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Race/ethnic differences in Alzheimer disease survival in US Alzheimer Disease Centers

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Abstract

Objective—Survival after Alzheimer disease (AD) is poorly understood for patients of diverse race/ethnic groups. We examined whether nonwhite AD patients (African American, Latino, Asian, American Indian) had different rates of survival compared with white AD patients.

Methods—The National Alzheimer's Coordinating Center (NACC) cataloged data from more than 30 AD centers in the United States from 1984 to 2005. Patients aged 65 years or older with a diagnosis of possible/probable AD were included (n = 30,916). Survival was calculated using Cox proportional hazards models with a primary outcome of time to death. Secondary outcomes of this study were neuropathologic characteristics on an autopsied subsample (n = 3,017).

Results—The 30,916 AD patients in the NACC were followed up for 2.4 ± 2.9 years (mean age 77.6 ± 6.5 years; 65% women; 19% nonwhite [12% African American, 4% Latino, 1.5% Asian, 0.5% American Indian, and 1% other]). Median survival was 4.8 years. African American and Latino AD patients had a lower adjusted hazard for mortality compared with white AD patients (African American hazard ratio [HR] 0.85, 95% CI 0.74 to 0.96; Latino HR 0.57, 95% CI 0.46 to 0.69). Asians and American Indians had similar adjusted hazards for mortality compared with white AD patients ($p > 0.10$ for both). African American and Latino autopsied AD patients had similar neuropathologic characteristics compared with white AD patients with similar clinical severity.

Conclusions—African American and Latino Alzheimer disease (AD) patients may have longer survival compared with white AD patients. Neuropathology findings did not explain survival differences by race. Determining the underlying factors behind survival differences may lead to longer survival for AD patients of all race/ethnic backgrounds.

Estimates of survival after a diagnosis of dementia vary, ranging from 3 to 9 years.^{1–6} Survival has been estimated from both the time of first symptom onset and the time of diagnosis. Median survival time has been shown to decrease with age and is longer for women.^{5,7}

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It is estimated that more than 3 million dementia patients in the United States will be of nonwhite race/ethnicity (African American, Latino, Asian or Pacific Islander, and American Indian) by 2050.⁸ A growing literature suggests that some nonwhite older adults have higher rates of dementia.^{9,10} Because the populations of nonwhite race/ethnic groups are increasing and there is potentially a higher incidence of Alzheimer disease (AD) in these groups, estimating survival for African Americans, Latinos, Asians, and American Indians will be useful for health planning, to identify differences in disease course, and to explore new approaches to treatment.

The few studies on race/ethnicity in dementia patients and their rates of all-cause mortality differ in their outcomes. Some studies suggest that Latino and African American AD patients have similar survival compared with white AD patients.^{4,6,7} However, the National Follow Back Survey¹¹ and the Cardiovascular Health Study¹² suggest that African American patients of the same age have shorter survival times. Few previous studies have determined estimates for Asian or American Indian participants. In addition, few previous epidemiologic studies have had neuropathology data to help understand whether persons of nonwhite race/ethnicity have less advanced neuropathology compared with white AD patients.

The National Alzheimer's Coordinating Center (NACC) gathered data from more than 30 AD Centers (ADCs) across the United States. Using these data, we estimated the differential survival rates of nonwhite AD patients (African American, Latino, Asian, and American Indian) compared with white AD patients. Further, we evaluated potential determinants of these differences, such as the influence of demographic characteristics or clinical severity on these findings. Last, we determined whether neuropathologic data exhibit any differences upon autopsy for patients of nonwhite race/ethnicity with similar clinical severity.

Methods

Setting

The NACC data sets are collected from more than 30 ADCs throughout the United States. A full description is provided in two previous reports.^{13,14} ADCs are National Institute on Aging (NIA)-funded centers, many of which are university-affiliated memory disorders clinics in urban settings; patients are enrolled via self-referral or via referral from a community health care provider. Some centers had specific mandates to develop outreach programs to people of nonwhite race/ethnicity. These centers have been collecting data on their patients using a data form from 1984 to 2005. The NACC has been a central repository since 1999. The current study using NACC data was approved by the institutional review board at the University of California, San Francisco.

Patients

This study included patients who were aged at least 65 years, had a diagnosis of possible or probable AD according to the National Institute of Neurological Diseases and Stroke-Alzheimer's Disease and Related Disorders Association criteria or its equivalent,¹⁵ and were seen at the ADC between January 1, 1984, and April 1, 2005. A total of 31,060 participants were selected. Decedents who were missing a date of death ($n = 144$) were excluded, yielding a final analytic cohort of 30,916 AD patients (99.5%).

Measures

Patient race and ethnicity were identified through patient self-report to the clinical staff at the ADCs. Race was categorized into the following groups: white, African American, Asian/Pacific Islander, American Indian, or other race. Ethnicity was categorized into Hispanic and

non-Hispanic. For the purposes of this study, we categorized individuals into the following mutually exclusive race/ethnic categories: Latino (all races), and non-Latino individuals who were white (white), African American, Asian/Pacific Islander (Asian), American Indian, and people of other/unknown race. Other predictors assessed included demographic characteristics such as age, sex, education, ADC site, marital status at the time of the ADC evaluation, and residence at the time of the ADC evaluation (living in a skilled nursing facility or assisted living in comparison with residing at home or in a retirement community). In addition to patient demographics, cognitive function test performance was evaluated at the patient's first evaluation at an ADC using the Mini-Mental State Examination (MMSE).¹⁶ Patients or their caregivers were asked to report when the dementia symptoms first began.

Vital status and neuropathology

Vital status was recorded for AD patients on an ongoing basis using follow-up procedures at each ADC. For a subset of patients (n = 3,017), neuropathology data were obtained upon autopsy. Braak staging was assessed for these patients; it includes seven pathologic stages (0 through 6) based on the evolution and spread of neurofibrillary pathology throughout the brain.¹⁷ For the purposes of this study, we compared AD patients with advanced Braak and Braak (Stages 5 and 6 compared with Stages 0 through 4). We also examined hallmark characteristics of AD (moderate or frequent neuritic or diffuse plaques) in comparison with those patients who had no plaques or who only had sparse plaques. This dichotomization is based on previous work with the NACC database.¹⁸ In addition, we examined neurovascular pathology as defined by presence of ischemic, hemorrhagic, vascular, or atherosclerotic vascular pathology (of the circle of Willis). We also examined the presence of an ApoE-ε4 allele in patients who were seen at autopsy.

Statistical analyses

First, patient characteristics and clinical markers were evaluated for the entire group of NACC patients and separately by race/ethnicity using χ^2 tests for dichotomous and *t* tests for continuous characteristics. Patient characteristics were also described for those who died under the period of observation and the subset of patients who had an autopsy performed. These calculations were also conducted separately for each race/ethnic group. Then, a Cox proportional hazards model was calculated using time of first evaluation at an ADC to the time of mortality as the outcome with separate indicator variables for African American, Latino, Asian, and American Indian AD patients contrasted to white AD patients as the reference; this model will be referred to as the base model. For the base model and each subsequent Cox proportional hazard model built, model assumptions were assessed.

To assess potential confounders of the relationship between race/ethnicity and survival, the following factors were added to the base model: age (as the time scale), sex, education, ADC (as a clustering variable), marital status at the time of ADC evaluation, and living situation at the time of ADC evaluation. To assess whether markers of disease severity such as the patients' MMSE score at first ADC evaluation or age when dementia symptoms began to mediate the association between ethnicity and survival, these terms were also added sequentially to the model. To assess potential effect modification based on age cohort or different levels of cognitive impairment, a series of subgroup analyses were conducted. For example, Cox proportional hazards models were calculated separately for ages above and below the median (77 years), for each sex group (men and women separately), and for MMSE scores in tertiles. For each subgroup analysis, a race/ethnicity interaction was tested. Information gathered at the ADCs did not include whether the patients were newly diagnosed AD patients. Therefore, patients could have been diagnosed by their primary care physician or by a community neurologist. If this resulted in a referral bias whereby nonwhite

patients with more progressed disease were referred, our findings may exhibit a false survival advantage. Therefore, we conducted a sensitivity analysis where we repeated our main analyses with only nonwhite and white AD patients whose dementia symptoms had started within 5 years of their first evaluation at the ADC.

For the patients who underwent autopsy, we examined the association between race/ethnicity and presence of advanced Braak and Braak staging (Stages 5 and 6), brain vascular (neurovascular) pathology, presence of frequent or moderate diffuse and neuritic plaques, and frequency of the ApoE- ϵ 4 allele. For each neuropathology outcome, we calculated odds ratios (ORs) to examine the association between with race/ethnicity and neuropathology for patients with similar MMSE scores (defined by tertile) at their first evaluation at an ADC. A test for trend across the MMSE scores was calculated separately for each race/ethnic group.

Results

Sample characteristics

Of the 30,916 AD patients from the ADCs, 12% were African American, 4% were Latino, 1.5% were Asian, 0.5% were American Indian, and 1% were of other race. The remaining 81% were white. The patients were 77.7 ± 6.4 years old on average, 65% were women, and they had on average 12.4 ± 3.9 years of education. Nonwhite patients were more often women and had lower average levels of education than white patients ($p < 0.001$ for each comparison). They were less often married and less likely to live in a skilled nursing or assisted living facility. On average, nonwhite patients had lower average MMSE scores at their first evaluation (table 1).

Overall, 38.5% died within the period of observation. Patients who died were older, were less often women, were similar in educational level, and had lower mean MMSE scores at their first evaluation ($p < 0.001$ for all). One quarter of the patients who died had an autopsy performed ($n = 3017$, 25.4%). Patients seen at autopsy were different from the other deceased patients in that they were more often white, were slightly younger, were less often women, and had a higher average educational level.

Mortality

Mortality varied by race/ethnicity. Forty-one percent of white patients died; all nonwhite groups had lower mortality (African Americans 30%, Latino 21%, Asian 17%, American Indian 38%; table 2). White patients were followed up on average 2.6 ± 2.9 years since ADC diagnosis, African American patients 1.9 ± 2.5 years, Latino patients 1.9 ± 2.4 years, Asian patients 1.2 ± 1.9 years, and American Indian patients 2.0 ± 2.3 years. Overall survival since their initial ADC visit was a median of 4.8 years, with a standard error of 0.03 years. After adjusting for age (as the time scale), sex, educational level in years, the ADC site where the patient was seen (as a clustering variable), their living situation, their MMSE score at first evaluation, and the age when dementia symptoms first began, both African Americans and Latino AD patients had a lower hazard for mortality compared with white patients (African American hazard ratio [HR] 0.85, 95% CI 0.74 to 0.96; Latino HR 0.57, 95% CI 0.46 to 0.69). Asian and American Indian AD patients had a similar hazard for mortality as white patients (Asian HR 1.06, 95% CI 0.81 to 1.39; American Indian HR 1.13, 95% CI 0.91 to 1.40). These results were not altered appreciably when the set of patients was limited to the AD patients who had their first symptoms of dementia within 5 years before their first evaluation (African American HR 0.91, 95% CI 0.77 to 1.1; Latino HR 0.53, 95% CI 0.44 to 0.64; Asian HR 0.86, 95% CI 0.61 to 1.2; American Indian HR 1.2, 95% CI 0.94 to 1.5).

To examine whether effect modification was present by age cohort, sex, or level of cognitive test score at first examination, three stratified Cox proportional hazards models were

calculated after adjustment (table 3). Both African American AD patients and Latino AD patients who were aged 77 years or older had a lower hazard for mortality than their younger counterparts (African American $p = 0.001$; Latino $p = 0.002$). Latino women patients had a stronger mortality advantage than Latino men ($p = 0.03$). Last, mortality advantage for both Latino and African American groups was stronger for patients with lower MMSE scores; however, this association did not reach significance at $\alpha = 0.05$.

Neuropathology

We examined whether nonwhite race/ethnicity was associated with advanced Braak staging, neurovascular pathology and more frequent dense and diffuse neuritic plaque formation, and ApoE- $\epsilon 4$ allele frequency. Percentages are shown in table 4. Both Latino and African American AD patients were equally likely to have advanced Braak and Braak staging (Stages 5 and 6) as compared with white AD patients (OR 1.5, 95% CI 0.9 to 2.5; OR 0.8, 95% CI 0.5 to 1.4). Latino patients were more likely to have neurovascular pathology than white AD patients (OR 2.1, 95% CI 1.0 to 4.4), whereas African American AD patients had similar rates of neurovascular pathology on autopsy (OR 1.0, 95% CI 0.6 to 1.6) compared with white AD patients. Latino and African American patients had similar frequency of neuritic plaques, diffuse plaques, and ApoE- $\epsilon 4$ allele presence compared with white patients ($p > 0.10$). We also examined these neuropathologic characteristics for race/ethnic groups stratified by tertile MMSE at their first evaluation (table 4). After stratification by MMSE, white, African American and Latino groups were similar on neuropathologic outcomes for most comparisons. Compared with patients with MMSE scores over 21, the OR for having more advanced Braak and Braak staging was 2.5 (95% CI 2.0 to 3.2) for patients with MMSE scores between 0 and 15 and 1.8 (95% CI 1.4 to 2.3) for patients with scores between 16 and 21. This relationship was slightly stronger for nonwhite AD patients: in nonwhites, the OR for MMSE scores between 0 and 15 compared with those with scores over 21 (OR 3.6, 95% CI 1.3 to 10.4); MMSE scores between 16 and 21 compared with those over 21 (OR 2.1, 95% CI 0.7 to 6.3). However, the interaction between race and MMSE score did not reach significance ($p = 0.49$). Lower MMSE score was associated with increased odds of advanced Braak and Braak staging for both nonwhites and whites (p trend in nonwhites 0.01; p for trend in whites < 0.001).

Discussion

We found that African American and Latino patients with AD had longer survival compared with their white AD counterparts. These findings were not explained by patient age, sex, educational level, marital status, living situation, and cognitive test score measured at their first evaluation at the ADC. The current study included a sample of more than 30,000 patients from more than 30 NIA funded centers across the United States. Because of the clinic-based nature of the NACC, this study should be viewed as a large case series and is not necessarily representative of the population at large. Thus, results should be viewed as generalizable to ADCs and may not be representative of all AD patients in the United States. Race and ethnic classification were detailed enough to perform analysis by race/ethnic groups. In addition, this study incorporated data on more than 3,000 autopsied individuals.

Our results were in accord with one previous study that reported longer survival estimates for Latino AD patients compared with white AD patients seen at the Baylor ADC.⁷ Although the authors suggested that their finding might be due to chance given the small number of Latino patients available for analysis, our results using data from more than 30 ADCs across the United States suggest a survival advantage in Latino AD patients. Similarly, another study of the onset of AD in Latinos suggested that this patient group had a mean age of symptom onset 6.8 years earlier than white patients with AD, after adjustment.

¹⁹ Earlier onset, coupled with our finding of a survival advantage in Latinos, suggests that Latino individuals may live longer with AD symptoms than their white counterparts.

Additionally, our results were also in accord with a study of approximately 400 AD patients who found a trend toward survival benefit for African Americans compared with whites in their base model and more survival benefit for African Americans compared with whites whose MMSE scores were greater than 8 points (HR 0.6).⁶ Interestingly, the authors interpreted these findings as there being “no survival difference by ethnicity.” Our similar results suggest that African American AD patients have a survival benefit over their white counterparts. Our results differ from an analysis of the Cardiovascular Health Study¹² and the National Follow Back Survey,¹¹ possibly because these data were population based. To our knowledge, this study is one of the few that has examined Asian and American Indian subgroups. Their survival was not different from that of white AD patients.

This study has as a unique feature that neuropathology data were available for a subset of the patients who underwent autopsy. The autopsied individuals included a select group of patients who were seen at the ADC and who consented to donate their brains; thus, they also can be viewed as a large case series who are not necessarily representative of all AD patients. However, in a post hoc analysis, we examined race/ethnic differences in survival for autopsied patients and nonautopsied patients, and the findings were similar. If Latino or African American populations had less extensive neuropathology at the time of death, this may suggest that these patient groups were dying of non-AD processes. However, our data suggest that Braak and Braak staging and diffuse plaque frequency was similar among whites, African Americans, and Latino patients. Our result was similar to findings from a previous neuropathologic study of 80 African Americans and white AD patients.²⁰ It is of interest that in our study, neurovascular pathology and neuritic plaque frequency were more common in Latinos compared with whites. However, upon closer examination of the data, these findings seem to be in part confounded by MMSE scores. Latinos had lower MMSE scores on average and lower MMSE score was associated with increased neurovascular pathology and neuritic plaque frequency. Future studies including neuropathologic data should confirm these findings and explore the relationship between these neuropathologic markers and clinical diagnoses for these patients.

Whereas our topic is of interest for characterizing the burden of dementia in ethnically diverse segments of the population, it may also be of interest to those who want to understand survival after an onset of dementia in general. One study suggests that after taking into account length bias (i.e., failing to consider persons with rapidly progressive illness who die before their inclusion in the study), the duration of survival after a diagnosis of dementia is as short as 3.3 years. If our results hold, and minority older adults have a survival advantage, identifying the reasons behind this survival advantage may lead to improved survival for all persons with AD. If AD patients who are nonwhite live longer with dementia, and their autopsy data suggest similar brain pathology, this may suggest that nonwhite AD patients have a “buffer” against dying with this disease.

The mechanism that leads to the survival advantage for certain nonwhite groups with AD is not clear. Possible reasons for this difference may be due to underlying genetic or cultural factors in nonwhite groups. In terms of genetics, the most frequently studied is the ApoE-ε4 allele, which was not more frequent in nonwhites compared with white patients in the NACC sample of autopsied individuals. However, there may be genes other than ApoE that mediate these differences in survival such as the angiotensin-1 converting enzyme, sortilin-related receptor SORL1, or the low-density lipoprotein receptor. Another possibility is that genetic admixture may play a role in race-based differences found in our study. Future

studies should examine any survival differences according to genetic admixture and compare them with survival differences found for self-report of race.

Possible cultural differences explaining the survival advantage could be differences in living environment or patients' level of comorbidity. One set of cultural differences between Latino and African American AD patients is the lower rate of nursing home placement or, conversely, that white patients may have more access to nursing homes. This cultural difference may lead to a survival benefit overall because nursing home placement is associated with increased mortality.²¹ However, our crude adjustment for living situation did not change the findings. Second, educational quality may account for the differences seen in our study, because previous work suggests that adjustment for educational attainment using years of education may not account for differences in the meaning or quality of education received by the race/ethnic groups in our study.²² Duration of stay in the United States may also influence survival in the AD patients who were foreign born.²³ Our study included Latino and Asian patients. We recognize that these broad categories include people from several countries of origin and that the rates of disease and duration of AD may vary according to these countries of origin. Latinos who were seen at an ADC could have had more supportive networks (i.e., social support from extended family), and this could have influenced their survival. Another possibility is that the families of Latino and African American patients may have made decisions to preserve life in AD patients with advanced comorbidity profiles compared with their white counterparts. Perhaps the most likely reason for our findings is that African American and Latino patients who were seen at the ADCs may have had a different comorbidity profile than the white AD patients seen at the ADCs or that they possibly received more aggressive treatment of their comorbidities at advanced stages of dementia; either of these could explain the longer survival. Our data were limited in that we could not examine several comorbidities known to be associated with both AD diagnosis and death, such as delusions and hallucinations,²⁴ falls and gait problems,⁶ and aphasia.²⁵ Whereas we could not address this directly, we could examine the duration of dementia symptoms, which may be correlated with comorbidity. Our sensitivity analysis, limited to patients with shorter dementia symptoms, did not alter the results appreciably.

Some limitations of our study deserve comment. First, these data are not truly population based and thus may not be generalizable to the population of patients with AD. Second, a few notable selection biases might be present. The information submitted from the ADCs to the central NACC repository was supposed to go through uniform procedures for data acquisition and entry; however, the efforts to maintain uniformity are relatively recent, and the acceptance of these procedures may vary across the ADCs. In fact, most of the data were collected before the MDS were established, and it may have not been possible for ADCs to apply uniform procedures for data coding and data entry for data that had already been acquired. One specific example where this may have been problematic is in the reporting of the age when dementia symptoms began, because recall bias may have been present. In addition, some ADCs had a mandate to outreach to specific race/ethnic groups; thus, these patients might be different from patients who self-referred or who were referred by a community physician for these ADCs. Hypothetically, it is also possible that referrals to the ADCs are biased toward whites with shorter survival and nonwhite patients with longer survival. For example, a selection bias may have been present where sicker Latino or African American patients with memory loss were more frequently cared for at home without referral to an ADC; therefore, the pool of individuals arriving at the AD would exhibit longer survival. Another possibility is that differential dropout accounts for the lower hazard for mortality in African American and Latino patients. As information on dropout was not systematically collected at the ADCs, the effects of differential dropout could not be examined in this study. We are also unable to address whether AD patients were seen at the

ADCs at comparable starting points in the biologic disease process (i.e., amyloid load). That is, the Latino patients could have been earlier in their disease process for any given MMSE score. Future studies could address some of these concerns this by using longitudinal assessment of change in cognitive score with particular attention to selective dropout to address whether survival differences are related to disease progression or other factors (comorbidity, supportive care). Another main limitation is the use of the MMSE to characterize cognitive status in this study. Previous literature suggests that the MMSE is highly language and education dependent. Thus, the characterization of cognitive status for this diverse group of patients may be suboptimal. Future studies should use tests with less differential-item and differential-test functioning across language, education, and race/ethnic groups to correctly classify cognitive status.

Regardless of the underlying mechanism for the survival advantage of African American and Latino AD patients, this finding may have implications for health care planning for diverse AD patients, particularly for the large group of AD patients seen at ADCs across the United States. In summary, our findings indicate that there may be a survival advantage for persons of Latino and African American race/ethnicity who have AD. Reasons for this survival difference are unknown. Further evaluation of culture and race/ethnicity specific factors including caregiving relationships, patient preferences, and treatment for persons with AD may shed light on the underlying cause of this important difference.

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Glossary

AD	Alzheimer disease
ADC	Alzheimer's Disease Center
HR	hazard ratio
MMSE	Mini-Mental State Examination
NACC	National Alzheimer's Coordinating Center
NIA	National Institute on Aging

OR odds ratio

Table 1
Characteristics of patients with either possible or probable Alzheimer disease seen at the 30 Alzheimer's Disease Centers between 1984 and 2005 (n = 30,916)*

Characteristic [†]	White (n = 25,160)	African American (n = 3,563)	Latino (n = 1,301)	Asian (n = 451)	American Indian (n = 162)
Mean age (SD), y	77.6 (6.4)	78.1 (6.7)	77.0 (6.6)	79.3 (6.2)	78.2 (7.0)
% Women	63	74	72	67	68
Mean educational level (SD), y	13.0 (3.5)	10.7 (3.9)	7.4 (4.8)	11.3 (5.6)	11.1 (3.5)
% Not married at the ADC evaluation	43	68	63	54	55
% Residing in a skilled nursing facility or in assisted living	23	14	9	9	25
Mean Mini-Mental State Examination score at first evaluation (SD)	18.1 (7.2)	15.3 (6.7)	14.9 (7.0)	16.2 (7.1)	16.5 (7.2)

* $p < 0.001$ for all characteristics when white Alzheimer disease (AD) patients compared with nonwhite AD patients.

[†] 279 AD patients who were identified as "other" race were not included.

ADC = Alzheimer's Disease Center.

Table 2
Race/Ethnicity and mortality, follow-up time, and adjusted hazard for mortality

Race/Ethnicity *	% Mortality	Mean (SD) duration of follow-up, y	Mortality hazard ratio (95% CI), adjusted model *
African American	30	1.9 ± 2.5	0.85 (0.74–0.96)
Latino	21	1.9 ± 2.4	0.57 (0.46–0.69)
Asian	17	1.2 ± 1.9	1.06 (0.81–1.39)
American Indian	38	2.0 ± 2.3	1.13 (0.91–1.40)
White	41	2.6 ± 2.9	1.00 (Reference)

* Adjusted for demographics (age as the time scale, sex, educational level, Alzheimer's Disease Center [ADC] site as a clustering variable, marital status at the ADC evaluation, living situation at the ADC evaluation), Mini-Mental State Examination Score, and age at first dementia symptom.

Table 3
Hazards of mortality for African American and Latino patients and subgroups according to age cohort, sex, and MMSE score

Subgroup	Race/Ethnicity	% Mortality	Hazard ratio*	95% CI	Interaction <i>p</i> value
Age < 77 y	African American	28	0.95	(0.80–1.1)	0.001
	Latino	19	0.59	(0.48–0.72)	0.002
	White	39			
Age ≥ 77 y	African American	31	0.82	(0.72–0.92)	
	Latino	23	0.57	(0.47–0.69)	
	White	43			
Men	African American	35	0.84	(0.74–0.95)	0.89
	Latino	27	0.72	(0.54–0.96)	0.03
	White	47			
Women	African American	28	0.85	(0.72–1.01)	
	Latino	18	0.50	(0.40–0.63)	
	White	38			
MMSE 0–15	African American	37	0.84	(0.70–1.0)	0.74
	Latino	24	0.55	(0.44–0.70)	0.41
	White	55			
MMSE 16–21	African American	25	0.82	(0.68–0.99)	
	Latino	17	0.53	(0.40–0.71)	
	White	41			
MMSE 22–30	African American	22	0.92	(0.71–1.2)	
	Latino	12	0.67	(0.48–0.92)	
	White	31			

* Each model in this table is additionally adjusted for age as the time scale, Asian race, American Indian race, sex, educational level, Alzheimer's Disease Center site (as a clustering variable), Mini-Mental State Examination (MMSE) score at first evaluation, and age when first dementia symptoms began.

Table 4
Neuropathologic characteristics by race/ethnic group and tertile Mini-Mental State Examination score at first evaluation (n = 3,017)

Neuropathologic characteristic	MMSE score	Race/Ethnic group			p Value
		White	African American	Latino	
% Braak and Braak Stages 5 and 6	Mild (22–30)	58	70	17	0.09
	Moderate (16–21)	72	79	73	0.85
	Severe (0–15)	77	84	80	0.60
% with neurovascular pathology	Mild (22–30)	80	69	100	0.23
	Moderate (16–21)	72	94	88	0.07
	Severe (0–15)	71	70	78	0.73
% with frequent neuritic plaques	Mild (22–30)	86	90	88	0.92
	Moderate (16–21)	89	87	75	0.24
	Severe (0–15)	88	85	86	0.88
% with frequent diffuse plaques	Mild (22–30)	75	64	88	0.49
	Moderate (16–21)	75	83	50	0.08
	Severe (0–15)	72	75	89	0.28

MMSE = Mini-Mental State Examination.