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RESEARCH ARTICLE

Chronic stress, social support, and Alzheimer's blood-based biomarkers in the HABS-HD study

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Abstract

INTRODUCTION: High levels of chronic stress and low social support have been associated with worse cognition among older adults, but the underlying mechanisms remain unclear.

METHODS: We included 2117 older adults (mean age 65.5 years) enrolled in the Health and Aging Brain Study – Health Disparities (HABS-HD). Linear regression models evaluated the associations between social support or chronic stress and Alzheimer's-related blood-based biomarkers (BBMs), including amyloid beta (Aβ) 42/40 ratio, neurofilament light chain (NfL), phosphorylated tau (p-tau)181, and total tau (t-tau). Interactions between chronic stress or social support and gender or race/ethnicity in relation to BBMs were assessed.

RESULTS: Higher chronic stress was associated with higher levels of t-tau. Higher social support was associated with lower levels of NfL. Neither gender nor race/ethnicity modified the associations between chronic stress or social support and BBM levels.

DISCUSSION: Chronic stress and social support are associated with BBMs of neurodegeneration.

KEYWORDS
Alzheimer's disease, blood-based biomarkers, chronic stress, epidemiology, ethnicity, gender, race, social support

Highlights

- Higher chronic stress was associated with higher levels of plasma total tau.
- Higher social support was associated with lower levels of plasma neurofilament light chain.
- Neither gender nor race/ethnicity modified the associations between chronic stress or social support and levels of blood-based biomarkers.
- Chronic stress and social support affect pathways related to neurodegeneration.

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1 | BACKGROUND

Alzheimer's disease (AD) and Alzheimer's disease and related dementias (ADRD) are a significant burden on the health care system.[1] By 2050, an estimated 13 million individuals in the United States will be living with AD/ADRD.[2] Therefore, there is an urgent need to identify risk and protective factors for AD/ADRD. We and others have reported that high levels of chronic stress[3–6] and low social support[7–9] are associated with worse cognition among older adults.[10] However, the mechanisms through which chronic stress and social support affect cognition remain unknown. More specifically, it is unclear whether chronic stress and/or social support affect cognition through vascular pathways,[11] AD-specific pathways,[12] neurodegeneration pathways,[13] a combination of these, or other pathways. The availability of blood-based biomarkers (BBMs) of Alzheimer's pathology and neurodegeneration can now provide insights into mechanisms using large, diverse epidemiological studies. Widely used AD/ADRD BBMs include amyloid beta (Aβ) 42/40 ratio,[14–17] neurofilament light chain (NfL),[18,19] phosphorylated tau (p-tau)181,[20–22] and total tau (t-tau).[23,24]

Notably, both perceived stress and social support vary by gender and race/ethnicity. Women generally report higher levels of both stress[25] and social support.[26] Multiple studies suggest that racial/ethnic minority groups report higher levels of stress.[27,28] However, studies of perceived social support are inconsistent; some report lower levels of perceived social support among racial/ethnic minority groups,[29] whereas others do not.[30] However, studies have not examined whether gender or race/ethnicity modify the associations between either perceived chronic stress or social support and the AD/ADRD BBMs.

In the current study, we examined the association between chronic stress, social support, and BBMs of AD/ADRD in the Health and Aging Brain Study – Health Disparities (HABS-HD), a large cohort study of African American (AA), Mexican American (MA), and non-Hispanic White (NHW) participants. Given disparities in stress and support, we also assessed whether gender or race/ethnicity modified the association between chronic stress or social support and the BBMs. We hypothesized that higher levels of chronic stress and lower levels of social support would be associated with AD/ADRD BBMs, and that these associations would be modified by gender and race/ethnicity, particularly showing stronger effects for women, AAs, and MAs.

RESEARCH IN CONTEXT

1. **Systematic review**: We reviewed the literature using traditional search engines (e.g., PubMed and Google Scholar). Few studies have examined whether chronic stress and/or social support are associated with blood-based biomarkers (BBMs) of Alzheimer's disease (AD) and Alzheimer's disease and related dementias (ADRD), and whether gender or race/ethnicity modify these associations.
2. **Interpretation**: Higher chronic stress was associated with higher levels of total tau. Higher social support was associated with lower levels of neurofilament light chain. Results suggest that chronic stress and social support may be associated with markers of neurodegeneration.
3. **Future directions**: Longitudinal studies are needed to determine whether chronic stress and social support are associated with changes in AD/ADRD biomarkers and incident cognitive impairment and dementia.

2 | METHODS

2.1 | Study population

The Health and Aging Brain Study – Health Disparities (HABS-HD; formally the Health and Aging Brain Study among Latino Elders, HABLE study) is a large, ongoing, community-based study of cognitive aging among individuals living in Dallas or Fort Worth, Texas. The methodology of HABS-HD has been published[31] and is briefly outlined below.

HABS-HD participants were recruited using a community-based participatory research approach.[31] HABLE enrollment began in 2017 for MA and NHW individuals who were 50 years of age or older, willing to provide blood samples, and capable of undergoing neuroimaging studies. In 2020, the cohort expanded to include AA and was renamed the HABS-HD cohort.[31]

Participants undergo a series of assessments over multiple appointments completed within a 4-month period at the Institute for Translational Research (ITR). Follow-up evaluations are conducted in scheduled waves, with ≈24-month intervals between visits. For example, visit 2 typically occurs between 24 and 30 months from baseline, and visit 3 between 48 and 60 months from baseline. Due to the coronavirus disease 2019 (COVID-19), the completion of wave 1 and wave 2 assessments was longer.[31]

The entire study protocol was completed in either Spanish or English in accordance with the participant's preferred language. The HABS-HD study is conducted under institutional review board (IRB)–approved protocols, with each participant (or their legal representative) providing written informed consent. Details on exclusion criteria have been described previously.[32]

2.2 | Diagnostic classification

Cognitive diagnoses were algorithmically determined (decision tree) and then verified by an interdisciplinary clinical consensus group. An informant interview was conducted for completion of the Clinical

Dementia Rating (CDR) scale[33] by clinicians with expertise in dementia to evaluate functional decline. Both the neuropsychological tests and the CDR scale were considered when making the cognitive diagnosis. Participants were considered cognitively unimpaired (CU) if they had a CDR Sum of Boxes (CDR-SB) score of 0,[34,35] their neuropsychological tests were broadly within normal limits (i.e., demographically adjusted *z*-score was 1.5 standard deviation [SD] below the normative range), and there were no self- or informant reports of cognitive decline. Participants were diagnosed with mild cognitive impairment (MCI) if they had a CDR-SB between 0.5 and 2.0,[34,35] one or more neuropsychological test scores falling ≤1.5 SD below the normative range, and self- or informant reports of cognitive decline. Participants were diagnosed with dementia if they had a CDR-SB score ≥2.5[34,35] and had two or more neuropsychological test scores that were 2 SD below the normative range. Participants with a dementia diagnosis ($n = 219$) were excluded from the present analysis.

2.3 | Clinical assessment

Perceived social support was characterized using the Interpersonal Support and Evaluation List (ISEL).[36] The ISEL is a self-report questionnaire that measures overall perceived ability and perceived social support across four dimensions: (1) belonging, (2) appraisal, (3) tangible, and (4) self-esteem. Chronic stress was assessed using the Chronic Burden Scale.[37]

2.4 | Covariates

Covariates were initially chosen based on variables in the literature associated with chronic stress or social support and BBMs. Self-reported demographic characteristics included age, gender, race/ethnicity, education, and marital status. Depression was assessed using the Geriatric Depression Scale (GDS).[38] Worrisome and symptoms of anxiety were assessed using the Penn State Worry Questionnaire.[39] Self-reported medical history, including history of diabetes, myocardial infarction, kidney disease, and stroke, were collected from the interview.

2.5 | BBMs

Previously published international guidelines were used for the collection, processing, and storage of fasting blood samples.[40] Assay preparation was completed using custom automated StarPlus system from Hamilton Robotics (Reno, NA).[41] Plasma Aβ42/40 ratio, p-tau181, t-tau, and NfL were assayed using the ultra-sensitive Simoa (single molecule array technology) on the HD-X platform (Quanterix.com, Billerica, MA) according to the manufacturer's instructions.[31,42,43] Coefficients of variation for all assays were ≤5%.

2.6 | Statistical analyses

All analyses were performed using R version 4.3.1 (https://cran.r-project.org/) with a two-sided p-value < 0.05 considered statistically significant. Participant characteristics were summarized using frequencies and percentages for categorical variables and mean and SDs for continuous variables. The association between chronic stress and social support was examined using Spearman rank correlation (r_s), with additional analyses to determine whether the correlation differed by gender or race/ethnicity. Linear regression models evaluated the cross-sectional associations of social support or chronic stress with the AD/ADRD BBMs (Aβ42/40 ratio, p-tau181, t-tau, and NfL). Model 1 did not adjust for any covariates. Model 2 adjusted for age, education, gender, race/ethnicity, marital status, symptoms of anxiety, and depression. Model 3 adjusted for covariates in Model 2, as well as self-reported history of diabetes, myocardial infarction, kidney disease, and stroke. Interactions between chronic stress or social support and gender or race/ethnicity were also assessed in relation to BBMs.

In additional sensitivity analyses, we examined the interrelationship between chronic stress, social support, and BBMs. We incorporated both chronic stress and social support in the same model, thus assessing whether controlling for one variable attenuated the association of the second variable with BBMs.

3 | RESULTS

3.1 | Participant characteristics

The baseline demographic characteristics of the 2117 HABS-HD study participants with BBMs and without dementia (81.3% CU and 18.7% MCI) are provided in Table 1. The mean age of the participants was 65.5 years, mean years of education was 13.2 years, 62.9% were women, 44.2% were NHW, 12.8% were AA, and 43.0% were MA. Of the participants, 21.4% had diabetes, and ≈4% of participants had a history of kidney disease, myocardial infarction, or a stroke. Chronic stress and social support were weakly correlated (Spearman rank $r_s = -0.218$; $p < 0.001$). We did not observe a difference in correlation between the two measures by gender or race/ethnicity.

Compared to NHW participants, AA and MA participants were younger, reported higher levels of depression, and had lower levels of plasma NfL and p-tau181 (eTable 1). MAs achieved fewer years of formal education and reported lower levels of chronic stress and social support than AA or NHW (eTable 1). Compared to men, women were younger, achieved fewer years of formal education, reported higher levels of chronic stress and depression, and had more symptoms of anxiety. In addition, women had lower p-tau181 levels but higher levels of t-tau compared to men (eTable 2). Compared to CU individuals, participants with MCI achieved fewer years of formal education, reported higher levels of depression, had higher levels of social support, and had higher levels of p-tau181 and NfL (eTable 3). No significant differences were observed in the associations between chronic stress, social

TABLE 1 Baseline demographics of participants.

	N = 2117
Demographics, Mean (SD)	
Age (years)	65.5 (8.54)
Education (years)	13.2 (4.51)
Chronic stress total	7.4 (6.65)
Social support total	41.1 (6.08)
PSWQ	38.8 (14.4)
GDS	5.36 (5.48)
Gender, No. (%)	
Women	1332 (62.9)
Race, No. (%)	
NHW	935 (44.2)
AA	272 (12.8)
MA	910 (43.0)
Marital Status, No. (%)	
Married	1262 (59.6)
Cognitive Status, No. (%)	
CU	1721 (81.3)
MCI	396 (18.7)
Comorbidities, No. (%)	
Diabetes	453 (21.4)
Chronic kidney disease	83 (3.9)
Myocardial infarction	92 (4.3)
Stroke	93 (4.4)
Plasma Biomarkers, Mean (SD)	
Aβ42/40 ratio	0.05 (0.013)
NfL	16.6 (19.1)
p-tau181	2.05 (1.20)
t-tau	2.19 (0.882)

Abbreviations: AA, African American; Aβ, amyloid beta; CU, cognitively unimpaired; GDS, Geriatric Depression Scale; MA, Mexican American; MCI, mild cognitive impairment; NfL, neurofilament light; NHW, non-Hispanic White; PSWQ, Penn State Worrisome Questionnaire; p-tau, phosphorylated tau; t-tau, total tau; SD, standard deviation.

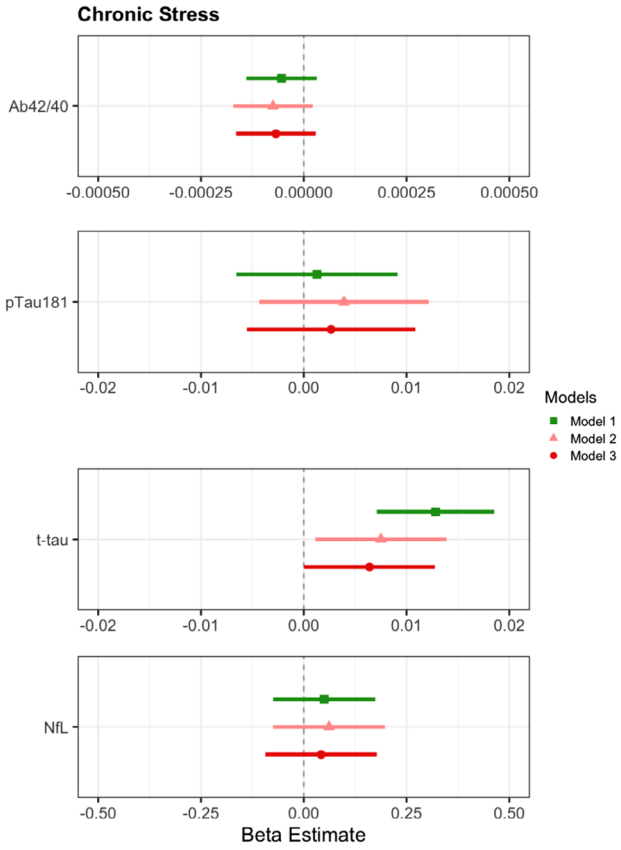

FIGURE 1 Cross-sectional association between chronic stress and BBMs using linear regressions among all participants (*n* = 2117). Green lines (Model 1) indicate unadjusted associations; pink lines (Model 2) indicate associations after adjustment for age, education, gender, race/ethnicity, marital status, symptoms of anxiety, and depression; and red lines (Model 3) indicate associations after adjusting for covariates in Model 2, as well as self-report history of diabetes, myocardial infarction, kidney disease, and stroke. Aβ42/40, amyloid beta 42/40 ratio; BBMs, blood-based biomarkers; NfL, neurofilament light chain; p-tau181, phosphorylated tau-181; t-tau, total tau.

support, and BBMs when comparing CU participants with those diagnosed with MCI.

3.2 | Association of chronic stress and BBMs

Unadjusted and adjusted associations of chronic stress with BBM levels (Aβ42/40 ratio, NfL, p-tau181, and t-tau) are shown in Figure 1 and Table 2. Each point increase in chronic stress was associated with higher levels of t-tau (β = 0.007, p = 0.021) in Model 2, adjusting for age, education, gender, race/ethnicity, marital status, symptoms of anxiety, and depression (Table 2). In Model 3, after additionally adjusting for comorbidities (i.e., diabetes, myocardial infarction, kidney disease, and stroke), the association between chronic stress and t-tau remained (β = 0.006, p = 0.049). In additional analyses, neither gender nor race/ethnicity modified the association between chronic stress and p-tau181, t-tau, or NfL (eTables 4 and 5). There was an interaction between chronic stress and race/ethnicity such that with increasing chronic stress, AA had lower levels of Aβ42/40 compared to NHWs (eTable 5). However, the effect size is very small (β = –0.0003).

3.3 | Association of social support and BBMs

Unadjusted and adjusted associations of social support with BBM levels are shown in Figure 2 and Table 3. Each point increase in social support was associated with lower levels of NfL (β = –0.247, p = 0.002) in the model adjusting for age, education, gender, race/ethnicity, marital status, symptoms of anxiety, and depression. In Model 3, after additionally adjusting for comorbidities, the association between social

TABLE 2 Cross-sectional associations of chronic stress and BBMs of ADRD.

		Model 1		Model 2		Model 3	
	N	*β* Estimate (SE)	*p*-value	*β* Estimate (SE)	*p*-value	*β* Estimate (SE)	*p*-value
Aβ42/40 Ratio	2027	−0.0001 (0.00004)	0.216	−0.0001 (0.00005)	0.130	−0.0001 (0.00005)	0.171
NfL	2063	0.050 (0.063)	0.433	0.061 (0.069)	0.377	0.042 (0.069)	0.543
p-tau181	2038	0.001 (0.004)	0.750	0.004 (0.004)	0.352	0.003 (0.004)	0.526
t-tau	2052	0.013 (0.003)	**<0.001**	0.007 (0.003)	**0.021**	0.006 (0.003)	**0.049**

Note: Model 1 was unadjusted. Model 2 was adjusted for age, education, gender, race/ethnicity, marital status, symptoms of anxiety, and depression. Model 3 adjusted for covariates in Model 2, with additional adjustments for comorbidities such as diabetes, myocardial infarction, kidney disease, and stroke.
Abbreviations: Aβ, amyloid beta; ADRD, Alzheimer's disease and related dementias; BBMs, blood-based biomarkers; NfL, neurofilament light chain; p-tau, phosphorylated tau; SE, standard error; t-tau, total tau.
Bolded values indicate statistically significant results ($p < 0.05$).

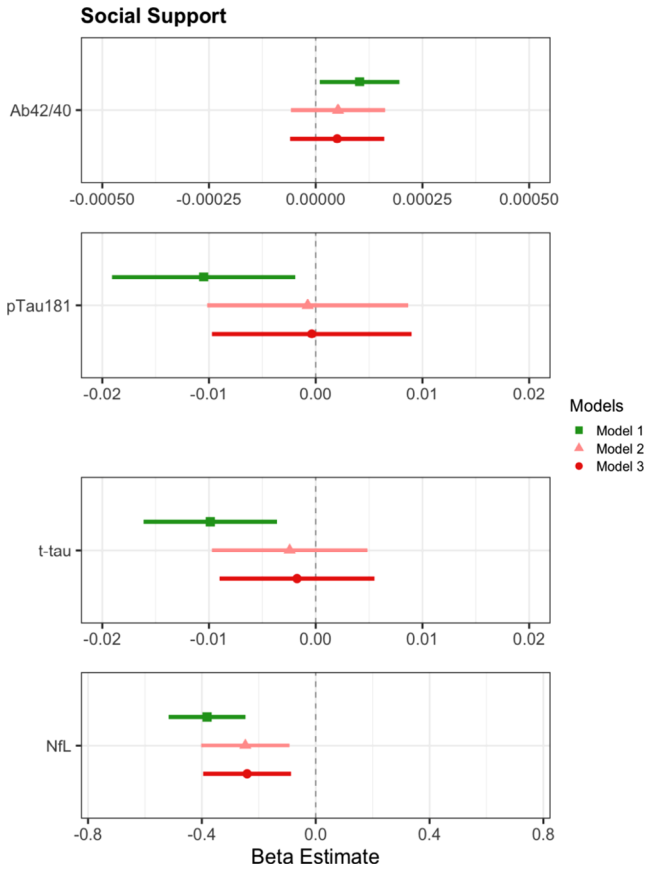

FIGURE 2 Cross-sectional association between social support and BBMs using linear regressions among all participants ($n = 2117$). Green lines (Model 1) indicate unadjusted associations; pink lines (Model 2) indicate associations after adjustment for age, education, gender, race/ethnicity, marital status, symptoms of anxiety, and depression; and red lines (Model 3) indicate associations after adjusting for covariates in Model 2, as well as self-report history of diabetes, myocardial infarction, kidney disease, and stroke. Aβ42/40, amyloid beta 42/40 ratio; BBM, blood-based biomarker; NfL, neurofilament light chain; p-tau181, phosphorylated tau-181; t-tau, total tau.

support and NfL remained ($\beta = -0.241$, $p = 0.002$) (Table 3). However, there were no associations between social support and Aβ42/40, p-tau181, or t-tau after adjusting for comorbidities added to Model 3. Neither gender nor race/ethnicity modified the association between social support and any of the BBMs (eTables 6 and 7).

3.4 | Examining the interrelationship between social support, chronic stress, and BBMS

We conducted additional analyses to examine the interrelationship between social support, chronic stress, and BBMs (eTable 8). We included both social support and chronic stress in the same multivariable model. The beta-estimates for social support generally did not change, indicating that adjusting for chronic stress did not attenuate the association between social support and any of the BBMs. In contrast, the *p*-value for chronic stress was slightly attenuated when social support was added to the model, particularly in relationship to NfL and t-tau (eTable 8).

4 | DISCUSSION

This study examined the cross-sectional associations of chronic stress and social support with BBMs in a large, diverse cohort of older adults, and determined whether gender or race/ethnicity modified the associations. Higher chronic stress was associated with higher levels of t-tau, whereas higher social support was associated with lower levels of NfL. Neither gender nor race/ethnicity modified these associations. These results suggest that chronic stress and social support are associated with higher levels of neurodegeneration-related BBMs.

4.1 | Chronic stress

The effects of chronic stress in relation to cognition have been previously characterized.[5,6] We and others have shown that stress impairs multiple cognitive domains, including attention, memory, language, executive function, problem-solving, and decision-making.[44,45]

TABLE 3 Cross-sectional associations of social support and BBMs of ADRD.

		Model 1		Model 2		Model 3	
	N	*β* Estimate (SE)	*p*-value	*β* Estimate (SE)	*p*-value	*β* Estimate (SE)	*p*-value
Aβ42/40 Ratio	2027	0.0001 (0.00005)	**0.031**	0.0001 (0.0001)	0.354	0.0001 (0.0001)	0.372
NfL	2063	−0.382 (0.069)	**<0.001**	−0.247 (0.079)	**0.002**	−0.241 (0.079)	**0.002**
p-tau181	2038	−0.011 (0.004)	**0.017**	−0.001 (0.005)	0.876	−0.0004 (0.005)	0.938
t-tau	2052	−0.010 (0.003)	**0.002**	−0.002 (0.004)	0.512	−0.002 (0.004)	0.636

Note: Model 1 was unadjusted. Model 2 was adjusted for age, education, gender, race/ethnicity, marital status, symptoms of anxiety, and depression. Model 3 adjusted for covariates in Model 2, with additional adjustments for comorbidities such as diabetes, myocardial infarction, kidney disease, and stroke.
Abbreviations: Aβ, amyloid beta; ADRD, Alzheimer's disease and related dementias; BBMs, blood-based biomarkers; NfL, neurofilament light chain; p-tau, phosphorylated tau; SE, standard error; t-tau, total tau.
Bolded values indicate statistically significant results ($p < 0.05$).

However, the mechanisms through which stress is associated with cognition, whether through vascular pathways, AD-related pathways, neurodegeneration, or a combination of these, remains uncertain. Current biomarkers, such as p-tau181 and an elevated Aβ42/40 ratio, are useful for understanding AD-related pathways and serve as potential surrogates of AD pathology.[46,47] In contrast, elevated levels of NfL and t-tau are nonspecific markers of neurodegeneration.[19,24]

Our study found that higher chronic stress was associated with higher levels of plasma t-tau, a nonspecific biomarker of neurodegeneration. Similarly, another study reported that longstanding stress in midlife was associated with higher levels of cerebrospinal fluid (CSF) t-tau, indicating that stress stimulates unspecific neurodegenerative processes and emerges early in the disease process.[48]

One theory proposes that stress-induced activation of the hypothalamic–pituitary–adrenal (HPA) axis may increase levels of glucocorticoid hormones, leading to structural and functional changes, contributing to brain atrophy and reduced cortical thickness.[49–51] It has also been reported that stress-induced activation of the HPA axis may activate tau-dependent pathways.[52–54] However, we did not observe a difference in any of the models examining the association between chronic stress and the Aβ42/40 ratio or p-tau181. If stress is more associated with tau pathology than with amyloid pathology, we may not have observed a significant association because both the Aβ42/40 ratio and p-tau181 are more specific markers of amyloid plaque pathology than tau neurofibrillary tangle pathology.

In this study, MA reported lower levels of chronic stress compared to AA and NHW. Conversely, AA reported higher levels of chronic stress than both MA and NHW. These results are inconsistent with previous literature that reported higher chronic stress levels among Latinos and AAs.[55] This discrepancy may be attributed to differences in population sampling: our study specifically focused on MA, whereas prior research examined a broader Latino demographic.[55]

Despite known differences in chronic stress across gender and racial/ethnic groups, our study did not find that the associations between chronic stress and the AD/ADRD BBMS were modified by gender or race/ethnicity. This suggests that the association between chronic stress and these particular AD/ADRD biomarkers does not vary as a function of social identity or gender. However, we did not examine other biomarkers that also differ by gender and race/ethnicity, including biomarkers of inflammation, synaptic dysfunction, or vascular function,[56] which are important to examine in the future.

4.2 | Social support

Social support has been associated with improved overall health and better cognitive performance.[7,57,58] In our previous study, we found that social support mitigated declines in cognitive performance, regardless of chronic stress levels.[10] However, the exact mechanisms through which social support is associated with cognition remain unclear. In this study, higher levels of social support were associated with lower levels of plasma NfL, a well-established marker of large-caliber axonal neurodegeneration.[59,60] One possible explanation is that social support may promote better health[61] and reduce the stress-related biological response,[62] thereby reducing the rate of neurodegeneration.[63]

In this study, social support was not associated with t-tau in either Model 2, which adjusted for demographics, or Model 3, which further adjusted for comorbidities. Although t-tau and NfL are both established markers for neurodegeneration, additional research is needed to determine whether these markers are associated with distinct pathways or pathological mechanisms. Moreover, studies are needed to determine how social support and chronic stress differentially affect neurodegenerative pathways related to these biomarkers. For instance, t-tau is considered a marker of neuronal cell body damage, whereas NfL is thought to be a marker of axonal damage.[64]

In additional analyses, neither gender nor race/ethnicity modified the association between social support and the BBMs. This uniformity suggests that the protective effects of social support against neurodegeneration may be broadly applicable across diverse populations and men and women. Nevertheless, additional research is needed to fully understand these interactions and to confirm that the benefits of social support extend across gender and race/ethnicities.

4.3 | Strengths and limitations

The strengths of this study include the large, diverse sample of AA, MA, and NHW participants and the investigation of gender and

race/ethnicity as potential effect modifiers. It is notable that this is the first study to explore these associations in the context of BBMs and to examine the underlying pathways associated with chronic stress and social support.

Limitations also warrant consideration. First, the cross-sectional design of the study limits our ability to assess causality. Second, the lack of biological measures of stress, such as cortisol levels, limits our understanding of the direct physiological mechanisms through which stress affects biomarker levels. Third, there is a relatively small number of blood samples from AA participants due to their inclusion starting at later visits. Finally, direct measures of personality traits, which could significantly affect both perceived stress and social support, were not available.

5 | CONCLUSIONS

This cross-sectional study investigated the association between chronic stress and social support and biomarker levels, providing insights into the underlying mechanisms driving cognitive changes associated with these risk factors. Specifically, higher chronic stress was associated with higher levels of t-tau, whereas higher social support was associated with lower levels of NfL, both of which are nonspecific markers of neurodegeneration. These findings suggest that chronic stress may negatively affect mechanisms related to neurodegeneration, whereas social support could act as a protective factor, potentially attenuating these effects.

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CONFLICT OF INTEREST STATEMENT

Dr. Mielke has served on scientific advisory boards and/or has consulted for Acadia, Althira, Biogen, Eisai, Lilly, Merck, Novo Nordisk, and Roche; received speaking honorariums from Novo Nordisk, PeerView Institute, and Roche; and receives grant support from the National Institutes of Health, Department of Defense, Alzheimer's Association, and Davos Alzheimer's Collaborative. Dr. Bateman has received honoraria from Novo Nordisk, PeerView Institute, and Spear Bio, Inc., which received grant support from the National Institutes of Health, Alzheimer's Association, and the Dementia Alliance of North Carolina. Dr. O'Bryant has multiple patents on precision medicine for neurodegenerative diseases and is the founding scientist of Cx Precision Medicine. Dr. Barnes reports no conflicts of interest but does disclose her role as deputy editor for the *Alzheimer's & Dementia*. All other authors report no conflicts of interest. Author disclosures are available in the Supporting Information.

DATA AVAILABILITY STATEMENT

All Health and Aging Brain Study – Health Disparities (HABS-HD) data are available to the scientific community through the University of North Texas Health Science Center Institute for Translational Research website (https://apps.unthsc.edu/itr/research).[65]

CONSENT STATEMENT

The Health and Aging Brain Study – Health Disparities (HABS-HD) study is conducted under institutional review board (IRB)–approved protocols, with each participant (or their legal representative) providing written informed consent.

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