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Metabolic Factors Are Related to Brain Amyloid Among Mexican Americans: A HABS-HD Study

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Abstract

Background: Despite the tremendous amount of research on Alzheimer's disease (AD) biomarkers, very little data is available regarding the fundamental biomarkers of AD among Mexican Americans.

Objective: Here we sought to examine the link between metabolic markers and brain amyloid among Mexican Americans as compared to non-Hispanic whites from the Health & Aging Brain Study – Health Disparities (HABS-HD) cohort.

Methods: PET amyloid (florbetaben) data was analyzed from 34 Mexican American and 22 non-Hispanic white participants.

Results: Glucagon ($t=3.84$, $p<0.001$) and insulin ($t=-2.56$, $p=0.02$) were both significantly related to global SUVR levels among Mexican Americans. Glucagon and insulin were both related to most ROIs. No metabolic markers were significantly related to brain amyloid levels among non-Hispanic whites.

Conclusion: Metabolic markers are related to brain amyloid burden among Mexican Americans. Given the increased risk for diabetes, additional research is needed to determine the impact of diabetes on core AD biomarkers among this underserved population.

Keywords

Alzheimer's Disease; amyloid; health disparities; Hispanic

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Conflict of Interest/Disclosure Statement

The authors report no conflict of interest to report related to this data. SEO holds multiple patents on precision medicine approaches to neurodegenerative diseases and holds an interest in Cx Precision Medicine.

INTRODUCTION

Alzheimer's disease (AD) is the most common neurodegenerative disease, which disproportionately impacts underserved communities[1]. In fact, Hispanics/Latinos are expected to experience the largest increase in AD and AD-related dementias (ADRDs) by 2060[2]. However, the reasons for this health disparity remain largely unknown, which is likely largely due to the substantial underrepresentation of Hispanics in AD research[3].

Both clinical research and epidemiological studies have linked diabetes to AD. In clinic samples, AD cases have been shown to have higher blood glucose levels[4], and diabetics with AD have been found to have increased rates of decline[5] as well as significantly greater cortical atrophy than non-diabetic AD cases[6]. The increased risk for AD and cognitive dysfunction among those with diabetes has been shown in numerous epidemiological studies, including the Rotterdam Study[7,8], the Canadian Study of Health and Aging[9], Framingham Heart Study [10], the Washington Heights Inwood Columbia Aging Project (WHICAP)[11], the Honolulu-Asia Aging Study [12], the Religious Orders Study [13] and the Sacramento Area Latino Study on Aging (SALSA) [14].

In our prior work, we have demonstrated that Mexican Americans (65% of the U.S. Hispanic population) experience a disproportionate burden of diabetes across the cognitive spectrum[15–17]. Additionally, we have also shown that metabolic markers are altered among Mexicans as compared to non-Hispanic whites[15] and metabolic markers are of importance in the proteomic profile of MCI among Mexican Americans[18]. However, to our knowledge, no prior study has examined the link between blood metabolic markers and brain amyloid PET levels among Mexican Americans. Therefore, here we sought to conduct a pilot study of the link between blood markers of metabolic function and brain amyloid levels among Mexican Americans as compared to non-Hispanic whites from the Health & Aging Brain – Health Disparities Study (HABS-HD)[15,18–20].

METHODS

Participants & Assessment

Data was analyzed from n= 34 Mexican American and n=22 non-Hispanic white participants from the Health & Aging Brain Study – Health Disparities (HABS-HD; formally the Health & Aging Brain study among Latino Elders, HABLE study) that had undergone PET amyloid scans. HABS-HD study is an ongoing, longitudinal, community-based project examining health disparities in MCI and AD among Hispanic, Mexican Americans (MA) as compared to non-Hispanic whites (NHW) [15,18,19,21] with recent expansion currently enrolling African Americans. HABS-HD methods have been published elsewhere[15] and are briefly outlined below. The data included in this study encompasses Mexican American and non-Hispanic white participants since the recruitment of the African American participants is ongoing. Inclusion criteria for the study includes 1) self-reported ethnicity of African American, Mexican American or non-Hispanic white, 2) willingness to provide blood samples, 3) capable of undergoing neuroimaging studies, 4) age 50 and above, and 5) fluent in English or Spanish. Exclusion criteria includes 1) Type 1 diabetes, 2) presence of active infection, 3) current/recent (12 month) cancer (other than skin cancer),

4) current severe mental illness that could impact cognition (other than depression), 5) recent (12 months) traumatic brain injury with loss of consciousness, 6) current/recent alcohol/substance abuse, 7) active severe medical condition that could impact cognition (e.g., end stage renal failure, chronic heart failure, chronic obstructive pulmonary disease) and 8) current diagnosis of dementia other than AD. Participant recruitment for HABS-HD includes a community-based participatory research (CBPR) approach [22,23]. The CBPR approach has been used successful as a recruitment modality for reaching underserved and minority populations. It involves collaborating with local communities through outreach (holding community events, seminars), word of mouth, marketing modalities (newspaper, television, radio), and providing back information (clinical lab work, MRI clinical reads, neuropsychological test results) to the participants and their health care providers. The HABS-HD protocol includes an interview, functional exam, blood draw for clinical labs and biobanking, neuropsychological testing and 3T MRI of the brain. Amyloid and tau PET scans are ongoing for the full cohort. All aspects of the study protocol can be conducted in Spanish or English. The HABS-HD study is conducted under IRB approved protocols and each participant (or his/her legal representative) signs written informed consent. The data is available to the scientific community through the UNTHSC Institute for Translational Research (ITR) website[24].

Interview and Neuropsychological Assessment

An interview is conducted as part of the HABS-HD protocol, which includes an interview and neuropsychological testing with the following battery: Mini Mental Status Exam (MMSE)[25], Wechsler Memory Scale- Third Edition (WMS-III) Digit Span and Logical Memory[26], Digit Symbol Substitution, Trail Making Test Parts A and B[27], Spanish-English Verbal Learning Test (SEVLT)[28], Animal Naming (semantic fluency)[28], FAS (phonemic fluency)[29] as well as the American National Adult Reading Test (English-speakers)[30], and Word Accentuation Test (Spanish-speakers)[31]. An informant interview is also conducted for completion of the Clinical Dementia Rating (CDR) Scale[32] by clinicians with expertise in dementia to evaluate for functional declines.

Blood Biomarkers

Fasting blood samples were collected, processed and stored per previously published international guidelines. Hemoglobin A1c (HbA1c) and glucose were collected as part of the clinical labs via Quest laboratories. Blood samples were collected in special glucagon-like peptide 1 (GLP-1) plasma tube for proteomic assays. All blood biomarker assays were conducted in the Institute for Translational Research (ITR) Biomarker Core. Preparation of samples for proteomic assay was conducted using the Hamilton Robotics StarPlus system, which facilitates substantially improved quality of assays, increased QA/QC monitoring, as well as increased proteomic capacity in the laboratory. Any re-aliquoting was conducted via the Hamilton easyBlood robotic system. Plasma samples were assayed via multi-plex biomarker assay platform using electrochemiluminescence (ECL). All plasma samples were assayed for targeted metabolic markers using a commercially available kit for GLP-1, insulin and glucagon. Due to degradation of protein levels in long-term storage, GLP-1 kits were run every two weeks to avoid long-term storage. APOEε4 genotyping was performed

using commercially available TaqMan assays and APOE variants were in Hardy-Weinberg equilibrium.

Diagnostic Classification

Cognitive diagnoses were assigned algorithmically (decision tree) and verified at consensus review as follows: Normal Control (NC) = no cognitive complaints, CDR sum of boxes score of 0 and cognitive tests scores broadly within normal limits (i.e. performance greater than that defined as meeting diagnostic criteria for MCI [i.e. <=1.5 standard deviations below the normative range]); Mild Cognitive Impairment (MCI): cognitive complaint (self or other), CDR sum of boxes score between 0.5– 2.0 and at least one cognitive test score falling <=1.5 standard deviation below normative ranges; Dementia: CDR sum of boxes score >=2.5 and at least two cognitive test scores 2 standard deviation below normative ranges.

Neuroimaging

PET Amyloid (Neuraceq; aka florbetaben). Beginning with Visit 2, all study subjects undergo PET amyloid imaging using Siemens Biograph Vision 450 whole-body PET/CT scanner following the ADNI3 protocol for Neuraceq scans. Briefly, participants are injected with an 8.1 mCi (±10%) bolus of Neuraceq. A 4-frame by 5-min (20min total) dynamic emission acquisition is started 90min post injection following the acquisition of a low dose CT scan used for attenuation correction. The emission images are processed by iterative reconstruction, 4 iterations and 16 subsets. FreeSurfer-defined regions (frontal, anterior/posterior cingulate, lateral parietal, lateral temporal cortex) were used to derive a summary cortical region of interest (ROI). Normalization to whole cerebellum reference region was conducted to obtain global standardized update value ratios (SUVR). An SUVR of 1.08 was used to define positivity.

Statistical Analyses

Statistical Analyses were conducted in SPSS 25 (IBM). Chi-square and ANOVA were utilized to compare groups on demographic and clinical variables. Linear regression was used to examine the link between all metabolic markers and SUVR values including age, gender, education and APOE4 as covariates. All metabolic markers were entered simultaneously to minimize the number of regression models. Analyses were conducted split by ethnicity and statistical significance was set at $p<0.05$.

RESULTS

Demographic characteristics of the sample are provided in Table 1. There was no difference between groups in age or any SUVR values. Mexican Americans achieved significantly lower levels of education and had lower MMSE scores. There were more females in the Mexican American group.

In linear regression models with age, gender, education, and APOE4 positivity as covariates, glucagon ($t=3.84$, $p<0.001$) and insulin ($t=-2.56$, $p=0.02$) were both significantly related to global SUVR levels among Mexican Americans (overall model $F=7.52$, $p<0.001$). With

regards to regional values, glucagon was related to frontal SUVR (overall model F=5.93, p<0.001; glucagon t=2.93, p=0.008), anterior/posterior cingulate SUVR (overall model F=5.79, p<0.001; glucagon t=3.06, p=0.006), lateral parietal SUVR (overall model F=7.14, p<0.001; glucagon t=3.99, p<0.001), and lateral temporal SUVR (overall model F=10.65, p<0.001, glucagon t=5.34, p<0.001). Insulin was significantly related to frontal SUVR (t=−2.56, p=0.02), lateral parietal SUVR (t=−2.18, p=0.04) and lateral temporal SUVR (t=−3.52, p=0.002) while the link between insulin and anterior/posterior cingulate SUVR (t=−1.92, p=0.07) approached significance.

None of the overall models or individual metabolic markers were significantly related to cerebral amyloid burden among non-Hispanic whites.

A total of four Mexican American participants were amyloid positive with a global SUVR cut-score of 1.08 whereas 7 non-Hispanic white participants were positive. For preliminary analyses, logistic regression was run using only glucagon and insulin as the predictors (including demographic characteristics resulted in perfectly identified model), glucagon was marginally significant (OR=1.08, p=0.05) only for Mexican Americans.

DISCUSSION

The current team has previously demonstrated that Mexican Americans develop cognitive loss[15] and neurodegeneration[20] at significantly younger ages when compared to non-Hispanic whites. We have also shown that Mexican Americans have lower overall amyloid positivity rates across cognitive classifications[15] as well as lower rates of the APOE4 genotype[15–17]. Therefore, it is imperative that the underlying contributions to AD, as well as AD health-disparities facing Mexican Americans be understood. Here, we demonstrate that metabolic factors are significantly associated with amyloid burden among Mexican American adults.

In our prior work, we have shown that metabolic factors are heavily weighted in the proteomic profile of MCI among Mexican Americans[18]. Additionally, we have recently demonstrated that metabolic factors (e.g., glucose, HbA1c, duration of diabetes) are associated with MRI-based neurodegeneration (N) from the AT(N) framework among Mexican Americans[20]. In light of our work demonstrating that proteomic profiles can be used to identify the specific AD patients most likely to respond to anti-diabetic medications[33], it is possible that anti-diabetic interventions may have particular relevance and importance for Mexican Americans with regards to treating and preventing AD, neurodegeneration and/or cognitive loss.

Few studies have examined the link between medical comorbidities and brain amyloid among underserved populations. In the Atherosclerosis Risk in Communities (ARIC) PET study, the link between white matter hyperintensity appeared to be more strongly associated with amyloid among Black participants[34]. Also, in the ARIC study, midlife vascular risk factors, including midlife BMI, were associated with late-life brain amyloid[35], but there was no race interaction. The metabolic syndrome was not associated with brain amyloid among Hispanics in Yew York City though elevated glucose was associated with lower brain

amyloid burden[36]. The current findings expand on the current literature and point to the need to carefully study the impact of medical comorbidities as they vary among populations.

There are weaknesses to the current study. First, the sample size is small. However, this is one of the only studies to specifically examine brain amyloid among Mexican Americans. Additionally, the current sample size of Hispanics is larger than all Hispanics enrolled in the Alzheimer's Disease Neuroimaging Initiative (ADNI). Additionally, the entire HABS-HD cohort is currently undergoing brain amyloid scans and, therefore, large-scale follow-up studies will soon be underway. Another weakness is the cross-sectional nature of the data; however, HABS-HD is a longitudinal study and therefore, follow-up studies will be conducted in the future. In spite of these limitations, this is, to our knowledge, one of the only and largest studies to specifically examine the impact of metabolic factors and brain amyloid levels among Mexican Americans. The findings, particularly within the context of our prior work, strongly points to the need to understand metabolic factors as they relate to cognitive loss and AD among Mexican Americans.

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Table 1:

Characteristics of Sample

	Mexican American	Non-Hispanic white	Statistic	p-value
Age	65.71 (11.12)	67.01 (8.81)	F=0.25	>0.05
Gender (% male)	35%	64%	χ^2=4.31	0.04
Education	8.91 (5.08)	15.55 (2.48)	F=32.32	<0.001
Diabetes (% yes)	47%	27%	χ^2=2.19	0.14
MMSE	25.35 (4.01)	27.73 (3.43)	F=5.23	0.03
CDR-SB	1.24 (1.81)	1.70 (2.48)	F=0.67	0.42
Trails A	54.51 (24.66)	44.77 (25.80)	F=1.99	0.17
Trails B	177.63 (93.52)	108.71 (70.69)	F=8.35	0.006
WMS-III LM1	25.29 (10.91)	35.05 (15.21)	F=7.80	0.007
WMS-III LM2	13.18 (8.39)	19.64 (10.27)	F=6.64	0.01
FAS	22.41 (9.48)	29.09 (6.67)	F=8.25	0.006
Animals	13.65 (5.62)	16.91 (5.79)	F=4.39	0.04
SEVLT Trials 1–5	24.29 (7.83)	25.91 (9.79)	F=0.47	0.50
SEVLT Delayed Recall	5.29 (3.25)	6.65 (3.77)	F=1.88	0.18
Glucose mg/dL	109.18 (30.07)	96.09 (15.54)	F=3.52	0.07
HbA1c	6.28 (1.32)	5.38 (0.47)	F=9.31	0.004
GLP1 pg/mL	1.48 (1.82)	2.67 (7.71)	F=0.73	0.40
Glucagon pg/mL	68.65 (44.33)	71.87 (66.59)	F=0.04	0.84
Insulin pg/mL	328.99 (347.29)	199.26 (180.84)	F=2.49	0.12
Global SUVR	1.02 (0.16)	1.07 (0.20)	F=1.01	>0.05
Frontal SUVR	1.01 (0.17)	1.06 (0.20)	F=1.73	0.19
Anterior/Posterior SUVR	1.09 (0.18)	1.16 (0.21)	F=0.94	0.34
Lateral Parietal SUVR	1.02 (0.16)	1.07 (0.20)	F=1.83	0.18
Lateral Temporal SUVR	0.95 (0.13)	1.01 (0.19)	F=1.36	0.25

SUVR = standardized uptake ratio value; MMSE = mini mental status examination; CDR-SB = clinical dementia rating scale sum of boxes score; Trails A = Trail Making Test Part A; Trails B = Trail Making Test Part B; WMS-III LM1 = Weschler Memory Scale 3rd edition, Logical Memory I; WMS-III LM2 = Wechsler Memory Scale 3rd edition, Logical Memory II; FAS = verbal fluency for letters F, A and S; Animals = animal naming test; SEVLT 1–5 = Spanish – English Verbal Learning Test sum of trials 1 – 5; HbA1c = hemoglobin A1c %; pg/mL = picogram per milliliter