at the time of initial evaluation in the setting of low average education and socioeconomic status. There was more depression in the Latinos compared with African Americans and WNHs. Greater severity of symptoms was not accounted for by a difference in lag time between onset of symptoms and initial evaluation. The apolipoprotein E-4 genotype was not associated with AD in the Latino cohort.

Conclusions—Minority groups in Philadelphia, especially Latinos, exhibit a more severe profile of AD at the time of presentation than WNHs. Important potential confounds need to be considered and future research comparing immigrant and nonimmigrant Latino groups will be necessary to elucidate the highly significant differences reported.

Keywords

Alzheimer disease; APOE; dementia; ethnoracial differences

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There are more than 5 million people with a clinical diagnosis of Alzheimer disease (AD) in the United States, a number expected to quadruple by 2050.[1] Considerable effort is being made to understand how genetic, developmental, environmental, medical, and psychiatric factors contribute to the pathophysiology of the disease and its clinical manifestations. Comparing AD in different ethnic and racial groups can help begin to address questions including how APOE allelic variants differentially influence disease expression in ethnoracial groups, to what degree differing rates of AD or ages of onset can be explained by factors such as socioeconomic status, education, or access to healthcare, and how comorbid conditions contribute to differences in rates and phenomenology of dementia.

Several epidemiologic studies have reported rates of dementia among African American, Latino, and white non-Hispanic ("WNH") groups.[2–5] While studies have not been fully consistent, a higher prevalence of AD has been reported among African Americans and Hispanics compared with WNHs.[3,4,6,7] One study concluded that when factors such as age, gender, years of education, and APOE status are considered, the higher rate of dementias of all types found in African American communities is eliminated.[1] In contrast, AD research with a major focus on Latinos of Caribbean background has reported a disparity in the rates of dementia among WNHs, African Americans, and Latinos.[3,4,7] In the North Manhattan Aging Project, the rate of developing AD is increased among African American and Caribbean Hispanic individuals compared with WNHs.[3,8] Some studies hypothesize that the higher prevalence of AD can be attributed to the higher rates of vascular disease and risk factors for vascular disease, lower rates of education or occupational attainment,[3] or differences in frequency of AD pathologic lesions.[9] Differences in the presence of the APOE-e4 allele and variations in clinical expression of AD in relation to APOE-e4 status among different ethnoracial groups are also well documented.[10–13]

In this study, we compared demographic, clinical, and genetic features among African Americans, Latinos, and WNHs who were diagnosed with AD or no cognitive impairment (NCI) at their initial evaluation at an NIA-supported Alzheimer Disease Center (ADC) in Philadelphia, PA.

METHODS

The University of Pennsylvania ADC works alongside a university-based primary care practice in West Philadelphia, the setting of both the university, and a lower income, primarily African American community. Patients are self-referred, referred by their primary care providers, or recruited through outreach efforts, including primary care settings in North Philadelphia, a neighborhood with the largest concentration of Latinos in the city.[14] Additional efforts to include minority participants were made through increased screenings at the university-based primary care clinic, as well as at a satellite primary care clinic in a community more heavily populated by members of the Latino community. Data from healthy older subjects from each ethnoracial group who were without cognitive, psychiatric, or neurologic illness were also assessed. These healthy comparison subjects had also been recruited and assessed identically to the patients with AD. Informed consent for the use of clinical, psychometric, and biomarker data was obtained from all persons, in accord with university institutional review board–approved protocols. Assessments included history, physical, and neurologic examinations conducted by clinicians experienced in the evaluation of dementia and review of neuroimaging and laboratory data. On the basis of these data, a consensus diagnosis was established using standardized clinical criteria. Between 1989 and January 2008, 1,341 people (1,128 AD, 213 healthy comparison subjects) met minimum criteria for inclusion in analyses. Inclusion criteria included a primary consensus diagnosis of AD or NCI, age greater than 50 years, known ethnoracial category, known age of onset if cognitively impaired, and completion of a standardized assessment at the time of initial

evaluation (Table 1). Of the 1,615 patients in the database meeting consensus criteria for AD, 487 (30%) were excluded because of missing or insufficient data. Similarly, of the 281 individuals with NCI, 68 (25%) were excluded for the same reasons. Analyses of basic demographic information including age, sex, and education revealed no differences between the participants included and those excluded from these analyses. Of the 213 healthy comparison cases, only 28 were related to an AD proband either as spouse or as first-degree relative. Findings from analyses including or excluding these first-degree AD and healthy relative pairs were similar and only results from the whole cohort are presented.

Clinical Assessments

All data presented here were collected at the initial visit to the Penn ADC.

Demographics and clinical characteristics—Variables included age at first visit, sex, ethnoracial group membership (per self-report), years of formal education, age of onset of cognitive impairment, interval between symptom onset and first visit, and documented history of diabetes mellitus, hypertension, hyperlipidemia, and/or cardiovascular disease (including cerebrovascular or peripheral vascular disease).

Cognition and dementia severity—Measures of cognition included the Mini-Mental State Exam[15] and a composite global cognition index, calculated as an average of *z*-transformed scores of six tests taken from the National Alzheimer's Disease Coordinating Center Unified Data Set[16] measuring language,[17] category fluency,[18] verbal learning and recall,[18] visuoconstruction,[18] and psychomotor speed of processing.[19] The Dementia Severity Rating Scale (DSRS)[20,21] served as a global rating of dementia severity. All tests were administered in the participants' preferred language, either English or Spanish.

Mood—Depression was assessed using the Geriatric Depression Scale (GDS).[22]

Genetic risk factors—DNA was collected via blood draw, extracted from peripheral leukocytes, and apolipoprotein E genotyping was performed.[23]

Data Analyses

We examined ethnoracial group differences in the continuous demographic variables, using linear regression. We assessed ethnoracial group differences in categorical demographic variables (eg, sex), using a Fisher's exact test because some frequencies in certain categories were low. The ethnoracial group differences were examined for age of onset, cognition, dementia, and depression variables by using linear regression after controlling for age, sex, and education. The ethnoracial group differences were examined for APOE by using logistic regression after controlling for age, sex, and education. For continuous outcomes, normality and equal variance assumptions were checked using graphs such as histograms and residual plots. To adjust for multiple comparisons and testing, statistical significance level α was set at 0.01. All statistical tests were two-sided. Data were analyzed with the JMP7 statistical software and SAS version 9.1 (SAS, Cary, NC).

RESULTS

Demographic and Clinical Characteristics

A consensus diagnosis of AD was established in 1,128 participants and NCI in 213. Demographic differences were detected among participants of specific ethnoracial groups. Latinos were younger than African Americans and WNHs when first evaluated (Table 1). Women were overrepresented in all ethnoracial groups. There were significant differences among the three ethnoracial groups for the years of formal education completed in both the

AD and NCI categories. The Latino group had fewer years of education than their WNH or African American counterparts, and African Americans had fewer years of education than WNHs.

Significant differences in age of onset of cognitive impairment were observed among ethnoracial groups (Table 2). Age of onset was younger in Latinos than in WNHs or African Americans, consistent with previous research that included a smaller portion of this sample.[24] We also considered whether there were differences between groups in the time interval between onset of cognitive symptoms and ADC evaluation, which might reflect disparity in access to healthcare. Contrary to expectations, we found no significant differences (Table 2).

In accord with known ethnoracial differences in the prevalence of metabolic and vascular risk factors,[25–27] we observed significant differences among the three groups in both the AD and NCI diagnostic categories (Table 1). Diabetes mellitus was especially common in Latinos and hypertension was more prevalent in African Americans. In comparing the presence of diabetes mellitus, hypertension, hyperlipidemia, and vascular disease within each ethnoracial group, we saw no significant differences in the prevalence of these disorders in AD compared with NCI and in regression models adjusting for these variables, we saw no effects of any of these disorders on age of onset of AD or the other cognitive and dementia variables presented below (data not shown).

Cognition

The MMSE was administered to all subjects and highly significant differences were noted among the groups (Table 2). Latinos had significantly lower scores than both WNHs and African Americans for both AD and NCI diagnoses, indicating lower levels of cognitive performance at first presentation to the ADC. African Americans with AD also scored lower than WNHs with AD at presentation, but there were no differences between healthy African Americans and WNHs.

As with the MMSE, significant differences in the composite global cognition index scores were seen among ethnoracial groups for both AD and NCI (Table 2). Post-hoc analyses in subjects with AD showed significantly lower global cognition index scores in both African Americans and Latinos than in WNHs. In the NCI category, Latinos had lower scores than both African Americans and WNHs. There was no difference between African Americans and WNHs.

Effect of Education on Ethnoracial Differences in Cognition

Education is an important variable in understanding cognition and often serves as a proxy for a host of other factors (eg, socioeconomic status, nutrition, healthcare) that affect health in general and cognitive health in particular.[28–31] For all subjects (AD and NCI), years of education were significantly correlated with MMSE ($r = 0.5$, $p < 0.0001$), the global cognition index ($r = 0.4$, $p < 0.0001$), DSRS ($r = 0.4$, $p < 0.0001$), and CDR ($r = -0.3$, $p < 0.0001$).

We examined the effect of education by comparing the parameter estimates of ethnoracial group to MMSE, global cognition index, DSRS and CDR sum of boxes in linear regression models with (Table 2) and without inclusion of education as an additional term. For participants with AD, there continued to be significant differences among groups after adjusting for years of education in the MMSE (along with age and sex, $F_{[2,1113]} = 10.8$, $p < 0.0001$) and the global cognition index ($F_{[2,1040]} = 10.6$, $p < 0.0001$). Adjustment for education attenuated the ethnoracial differences in DSRS in the AD group, although the overall effect of ethnoracial group remained significant ($F_{[2,1092]} = 4.3$, $p = 0.01$), as did the CDR sum of boxes ($F_{[2,679]} = 8.1$, $p = 0.0003$).

MMSE—For African Americans with AD in reference to Latinos, adjusting for education attenuated the effect of ethnoracial group for MMSE, reducing the parameter estimate from 2.8 to −0.3 and this was no longer significant ($t_{[1113]} = -0.5$, p = 0.64). For African Americans with AD in reference to WNHs with AD, adjusting for education in the regression models examining MMSE reduced the parameter estimate from −2.9 to −−1.8, but the effect of ethnoracial group remained significant ($t_{[1113]} = -4.4$, $p < 0.0001$). For Latinos compared with WNHs, adjusting for education attenuated the group effect for MMSE, greatly reducing the parameter estimate from −5.8 to −1.5; but this effect also remained significant ($t_{[1113]} = -2.7$, p = 0.007).

Global Cognition Index—For African Americans with AD in reference to Latinos, adjusting for education attenuated the effect of ethnoracial group for the global cognition index, reducing the parameter estimate from 0.3 to −0.2 and this was no longer significant ($t_{[1040]} = -1.8$, p = 0.07). For African Americans compared with WNHs adjusting for education reduced the parameter estimate from −0.5 to −0.4, but the effect of ethnoracial group remained significant ($t_{[1040]} = -4.6$, $p < 0.0001$). For Latinos compared with WNHs, adjusting for education reduced the parameter estimate from −0.8 to −0.1 and the effect was no longer significant ($t_{[1040]} = -1.2$, p = 0.25).

Adjustment for education in the NCI participants—We also assessed the effects of adjustment for education in the healthy comparison groups with NCI. (Table 2). First, there were no differences between African Americans and WNHs in MMSE and the global cognition index, without (data not shown) or with adjustment for education (Table 2). For MMSE in Latinos compared with African Americans, age adjustment slightly reduced the parameter estimate from −2.0 to −1.5, but the effect remained significant ($t_{[206]} = -4.4$. $p < 0.0001$). For the global cognition index in Latinos compared with African Americans, the parameter estimate was reduced from −0.8 to −0.5, and the education adjusted effect also remained significant ($t_{[203]} = -3.6$, p = 0.0004). Comparing Latinos with WNHs, education adjustment for MMSE reduced the parameter estimate from −2.0 to −1.2, which remained significant ($t_{[206]} = -3.8$, p = 0.0002). For the global cognition index, the parameter estimate was reduced from −1.04 to −0.55 and also remained significant ($t_{[203]} = -4.5$, $p < 0.0001$).

Dementia Severity

We used two scales, the DSRS and the CDR, to rate the severity of functional signs and symptoms of dementia. Scores on both differed among ethnoracial groups (Table 2), with comparisons of individual groups showing higher severity in African Americans compared with WNHs, and a significantly greater severity in Latinos compared with African Americans for CDR. There was a trend of higher severity in Latinos than in WNHs for both scores, although it did not reach statistical significance after adjustment of education. We also applied these scales in the NCI subjects with normal cognition and there was a modest difference among groups in the DSRS, with African Americans having fewer endorsed symptoms than either WNHs or Latinos, although all scores were very low.

Depression

Differences in depressive symptoms, as measured by the GDS, were observed among the three ethnoracial groups (Table 2). Post-hoc analyses found that the Latino group endorsed more depressive symptoms than the African American and WNH groups in both AD and NCI diagnostic categories, although there were no differences between African Americans and WNHs.

In consideration of possible differences in expressivity of emotion among groups, we also created more conservative categories of “major” depression and “low” depression as defined

by GDS scores of 7 or more and 3 or less, respectively. Significant between-group differences were found in AD ($\chi^2_{[509]} = 18.9$, $p < 0.0001$) and NCI ($\chi^2_{[142]} = 16.3$, $p < 0.0001$). Among those with AD, a significantly higher frequency of depression was found among Latinos (31%) than either African Americans (12%, Fisher's exact test, $p = 0.008$) or WNHs (8.2%, Fisher's exact test, $p < 0.0001$). Similarly, among those with normal cognition, Latinos had a higher frequency of depression (28%) than African Americans (0.0%, Fisher's exact test, $p = 0.01$) and WNHs (3.3%, Fisher's exact test, $p = 0.0007$).

Apolipoprotein E

Highly significant ethnoracial differences were found in the frequencies of the APOE-e4 allele in AD ($\chi^2_{[917]} = 24.1$, $p < 0.0001$) but not NCI ($\chi^2_{[173]} = 3.1$, $p = 0.20$). Post-hoc logistic regression analyses found that among African Americans, the APOE e4 allele was present in 67% of those with AD and 35% of those with NCI (OR: 3.8, $p = 0.001$) and among WNHs, the e4 allele was present in 59% of those with AD compared with 25% of those with NCI (OR: 4.3, $p < 0.0001$). However, in Latinos, the e4 allele was present in only 38% of those with AD and 41% of those with NCI (OR:= 0.87, $p = 0.75$). APOE genotype was also associated with age of onset.[10] In our subjects with AD, the presence of least one e4 allele was associated with younger age of onset in African Americans by an average of 3.9 years (76.0 [SD:7.9] versus 72.1 [7.1], $t_{[167]} = 3.3$, $p = 0.001$) and in WNHs by an average of 2.9 years (74.0 [9.3] versus 71.1 [7.7], $t_{[639]} = 4.3$, $p < 0.0001$), but not in Latinos (67.6 [10.3] versus 66.8 [9.4], $t_{[105]} = 0.41$, $p = 0.69$).

DISCUSSION

Differences among the three ethnoracial groups were found for nearly all study variables. In particular, the Latino group had younger age at onset of AD, more cognitive impairment and dementia at the time of initial presentation, and more depressive symptoms. African Americans had a slightly older age of onset than WNHs, but levels of dementia severity and cognitive impairment were intermediate between WNHs and Latinos. Also of interest is the apparent absence of apolipoprotein E e4 genotype as a risk factor for AD in our Latino cohort.

Many sociocultural factors contribute to general health and limit access to healthcare and could delay evaluation, which might skew the clinical profile at initial presentation. We considered if this delay contributed to differences in severity. We found no statistically significant differences between any of the groups in the interval between first onset of symptoms and evaluation in our ADC. This suggests that in this population, perhaps due to effective outreach and recruitment, there is equivalent access. Other studies explain greater symptom severity as being related to a lack of linguistically and culturally appropriate care providers.[32] It is also possible that differences in how members of different ethnoracial groups recognize cognitive decline and assign a time of onset could be responsible for delays in evaluation, thus explaining the greater severity of symptoms at first visit. In a study in which Latinos and WNHs were queried about their understanding of the cause of AD both groups reported "genes" as a major cause; however, Latinos were more likely to report "stress" as a cause, whereas WNHs reported an idea about "plaques" or "brain plasticity."[33]

Support for the hypothesis that lower education is associated with higher levels of cognitive impairment, increased risk of dementia, and more rapid cognitive decline is mixed, particularly when other variables including pathophysiology of the disease and symptom severity are taken into consideration.[30,34] In our sample, there were considerable differences in education between the groups, especially notable for low education levels in many Latinos. Education is considered a proxy for many factors (eg, poverty, nutrition, healthcare

access) that affect health as well as the expression of disease, particularly cognitive disorders. We attempted to control for education statistically by using years of education as a regressor in statistical analyses. Although these adjustments attenuated the ethnoracial differences, significant associations between ethnoracial group and cognition persisted. In some comparisons however (eg, Latinos versus WNHs for global cognition), statistical adjustment for education eliminated the ethnoracial group effect, even when the difference between groups was extremely large.

Another variable often associated with functional decline and poorer cognitive performance is depression.[25,35,36] Greater endorsement of depressive symptoms by Latinos in both AD and NCI groups could reflect higher distress levels in this largely poor, linguistically and culturally isolated immigrant group. Alternatively, it could indicate a culturally specific reflection of disease expression or a different degree of endogenous mood symptoms. Our data highlight the need to better understand how depression contributes to or is associated with cognitive impairment.

Finally, this study found even stronger evidence than the North Manhattan project[12] that APOE-e4 is not as highly associated with AD diagnosis in Caribbean Latinos as it is in African Americans and WNHs. In our study population, there was no association of APOE-e4 genotype with AD diagnosis or cognition in the Latino cohort, while this association was strong in the African American and WNH groups. Furthermore, and in contrast to recently reported findings,[37] we found no difference in age of onset in Latino APOE e4 carriers than noncarriers, unlike WNH or African American groups.

Normative data are lacking for language-translated study measures, posing a challenge to ADCs serving diverse communities in the United States.[38] This allows for potential confounds related to cultural bias and inadequate reference groups. One way to assess the contributions of cultural bias in the present study was to look for potential differences between the performances of people in different ethnoracial groups with NCI. For instance, we observed strong differences among the groups in the Boston naming task and digit symbol substitution task, but not the clock draw or 10-item word list learning and recall tests. Future studies using measures less susceptible to cultural bias will be important. Other studies have found that even when demographic variables such as age, education, gender and socioeconomic background are held constant disparities remain in scores on commonly used neuropsychological testing measures.[39]

There are important limitations to acknowledge in this study. As a descriptive study using a convenience sample, it cannot draw causal inferences about the variables under consideration and the onset and progression of dementia. Furthermore, the nature of our sample, which likely includes a degree of self-selection bias, may preclude generalization to the general population. Also, the Latino community in the United States is very diverse and our cohort principally included people of Puerto Rican heritage, resident of a culturally secluded and economical deprived area of Philadelphia. Therefore, caution must be heeded when attempting to generalize data from this study to other Latino communities or even other immigrant or nonimmigrant Puerto Rican communities. Furthermore, selection into ethnoracial group was determined by self-report. It can be argued that these classifications are socially constructed classifications and therefore subjective. Finally, the Latino individuals included in this study represent a fairly restricted range of education and socioeconomic level. The especially low levels of education reported by the Latino members of this cohort are noteworthy as a significant confound. They are also mostly immigrants and have had the added stress of relocation and acculturation. Finally, the recruitment methods for many of the Latino and African American participants involved targeted outreach efforts that were different from the way in which we recruited the WNH subjects

into the cohort. The African American and Latino participants were approached in a primary care health clinic in North Philadelphia or screened through other outreach endeavors at the West Philadelphia Primary Care office. Furthermore, studies comparing immigrant and nonimmigrant Latino communities will be important to identify the contributions of these many factors.

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TABLE 1

Demographic and Medical History Information

Variable	African American (N = 203)	Hispanic (N = 154)	White Non-Hispanic (N = 771)	Total (N = 1128)	Overall p Value	AA vs WNH, p Value	Hispanic vs WNH, p Value	AA vs Hispanic, p Value
Alzheimer disease participants								
Age at initial evaluation (years, STD)	76.6 (7.37)	71.3 (9.69)	75.7 (8.16)	75.3 (8.40)	$F_{[2,1125]} = 21.8054$, $p < 0.0001$	$t_{[1125]} = 1.3558$, $p = 0.1754$	$t_{[1125]} = -6.0975$, $p < 0.0001$	$t_{[1125]} = 6.0371$, $p < 0.0001$
Gender (% female)	149 (73.4)	97 (63.0)	485 (62.9)	731 (64.8)	p = 0.0164	p = 0.0049	p = 1.0000	p = 0.0383
Education (mean years, STD)	11.2 (3.68)	5.8 (4.35)	13.4 (3.55)	11.9 (4.50)	$F_{[2,1116]} = 272.3834$, $p < 0.0001$	$t_{[1116]} = -7.4612$, $p < 0.0001$	$t_{[1116]} = -23.1144$, $p < 0.0001$	$t_{[1116]} = 13.5138$, $p < 0.0001$
Diabetes (%)	32 (15.8)	31 (20.1)	41 (5.3)	104 (9.2)	p < 0.0001	p < 0.0001	p < 0.0001	p = 0.3269
Hypertension (%)	121 (59.6)	59 (38.3)	281 (36.4)	461 (40.9)	p < 0.0001	p < 0.0001	p = 0.7143	p = 0.0001
Hyperlipidemia (%)	14 (6.9)	9 (5.8)	62 (8.0)	85 (7.5)	p = 0.6473	p = 0.6609	p = 0.4100	p = 0.8285
Cardiovascular (%)	39 (19.2)	24 (15.6)	175 (22.7)	238 (21.1)	p = 0.1118	p = 0.3406	p = 0.0532	p = 0.4028
Variable	**African American (N = 33)**	**Hispanic (N = 44)**	**White Non-Hispanic (N = 136)**	**Total (N = 212)**	**Overall P value**	**AA vs WNH p Value**	**Hispanic vs WNH p Value**	**AA vs Hispanic p Value**
No cognitive impairment participants								
Gender (% female)	30 (90.9)	37 (84.1)	80 (58.8)	147 (69.0)	p < 0.0001	p = 0.0004	p = 0.0020	p = 0.5017
Education (mean years, STD)	13.8 (3.07)	9.1 (5.01)	16.4 (3.08)	14.5 (4.59)	$F_{[2,209]} = 70.5785$, $p < 0.0001$	$t_{[209]} = -3.6664$, $p = 0.0003$	$t_{[209]} = -11.8305$, $p < 0.0001$	$t_{[209]} = 5.8264$, $p < 0.0001$
Diabetes (%)	9 (27.3)	7 (15.9)	7 (5.1)	23 (10.8)	p = 0.0005	p = 0.0006	p = 0.0450	p = 0.2643
Hypertension (%)	21 (63.6)	21 (47.7)	59 (43.4)	101 (47.4)	p = 0.1191	p = 0.0512	p = 0.7273	p = 0.2475
Hyperlipidemia (%)	2 (6.1)	8 (18.2)	13 (9.6)	23 (10.8)	p = 0.2067	p = 0.7381	p = 0.1735	p = 0.1741
Cardiovascular (%)	3 (9.1)	6 (13.6)	27 (19.9)	36 (16.9)	p = 0.3291	p = 0.2049	p = 0.5014	p = 0.7245

Notes. p values are based on Fisher's exact test for categorical variables and linear regression for continuous variables.

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TABLE 2

Cognitive, Psychiatric, and Genetic Characteristics

Variable	African American (N = 203)	Latino (N = 154)	White Non-Hispanic (N = 771)	Total (N = 1128)	Overall p Value	AA vs WNH, p Value	Latino vs WNH, p Value	AA vs Latino, p Value
Participants with Alzheimer disease								
AGE of onset (STD)	73.5 (7.59)	68.0 (0.09)	72.4 (8.47)	72.0 (8.71)	$F_{[2,1114]} = 34.5557$, p < 0.0001	$\beta = 0.2070$, $t_{[1114]} = 0.3006$, p = 0.7638	$\beta = -7.23$, $t_{[1114]} = -7.9249$, p = < 0.0001	$\beta = 7.441$, $t_{[1114]} = 7.5480$, p < 0.0001
Interval between onset and initial presentation (STD)	3.1 (2.12)	3.3 (3.32)	3.4 (2.29)	3.3 (2.43)	$F_{[2,1093]} = 1.8804$, p = 0.1530	$\beta = --0.380$, $t_{[1093]} = -1.9278$, p = 0.0541	$\beta = -0.081$, $t_{[1093]} = -0.2994$, p = 0.7647	$\beta = -0.299$, $t_{[1093]} = -1.0347$, p = 0.3010
MMSE (STD)	17.6 (5.16)	15.1 (5.91)	20.7 (5.34)	19.3 (5.76)	$F_{[2,1113]} = 10.7667$, p < 0.0001	$\beta = -1.78$, $t_{[1113]} = -4.3549$, p < 0.0001	$\beta = -1.50$, $t_{[1113]} = -2.6819$, p = 0.0074	$\beta = -0.284$, $t_{[1113]} = -0.4738$, p = 0.6358
Global cognition index (STD)	−2.8 (0.98)	−3.0 (1.05)	−2.2 (1.04)	−2.4 (1.07)	$F_{[2,1040]} = 10.5919$, p < 0.0001	$\beta = -0.373$, $t_{[1040]} = -4.6011$, p < 0.0001	$\beta = -0.140$, $t_{[1040]} = -1.1637$, p = 0.2448	$\beta = -0.233$, $t_{[1040]} = -1.8009$, p = 0.0720
DSRS (STD)	18.0 (9.31)	19.0 (10.87)	14.6 (8.03)	15.8 (8.87)	$F_{[2,1092]} = 4.3395$, p = 0.0133	$\beta = 2.042$, $t_{[1092]} = 2.9460$, p = 0.0033	$\beta = 0.7320$, $t_{[1092]} = 0.7700$, p = 0.4415	$\beta = 1.310$, $t_{[1092]} = 1.2798$, p = 0.2009
CDR sum of boxes (STD)	7.4 (3.68)	7.1 (4.05)	5.6 (3.31)	6.1 (3.55)	$F_{[2,679]} = 8.1149$, p = 0.0003	$\beta = 1.420$, $t_{[679]} = 3.8994$, p = 0.0001	$\beta = -0.010$, $t_{[679]} = -0.0190$, p = 0.9848	$\beta = 1.429$, $t_{[679]} = 2.6125$, p = 0.0092
GDS (STD)	2.6 (2.89)	4.1 (2.73)	2.3 (2.40)	2.5 (2.59)	$F_{[2,606]} = 10.2883$, p < 0.0001	$\beta = 0.3820$, $t_{[606]} = 1.3329$, p = 0.1831	$\beta = 1.826$, $t_{[606]} = 4.5327$, p < 0.0001	$\beta = -1.44$, $t_{[606]} = -3.3534$, p = 0.0008
APOE e4 (% yes)	113 (55.7)	40 (26.0)	377 (48.9)	530 (57.8)	$\chi^2_{[2]}=21.8361$, p < 0.0001	OR: 1.53, $\chi^2_{[1]}=5.0682$, p = 0.0244	OR: 0.38, $\chi^2_{[1]}=12.1393$, p = 0.0005	OR: 3.99, $\chi^2_{[1]}=21.7623$, p < 0.0001
Variable	**African American (N = 33)**	**Latino (N = 44)**	**White Non-Hispanic (N = 136)**	**Total (N = 213)**	**Overall p Value**	**AA vs WNH, p Value**	**Latino vs WNH, p Value**	**AA vs Latino, p Value**
Participants with no cognitive impairment								
MMSE (STD)	29.2 (0.87)	27.4 (1.73)	29.0 (1.38)	28.7 (1.53)	$F_{[2,206]} = 10.1707$, p = 0.0001	$\beta = 0.3000$, $t_{[206]} = 1.1078$, p = 0.2693	$\beta = -1.21$, $t_{[206]} = -3.7792$, p = 0.0002	$\beta = 1.508$, $t_{[206]} = 4.3619$, p < 0.0001
Global cognition index (STD)	0.0 (0.42)	−0.6 (0.58)	0.2 (0.64)	0.0 (0.67)	$F_{[2,203]} = 10.4924$, p < 0.0001	$\beta = -0.078$, $t_{[203]} = -0.7643$, p = 0.4456	$\beta = -0.551$, $t_{[203]} = -4.5244$, p < 0.0001	$\beta = 0.4730$, $t_{[203]} = 3.5990$, p = 0.0004
DSRS (STD)	0.4 (1.12)	1.6 (1.88)	1.4 (2.06)	1.3 (1.95)	$F_{[2,148]} = 2.7690$, p =0.0660	$\beta = -0.764$, $t_{[148]} = -1.6638$, p = 0.0983	$\beta = 0.6230$, $t_{[148]} = 1.1646$, p = 0.2460	$\beta = -1.39$, $t_{[148]} = -2.2891$, p = 0.0235
CDR Sum of boxes (STD)	0.1 (0.21)	0.1 (0.34)	0.3 (1.48)	0.2 (1.19)	$F_{[2,148]} = 1.5337$, p = 0.2192	$\beta = -0.380$, $t_{[148]} = -1.3612$, p = 0.1755	$\beta = -0.534$, $t_{[148]} = -1.5286$, p = 0.1285	$\beta = 0.1530$, $t_{[148]} = 0.4291$, p = 0.6685

Variable	African American (N = 203)	Latino (N = 154)	White Non-Hispanic (N = 771)	Total (N = 1128)	Overall p Value	AA vs WNH, p Value	Latino vs WNH, p Value	AA vs Latino, p Value
GDS (STD)	1.2 (1.75)	3.6 (3.33)	1.3 (1.96)	1.7 (2.44)	$F_{[2,150]} = 5.7955$, p = 0.0038	$\beta = -0.089$, $t_{[150]} = -0.1692$, p = 0.8659	$\beta = 2.007$, $t_{[150]} = 3.0799$, p = 0.0025	$\beta = -2.10$, $t_{[150]} = -3.1563$, p = 0.0019
APOE e4% (STD)	10 (30.3%)	11 (25.0%)	29 (21.3%)	50 (28.9%)	$\chi^2_{[2]}=3.0019$, p = 0.2229	OR: 2.05, $\chi^2_{[1]}=2.1711$, p = 0.1406	OR: 2.27, $\chi^2_{[1]}=1.8971$, p = 0.1684	OR:= 0.91, $\chi^2_{[1]}=0.0252$, p = 0.8738

Notes. p values are based on logistic regression for categorical variables and linear regression model for continuous variables after adjustment for age, sex, and education.