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# Ethnoracial differences in Lewy body diseases with cognitive impairment

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## Abstract

**Background—**Increasing research focuses on ethnic differences in Alzheimer's disease, but such efforts in other neurodegenerative dementias are lacking. Currently, data on the ethnic profile of cognitive impaired persons with Lewy body disease (LBD) is limited, despite Lewy body dementia being the second most common neurodegenerative dementia.

**Objective—**The study aimed to investigate presenting characteristics among ethnoracially diverse individuals with cognitive impairment secondary to LBD using the National Alzheimer's Coordinating Center database.

**Methods—**Participants self-identified as African American, Hispanic, or White. We used Kruskal-Wallis and Pearson $\chi 2$ analyses to investigate group differences in presenting characteristics and linear regression to compare neuropsychological test performance.

**Results—**Presentation age was similar between groups (median 74–75 years). Compared to Whites (n=1782), African Americans (n=130) and Hispanics (n=122) were more likely to be female and single, have less educational attainment, report more cardiovascular risk factors, describe less medication use, and perform worse on select cognitive tests. Hispanics reported more depressive symptoms.

**Conclusion—**Cohorts differences highlight the need for population-based LBD studies with racial-ethnic diversity. Culturally-sensitive neuropsychological tests are needed to determine whether observed differences relate to cultural, social, testing, or disease-related factors. More research is needed regarding how social and biological factors impact LBD care among diverse populations.

### Keywords

Lewy body disease; dementia; mild cognitive impairment; demography; ethnic groups; Alzheimer's disease centers

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## INTRODUCTION

Alzheimer's disease (AD) research highlights ethnoracial differences in prevalence rates [1–3], neuropathological disease characteristics [4–6], progression [7, 8], and treatment [3, 9]. Data suggests that African Americans are twice as likely to have AD compared to White non-Hispanics, while Hispanics are 1.5 times more likely to have AD than White non-Hispanics [3]. Further, Hispanics and African Americans often show a more severe AD profile compared to White non-Hispanics, including earlier age of onset and greater severity of cognitive impairment at initial presentation [10]. Efforts to explain observed differences suggest that differences can be attenuated, or even eliminated, when adjusting for factors that may influence and/or interact with ethnicity in AD risk and presentation [1, 3, 11]. Research shows that disparities in the prevalence, presentation, and disease progression among Hispanics and African American can be impacted by biological risk factors, such as genetics (e.g. APOE ε4 status) and greater cardiovascular disease, as well as sociodemographic variables and social determinants of health (e.g. lower educational attainment, less access to quality of care, etc) [2, 3, 12].

Several organizations, including the National Institute of Health [13] and the Alzheimer's Association International Society to Advance Alzheimer's Research and Treatment [12], identify increasing diversity as an immediate need for advancing AD/AD related dementia (ADRD) research. Although understanding of how ethnoracial factors interfaces with AD expression and treatment is incomplete, such knowledge exceeds data on ethnoracial influence in other neurodegenerative diseases. The most recent ADRD Summit acknowledged that data regarding the epidemiology and clinical course of ADRD subtypes (e.g. Lewy body diseases, frontotemporal dementia) are limited within underrepresented minorities [14].

Lewy Body Diseases (LBDs) are neurodegenerative diseases associated with alpha-synuclein protein aggregations. LBDs with cognitive impairment include dementia with Lewy bodies (DLB), a prodromal DLB state with mild cognitive impairment (MCI; DLB-MCI), Parkinson's Disease (PD) with MCI, and PD with dementia (PDD). Together, DLB and PDD are termed Lewy body dementia. As a single entity, Lewy body dementia is the second most common neurodegenerative dementia after AD [15]. The associated MCI populations are sometimes termed "pre-dementia LBD" [14].

The ADRD Summit 2019 Report [14] identifies the need for observational studies with diverse populations across ADRDs and increased availability and use of culturally- and linguistically-valid assessment tools in aging populations. Additionally, the Report promotes diverse longitudinal clinical cohorts and clinical trial populations in Lewy body dementia. Currently, LBD ethnic profile data is scant, limiting understanding of risk factors, etiology, prognosis, and other variables that may affect disease expression among diverse populations. In view of national priorities targeting increased diversity in LBD cohorts, we aimed to identify the presentation of diverse ethnoracial groups diagnosed with cognitive impairment suspected secondary to LBD using data from the National Alzheimer's Coordinating Center (NACC) Uniform Data Set (UDS) (NACC-UDS).

# METHODS

## Setting

Data for the current study were obtained from the NACC-UDS, a database composed of longitudinal data that comprised of over 35,000 participants at the time we received our file from Alzheimer's Disease Center (ADC) programs in the United States [16]. Study approval was obtained from the Institutional Review Board for each participating NACC site. NACC recruitment and data collection has been described previously [16–19]. Data for the current analysis represents data collected from September 2005 to the December 2018 data freeze.

## Participants

Inclusion criteria for the current analysis were: (1) clinical diagnosis of MCI or dementia, (2) presumptive etiologic diagnosis of LBD as the primary or contributing cause of cognitive impairment at the participant's most recent study visit, (3) age 45 years or older, and (4) participant self-identification as Hispanic, White, or African American. We extracted data from the first visit associated with a diagnosis of MCI or dementia. For NACC participants who were cognitively healthy at baseline and progressed to MCI/dementia, we utilized data from the first visit where a diagnosis of MCI or dementia (for those who went from cognitively normal to dementia, skipping a MCI diagnosis) was entered.

## Demographics & Measures

Participants in the current analysis participated in data collection through NACC-UDS versions 1–3. Participants were classified as White non-Hispanic, African American non-Hispanic, or Hispanic (associated with White or African-American race) based on self-report. Other collected demographics included: age at visit, sex, educational attainment, visit year, marital status, and additional lifestyle factors (e.g. living situation, type of residence, and independence status). Extracted medical data included self/informant-report of recent or remote history of diabetes, stroke, hypertension, and hypercholesterolemia and current antidepressant, antipsychotic, antiparkinson agent, cholinesterase inhibitor, and memantine use.

Clinical variables of interest included functional, psychiatric, and cognitive data. The Functional Activities Questionnaire (FAQ) [20] informed functional status; the Unified Parkinson Disease Rating Scale (UPDRS) motor subscale [21] assessed parkinsonism. The Neuropsychiatric Inventory- Questionnaire (NPI-Q) [22] and the Geriatric Depression Scale (GDS) [23] assessed psychiatric symptoms. Cognitive measures included the CDR® Dementia Staging Instrument [24], Mini-Mental State Examination (MMSE) [25], Boston Naming Test (BNT) [26], Wechsler Memory Scale-Revised Logical Memory subtest [27], Wechsler Adult Intelligence Scale – R (WAIS-R) Digit Span Forward and Backward subtests [28], WAIS-R Digit Symbol Coding subtest [28], Trail Making Test (TMT) A & B [29], and Animals and Vegetable Fluency [30].

## Statistical analyses

Continuous and categorical variables were summarized by medians and proportions, respectively. Kruskal-Wallis and Pearson $\chi 2$ analyses assessed between-group differences

for continuous and categorical variables. Significant main effects were followed up by pairwise comparisons using Bonferroni-correction. We conducted simultaneous linear regressions to explore associations of ethnoracial differences on neuropsychological performance adjusting for significant group demographic differences, including education (continuous), sex (female/male; reference group: male), and cardiovascular disease risk factors (hypertension, diabetes; reference group: no reported history of risk factor). To account for proportion differences in diagnostic severity (MCI vs. dementia) between group, we used the CDR® Dementia Staging Instrument global score (continuous, 0–3) to control for cognitive severity. Neuropsychological scores were modeled as unstandardized continuous variables. Ethnoracial status was dummy-coded prior to analysis and "White" was the comparison group. Multicollinearity was checked for all study variables by using correlations, tolerances, and variance inflation factors (VIF). Correlations were sufficiently low ($r < .31$), tolerance scores were greater than .8, and VIF scores were below 2. Statistical significance was set at $p \leq 0.0001$ to adjust for multiple comparisons. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 25.0 (Armonk, NY).

## RESULTS

Data from 1782 Whites, 130 African Americans, and 122 Hispanics were available for analysis. Hispanics with dementia self-identified from Mexican origin (29%), followed by Puerto Rican (15%), Cuban (14%), Dominican (12%), South American (11%), and Central American (8%) origin; 11% of the sample did not specify country of origin. Hispanics with MCI identified themselves from Mexican (26%), Cuban (23%), Dominican (16%), Central American (13%), South American (13%), Puerto Rican (3%), and unspecified (6%) origin. Seventy two percent of Hispanics (N=88) identified Spanish as their primary language, compared to 99% of African Americans and Whites who identified English as their primary language. Fifty percent of (N=62) Hispanics were administered the Spanish version of the UDS neuropsychological test battery. Of the total sample, 65% had a dementia diagnosis. Table 1 includes breakdown of cognitive severity for each ethnoracial group. ADC site stratified by ethnoracial group is available in Supplemental Table 1.

### Sample characteristics

The average age at presentation with a cognitive diagnosis was similar across cohorts (Table 1). African Americans and Hispanics were more likely to be female than White participants. African Americans and Hispanics also had less educational attainment, were more likely to be single (widowed or divorced/separated), and were more likely to live alone than White participants, though functional status was not statistically different between groups. African-Americans and Hispanics reported a history of diabetes and hypertension more commonly than White participants.

### Psychiatric symptoms

Ethnoracial groups did not significantly differ across individual symptoms or total symptom severity measured by the NPI-Q (Table 2). However, Hispanics self-reported more depressive symptoms on the GDS compared to African Americans and Whites. Additionally,

a larger percentage of Hispanics fell within the range of suggestive depression (GDS score of ≥6) than Whites and African Americans.

### Cognition

After adjusting for sex, education, hypertension, diabetes, and cognitive severity, Hispanics performed worse on Digit Span Forward and the BNT, whereas African Americans performed worse on Digit Symbol, TMT A, BNT, and animal fluency compared to Whites (Table 3). Full linear regression model results are available in Supplemental Table 2. Stratifying the sample using clinical diagnosis (MCI or dementia), rather than using the CDR® Dementia Staging Instrument to account for proportion differences in clinical diagnosis categories, did not meaningful change outcomes, except for having less precision relating to smaller sample sizes (Supplemental Tables 3, 4).

### Other symptoms

No ethnoracial differences were evidence across everyday functional abilities (FAQ) (median scores: White-16, African-American-17, Hispanic-17; $p$=.468), or degree of motor symptoms (UPDRS) (median scores: White-16, African-American-15, Hispanic-14; $p$=.845).

### Treatment

The only treatment difference observed between groups was that Whites were significantly more likely to report current use of an antiparkinson agent (Table 4).

## DISCUSSION

The purpose of this study was to describe how ethnically diverse NACC participants diagnosed with LBD compare sociodemographically and clinically. Compared to Whites, African Americans and Hispanics were more likely to be female and single, and have less educational attainment, higher self-reported rates of diabetes and hypertension, less Parkinson medication use, and worse performance on attention, naming and executive functioning measures. In addition, Hispanics were more likely to report depressive symptoms. Group differences were present between Hispanics and African Americans, but more frequently observed between White and ethnic minority participants.

Sex differences varied dramatically by ethnoracial background, with females representing 60% of the African American NACC LBD cohort, 41% of the Hispanic cohort, and only 26% of the White cohort. A less prominent sex difference was reported in a NACC pathology study including non-Hispanic African American and White cohorts with various dementias (64.5% and 42.5% female, respectively) [31]. However, our finding is in contrast to prior data reporting that LBD is more common in men in both clinical [32] and pathological [15, 33, 34] cohorts, largely reflecting White populations [15, 32, 34]. Differences in our findings compared to LBD literature could reflect our inclusion of all cases where LBD was suspected as a primary or contributing cause of cognitive impairment. Females are 1.5 times more likely than males to have a documented diagnosis of probable AD [35] and African-Americans and Hispanics are more frequently diagnosed with AD [3],

potentially skewing our study demographics. Gender differences in dementia are partly attributed to the longer life expectancy of women in the United States (81.3 vs 76.3 years), differences which vary by racial-ethnic background (White 81.5 vs 76.7 years; African-American 78.5 vs 72.3 years; Hispanic 85.4 vs 80.2 years) [36]. Particularly given the average age of participants in our cohorts, survival differences alone are an unlikely explanation for the marked gender differences between groups, but may contribute. Additionally, these findings may reflect that NACC is a convenience rather than population-based sample, and thus differences may occur for non-biologic reasons. Existing research shows no clear link between subject or caregiver gender and willingness to enroll an individual in research,[37, 38] but this area of research remains largely unexplored. While the observed sex differences may be partly explainable by these or other factors, the observed differences emphasize the need for diverse enrollment in LBD cohorts and for population-based studies performed in diverse communities [14].

While our study did not investigate co-informant type, we found that Hispanic and African American participants were more likely to be single and living alone. This is similar to a study of dementia caregiving across racial-ethnic groups, where the frequency of spouse caregivers was highest for White participants. Adult children and other family were more common caregivers for Mexican American and African American participants [39]. Our observation of lower educational attainment in Hispanic and African American populations compared to White populations is consistent with prior clinical and population-based studies [10, 40]. Similarly, our finding that Hispanics and African Americans have higher reported rates of hypertension and diabetes compared to White non-Hispanics matches prior reports [1, 41, 42].

Hispanics had slightly higher GDS scores and more individuals who fell within the suggestive depression range using GDS cutoff score >5. While qualitative inspection of data revealed that the degree of difference was relatively small, these results are consistent with prior data that Hispanics with AD experience more depressive and neuropsychiatric symptoms than White and African American populations [43]. We found no difference in NPI-Q total and subscale scores between groups, but a prior NACC analysis performed using data from Hispanic and White non-Hispanic participants with normal cognition or dementia suggested that NPI-Q scores may not be comparable across these ethnic groups due to lack of ethnic-group scalar invariance [44]. NPI-Q scores were higher in Hispanic than White participants in that study and authors theorized differences could relate to ethnocultural differences (including perceived stigma, informant reporting), differences in education, and/or language barriers [44].

Cohort differences in antidepressant use were not statistically significant, though the point frequency for antidepressant use was much lower for African-Americans than the other cohorts. Antipsychotic medication use was also similar between cohorts, in contrast to a prior NACC study where Hispanics had a greater use of antipsychotic medications compared to White non-Hispanics [45]. White participants had a statistically higher frequency of antiparkinsonian medication use and a non-significant higher use of medications for dementia in our cohorts. These results are consistent with studies suggesting that ethnic

minorities, particularly African Americans, are less likely to receive anti-dementia pharmacological treatment than White patients [9, 46–48].

Hispanics and African Americans had poorer performance in naming, attention, and executive functioning compared to White participants after adjusting for sex, educational attainment, hypertension, diabetes, and cognitive severity. Whether these differences relate to test characteristics, analysis, biopsychosocial factors, comorbidities, or differences in LBD expression is uncertain. Research suggests that adjusting for years of education has limited utility in correcting for educational inequities across ethnic groups [11]. One test with notably lower performance across Hispanics and African Americans compared to Whites was the BNT. BNT performance is significantly influenced by culture [49], a construct for which race and ethnicity are often a proxy. Based on our findings that African Americans and Hispanics reported more frequent cerebrovascular risk factors, it is plausible that cerebrovascular disease played a role in performance on processing speed and executive function measures such as TMT and Digit Span backwards [50]. However, including reported history of hypertension and diabetes did not eliminate the effect of ethnicity on performance. While reported history of cardiovascular disease risk factors may be a weak proxy of cerebrovascular burden, inclusion of neuroimaging parameters was outside the scope of the current analysis. It is also possible that poorer performance on attention and executive functioning among African Americans and Hispanics is indicative of the limits on statistical adjustments for disease severity since the ethnic minority groups had more individuals presenting at dementia vs MCI stages, though the differences between groups were non-significant (see Table 1).

To our knowledge, this study is the first to examine differences in ethnically diverse individuals diagnosed with LBD. Study results emphasize how sociodemographic variables may influence the clinical presentation of LBD among ethnically diverse populations. However, this is a largely descriptive study based on NACC data. NACC represents a convenience sample from 39 ADCs serving various populations and with different local foci, rather than a representative sample of dementia cases in the U.S. Location, specialty, gender, race/ethnicity, and other factors may affect recruitment patterns. Ethnic minorities account for only 10–11% of the total sample, hindering statistical analysis and the generalizability of the study findings. In addition, 72% of Hispanics reported Spanish as their primary language, but only 50% of Hispanics completed the neuropsychological evaluation in Spanish. It is possible that this affected performance on some neuropsychological tasks, particularly relating to fluency. We used categorized groups based off self-reported ethnoracial status, yet race/ethnicity can be a proxy for biological and social variables, like acculturation, genetic markers, and geographic factors. Categorizing Hispanics as a homogenous group does not account for the various countries of origin represented that might better characterize homogenous subgroups. Additionally, diagnosing LBD is a challenge that can require multiple physicians over multiple office visits to render an accurate diagnosis [51] and we did not limit our study to pathologically-confirmed cases. Lastly, although NACC published a LBD module in 2017, the data from that module remain unavailable for analysis due to a small sample size. Similarly, Version 3 of the UDS is currently in use, which adopted four new non-proprietary neuropsychological tests to replace similar tests that were previously used under licensing restrictions. Although the four new

measures can be equated to their previous counterparts using equipercentile equating [52], we did not compare cognitive performance on measures implemented in Version 3 since only 14% Whites, 6% African Americans, and 19% Hispanics had these data available. As available data increases, additional analyses can explore group differences using more sensitive or appropriate variables for LBD cohorts.

Despite limitations, this study is highly relevant to ADCs, whose research is critical for advancing the detection, diagnosis, treatment, and prevention of neurodegenerative diseases. Differences between cohorts, such as sex, highlight the need for population-based studies in LBD engaging individuals from diverse racial-ethnic backgrounds [14]. Future research should also explore whether observed sex difference relate to NACC recruitment, social/cultural differences between different racial/ethnic backgrounds, or LBD differences between racial-ethnic groups. Research is needed regarding optimal culturally-sensitive neuropsychological testing and whether observed differences relate to cultural, social, testing, or disease-related factors. Overall, there is a need for more research in investigating how social and biological factors interplay, ultimately impacting the detection, evaluation, and treatment of LBD among ethnically diverse populations.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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**Table 1.**

Sociodemographic characteristics by ethnoracial background

| | White | African American | Hispanic | *P* value |
|---|---|---|---|---|
| Female, % | 26[a] | 60[b] | 41[c] | <0.0001 |
| Age at visit, median (range) | 74(46–100) | 75(52–91) | 75(50–93) | .553 |
| Visit year, median (range) | 2008(2005–2018) | 2009(2005–2018) | 2009(2005–2018) | .203 |
| Education, % | | | | <0.0001 |
| <12 | 4[a] | 22[b] | 41[c] | |
| 12 | 18 | 22 | 22 | |
| >12 | 77[a] | 56[b] | 37[c] | |
| Cognitive status, % | | | | .005 |
| mild cognitive impairment | 36.5 | 28 | 25 | |
| dementia | 63.5 | 72 | 75 | |
| Marital status, % | | | | <0.0001 |
| Married | 80[a] | 43[b] | 57[b] | |
| Widowed | 11[a] | 34[b] | 18[a] | |
| Divorced/separated | 4[a] | 16[b] | 16[b] | |
| Other or unknown | 5 | 7 | 9 | |
| Living situation, % | | | | <0.0001 |
| alone | 9[a] | 23[b] | 12[a] | |
| with spouse | 78[a] | 41[b] | 56[b] | |
| with relative or friend | 5[a] | 30[b] | 22[b] | |
| group | 4 | 3 | 7 | |
| other | 5 | 2 | 4 | |
| Type of residence, % | | | | 0.848 |
| single | 90 | 90 | 88 | |
| other | 10 | 10 | 12 | |
| Independence status, % | | | | .005 |
| independent | 31 | 30 | 24 | |
| assistance w/ IADLS | 40 | 28 | 37 | |
| assistance w/ ADLS | 20 | 30 | 23 | |
| completely dependent | 10 | 13 | 17 | |
| Health history, % | | | | |
| diabetes | 10[a] | 26[b] | 21[b] | <0.0001 |
| stroke | 5 | 10 | 10 | .008 |
| hypertension | 48[a] | 78[b] | 62[c] | <0.0001 |
| hypercholesterolemia | 49 | 54 | 56 | .245 |

*Note:* White education n = 1773, Hispanic education n = 121, White diabetes n = 1754, African American diabetes n = 129, Hispanic diabetes n = 118, White stroke n = 1743, African American stroke n = 129, Hispanic stroke n = 118, White hypertension n = 1744, Hispanic hypertension n = 117, White hypercholesterolemia n = 1735, African American hypercholesterolemia n = 127, Hispanic hypercholesterolemia n = 117, White living situation n = 1775, African American living situation n = 129, Hispanic living situation n = 121, White type of residence n = 1749, African American type of residence n = 125, Hispanic type of residence n = 119, White independence status n = 1765, African American independence status n = 128, Hispanic independence status n = 119

*Note*: Proportions with different alphabetic superscript are statistically significant using post hoc Bonferroni-correction. Identical alphabetic superscript means they did not significantly differ

**Table 2.**

Psychiatric symptoms by ethnoracial background

| | White n = 1722 | African American n=126 | Hispanic n = 116 | *p* value |
|---|---|---|---|---|
| NPI-Q total severity, *median (range)* | 3(0–28) | 4(0–25) | 5(0–20) | .002 |
| NPI-Q =>4, *%* | 46 | 50 | 58 | .037 |
| NPI symptom | | | | |
| Delusion, *%* | 18 | 25 | 28 | .016 |
| Hallucination, *%* | 25 | 29 | 35 | .027 |
| Agitation, *%* | 31 | 37 | 38 | .111 |
| Depression, *%* | 44 | 44 | 60 | .006 |
| Anxiety, *%* | 42 | 33 | 55 | .002 |
| Euphoria, *%* | 4 | 6 | 6 | .543 |
| Apathy, *%* | 46 | 44 | 44 | .848 |
| Disinhibition, *%* | 18 | 18 | 16 | .927 |
| Irritability, *%* | 39 | 37 | 40 | .866 |
| Motor, % | 19 | 27 | 26 | .035 |
| Sleep, % | 47 | 48 | 51 | .735 |
| Appetite and eating, *%* | 28 | 33 | 35 | .095 |
| GDS total, *median (range)* | 3(0–15)[a] | 2(0–15)[a] | 4(0–15)[b] | <0.0001 |
| GDS => 6; % | 21[a] | 19[a] | 39[b] | <0.0001 |

Abbreviation: NPI, Neuropsychiatric Inventory- Questionnaire; GDS, Geriatric Depression Scale.

White NPI-Q total n = 1708; White delusions, hallucination, euphoria n = 1720; White agitation, disinhibition, irritability, appetite n = 1721; White sleep n = 1719; White GDS n = 1548; African American GDS n = 108, Hispanic GDS n = 108

*Note*: Proportions with different alphabetic superscript are statistically significant using post hoc Bonferroni-correction. Identical alphabetic superscript means they did not significantly differ

**Table 3.**

Neuropsychological scores by ethnoracial background; adjusted for sex, cognitive severity, and education

| Measure | Group* | *B* | SE *B* | β | *p* value |
|---|---|---|---|---|---|
| MMSE | African American | −.751 | .406 | −.030 | .065 |
| | Hispanic | −.301 | .451 | −.011 | .504 |
| DSF | African American | −.245 | .229 | −.025 | .284 |
| | Hispanic | −1.588 | .246 | −.157 | <0.0001 |
| DSB | African American | −.630 | .208 | −.072 | .003 |
| | Hispanic | −.501 | .222 | −.055 | .024 |
| Digit Symbol | African American | −5.384 | 1.396 | −.097 | <0.0001 |
| | Hispanic | −2.442 | 1.518 | −.042 | .108 |
| TMTA | African American | 16.430 | 3.928 | ..094 | <.0001 |
| | Hispanic | 9.954 | 4.293 | .053 | .021 |
| TMTB | African American | 28.955 | 11.142 | .073 | .009 |
| | Hispanic | 21.908 | 11.897 | .051 | .066 |
| BNT | African American | −5.633 | .531 | −.217 | <.0001 |
| | Hispanic | −3.799 | .576 | −.139 | <.0001 |
| Animals fluency | African American | −2.068 | .510 | −.084 | <0.0001 |
| | Hispanic | −.781 | .543 | −.031 | .151 |
| Vegetable fluency | African American | −.518 | .359 | −.030 | .149 |
| | Hispanic | −1.045 | .385 | −.059 | .007 |
| Immediate memory | African American | −.204 | .434 | −.011 | .639 |
| | Hispanic | −.074 | .467 | −.004 | .874 |
| Delayed memory | African American | −.341 | .435 | −.019 | .433 |
| | Hispanic | −.195 | .473 | −.010 | .680 |

Abbreviation: MMSE, Mini-Mental Status Exam; DSF, Digit Span Forward; DSB, Digit Span Backward; TMTA, Trail Making Test A, TMTB, Trail Making Test B; BNT, Boston Naming Test

*White cohort was inputted as the referent group

**Table 4.**

Medication use by ethnoracial background

| | White (n =1769) | African American (n=127) | Hispanic (n=120) | *p* value |
|---|---|---|---|---|
| Antidepressant | 45 | 28 | 46 | .001 |
| Antipsychotic | 14 | 17 | 21 | .121 |
| Alzheimer's medications | 55 | 41 | 46 | .001 |
| Antiparkinson agent | 34[a] | 18[b] | 23[b] | <0.0001 |

*Note*: Proportions with different alphabetic superscript are statistically significant using post hoc Bonferroni-correction. Identical alphabetic superscript means they did not significantly differ