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RESEARCH ARTICLE

Temporal associations between treated and untreated hearing loss and mild behavioral impairment in older adults without dementia

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Abstract

INTRODUCTION: Hearing loss (HL) and mild behavioral impairment (MBI) are non-cognitive markers of dementia. This study investigated the relationship between hearing and MBI and explored the influence of hearing aid use on the treatment of hearing loss, both cross-sectionally and longitudinally.

METHODS: Data were analyzed from National Alzheimer's Coordinating Center participants, age ≥50, dementia-free at baseline, collected between 2005 and 2022. Three self-report questions were used to generate a three-level categorical hearing variable: No-HL, Untreated-HL, and Treated-HL. MBI status was derived from the informant-rated Neuropsychiatric Inventory Questionnaire (NPI-Q) using a published algorithm. At baseline (n = 7080), logistic regression was used to examine the association between hearing status (predictor) and the presence of global and domain-specific MBI (outcome), adjusting for age, sex, cognitive diagnosis, and apolipoprotein E4 (APOE4). Cox proportional hazard models with time-dependent covariates were used to examine the effect of (1) hearing status as exposure on the rate of incident MBI (n = 5889); and (2) MBI as exposure on the rate of incident HL in those with no HL at baseline (n = 6252).

RESULTS: Cross-sectionally, participants with Untreated-HL were more likely to exhibit global MBI (adjusted odds ratio (aOR) = 1.66, 95% CI: 1.24–2.21) and individual MBI domains of social inappropriateness (aOR = 1.95, 95% CI: 1.06–3.39), affective dysregulation (aOR = 1.71, 95% CI: 1.21–2.38), and impulse dyscontrol (aOR = 1.71, 95% CI: 1.21–2.38), compared to those with No-HL. Participants with Treated-HL (i.e., hearing aid use) did not differ from No-HL for odds of global or most MBI domains, except for impulse dyscontrol (aOR = 1.38, 95% CI: 1.05–1.81). Longitudinally, we found relationships between Treated-HL and incident MBI (adjusted hazard ratio (aHR) = 1.29, 95% CI: 1.01–1.63) and between MBI and incident Untreated-HL (aHR = 1.51, 95% CI: 1.19–1.94).

Penny Gosselin and Dylan X. Guan are co-first authors.

DISCUSSION: Our cross-sectional results support that hearing aid use is associated with lower odds of concurrent global MBI in dementia-free participants. Longitudinally, relationships were found between MBI and HL. The severity of HL was not assessed, however, and may require further exploration.

KEYWORDS
dementia, dementia prevention, hearing aid, hearing loss, MBI, mild behavioral impairment, neuropsychiatric symptoms, risk factor

Highlights

- Hearing Loss (HL) and mild behavioral impairment (MBI) are markers of dementia
- Cross-sectionally: Untreated-HL was associated with global MBI burden, but
- HL treated with hearing aids was not
- We found associations between MBI and incident Untreated-HL

1 | BACKGROUND

Dementia is a global health priority with significant economic, societal, and personal costs to those affected and their carers. Dementia represents a major cause of disability, now the 7th leading cause of mortality amongst all diseases.[1] While disease-modifying drugs show promise, a large focus of the World Health Organization action plan is on prevention and risk reduction.[2] Hearing loss (HL) and behavioral change can occur in advance of cognitive change, which more typically signal dementia risk; leveraging these markers may help identify people at preclinical and prodromal disease stages for earlier intervention.[3–7]

Hearing loss is the single greatest modifiable risk factor for dementia and the third most prevalent chronic condition among older adults.[8,9] In the life-course model of modifiable risk factors for dementia, of 12 risk factors, HL accounts for more than 8% of the population attributable risk in mid-life for later-life incidence of dementia.[8] The Lancet Commission advocates that taking care of hearing health—by addressing HL and limiting excessive noise exposure—can influence brain health and help mitigate the risk of developing dementia.[8] In addition to dementia risk and links to dementia biomarkers, HL is associated with higher rates of hospitalization/rehospitalization, with longer hospital stays, frailty, depression, loneliness, social isolation, and behavioral symptoms in long-term care.[10–16] Despite the prevalence and negative consequences of HL, treatment uptake is surprisingly low at 17%.[17] Among adults over 70 that could benefit from using a hearing aid to treat HL, less than 1/3 have done so.[18]

Behavioral symptoms are almost ubiquitous in dementia, and neuropsychiatric symptoms (NPS) are part of core clinical criteria.[19] While behavioral symptoms are common with dementia diagnosis, they can also represent dementia risk, especially when later-life emergent and persistent. The construct of mild behavioral impairment (MBI) has been described and validated as a neurobehavioral syndrome that leverages later-life symptom emergence (≥50 years) and symptom persistence (≥6 months) to identify a high-risk group for incident cognitive decline and dementia.[4,20–22] MBI criteria are applied in conjunction with cognitive diagnosis (e.g., cognitively normal/subjective cognitive decline [CN/SCD], or mild cognitive impairment [MCI]) to identify the risk group with greater specificity than conventional approaches to incorporating behavior into risk modeling.[23–25] For some, MBI may be a proxy marker of neurodegenerative disease occurring in advance of or concurrently with cognitive impairment, as evidenced by associations with AD biomarkers.[26–33]

While HL has been associated with depression, the influence on this association with hearing aids as a complementary treatment is less clear.[11,34–36] Further, few studies have investigated the influence of HL and hearing aid treatment on MBI symptoms beyond depression.[37,38] Understanding the potential role of hearing aid use as a non-pharmacological intervention for a larger spectrum of MBI symptom management and HL treatment could help optimize health outcomes.

Here, we investigated the relationship between hearing and MBI in dementia-free participants and explored the influence of hearing aid use for the treatment of HL, both cross-sectionally and longitudinally. To address the unanswered question on the directionality of the association between HL and MBI, we examined the effect of (1) hearing status as exposure on incident MBI; and (2) MBI as exposure on incident HL in those with no HL at baseline. We hypothesized that Treated-HL (hearing aid use) would have a protective effect, associating with lower MBI symptom burden both cross-sectionally and longitudinally.

2 | METHODS

2.1 | Participants

Participant demographic and clinical data were obtained from the National Alzheimer's Coordinating Center Uniform Dataset

(NACC-UDS), collected between 2005 and 2022. NACC was established by the National Institute on Aging (NIA) and consists of multiple NIA-funded Alzheimer's Disease Research Centers (ADRCs) recruiting and collecting data from individuals with cognitive function ranging from normal cognition to dementia. The UDS is a large prospective and longitudinal clinical evaluation that includes demographic and standardized clinical data collected approximately annually. All contributing ADRCs were required to administer standardized forms, obtain informed consent from all participants and their informants, as well as IRB approvals prior to submitting data to NACC. Detailed information on the cohort and neuropsychological battery of tests included in the UDS is described elsewhere.[39–41]

Samples for cross-sectional and longitudinal analyses were drawn from a pool of 7080 participants (age ≥50) with no history of chronic psychiatric conditions or dementia diagnosis at baseline, complete demographic, apolipoprotein E4 (APOE4), NPI-Q, and hearing data, consistent self-reported hearing and hearing aids use, and available longitudinal data (Figure 1).

2.2 | Hearing assessment

Self-reported hearing-related data were extracted from three questions on the NACC-UDS Physical Evaluation form at baseline and at each follow-up visit. The first question gauged the presence or absence of HL: "Without a hearing aid(s), is the subject's hearing functionally normal?". Hearing aid use and function were determined by two questions: (1) "Does the subject usually wear a hearing aid(s)?" and (2) "If the subject usually wears a hearing aid(s), is the subject's hearing functionally normal with a hearing aid(s)?". Results from combinations of these three questions were used to generate a categorical hearing variable with three levels: (1) "No-HL" for participants who reported functionally normal hearing and no hearing aid use; (2) "Untreated-HL" for participants who reported functionally impaired hearing and no hearing aid use or hearing aid use that did not restore functionally normal hearing; and (3) "Treated-HL" for participants who reported functionally impaired hearing and hearing aid use that restored functionally normal hearing.

2.3 | MBI assessment

The informant-rated Neuropsychiatric Inventory Questionnaire (NPI-Q) includes 12 NPS domains scored for frequency and severity over a 1-month reference period. Global and domain-specific MBI scores were generated from 10 NPI-Q domains (sleep and appetite abnormalities were excluded) using a published algorithm to establish the five MBI domains: decreased motivation, affective dysregulation, impulse dyscontrol, social inappropriateness, and psychosis.[42] In accordance with the MBI symptom persistence criterion, participants were considered MBI+ in a domain if they scored >0 for any symptom at two consecutive visits prior to dementia diagnosis or MBI- if they scored 0 in either of the two visits.

RESEARCH IN CONTEXT

1. **Systematic review**: We reviewed the literature using PubMed and Google Scholar. Little is known about the influence of hearing aids on mild behavioral impairment (MBI).
2. **Interpretation**: Our findings show that hearing aid use is associated with lower odds of global MBI symptomology in dementia-free persons. Longitudinally, we found associations between MBI and incident Untreated-HL and between Treated-HL and incident MBI reflecting what may be greater HL severity in those with Treated-HL.
3. **Future directions**: We describe potential mechanisms to account for the association between HL and MBI, but additional studies are required to extend this work with (1) measures of audiometric HL to account for HL severity and limitations of self-report; (2) data on the frequency and duration of actual hours of hearing aid use; (3) use of the MBI-Checklist to measure MBI; and (4) biomarker and neuroimaging data to better understand the relationship in the context of neurodegenerative disease burden.

2.4 | Statistical analysis

For participant characteristics, continuous variables were reported in mean (standard deviation; SD); range and categorical variables were reported in *n* (%). Groups were compared using analysis of variance (ANOVA), independent samples *t*-tests, or Mann-Whitney U tests for continuous variables, and chi-squared or Fisher's exact test for categorical variables as appropriate.

The cross-sectional analysis included participants with complete and consistent reports of HL and hearing aid use/non-use. Participants in the No-HL group never reported HL at any follow-up visit. For participants that reported HL, a participant-specific baseline was defined as the first visit they reported Treated-HL or Untreated-HL (with all subsequent visits consistent with baseline). Cross-sectional associations between hearing status as predictor (i.e., Treated- or Untreated-HL referenced to No-HL) and global and domain-specific MBI status as outcome (i.e., the presence or absence of global MBI or domain-specific MBI symptoms) were examined using multivariable logistic regression adjusted for age, sex, education, APOE4, and cognitive diagnosis (i.e., CN/SCD, MCI)[39–41] to estimate adjusted odds ratios (aORs).

Two survival analyses with baselines set at the first available participant visit were conducted to explore reciprocal associations between hearing loss (whether treated by consistent hearing aid use or not) and MBI (Figure 1). The first Cox proportional hazard model was used to examine the effect of hearing (exposure) on incident MBI (outcome) in a sample of 5889 participants with no MBI at baseline. Specifically, two hazard ratios adjusted for age, sex, education, APOE4, and cognitive diagnosis were calculated to report:

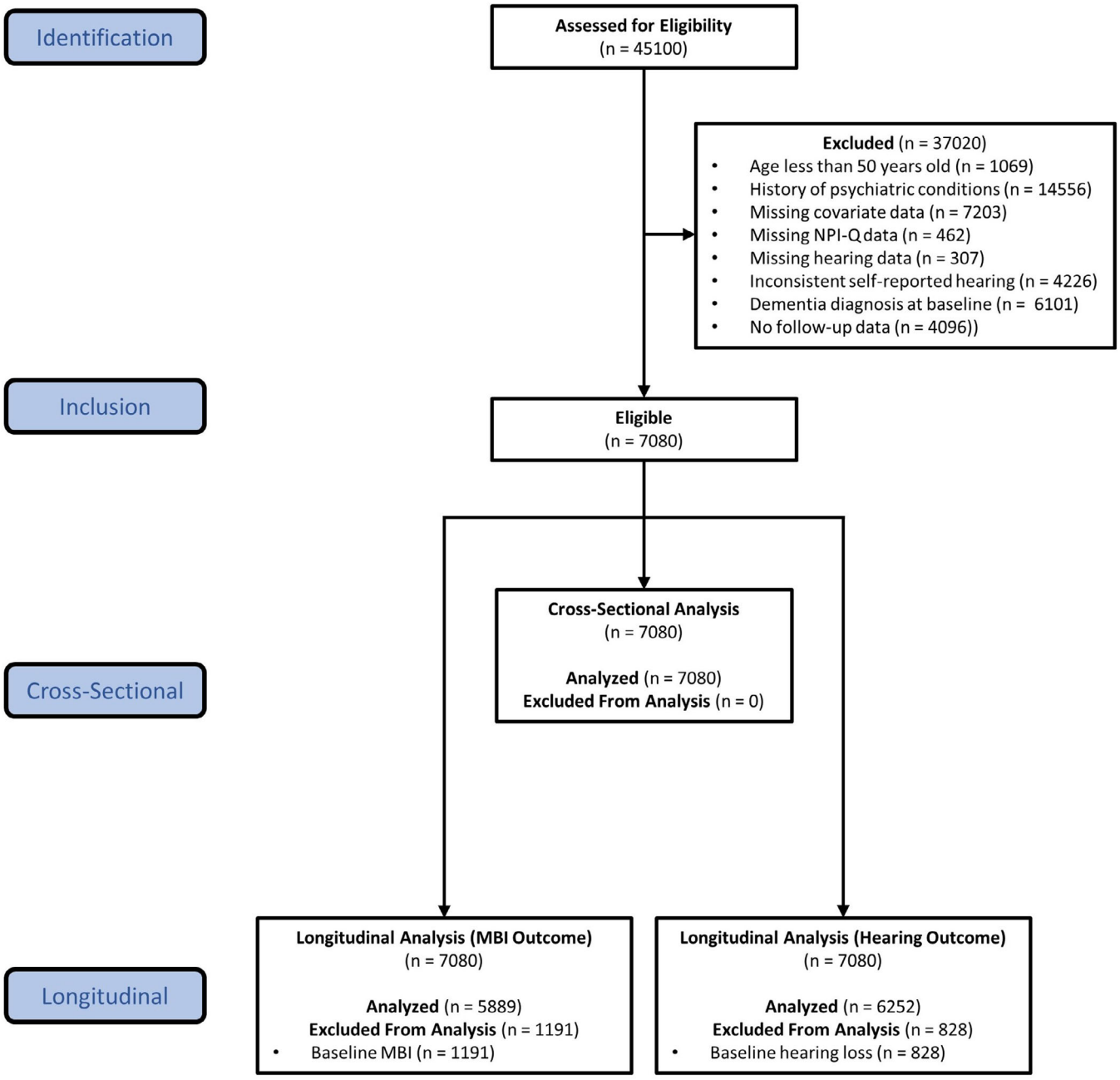

FIGURE 1 Participant flow chart. The cross-sectional analysis examined the relationship between hearing loss and global and domain-specific MBI status in older adults without dementia. Participants from the cross-sectional analysis, without MBI at baseline were used to examine the effect of hearing (exposure) on incident MBI as outcome in our first longitudinal analysis. Using a different subset of participants without HL at baseline, our second longitudinal analysis examined the effect of MBI (exposure) on incident Treated-HL or Untreated-HL as outcome. NPI-Q, Neuropsychiatric Inventory Questionnaire; MBI, mild behavioral impairment.

(1) the effect of Treated-HL as exposure (i.e., consistent hearing aid use) compared to No-HL on incident MBI; and (2) the effect of Untreated-HL as exposure (i.e., no hearing aid use) compared to No-HL on incident MBI. The hearing exposure and cognitive diagnosis variables were treated as time-dependent covariates in the models.

The second set of Cox proportional hazard models was used to examine the effect of MBI (exposure) on rates of incident HL, either Treated or Untreated, in a sample of 6252 participants with No-HL at baseline. Two hazard ratios adjusted for age, sex, education, APOE4, and cognitive diagnosis were calculated to report: (1) the effect of MBI+ status on incident hearing loss with hearing aid use (i.e., Treated-HL) compared to MBI-; and (2) the effect of MBI+ status on incident hearing loss without hearing aid use (i.e., Untreated-HL), compared to MBI-. The MBI exposure and cognitive diagnosis variables were treated as time-dependent covariates in these models.

TABLE 1 Baseline participant characteristics stratified by hearing loss status for the cross-sectional analysis.

Variable	Total	No hearing loss	Treated hearing loss	Untreated hearing loss	*p*-Value[a]	*p*-Value[b]
N	7080	6252	528	300		
Age (years)	71.6 (8.8), 50–100	70.6 (8.4), 50–100	79.3 (8.2), 58–100	78.3 (8.9), 53–100	<.001	.23
Sex (female)	4367 (61.7)	4014 (64.2)	212 (40.2)	141 (47.0)	<.001	.06
Education (years)	15.7 (3.0), 1–29	15.6 (3.0), 1–29	15.9 (2.9), 1–26	15.7 (3.2), 4–22	.09	.76
Diagnosis					<.001	<.001
CN	4926 (69.6)	4431 (70.9)	327 (61.9)	168 (56.0)		
SCD	381 (5.4)	319 (5.1)	26 (4.9)	36 (12.0)		
MCI	1773 (25.0)	1502 (24.0)	175 (33.1)	96 (32.0)		
APOE e4					<.001	.04
0	4575 (64.6)	3977 (63.6)	366 (69.3)	232 (77.3)		
1	2193 (31.0)	1986 (31.8)	145 (27.5)	62 (20.7)		
2	312 (4.4)	289 (4.6)	17 (3.2)	6 (2.0)		
MBI prevalence						
Any MBI	1191 (16.8)	1003 (16.0)	111 (21.0)	77 (25.7)	<.001	.13
Decreased motivation	296 (4.2)	244 (3.9)	30 (5.7)	22 (7.3)	.003	.35
Affective dysregulation	753 (10.6)	648 (10.4)	56 (10.6)	49 (16.3)	.005	.02
Impulse dyscontrol	774 (10.9)	641 (10.3)	83 (15.7)	50 (16.7)	<.001	.72
Social inappropriateness	169 (2.4)	137 (2.2)	17 (3.2)	15 (5.0)	.003	.20
Psychosis	63 (0.9)	53 (0.8)	5 (0.9)	5 (1.7)	.33	.36
MBI severity						
Any MBI	0.7 (1.6), 0–17	0.7 (1.6), 0–16	0.7 (1.7), 0–17	1.0 (2.0), 0–12	<.001	.03
Decreased motivation	0.1 (0.3), 0–3	0.1 (0.3), 0–3	0.1 (0.3), 0–2	0.1 (0.4), 0–3	.004	.21
Affective dysregulation	0.3 (0.7), 0–7	0.3 (0.7), 0–7	0.2 (0.7), 0–5	0.4 (0.8), 0–5	.08	.03
Impulse dyscontrol	0.3 (0.8), 0–7	0.3 (0.7), 0–7	0.3 (0.8), 0–7	0.4 (1.1), 0–6	<.001	.45
Social inappropriateness	0.0 (0.3), 0–3	0.0 (0.3), 0–3	0.1 (0.3), 0–3	0.1 (0.3), 0–2	.002	.09
Psychosis	0.0 (0.2), 0–6	0.0 (0.2), 0–4	0.0 (0.2), 0–4	0.0 (0.4), 0–6	.64	.36

Note: All values have been rounded to one decimal place, except for *p*-values which have been rounded to two or three decimal places, as appropriate. Continuous variables are shown in mean (standard deviation), range. Categorical variables are shown in *n* (%). Comparisons between hearing groups were tested using analysis of variance or independent samples *t*-tests or Mann-Whitney U tests for continuous variables and chi-squared or Fisher's exact test for categorical variables, as appropriate.

Abbreviations: APOE, apolipoprotein E; CN, cognitively normal; MBI, mild behavioral impairment; MCI, mild cognitive impairment; SCD, subjective cognitive decline.

[a]*p*-Value indicating the significance of the difference between no hearing loss, treated hearing loss, and untreated hearing loss groups.

[b]*p*-Value indicating the significance of the difference between treated and untreated hearing loss groups.

All statistical analyses were performed using R (version 4.0.2). Schoenfeld residuals were evaluated to confirm that the proportional hazards assumption was met ($p > .05$) for all Cox regression models. Given the number of statistical models, the false discovery rate (FDR) method was applied to all relevant regression *p*-values to adjust for multiple comparisons, resulting in adjusted *q*-values.

3 | RESULTS

3.1 | Cross-sectional analysis

Demographic characteristics of the 7080 participants in the cross-sectional analysis (62% female, mean age (±SD) 71.6 (±8.8) years) are included in Table 1. The sample comprised 75% CN/SCD and 25% MCI. Participants self-reporting No-HL were younger than those in the HL groups. The prevalence and severity of MBI symptoms tended to be greatest in participants with Untreated-HL, intermediate in those with Treated-HL, and lowest in those with No-HL (Figure 2). People with Untreated-HL had a higher prevalence of global MBI and affective dysregulation compared to those with Treated-HL whereas those with Treated-HL had slightly more APOE4 involvement than those with Untreated-HL. Table S1 shows a comparison between included participants and those excluded for missing or inconsistent NPI-Q and hearing data (i.e., participants whose self-report ratings changed from: HL to No-HL, Treated-HL to Untreated HL, and Untreated-HL to Treated-HL).

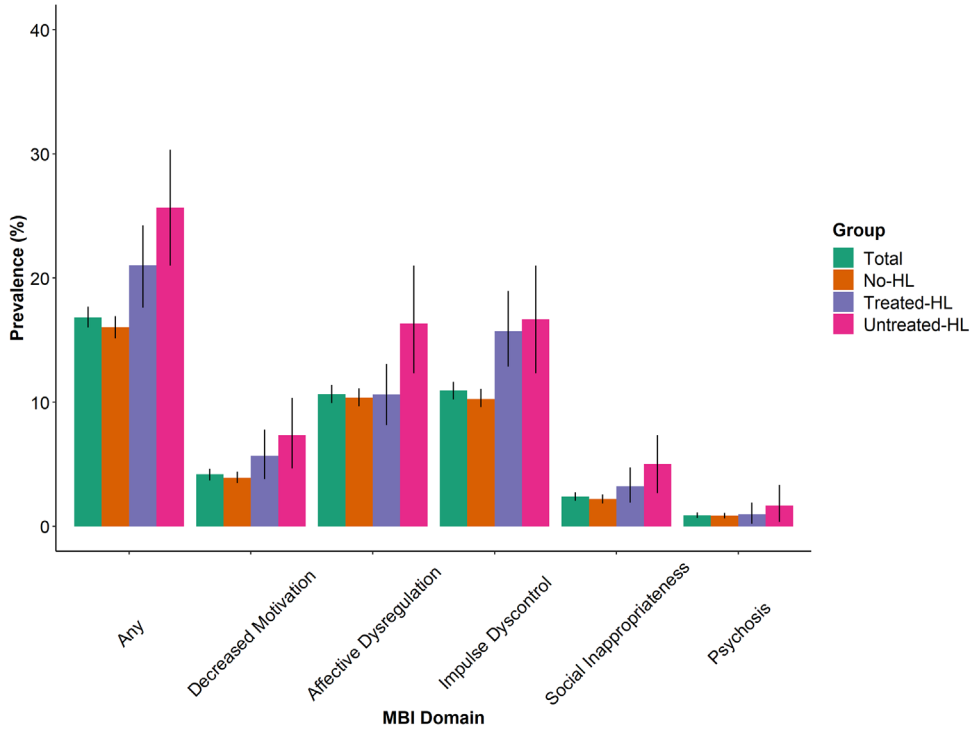

FIGURE 2 Prevalence of MBI across older adults with or without hearing loss. Vertical error bars indicate 95% confidence intervals obtained via bootstrapping. MBI, mild behavioral impairment; HL, hearing loss.

At baseline, participants with Untreated-HL (i.e., not wearing hearing aids) showed a 1.66-fold greater adjusted-odds of reporting symptoms of MBI than participants with No-HL (95% CI: 1.24–2.21, $q = .008$) (Table 2). MBI domain-specific analyses revealed that participants with Untreated-HL had a 1.95-fold greater adjusted-odds of reporting social inappropriateness (95% CI: 1.06–3.39, $p/q = .02/.06$), 1.71-fold greater adjusted-odds of reporting affective dysregulation (95% CI: 1.21–2.38, $q = .01$) and impulse dyscontrol (95% CI: 1.21–2.38, $q = .03$), relative to No-HL. For participants with no-HL or Treated-HL (i.e., consistent hearing aid use), no associations were observed for the presence of either global MBI and most individual MBI domains except impulse dyscontrol (aOR = 1.38, 95% CI: 1.05–1.81, $p/q = .02/.06$).

3.2 | Longitudinal analyses

Results of the first survival analysis of 5889 participants (64% female, mean age (±SD) 71.5 (±8.8) years) with a mean follow-up time of 4.2 (±3.2) years are included in Table 3. This analysis explored the effects of HL (whether treated by consistent hearing aid use or not) on the development of MBI, compared to No-HL. The No-HL group was younger, had more females, and more APOE4 involvement than the HL groups. A total of 962 (16%) participants developed MBI over the follow-up period. Participants with Treated-HL had a 1.29-fold higher adjusted hazard of developing MBI+ versus No-HL (95% CI: 1.01–1.63, FDR, $p/q = .04/.08$) whereas participants with Untreated-HL showed no significant difference in the hazard of developing MBI+ versus No-HL (aHR = 1.16, 95% CI: 0.80–1.70, $q = .43$) (Table 2).

Results of the second survival analysis of 6252 participants (64% female, mean age (±SD) 70.6 (±8.4) years) with a mean follow-up time of 4.1 (±3.1) years are included in Table 4. This analysis examined the effect of MBI (exposure) in hearing unimpaired participants on rates of incident HL, either Treated or Untreated. Participants with MBI at baseline were less educated, included more males, and were more likely to have MCI and APOE4 alleles than participants without MBI. A total of 662 (11%) participants developed HL over the follow-up period. Participants with MBI had a 1.51-fold higher adjusted hazard of developing Untreated-HL from No-HL compared to those without MBI (aHR = 1.51, 95% CI: 1.19–1.94, $q = .002$); participants with MBI showed no significant difference in the hazard of developing Treated HL from No-HL when compared to those without MBI (aHR = 1.24, 95% CI: 0.89–1.73, $q = .20$) (Table 2).

4 | DISCUSSION

In a cross-sectional sample of dementia-free older adults, Untreated-HL, compared to No-HL, was associated with greater odds of having MBI globally and in the domains of social inappropriateness, affective dysregulation, and impulse dyscontrol. In contrast, Treated-HL did not differ from No-HL for odds of global MBI and most individual MBI domains except impulse dyscontrol.

Previous research in tertiary memory care center participants with varied cognitive abilities (52% dementia, 27% MCI) found that cross-sectionally, hearing aid users had fewer and less severe NPS and less depressive symptomatology.[38] Half the participants had dementia, and

TABLE 2 Cross-sectional and longitudinal associations between treated and untreated hearing loss and MBI status.

	Treated hearing loss			Untreated hearing loss		
Variable		**95% CI**	***p/q***		**95% CI**	***p/q***
Cross-sectional logistic regression	aOR			aOR		
MBI status (outcome)						
Any MBI	1.19	0.93–1.51	.16/.29	1.66	1.24–2.21	<.001/.008
Decreased motivation	1.09	0.70–1.63	.70/.84	1.59	0.96–2.53	.06/.12
Affective dysregulation	1.00	0.73–1.35	.99/.99	1.71	1.21–2.38	.002/.01
Impulse dyscontrol	1.38	1.05–1.81	.02/.06	1.71	1.21–2.38	.002/.03
Social inappropriateness	1.14	0.64–1.92	.63/.84	1.95	1.06–3.39	.02/.06
Psychosis	1.09	0.36–2.64	.86/.94	1.88	0.63–4.52	.20/.30
Cox proportional hazard models	aHR			aHR		
Analysis 1						
MBI status (outcome)	1.29	1.01–1.63	.04/.08	1.16	0.80-1.70	.43/.43
Analysis 2						
MBI status (exposure)	1.24	0.89–1.73	.20/.20	1.51	1.19–1.94	<.001/.002

Note: The outcome variable for the cross-sectional logistic regression models ($n = 7080$) is the presence or absence of MBI and its domains after controlling for age, sex, education, cognitive status, and the number of apolipoprotein E (APOE) e4 alleles; the coefficient is adjusted odds ratios (aOR). For our first longitudinal analysis ($n = 5889$), a Cox proportional hazard model was used to examine the effect of hearing (exposure) on incident MBI as outcome. Specifically, adjusted hazard ratios (aHR) report the effect of Treated-HL (exposure) compared to No-HL (non-exposure) on incident MBI+ and the effect of Untreated-HL (exposure) compared to No-HL (non-exposure) on incident MBI+. For our second longitudinal analysis ($n = 6252$) Cox proportional hazard models were used to examine the effect of MBI (exposure) on incident Treated-HL or Untreated-HL as outcome. Specifically, adjusted hazard ratios (aHR) report the effect of the effect of MBI+ (exposure) compared to MBI- (non-exposure) on incident Treated-HL, and the effect of MBI+ (exposure) compared to MBI- (non-exposure) on incident Untreated-HL. All aHR are adjusted for age, sex, education, cognitive status, and the number of APOE e4 alleles. Hearing loss group, MBI status, and cognitive status were modelled as time-dependent covariates in the Cox regressions, as appropriate. All adjusted *p*-values, indicated by a *q*-value, were adjusted for multiple comparisons using the false discovery rate (FDR) method.
Abbreviations: 95% CI, 95% confidence interval; MBI, mild behavioral impairment.

TABLE 3 Baseline participant characteristics for the longitudinal analysis with incident MBI as the outcome.

Variable	**Total**	**No hearing loss**	**Treated hearing loss**	**Untreated hearing loss**	***p*-Value[a]**	***p*-Value[b]**
N	5889	5249	417	223		
Age (years)	71.5 (8.8), 50–100	70.6 (8.4), 50–100	79.4 (8.3), 58–100	78.6 (9.0), 56–100	<.001	.35
Sex (female)	3787 (64.3)	3501 (66.7)	180 (43.2)	106 (47.5)	<.001	.29
Education (years)	15.7 (3.0), 1–29	15.7 (3.0), 1–29	16.0 (2.9), 1–26	15.7 (3.2), 4–21	.24	.70
Diagnosis					<.001	.006
CN	4448 (75.5)	4014 (76.5)	288 (69.1)	146 (65.5)		
SCD	284 (4.8)	240 (4.6)	19 (4.6)	25 (11.2)		
MCI	1157 (19.6)	995 (19.0)	110 (26.4)	52 (23.3)		
APOE e4					<.001	.18
0	3852 (65.4)	3384 (64.5)	295 (70.7)	173 (77.6)		
1	1796 (30.5)	1638 (31.2)	112 (26.9)	46 (20.6)		
2	241 (4.1)	227 (4.3)	10 (2.4)	4 (1.8)		

Note: All values have been rounded to one decimal place, except for *p*-values which have been rounded to two or three decimal places, as appropriate. Continuous variables are shown in mean (standard deviation), range. Categorical variables are shown in *n* (%). Comparisons between hearing groups were tested using analysis of variance or independent samples *t*-tests or Mann-Whitney U tests for continuous variables and chi-squared or Fisher's exact test for categorical variables, as appropriate.
Abbreviations: APOE, apolipoprotein E; CN, cognitively normal; MCI, mild cognitive impairment; SCD, subjective cognitive decline.
[a]*p*-Value indicating the significance of the difference between no hearing loss, treated hearing loss, and untreated hearing loss groups.
[b]*p*-Value indicating the significance of the difference between treated and untreated hearing loss groups.

TABLE 4 Baseline participant characteristics for the longitudinal analysis with hearing loss as the outcome.

Variable	Total	MBI-	MBI+	*p*-Value
n	6252	5249	1003	
Age (years)	70.6 (8.4), 50–100	70.6 (8.4), 50–100	70.5 (8.2), 50–96	.93
Sex (female)	4014 (64.2)	3501 (66.7)	513 (51.1)	<.001
Education (years)	15.6 (3.0), 1–29	15.7 (3.0), 1–29	15.4 (3.3), 2–29	.03
Diagnosis				<.001
CN	4431 (70.9)	4014 (76.5)	417 (41.6)	
SCD	319 (5.1)	240 (4.6)	79 (7.9)	
MCI	1502 (24.0)	995 (19.0)	507 (50.5)	
APOE e4				.001
0	3977 (63.6)	3384 (64.5)	593 (59.1)	
1	1986 (31.8)	1638 (31.2)	348 (34.7)	
2	289 (4.6)	227 (4.3)	62 (6.2)	

Note: All values have been rounded to one decimal place, except for *p*-values which have been rounded to two or three decimal places, as appropriate. Continuous variables are shown in mean (standard deviation), range. Categorical variables are shown in *n* (%). Comparisons between hearing groups were tested using ANOVA or independent samples *t*-tests or Mann-Whitney U tests for continuous variables and chi-squared or Fisher's exact test for categorical variables, as appropriate.

Abbreviations: APOE, apolipoprotein E; CN, cognitively normal; MBI, mild behavioral impairment; MCI, mild cognitive impairment; SCD, subjective cognitive decline.

over 90% of this memory clinic sample presented with at least one NPS. Our baseline findings extend this work to a non-dementia population, where 17% had MBI symptoms. These findings suggest that hearing aid use may offer an early intervention associated with lower odds of concurrent global MBI symptomology in dementia-free participants. In contrast, with 219 dementia-free participants from the Canadian Comprehensive Assessment of Neurodegeneration and Dementia (COMPASS-ND) study, we did not find an association between the NPS of MBI and hearing aid use. A combination of a shorter single timepoint assessment of NPS (conferring lower specificity of NPS assessment for identifying behavioral symptoms secondary to neurodegenerative disease), low uptake of hearing aids, and a screening audiometry approach that identified normal hearing ability in much of our sample may explain this lack of association.[37] While a link between hearing and social inappropriateness is new to this study, our prior work has shown links with affective dysregulation and impulse dyscontrol.[37] As for the association between Treated-HL and impulse dysregulation, this may have been influenced by greater APOE4 and we suspect, greater hearing loss severity, in those using hearing aids compared to those without. However, it is also possible that hearing aids are as effective in targeting impulse dysregulation in people with HL. Hearing aids are designed to improve audibility and speech understanding which can enhance social interaction, and hearing aids have been shown to relieve loneliness and depressive symptoms.[34–36]

It is unclear whether the mechanisms linking HL and MBI involve a causal relationship and/or a common etiology. Of relevance to MBI are the causal mechanisms (i.e., social engagement, cognitive load, and structural brain changes) proposed to link HL with cognitive decline and with late-life depression.[43] Many older adults have difficulty hearing with background noise and will avoid or withdraw from situations where listening is too effortful. These hearing difficulties may result in social isolation with changes in mood, affect, and irritability. A previous model posits that to support effortful listening compensatory neuroplastic changes occur, which involve an increase in activation of the cognitive control network in response to degraded auditory input. In addition, and in agreement with other mechanistic research, owing to auditory deafferentation associated atrophy of the prefrontal cortex and anterior cingulate cortex, along with dysfunctional auditory-limbic connectivity there is greater executive and affective dysregulation, which can influence late-life depression with consequent cognitive decline.[44] Regarding potential common etiologies, HL and MBI—both prevalent in preclinical and prodromal disease—have shown associations with AD biomarkers like amyloid-β, p-tau, and tau, in advance of dementia-related cognitive and functional changes.[15,16,26,28,32] Reviews of neuroimaging studies of older adults with HL or depression have shown similar patterns of diminished activity in the limbic system, frontal cortex, and auditory cortex.[34,43,44] Additionally, research involving facial emotion recognition—a measure of social cognition commonly disrupted in people with dementia—identified atrophy of the right insula, right hippocampus, bilateral cingulate cortex, and multiple areas of the temporal cortex in people with HL but not in controls with No-HL.[45] This lack of facial emotional recognition may explain why Untreated-HL was associated with a higher score on the social inappropriateness domain of MBI. Taken together, the biomarker and neuroimaging evidence of common neural degeneration suggest that MBI and at least Untreated-HL are possible sequelae of a shared neurodegenerative disease etiology. The fact that cross-sectionally we did not find an association between Treated-HL and global MBI suggests that remediation of HL with hearing aid treatment may prevent MBI, warranting further investigation.

Longitudinally, we found associations between Treated-HL and incident MBI and between MBI and incident Untreated-HL. The results of our first longitudinal analysis were surprising as we predicted hearing aid use would have a protective effect on MBI development. However, in a study of cognitively unimpaired adults ≥60 years, Treated-HL rather than Untreated-HL was associated with greater risk for depression and incident dementia.[36] Deconstructing the HL component of our composite hearing variable offers insights. With age, prevalence and severity of HL increases, and people with more severe HL are more likely to use hearing aids.[18,46] Our participants with HL were 9–10 years older than those reporting No-HL. We speculate that the Treated-HL group had greater HL severity than the Untreated-HL group, requiring hearing aid use, and accounting for the association with Treated-HL. These interpretations align with studies that have found increasing degrees of HL associated with cognitive decline, incident dementia, lower social engagement, and poorer health outcomes.[4,6,20,24,43,47] Using five groups (i.e., No-HL, unaided mild-HL, aided mild-HL, unaided-severe-HL, and aided-severe-HL), one study found that unaided-severe-HL (but not unaided-mild-HL) was associated with poorer health outcomes; for those who were aided with severe-HL, hearing aids reduced the magnitude of the association with depression, low social support, and mobility limitations. This study lends support to the idea that to uncover the benefits of hearing aid use, the degree of HL needs to be considered.

The results of our second longitudinal analysis demonstrated that MBI was associated with incident Untreated-HL. It is possible that MBI may lead older adults who develop HL to be less likely to regularly wear hearing aids. In contrast, participants with or without MBI showed no significant difference in the hazard of developing Treated-HL. Overall, our participants with MBI were less educated, proportionally had more MCI, and more APOE4 involvement than those without MBI, suggestive of a group further along the neurodegenerative disease continuum.[4,24–33] Ultimately, randomized clinical trials will be needed to determine definitively whether hearing aids reduce the risk of MBI, and conversely, if treatment of MBI reduces the incidence and/or severity of HL.

The strength of our large sample size is offset by some limitations. The NACC-UDS data rely on a binary yes/no approach to self-reported HL. Research comparing audiometric data with self-report has shown that with greater levels of cognitive impairment, HL may go underreported and unaddressed (i.e., the sensitivity of self-rated hearing dropped from 71% for CN adults to 61% for adults with MCI to 53% for persons with dementia).[48] Hearing aid use could be a surrogate for socioeconomic factors, financial resources, access to healthcare, the ability and willingness to use amplification and availability of social support—variables not collected in the UDS. Further, to minimize the bias arising from self-reported HL and hearing aid use status, we included only participants with complete and consistent reporting of HL and hearing aid use/non-use. With regard to behavior, MBI was derived from the NPI-Q which uses a 1-month reference period. Although MBI was operationalized with two consecutive visits, this approach may still include some transient symptomology, less likely to be associated with underlying neurodegenerative disease.

Future studies investigating hearing and MBI should address these limitations by using objective audiometric measures to quantify HL, datalogging to detail frequency and duration of actual hours of hearing aid use, and the MBI-Checklist[21] as the validated case-ascertainment tool for MBI. In addition, onset of HL has also emerged as a factor that could have implications for future MBI work. Research investigating depressive symptoms and social network strength found that people with longstanding hearing problems had smaller social networks, more depressive symptoms at baseline, and a greater increase of depressive symptoms over time, whereas those with a new onset of HL were more likely to report a decline in their social network over time.[49] Further study is needed to investigate temporal relations to definitively determine if HL precedes the onset of MBI or vice versa. Last, including biomarkers and neuroimaging will help determine the neural mechanisms linking hearing with MBI and may illuminate how hearing aids influence this relationship.

Our cross-sectional findings, while statistically significant, are also clinically meaningful as they support the potential benefits of amplification to reduce the likelihood of global MBI in a dementia-free population. Longitudinally, we found associations between Treated-HL and MBI, which we feel are likely tempered by HL severity being greater in those with Treated-HL. We also found associations between MBI and incident Untreated-HL with our MBI+ sample identifying more severe neurodegenerative disease. Finally, we must emphasize that hearing aid treatment is only one aspect of a comprehensive program of aural rehabilitation. Due to poor help-seeking among people with HL, in addition to hearing aids targeted interventions (possibly offered online) could include use of assistive listening devices and communication aids, interventions to enhance communication skills and communication strategy use, environmental modifications to optimize listening in noisy and challenging situations and, psychosocial counseling to offer coping strategies, social support, and assistance to accept HL.[34,35,50]

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CONFLICT OF INTEREST STATEMENT

Zahinoor Ismail: Consulting/Advisory Boards for Otsuka/Lundbeck; consulting fees paid to institution by Roche. Eric E. Smith: Consulting/Advisory Boards for Eli Lilly. All remaining authors declare no conflicts. Author disclosures are available in the supporting information.

CONSENT STATEMENT

All contributing ADRCs were required to administer standardized forms, obtain informed consent from all participants and their informants, as well as IRB approvals prior to submitting data to NACC.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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