The Interaction Between Race/Ethnicity and Traumatic Brain Injury on Alzheimer's Disease Risk in Late Life

Matthew W. Thomson & Alexandra L. Clark, Ph.D.
[1]Department of Psychology, College of Liberal Arts, The University of Texas at Austin

The University of Texas at Austin
Department of Psychology
College of Liberal Arts

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INTRODUCTION

• Traumatic Brain Injury (TBI) is a known risk factor for Alzheimer's Disease (AD) (Zhang et al., 2021; Masel & DeWitt, 2010).

•TBI has been to shown to disproportionately affect racially/ethnically diverse populations, and negative health outcomes following injury may be exacerbated by the consequences of larger societal inequities (Penner et al., 2017; Zavala et al., 2021).

• Most TBI research focuses on the examining the long-term cognitive and neural effects of injury in Non-Hispanic White populations (NHW). However, there is a need to better understand whether these outcomes differ across racial/ethnic groups.

• The present study aimed to examine whether experiencing a TBI disproportionately increases AD risk in racially/ethnically diverse older adults relative to their NHW counterparts.

•We hypothesized that racial/ethnic minoritized older adults with a TBI would show poorer cognitive outcomes and worse plasma AD biomarkers relative to NHWs.

METHODS

Study Participants

- Baseline data from 1,649 community dwelling older adults who participated in the Health and Aging Brain Study – Health Disparities (HABS-HD)
- Self-reported racial groups were as follows:
  - 631 Non-Hispanic White (NHW)
  - 681 Hispanic/Latino (H/L)
  - 337 Non-Hispanic Black

Study Measures

- The Ohio State University TBI:
  - A semi-structured interview was used to assess whether any reported injury met clinical criteria for TBI; TBI was determined by an indication of a loss of consciousness (LOC); LOC duration was used to assess mild (<30 minutes), moderate (>30 minutes, <24 hours), and severe (>24 hour) injury severity. Given repetitive injury was common, a total number of TBI index was also calculated.
- AD Risk:
  - Cognition: Memory and executive function composite z-scores
  - Plasma Biomarkers: Amyloid Beta 42/40 ratio and total-tau (t-tau)

METHODS & RESULTS

Statistical Analyses:

- ANOVAs & chi-square tests examined racial group differences on sociodemographic, TBI injury, cognitive outcomes, and AD markers (cognitive and plasma biomarkers).
- ANCOVAs investigated TBI x Race interactions on AD cognitive and biomarker outcomes controlling for the effects of age, sex, income, TBI, and education.
- Follow-up regression analyses explored TBI injury severity x race and TBI quantity x race interactions on AD outcomes as well.

Table 1. Participant demographic & group comparisons

Variable	Total Sample	Non-Hispanic Whites	Hispanic/Latinos	African American/Black	Omnibus Test Result (by Race/Ethnicity)		
	n = 1649	*n* = 631	*n* = 681	*n* = 337	Test Statistic	p-value	*Effect Size (V or eta^2)*
Age, M(SD)	65.01 (8.50)	68.19 (8.50)	63.28 (8.06)	62.52 (7.51)	F = 79.839	p = <.001	.088
Income, M(SD)	66,082 (84,476)	93,363 (94,791)	37,759 (58,028)	72,237 (91,099)	F = 5.144E+11	p = <.001	.087
Education, M(SD)	12.93 (4.61)	15.64 (2.70)	9.48 (4.54)	14.81 (2.73)	F = 541.968	p = <.001	.397
APOE4					--	--	--
positive, *n* (%)	225 (13.6)	125 (19.8)	99 (14.5)	**			
negative, *n* (%)	776 (47.1)	335 (53.1)	441 (64.8)	**			
missing, *n* (%)	648 (39.3)	171 (27.1)	141 (20.7)	**			
Female, *n* (%)	1044 (63.3)	360 (57.1)	454 (66.7)	230 (68.2)	x2 = 17.480	p = <.001	.103
TBI Positive, *n* (%)	363 (22.0)	174 (27.6)	117 (17.2)	72 (21.4)	x2 = 20.717	p = <.001	.112
TBI Quantity					x2 = 29.942	p = <.001	.095
0 TBI, *n* (%)	1286 (78.0)	457 (72.4)	564 (82.8)	265 (78.6)			
1 TBI, *n* (%)	237 (14.4)	108 (17.1)	79 (11.6)	50 (14.8)			
≥ 2 TBIs, *n* (%)	126 (34.7)	66 (37.9)	38 (32.5)	22 (30.6)			
TBI Severity					x2 = 12.352	p = .015	.130
mild, *n* (%)	254 (70.0)	124 (71.3)	79 (67.5)	51 (70.8)			
moderate, *n* (%)	76 (20.9)	31 (17.8)	34 (29.1)	11 (15.3)			
severe, *n* (%)	33 (9.1)	19 (10.9)	4 (3.4)	10 (13.9)			
Cognitive Outcomes							
EF composite, M(SD)	.134 (.87)	-.01 (.81)	.25 (.92)	.15 (.84)	F = 15.193	p = <.001	.018
Memory composite, M(SD)	-.01 (.84)	.03 (.85)	.003 (.81)	-.09 (.86)	F = 2.416	p = .090	.003
AD Biomarker Outcomes							
Amyloid (Aβ40/42), M(SD)	-.03 (.81)	-.03 (.78)	-.05 (.78)	.04 (.96)	F = .737	p = .497	.001
Total Tau (t-tau), M(SD)	-.06 (.85)	-.11 (.88)	-.01 (.81)	-.08 (.88)	F = 2.035	p = .131	.003

Note. TBI = Traumatic Brain Injury; EF = Executive Function; EF composite was inverted to increase readability; EF & Memory composites' outliers (zscore > ± 3) were deleted; Income was log-transformed to improve normality of distribution; ** = APOE data was batched so AA data was not available; 1331 participants had t-tau data, 1316 participants had AB42/40 data, the participants missing this data were disproportionately AA.

Plasma AD Outcomes:

- ANCOVAs revealed there was a significant TBI x race interaction on the plasma AB42/40 ratio. See Figure 2. Black older adults had lower plasma AB42/40 levels (indicative of increased AD risk) relative to Hispanic (p = .06) and NHW (p = .19) adults.
- There was no significant interaction on plasma t-tau (F = 0.16, p = .85).

Exploratory Analyses:

- Within the TBI group, ANCOVAs revealed there was no significant TBI severity x race interaction on any cognitive or biomarker outcomes (ps > .071).
- There was a significant # of TBI x race interaction on memory. NHW older adults with multiple injuries (2+) had poorer memory performance relative to Black (p = .002) and Hispanic (p = .07) adults. No other interactions on outcomes were observed (ps > .05).

Cognitive Outcomes:

- ANCOVAs revealed there was no significant TBI x race interaction on the memory (F = 1.79, p = 0.16) or the executive function composite (F = .046, p = .955) composite. See Figure 1.

Figure 1. TBI x Race on memory outcomes

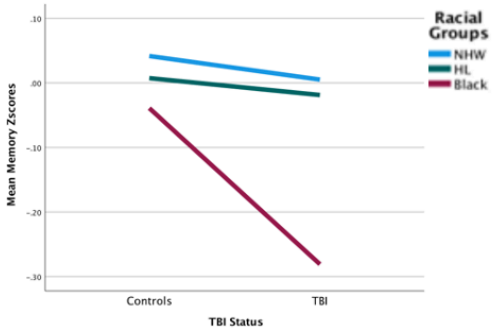
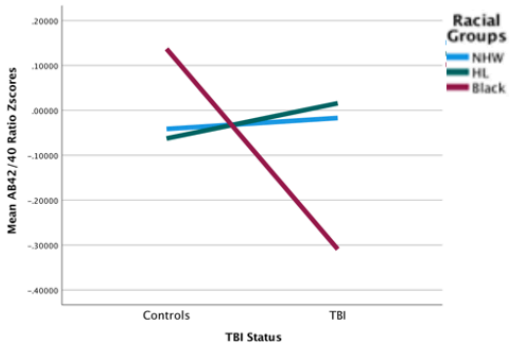


Figure 2. TBI x Race on plasma AB42/40



DISCUSSION

- Findings revealed that Black older adults with a history of TBI may demonstrate worse AD pathology as characterized by plasma AB 42/40 levels.
- Targeted interventions centered on reducing amyloid levels in Black older adults with TBI history may help prevent future progression to AD.
- Future analyses should explore more direct central nervous system markers of AD pathology using tau and amyloid PET neuroimaging methods.

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