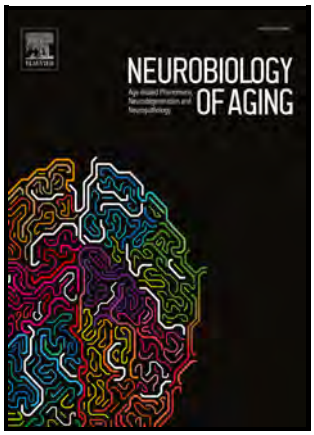

Age-related hearing loss associated with differences in the neural correlates of feature binding in visual working memoryRunning title: ARHL and neural correlates of memory

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Age-related hearing loss associated with differences in the neural correlates of feature binding in visual working memory

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Abstract

The underlying neural mechanisms underpinning the association between age-related hearing loss (ARHL) and dementia remain unclear. A limitation has been the lack of functional neuroimaging studies in ARHL cohorts to help clarify this relationship. In the present study, we investigated the neural correlates of feature binding in visual working memory with ARHL (controls = 14, mild HL = 21, and moderate or greater HL = 23). Participants completed a visual change detection task assessing feature binding while their neural activity was synchronously recorded via high-density electroencephalography. There was no difference in accuracy scores for ARHL groups compared to controls. There was increased electrophysiological activity in those with ARHL, particularly in components indexing the earlier stages of visual cognitive processing. This activity was more pronounced with more severe ARHL and was associated with maintained feature binding. Source space (sLORETA) analyses indicated greater activity in networks modulated by frontoparietal and temporal regions. Our results demonstrate there may be increased involvement of neurocognitive control networks to maintain lower-order neurocognitive processing disrupted by ARHL.

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Abbreviations: AD = Alzheimer's Disease; ARHL = age-related hearing loss; CPL = left centro-parietal; CPR = right centro-parietal; FCL = left fronto-central; FCR = right fronto-central; MCI = Mild Cognitive Impairment; MCI-FAD = Mild Cognitive Impairment - Familial Alzheimer's Disease; POL = left parieto-occipital; POR = right parieto-occipital; ROI = Region of Interest; sLORETA = standardised low-resolution brain electro-magnetic tomography; VSTMB = visual short-term memory binding which involved encoding and retrieval across two conditions shapes and binding giving four phases of interest (shapes-encoding, shapes-retrieval, binding-encoding, and binding-retrieval);

Introduction

Recent epidemiological evidence reports a significant association between age-related hearing loss (ARHL) and a greater risk of developing dementia (Livingston et al., 2020; Loughrey, Kelly, Kelley, Brennan, & Lawlor, 2018). It is estimated that by 2050, 2.5 billion people globally will suffer from hearing loss (WHO, 2021), making this association potentially consequential for global health policy. However, it remains unclear what the

neurocognitive mechanisms underpinning this relationship are as hearing loss has been understudied compared to other risk factors for dementia (Griffiths et al., 2020; Panza, Solfrizzi, & Logroscino, 2015). Clarifying this relationship is crucial for guiding future research and public health approaches that aim to reduce the prevalence of dementia through management of ARHL.

An important potential mechanism is the neural changes in the brain following onset of ARHL. From the early stages of ARHL, a functional reorganisation has been observed in frontal and temporal regions in response to simple visual and speech stimuli (Campbell & Sharma, 2013, 2014, 2020). Structural differences have also been observed in temporal regions of the brain important for memory that are affected in the initial stages of Alzheimer's Disease (AD) (Armstrong et al., 2019; Lin et al., 2014; Slade et al., 2022). Behavioural research suggests that ARHL may be linked initially with subtle differences in low-level automatic cognitive processing (Gillingham, Vallesi, Pichora-Fuller, & Alain, 2018; Loughrey, 2018). However, it is unclear if these cognitive differences are linked to the observed changes in neural function. There is currently a lack of neuroimaging studies in ARHL cohorts which examine the neural correlates of cognitive function that would help clarify this.

Prior research has indicated that ARHL may be associated with poorer binding of visual features of an object compared to single features in short-term memory (Loughrey, Parra, & Lawlor, 2019). Temporarily integrating visual perceptual features of an object (e.g., shapes and colours) is an automatic process in working memory that relies on communication between specialised neural regions (Didic et al., 2011; Luck et al., 2010; Parra et al., 2017; Staresina & Davachi, 2010) which may be disrupted with ARHL (Armstrong et al., 2019; Husain et al., 2011; Lin et al., 2014). It may also be sensitive to functional connectivity abnormalities related to AD (Parra et al., 2017), including its preclinical stages (Dubois et al., 2016; Parra et al., 2010). Therefore, feature binding may provide a potential marker that would be useful for future research endeavouring to elucidate the neural mechanisms underlying ARHL and dementia.

The aim of this preliminary study was to examine the behavioural and neural correlates of binding memory function with ARHL using the visual short-term memory binding (VSTMB) task and electroencephalography (EEG). Based on our prior study findings, we hypothesised that ARHL would be associated with maintained memory for single-features, but poorer

feature-binding compared to controls and that these differences would increase with greater severity of ARHL. We performed exploratory analysis of electrophysiological correlates of task performance to investigate differences in neurocognitive processing with ARHL. We included subgroup analyses comparing the electrophysiological correlates across groups stratified by level of feature binding maintenance performance. We also performed exploratory analysis of estimated cortical sources to investigate differences between groups in the activity of neural anatomical regions underlying VSTMB.

Materials and methods

Participants

Participants aged 65 years and over were recruited from the public and from a pool of volunteers who consented to be contacted for this study in Ireland. Volunteers were screened and were excluded if they had a history of psychiatric or neurological illness or injury, substance abuse, diabetes, stroke, severe motor impairment; were taking certain medications for a psychiatric condition; had a personal or family history of epilepsy or unexplained fainting or sensitivity to flickering light; or had hearing loss from the congenital/pre-lingual stage or due to injury or disease. Participants gave consent prior to testing. Testing was conducted between September and December 2019. Ethical approval for this study was granted by the School of Psychology Research Ethics Committee in Trinity College Dublin.

Background measures

Participants were administered the Montreal Cognitive Assessment (MoCA) (Nasreddine et al., 2005). Several tasks from the Cambridge Neuropsychological Test Automated Battery (CANTAB®) were also administered; Motor Screening Task, Multitasking Test, Paired Associates Learning; the Reaction Time simple and five-choice tests and Spatial Working Memory. Tasks were selected based on the function they assessed and their accessibility to a hearing-impaired sample. A self-report questionnaire was used to collect data on demographic and lifestyle factors, such as physical and mental health, alcohol consumption and smoking from participants. More details on the background and CANTAB measures are included in the Supplementary Materials.

Hearing measure

A measure of pure-tone air conduction hearing sensitivity was self-administered by participants using the uHear™ application (version 1.3.7, Unitron, Victoria, BC, Canada) on an iPad. The uHear™ app has been validated and it produces an audiogram making it among the most used hearing test apps in research (Barczik & Serpanos, 2018; Handzel et al., 2013; Irace, Sharma, Reed, & Golub, 2021; Shilo et al., 2022; Szudek et al., 2012). The app was administered to detect pure-tone decibel thresholds across a range of frequencies; 0.5, 1, 2, 4 & 6 kilohertz (kHz). The hearing measure was conducted in a testing room with an ambient noise level of less than 35 dB, as measured by a Casella CEL-240 Digital Sound Level Meter. Participants were instructed on how to self-administer the uHear™ app and wore noise-cancelling headphones. Participants were allocated to the control group (CG), mild hearing loss group (MLD), and moderate or greater hearing loss group (MOD+) according to their pure-tone average (PTA) of decibel hearing loss (dB HL) across these frequencies. After all measures were completed, those with a PTA of <26 dB HL were allocated to the CG, those with a PTA ≥26 dB HL to the MLD, and those with a ≥41 dB HL to the MOD+.

Visual Short-Term Memory Binding Task

As part of the VSTMB task, participants are asked to remember a visual array of objects during the encoding phase (500ms) and, after a brief pause (900ms), detect if a change has occurred when visually prompted again with another array of objects in the retrieval phase displayed until they provided a response (Figure 1). This is done across two conditions; a visual array of three shapes (shapes) is presented in the first condition and then three coloured shapes (binding) in the second condition. Across 50% of the trials there was a change and for the remaining trials there was no change. Changes consisted of new features replacing studied features (shapes) or features swapping across items (binding). The items changed location from the encoding to the retrieval phases so that the location could not be used as an assistive feature to aid recall. The VSTMB task uses no nameable geometric shapes and non-primary colours to avoid or reduce phonological coding of the stimuli, thus it was not checked if participants used verbal mediation.

Participant sat in front of a computer in a dimly lit room during the task. For both conditions, participants were given a brief practise session followed by 100 trials per condition (200 trials in total). Participants inputted their response on an Ergodex DX1 keyboard adapted. The board had two keys marked 'same' and 'different'. Participants pressed an additional key to start the next trial. Participants were offered short breaks halfway through each condition and between conditions. An EEG recording was synchronously conducted using a high-density array (ActiveTwo Biosemi™ electrode system, including 128 scalp electrodes). The sampling rate was set at 4096 Hz, and signals were bandpass filtered between 0.16 Hz (high pass) and 100 Hz (low pass). The test was presented using E-Prime 2.0©, (Psychology Software Tools, Inc, Sharpsburg, PA, USA).

Data analysis

Comparisons of background and behavioural data between groups were performed via using one-way analysis of variance (ANOVA) followed by Bonferroni-corrected post hoc tests or chi-squared test and their nonparametric alternatives (Howell, 2002). Normality of continuous data was examined using the Kolmogorov–Smirnov test (Howell, 2002). The VSTMB accuracy score within both (shapes and binding) conditions was calculated as the proportion of correct trials (i.e., correction recognition of change trials and rejection of same trials) (Parra et al., 2010; Pietto et al., 2016). VSTMB mean reaction time was reported in seconds. Additionally, for the primary behavioural outcome we calculated a weighted binding score (binding accuracy score minus shapes accuracy score) to examine the difference in binding function adjusted for performance on shapes condition. This was to examine differences between groups in binding function adjusted for differences in memory for single features. Analysis of this data was conducted in SPSS statistical software version 26.

Preparation and analyses of EEG data were conducted using MATLAB (version 2020a) and Brainstorm (Tadel, Baillet, Mosher, Pantazis, & Leahy, 2011), a documented and freely available software under the GNU general public license (http://neuroimage.usc.edu/brainstorm). The data preparation and analytical approach to the electrophysiological correlates was adapted from that followed by Pietto et al. (2016). There were four phases of interest in this analysis: shapes-encoding, shapes-retrieval, binding-encoding, and binding-retrieval. These phases were locked to onset of the encoding or

retrieval stimulus for the shapes and binding conditions. The data was band-pass filtered between 0.5 Hz (high-pass) and 30 Hz (low-pass) and down-sampled to 256 Hz. Bad channels were marked and removed. EEG activity was re-referenced to the mean across channels. Independent components analysis (ICA) was performed to detect and remove oculomotor artifacts. Epochs were imported (-200 to 1000 ms) for correct response trials only. Channels with artifacts +/- 100 μV were removed and trials were removed if necessary. Five trials from four subjects were rejected. There was no difference in rejected trials by group or outcome or their interaction ($p > 0.1$). Averages were computed for each of the four phases for all subjects.

The main analyses focused on differences in P1, N1, P2, P3 and late positive potential (LPP) ERP components between groups. These ERPs, apart from P1, have been explored in a previous study examining VSTMB in a group of patients with mild cognitive impairment (MCI) and a group in the MCI stages of familial AD (MCI-FAD) (Pietto et al., 2016). There were 6 regions of interest (ROI) with 14 electrodes allocated to each ROI (fronto-central/centro-parietal/parieto-occipital regions for left and right hemispheres – FCL, FCR, CPL, CPR, POL, and POR) (Supplementary Table S1). The ROIs and the electrodes allocated to each ROI were selected following the methodology by Pietto et al. (2016). Pietto et al. (2016). To identify significant differences between groups, nonparametric permutation tests were run in which the permutations were approximated using a Monte-Carlo approach (4,000 permutations). Significant sensors ($p<0.01$) were then identified within each ROI. These sensors were then averaged within each of the 6 ROIs and across four-time windows (80-140 ms, 140-250 ms, 250-550 ms and 550-900 ms). This averaged activity was compared independently for each ROI and time window using permutation tests with a Monte-Carlo approach (4,000 permutations) ($p <0.05$). This method does not depend on Gaussian data distribution assumptions and offers a straightforward solution for multiple comparison problems (Pietto et al., 2016). To adjust for multiple comparisons across the six prospectively assessed ROIs for each ERP time window of interest, we further applied a False Discovery Rate (FDR) correction for these six comparisons.

Analyses were conducted, *post hoc,* on the electrophysiological data comparing the groups stratified by high and low scorers and within each group between high and low scorers. Groups were divided into high and low performers according to the median weighted binding score of the entire sample. This was to explore differences in electrophysiological correlates between groups stratified by binding function ability adjusted for shapes score. These

analyses followed the same procedure as for the main analyses. An additional *post hoc* analysis was conducted to explore differences in the association of ERP activity with maintenance of binding function across groups. The mean amplitudes for ROIs across time windows that were associated with ERPs, calculated in the main analyses, were correlated with the weighted binding score using Spearman's rank correlation coefficient. For both the P1 and N1 components, we used the mean activity from the bilateral parieto-occipital regions during the first- and second-time windows (80-140 ms and 140-250 ms), respectively. For the P2 and P3 components, data was used from the bilateral fronto-central and centro-parietal regions during the second- and third-time windows (140-250 ms and 250-550 ms), respectively. For the LPP component, data from the bilateral fronto-central regions during the final time window (550-900 ms) was used.

EEG estimated source imaging was visualised using the default anatomy derived from a non-linear average of T1 MRI scans from 152 participants from the Montreal Neurological Institute (MNI) (Fonov et al., 2011) using the Brainstorm platform (Tadel et al., 2011). Forward head models of the cortex surface were formulated for each participant using the Boundary Element Method (BEM) from the open-source software OpenMEEG (Gramfort, Papadopoulo, Olivi, & Clerc, 2010; Kybic et al., 2005). For inverse modelling, standardised low-resolution brain electro-magnetic tomography (sLORETA) (Pascual-Marqui, 2002) was used to compute cortical maps for each participant. We used minimum norm imaging with unconstrained source orientations (15,002 sources in three orthogonal directions), depth weighting and a 3-dB signal-to-noise ratio. The noise covariance was calculated using the baseline period of the included trials and noise covariance regularization was set to 0.1. Cortical maps were created for the averaged outcome for each participant and were then normalized to baseline and flattened. Nonparametric testing, derived from 4000 randomizations, were conducted on the averaged differences between groups across the same four-time windows. This approach has demonstrated effectiveness in controlling Type I error in neuroimaging studies (Nichols & Holmes, 2002). Statistical maps of the cortex were then generated with t-values for each voxel ($p < 0.05$) across time windows. Regions where at least 20 voxels had a threshold of $p < 0.05$ were identified based on the parcellation scheme according to the Desikan-Killiany adult cortical atlas and the statistical data (peak t value) was tabulated along with the number of significant voxels and MNI coordinates (Desikan et al., 2006).

Data availability statement

The data in this study is available upon a reasonable request to the corresponding author.

Results

Participant characteristics

For this study, 60 people were assessed and two were excluded due to incomplete data and poor EEG data quality. There were 58 people included in the analyses; 14 people in CG, 21 people in MLD, and 23 people in MOD+. There were no significant differences between groups on background factors although the difference for age trended toward significance (Table 1). All groups were significantly different ($p < 0.01$) for PTA of dB HL in both the better and the worse ear. For self-reported hearing impairment (HHIE-S), MOD+ was significantly different from CG ($p < 0.01$). Three people in MLD and five people in MOD+ reported wearing hearing aids. Two people in the MLD and seven people in the MOD+ reported experiencing tinnitus. There were no significant differences between groups in the MOCA or on any CANTAB measure. The group data for the CANTAB measures and other background measures are reported in Supplementary Table S2.

VSTMB task behavioural results

The behavioural results from the VSTMB task are listed in Table 2. There were no significant group differences in change in accuracy from shapes to binding conditions (weighted binding score; $F = 0.32$, $p = 0.73$). There were no significant differences between groups on the VSTMB task on the shapes condition in accuracy score ($U = 1.44$, $p = 0.49$) or mean reaction time ($U = 0.79$, $p = 0.67$). There were also no differences between groups on the binding accuracy score ($F = 0.72$, $p = 0.49$) or mean reaction time ($U = 2.15$, $p = 0.34$).

Main electrophysiological findings

Summary

There were several differences between groups in electrophysiological outcomes. A summary of the results is listed in Table 3. Statistical data is listed in Table 4 and ERPs are displayed in Figures 2-4. In summary, for the MLD compared to the CG, differences were mainly for P2. For the MOD+ compared to controls, differences were mostly for P2 and P3. Differences were mostly due to greater amplitude from the HL groups. For the MOD compared to the MLD, differences were found for P1 and P2 across all phases and for LPP across the first three phases. The MOD+ had a greater amplitude for P1 across all phases and the MLD had greater amplitude for P2 and LPP except for shapes retrieval.

Mild HL group versus control group

During the shapes-encoding phase, there was a significant difference in P2 over CPL (t = 2.05, p = 0.04). During the binding-encoding phase, there was a significant difference in P2 over bilateral FC (left: t = 2.08, p = 0.046, right: t = 2.09, p = 0.04) regions and in P3 over POL (t = -2.18, p = 0.03). There was a significant difference in P2 during the binding-retrieval phase over CPR (t = 2.29, p = 0.03) and in LPP over FCR (t = 2.23, p = 0.03). Except for P3 during the binding-encoding phase, MLD had the greater mean amplitude in these comparisons. None of these results remained significant after the FDR adjustment.

Moderate or greater HL group versus control group

During the shapes-encoding phase, there was a significant difference in P2 over CPR (t = -2.36, p = 0.02), and bilateral FC (left: t = -2.13, p = 0.05, right: t = -2.8, p = 0.01, FDR p < .05) regions with CG having a greater positive going amplitude. There was also a difference in N1 over POR (t = -2.17, p = 0.04) with MOD+ having a greater mean amplitude. MOD+ also had a greater mean amplitude for several components during the shapes-retrieval phase. There was a significant difference in P2 over FCL (t = 2.08, p = 0.04); in P3 over FCL (t = 2.71, p = 0.01), bilateral CP (left: t = 2.2, p = 0.03, right: t = 2.05, p = 0.04) regions; and in LPP over bilateral CP (left: t = 2.55, p = 0.02, right: t = 2.92, p = 0.003, FDR p < .05) and FCL (t = 2.33, p = 0.02, FDR p < .05) regions. On the binding-encoding phase, there was a

significant difference in P3 due to greater amplitude from CG over the CPL (t = -2.31, p = 0.02) and POL (t = -2.24, p = 0.04) regions. In the POL region this extended into the 550-900ms time window (t = -2.29, p = 0.03).

Moderate or greater HL group versus mild HL group.

During the shapes-encoding phase, there was a significant difference in P1 over the POR (t = 2.16, p = 0.04); in P2 over the CPL (t = -2.11, p = 0.04); and in LPP over the CPL (t = -2.47, p = 0.01) and POL (t = -2.21, p = 0.03) regions. Apart from P1, MLD had the greater mean amplitude. As in comparisons with CG, MOD+ had a greater mean amplitude in several components for the shapes-retrieval phase. There was a significant difference in P1 over the POR (t = 2.63, p = 0.01); in P2 over the CPL (t = 2.19, p = 0.04), CPR (t = 2.37, p = 0.02) and FCL (t = 2.81, p = 0.01) regions; in P3 over the CPR (t = 2.33, p = 0.02) and FCL (t = 2.43, p = 0.02) regions; and in LPP over CPR (t = 3.04, p = 0.003, FDR p < .05).

During the binding-encoding phase, MOD+ had a significantly greater mean amplitude in P1 over the POR (t = 2.87, p = 0.005, FDR p < .05) and in LPP over bilateral CP (left: t = 2.95, p = 0.004, FDR p < .05, right: t = 3.01, p = 0.003, FDR p < .05) regions. MLD had a greater mean amplitude in P2 over FCR (t = -2.59, p = 0.02, FDR p < .05). During the binding-retrieval phase, MOD+ had a greater mean amplitude for P1 over bilateral PO regions (left: t = 2.48, p = 0.02, FDR p < .05, right: t = 2.96, p = 0.003, FDR p < .05). There was a significant difference in P2 over bilateral CP regions (left: t = -2.25, p = 0.03, FDR p < .05, right: t = -2.75, p = 0.01, FDR p < .05) with MLD having the greater mean amplitude.

Secondary electrophysiological findings

A summary of findings from the secondary analyses is provided here. The tabulated results (Table S3-S4) from these analyses along with figures (Figure S1.1 – S2.3) is available in the Supplementary Materials. Groups were split according to the overall sample median binding accuracy score adjusted for shapes score (-10.5). The numbers of participants for each group were as follows; CG high/low = 6/8, MLD high/low = 12/9, MOD+ high/low = 10/13. There were no significant differences between groups in age, sex, or education, or in VSTMB outcomes within both stratifications for performance level (Kruskal-Wallis test: p > 0.05). Within groups, there were no differences in sex or education. There was a significant

difference in age between high and low performers in CG only (Mann-Whitney U test: $p < 0.05$).

Comparisons were made between groups, stratified by high and low performers. There were more differences in electrophysiological activity between high performers than between low performers. For MLD vs CG high performers, differences were mainly in P2 across the last three phases with MLD having greater activity in the last two phases. For MOD+ vs CG high performers, there were differences mainly in P2 and P3 across all phases, and to a lesser extent, in P1 and LPP. MOD+ had greater activity in P2 across all phases (during shapes-retrieval both groups had greater activity in different regions). Most differences were due to MOD+ having less activity than CG in the first phase and greater activity in the retrieval phases. For MOD+ vs MLD group high performers, differences were mainly in P1 (across all phases) and P2 (across the first three phases). MOD+ group had greater activity in P1 and MLD in P2 except for during the shapes-retrieval phase. There were fewer differences among low performers. Compared to CG, differences were mainly in P1 for MLD and in P2 for MOD+ due to more activity with HL. Between HL groups, differences were mostly in P3 across the first three phases due to MOD+ having greater activity.

Comparisons were also made within groups between high and low performers. For the CG high vs low performers, differences were mainly in P2 and P3 across the first three phases. In the MLD group, differences were mainly in P1 across the last three phases and in P2 and P3 in the encoding phases. For the MOD+ group, differences were mainly in P2 across the last three phases and in N1 in retrieval phases. Apart from P1 in the MLD, differences were primarily due to high performers having greater mean amplitude.

ERPs and binding function

The mean amplitudes of ERPs were correlated with the weighted binding score for each group across comparisons (Table 5). For P1, there were no significant correlations for MLD and CG, but MOD+ trended to significance ($p < 0.1$) compared with both other groups. The N1 mean amplitude significantly correlated with maintained binding only for MOD+ in comparison with both CG and MLD. For P2, MLD showed a significant positive correlation when contrasted with CG and MOD+, whereas MOD+ only demonstrated a trend towards significance ($p < 0.1$) against CG, but a significant correlation with MLD. For P3, only MLD

showed a significant correlation with maintained binding against CG and MOD+. For LPP, increased mean amplitude was correlated with a decline in binding for CG compared to MOD+ and MLD, while MOD+ exhibited improved binding which trended towards significance ($p < 0.1$) compared to MLD only.

Source space analysis

Analysis was conducted on cortical sources to explore differences in neural activity underlying VSTMB between groups. A summary of findings is provided here. Figures of the cortical statistical maps (Figures S3-5) and tables (Tables S5-7) are available in the Supplementary Materials. There were differences in multiple neural regions for both HL groups compared to the CG and compared to each other. For all comparisons, differences occurred mostly in the first two phase before declining across the remainder of the task.

For MLD compared to CG, differences across phases were most consistently in bilateral frontal, parietal, and right occipital regions. There were some differences in left occipital and central regions in the shapes conditions. Differences emerged in bilateral temporal regions in the middle phases. There were few differences in the last phase. Most differences were due to greater activity from the MLD and primarily occurred during the second time window (140-250 ms – N1 and P2) across phases.

For MOD+ compared to CG, there were fewer differences. These were mostly in the second phase and in bilateral frontal, central, and parietal regions. There were also some differences in left occipital and temporal regions. In the third phase there was an increase in activity in bilateral prefrontal and right temporal regions during final time window (LPP). Most differences were due to greater activity from the MOD. Across phases there was no time window with consistently more pronounced activity.

For MOD+ compared to MLD, there were numerous differences in bilateral frontal, central, parietal, and occipital regions in the first phase due to greater activity from MLD. In the second phase there were numerous differences in bilateral frontal, central, parietal, left occipital and right temporal regions. Except for left central regions, these were primarily due to greater activity in MLD initially. The MOD+ demonstrated greater activity in later time windows in this phase. In the third phase, there were difference in left temporal, right occipital and parietal due to greater activity from MLD and in right frontal-central and

temporal regions due to greater activity from MOD+. In the final phase, most differences were in right frontal and parietal regions due to MLD and bilateral temporal regions due to MOD+. Across phases, the MLD appeared to demonstrate more activity in earlier time windows whereas the MOD+ had greater activity in later time windows.

Discussion

Summary of findings

To our knowledge, this is the first study investigating the neurocognitive correlates of feature binding in visual working memory with ARHL. There were no differences between groups in behavioural outcomes across VSTMB task conditions. There were multiple differences in ERPs which can detect subtle changes in neurocognitive processing in the absence of a significant deficit in behavioural performance (Golob et al., 2009; Palop & Mucke, 2016). Differences between HL groups and CG were primarily in the mean amplitude of the P2 and P3 components. For P2 this was mainly due to HL groups having the greater mean amplitude whereas for P3 it was less consistent. Between the HL groups there were more differences, primarily for P1, P2 and LPP. The MOD+ had a greater mean amplitude for P1 as did MLD primarily for P2 and LPP. We compared ERPs between and within groups, according to level of binding performance adjusted for shapes performance. These analyses indicated that electrophysiological differences between groups were due primarily to greater activity from high performers in the HL groups who better maintained accuracy across conditions (i.e., did not demonstrate poorer ability in visual working memory for binding features adjusted for baseline performance for single features). This was supported by correlational analysis of the mean amplitudes with the weighted binding score across groups. The results indicated that, for MLD, increased P2 and P3 amplitudes were linked to better maintained binding function in working memory. In MOD+, this relationship was observed in the P1, N1, and P2 components. For CG, an inverse relationship was observed with the LPP mean amplitude and the weighted binding score. Analyses of estimated cortical sources suggested that both HL groups may have utilised a frontoparietal network which became more active in the first phase for the MLD (particularly during the P2 time window) and in the second phase for the MOD+ before declining in difference across the remainder of the task.

No differences were observed in behavioural or neural outcomes that were specific to the VSTMB task condition (shapes vs binding) suggesting that the increased neural activity with HL was general to visual working memory and not specific to feature binding. A previous study had reported a behavioural difference in VSTMB with ARHL (Loughrey et al., 2019). The discrepancy may have been due to methodological differences. In the prior study, participants viewed an array of two items (Loughrey et al., 2019) whereas this study used a variant of the VSTMB task with three items. In patients with mild cognitive impairment (MCI), a selective binding deficit has been observed with a visual array of two but not three items, suggesting an observable deficit compared to controls may be contingent upon memory load (Parra et al., 2019; Parra et al., 2017). Additionally, the iPad-based app used in this study, while it has been validated, cannot provide the same level of accuracy in measurement of hearing sensitivity as a clinical audiological assessment (Barczik & Serpanos, 2018; Handzel et al., 2013; Irace et al., 2021; Shilo et al., 2022; Szudek et al., 2012). Furthermore, the HL group in the prior study had a higher HHIE-S score (Loughrey et al., 2019), suggesting a greater functional impact, or possibly a longer duration, of HL in that group. A greater perceived hearing impairment has been reported to be more closely linked with poorer health outcomes than objective (audiometrically measured) HL (Gopinath et al., 2012).

ARHL and neural correlates of VSTMB

Our analyses were exploratory to help address a gap in the literature, and our findings should be interpreted cautiously. Our findings indicate that there is increased activity with ARHL in the neural networks subserving attentional mechanisms that support feature binding in visual working memory. There also appeared to be a dose-response effect whereby more severe HL was linked with greater neural activity. In a study using the VSTMB task with participants with MCI and in the MCI stages of familial AD (MCI-FAD) samples, there was a behavioural difference in binding for these groups and reduced mean amplitudes in electrophysiological components compared to controls (Pietto et al., 2016). The authors reported that this indicated a reduced efficiency in attentional control due to decline in the frontoparietal attention network in the MCI groups (Pietto et al., 2016) which is thought to play a causal role in VSTMB performance (Birba et al., 2017). In contrast, in our study, there appeared to be increased activity in this network with the HL groups compared to controls

which may have reflected involvement of attentional resources in earlier perceptual processes, sensory filtering and selective attention (P1 and P2), rather than downstream higher order, working memory processes (P3 and LPP).

The P1 component is thought to be a neural signature of early attentional control and processing of low-level features (Aksoy, Ufodiama, Bateson, Martin, & Asghar, 2021; Pratt, Willoughby, & Swick, 2011; Verschooren, Schindler, De Raedt, & Pourtois, 2021) before scanning for target features as indexed by the P2 component prior to response selection (Luck & Hillyard, 1994; Potts, 2004). These components can be modulated by top-down attentional mechanisms (Gazzaley et al., 2008; Kotchoubey, 2006; Zanto, Toy, & Gazzaley, 2010) and differences in amplitude may reflect reallocation of cortical resources by these mechanisms (Pratt et al., 2011) or processing efficiency (George & Coch, 2011; Tong, Melara, & Rao, 2009). Our analyses of these components indicated that the two HL groups may have differences in efficiency during early-stage processing of task features or may have distributed limited cortical resources differently. The MLD group demonstrated greater mean amplitude primarily in P2 whereas the MOD+ had greater amplitude primarily in P1. However, our correlational analysis indicated that increased mean amplitude in the P2 component may have supported maintenance of binding function in both hearing loss groups, whereas P1 supported function in MOD+ only. Elevated amplitudes in P1 and P2 in response to simple visual stimuli has previously been observed with HL (Campbell & Sharma, 2014, 2020; Intartaglia, Prud'homme, Foster, Zeitouni, & Lehmann, 2022). A previous study reported that in response to passively viewed visual stimuli, a decreased P1 *latency* was observed with moderate compared to mild HL and a greater P2 *amplitude* with mild HL compared to moderate HL (Campbell & Sharma, 2020). Additionally, an increased amplitude with HL was observed in the P2 component on passive visual tasks involving increased activation of the auditory temporal cortex (Campbell & Sharma, 2014). This has been posited to reflect cross-modal connectivity modulated by frontal cortical networks via top-down mechanisms (Campbell & Sharma, 2014, 2020).

In this study, subgroup analyses indicated that elevated P2 was associated with better performance within groups. However, a greater P1 amplitude was associated with better performance in MOD+ but poorer performance in MLD. An increased P1 amplitude may have reflected an enhanced sensory gain amplification and greater earlier processing efficiency in MOD+ due to adaptation following a longer duration of HL (Campbell & Sharma, 2020). In MLD, it may reflect a decline in early-stage sensory processing or an

initial inefficiency in sensory gating. Reduction in amplitude of P1 with hearing rehabilitative therapy has been reported, indicating a mechanistic relationship with HL (Glick & Sharma, 2020). Possibly top-down attentional processes are focused on enhancing earlier, low-order information processing with more severe HL (P1 and N1) whereas in the initial stages of HL there is reliance on processing in subsequent stages only (P2 and P3). Other studies report that attention mechanisms associated with visual working memory may modulate sensory input across various stages of sensory processing including from the brainstem and peripheral levels (Marcenaro, Leiva, Dragicevic, López, & Delano, 2021; Sörqvist, Stenfelt, & Rönnberg, 2012). A recent 3T MRI study reported that ARHL was associated with an increased efficiency in the visual subnetwork suggesting there is longer-term functional reorganisation in associated cortical regions to possibly compensate for hearing abilities (Ponticorvo et al., 2022).

Differences between groups were less consistent for the N1, P3, and LPP components which are thought to reflect activity underpinning working memory (Kok, 2001; Pietto et al., 2016; Polich, 2007; Pratt et al., 2011; Smart, Segalowitz, Mulligan, & MacDonald, 2014) and memory encoding/post-retrieval processes respectively (Friedman & Johnson, 2000; Koenig & Mecklinger, 2008; Pietto et al., 2016). Increased P3 amplitude was associated with better performance within CG and MLD but for MOD+ it was less consistent. Increased mean amplitude in P3 was significantly correlated with better maintained binding function for MLD only. Differences in LPP also did not have any clear association with performance. It did have a significant inverse correlation with better preserved binding function for CG only. This may indicate efficient neurocognitive processing without the need for increased activity during the initial stages (P1 and P2). The lack of clear group differences due to HL at this later stage in cognitive processing is somewhat consistent with measures of higher-order cognitive functioning from the MoCA and CANTAB, for which there were no group differences in cognitive performance. While we have found evidence of a differing pattern of activity, more research is needed to fully understand the mechanisms underpinning these differences.

Causal mechanisms in ARHL and neurocognitive decline

ARHL has been posited as a risk factor for dementia that emerges primarily in midlife (Livingston et al., 2020; Livingston et al., 2017) and appears to be dose-dependent with an

exponentially increased risk of dementia with progressively severe HL (Lin et al., 2011). The pathophysiological relationship remains unresolved and there are several potential mechanisms including a common causal factor such as microvascular disease, a mechanistic relationship whereby ARHL negatively impacts cognition, or a mediating factor such as social isolation (Lin et al., 2011; Lindenberger & Baltes, 1994; Loughrey et al., 2018; Panza et al., 2015; Wayne & Johnsrude, 2015). These pathways may also co-occur and progressively increase risk of developing dementia through a cascade effect. A further consideration is that the potential role of ARHL across a continuum of neurocognitive decline has not yet been fully delineated and may alter across various stages. An important line of research comes from structural neuroimaging studies which have linked acquired HL with an increased rate of atrophy in whole brain volume (Lin et al., 2014) and the temporal lobe (Armstrong et al., 2019; Lin et al., 2014; Xu et al., 2019) comparable to that observed in those developing MCI (Lin et al., 2014). Longitudinal research from the Alzheimer's Disease Neuroimaging Initiative (ADNI) (Xu et al., 2019) and the Baltimore Longitudinal Study of Aging (BLSA) (Armstrong et al., 2019) report atrophy with HL of the hippocampus and entorhinal cortex which are important for memory and are affected in the early stages of AD (Braak & Braak, 1991). Such associations have remained after adjustment for demographic and cardiovascular factors suggesting that factors apart from broader physiological decline may contribute (Armstrong et al., 2019; Lin et al., 2014). Additionally, animal studies have indicated a causal relationship whereby induced HL leads to atrophy and altered neural function in the hippocampus, and impaired learning and memory, and with increased tau phosphorylation (Dong et al., 2018; Liu et al., 2016; Paciello et al., 2021; Park et al., 2018; Park et al., 2016; Yu, Zhai, Dai, & Hu, 2011).

A plausible mechanism is a maladaptive neural reorganization or atrophy with acquired HL which may deplete cognitive reserve or the brain's resilience against neuropathologies and the effects of ageing (Belkhiria et al., 2019; Belkhiria et al., 2020; Campbell & Sharma, 2013, 2014, 2020; Ha et al., 2020; Husain et al., 2011; Lin et al., 2014; Lin et al., 2011; Park et al., 2016; Qian, Chang, Moonis, & Lalwani, 2017; Ren et al., 2018; Rosemann & Thiel, 2019; Rudner, Seeto, Keidser, Johnson, & Rönnberg, 2019; Wang et al., 2022; Xu et al., 2019). Altered neural function has been observed even from the earlier, milder stages of ARHL (Bidelman et al., 2019; Campbell & Sharma, 2013, 2014) and may lead to increased neural connectivity (García-Cordero et al., 2015; Pasquini et al., 2015) and grey matter volume prior to a greater rate of atrophy (Xu et al., 2019). This may demonstrate initially as an over-

recruitment of brain regions on simple cognitive tasks that reflects inefficiencies in neural regions that support early multi-modal sensory processing (Bidelman et al., 2019; Reuter-Lorenz & Cappell, 2008). ARHL has also been associated with weaker lower-order automatic processes and increased involvement of higher-order cognitive functions on non-auditory cognitive tasks (Loughrey, Mihelj, & Lawlor, 2021; Loughrey, Pakhomov, & Lawlor, 2020). Another important consideration is the compensatory changes that may occur to support auditory processing whereby cortical resources are reallocated to frontal regions from other regions including the temporal lobes (Campbell & Sharma, 2013). There is also cross-modal reorganisation whereby the auditory cortices become more responsive to visual stimuli (Campbell & Sharma, 2014, 2020). These functional changes have been correlated with HL severity and may help maintain behavioural performance in speech perception (Bidelman et al., 2019; Campbell & Sharma, 2013, 2014) but possibly contribute to depletion of cognitive reserve and an increased risk of cognitive impairment (Lin et al., 2014; Lin et al., 2011). Further support comes from neuroimaging studies which report that hearing aids may moderate or even reverse cortical neuroplastic adjustment following hearing loss (Glick & Sharma, 2020; Vogelzang, Thiel, Rosemann, Rieger, & Ruigendijk, 2021).

Our findings suggest that ARHL may lead to a reliance on compensatory mechanisms modulated by higher-order neurocognitive resources to maintain behavioural performance. As these resources appeared to be allocated to earlier neural processes this might reflect a compensatory response to inefficiencies in regions modulating multi-modal lower-order processes (Loughrey et al., 2021; Loughrey et al., 2020). This compensatory response may occur earlier in the neurocognitive processing pipeline with increasing severity of ARHL (in P2 and then P1). There was greater activity in bilateral temporal regions, consistent with previous research indicating cross-modal activity with acquired HL (Campbell & Sharma, 2014). There was also greater activity in frontoparietal regions which may reflect increased involvement of a higher order neural network. A right frontoparietal network which underpins processes in visual attention and working memory has been hypothesised to play a role in the protective effects of cognitive reserve on function (Brosnan et al., 2018; Robertson, 2014) and may contribute to maintained feature binding in those at greater AD risk (Heneghan et al., 2022). Possibly, ARHL leads to an increased reliance on such a neural network to maintain cognitive function which may in turn deplete cognitive reserve. However, this is a speculative potential mechanism and further research is required to

examine the temporal relationship of ARHL to neural changes and whether such changes are associated with future cognitive differences.

Feature binding could provide an informative measure in future research that aims to clarify the association between ARHL and dementia. In AD studies, greater neural activity and functional network reorganization has been observed from the earlier stages of the disease process and can precede decreased connectivity and cognitive decline by several years (García-Cordero et al., 2015; Golob et al., 2009; Parra et al., 2017; Pasquini et al., 2015). Familial AD carriers with no cognitive deficits have demonstrated larger P2 amplitudes on an auditory discrimination task ten years before dementia onset (Golob et al., 2009). The VSTMB task may be sensitive to such maladaptive changes in AD cohorts from the prodromal stages (Parra et al., 2017). Additionally, performance on the VSTMB task has been correlated with tau in the entorhinal cortex and inferior temporal lobe, and with amyloid burden (Norton et al., 2020). ARHL has been associated with disruption in neural regions underlying feature binding (Armstrong et al., 2019; Husain et al., 2011; Lin et al., 2014) and other important functions such as the integration of perceptual and conceptual data in facial emotion processing (Belkhiria, Vergara, Martinez, Delano, & Delgado, 2021). Furthermore, ARHL has been linked with elevated higher amyloid burden and tau levels (Golub, Sharma, Rippon, Brickman, & Luchsinger, 2021; van 't Hooft et al., 2023; Wang et al., 2022; Xu et al., 2019; Zheng et al., 2022), possibly via altered expression of the SIRT1-PGC1a and LKB1-AMPK or vascular endothelial growth factor signal pathway, which has been observed in both ARHL and AD mouse models (Shen et al., 2018; Xu et al., 2019). Therefore, feature binding could be a useful measure to clarify the disease mechanisms linking ARHL to dementia.

Limitations and future directions

A limitation in our study was the small sample size, particularly among the subgroups. Additionally, we conducted many statistical tests and some of our findings could have been due to chance. However, we had no *a priori* hypotheses regarding neural outcomes, and these analyses were considered exploratory to elucidate potentially important findings to inform future studies. Further research with larger samples sizes and more robust methodology is warranted. More functional neuroimaging studies of working memory and attention in ARHL cohorts are required to assess how early neural processing differences may be related to

future cognitive decline. This research could assess if the increased electrophysiological activity observed here provides compensatory support in visual cognitive tasks or if there is another factor underlying the association such as differences in age. Differences in visuospatial function have been observed with ARHL (Bonmassar, Pavani, Spinella, Frau, & van Zoest, 2022; Loughrey et al., 2018; Rönnberg, Hygge, Keidser, & Rudner, 2014) and in AD (Laukka, Macdonald, Fratiglioni, & Backman, 2012; Williams et al., 2020). Additionally, research could examine how differences in measures of reserve and of AD biomarkers may mediate these outcomes. Cortical changes subserving adaptation with ARHL has been reported to be associated with limited effectiveness of rehabilitative therapies (Sandmann et al., 2012) making cognitive measures of such change important for future intervention trials.

Conclusion

We found no behavioural differences between hearing loss groups and controls in VSTMB outcomes. However, we found numerous differences in neural correlates of VSTMB which may precede cognitive differences. Our findings indicated increased involvement of a frontoparietal network, particularly in earlier sensory processing of task features which reflect a compensatory response to underlying neural inefficiencies. Additional research is warranted to investigate changes in neurocognitive processing on cognitive tasks with ARHL. Effective treatment of ARHL which promotes brain health could have enormous implications for the prevalence of dementia globally (Livingston et al., 2020; Livingston et al., 2017). Thus, it is a priority to specify the causal pathways through which ARHL and dementia may be linked and provide measures for optimising rehabilitative therapies.

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Competing interests

The authors declare that they have no competing interests.

Supplementary materials included

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Figure legends

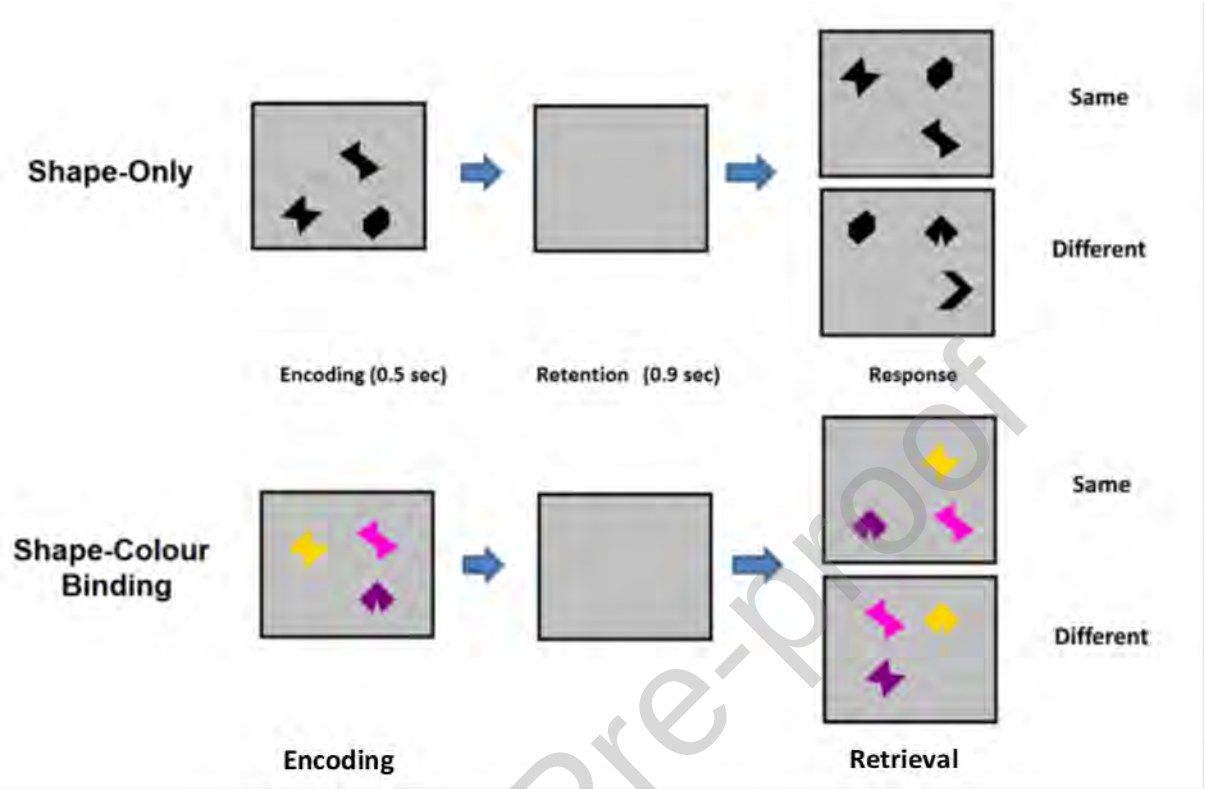


Figure 1: The Visual Short-Term Memory Binding task (Parra et al., 2017). The task has two conditions: shape-only and shape-colour binding. Participants are asked to remember a visual array of objects during the encoding phase (0.5 seconds) and, after a brief pause (0.9 seconds), detect if a change has occurred when visually prompted again with another array of objects in the retrieval phase displayed until they provided a response. Across 50% of the trials there was a change and for the remaining trials there was no change. Changes consisted of new features replacing studied features (shape-only) or features swapping across items (binding).

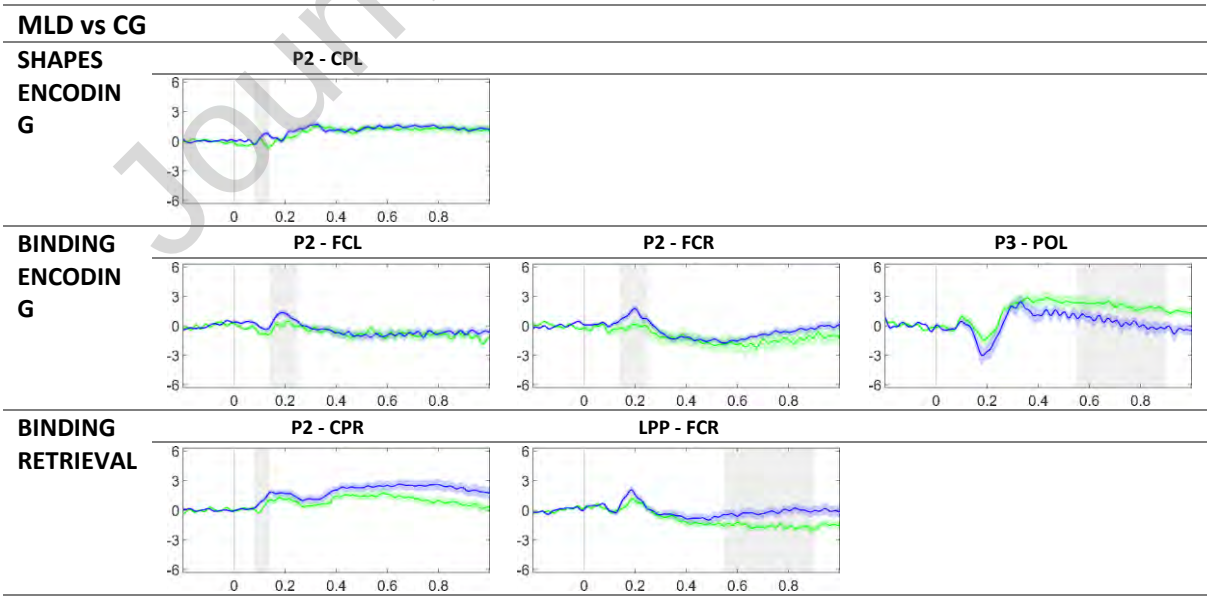


Figure 2: Significant differences (shaded area) in ERP activity (mean value and standard error) for MLD (blue) and CG (green) in the shape-only (encoding and retrieval) and shape-colour binding (encoding and retrieval) conditions. The x-axis is Time (in seconds), and the y-axis is microvolts (*uV*).

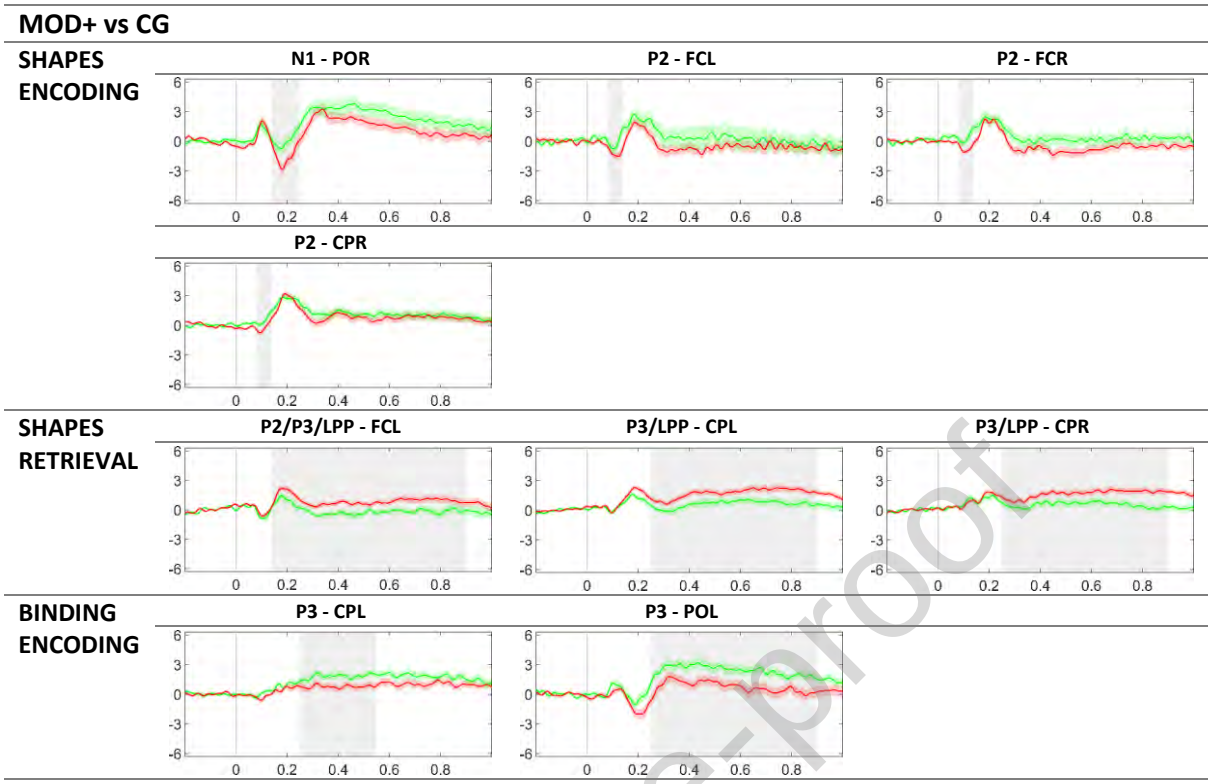
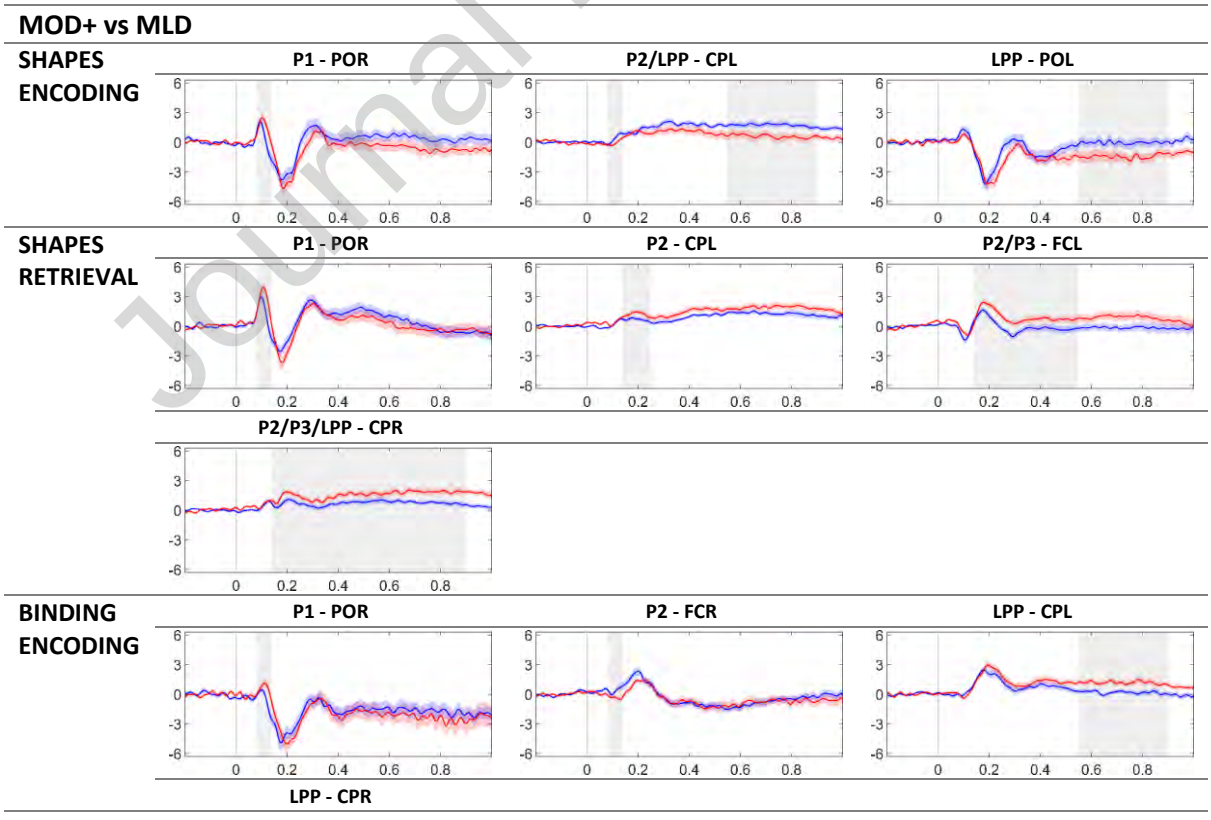


Figure 3: Significant differences (shaded area) in ERP activity (mean value and standard error) for MOD+ (red) and CG (green) in the shape-only (encoding and retrieval) and shape-colour binding (encoding and retrieval) conditions. The x-axis is Time (in seconds), and the y-axis is microvolts (*uV*).



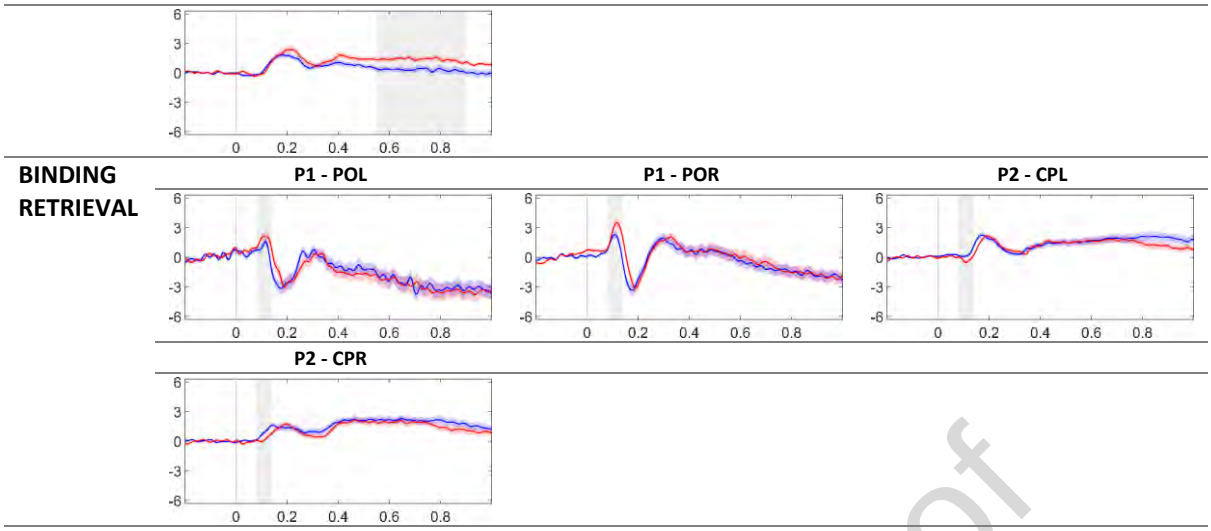


Figure 4: Significant differences (shaded area) in ERP activity (mean value and standard error) for MOD+ (red) and MLD (blue) in the shape-only (encoding and retrieval) and shape-colour binding (encoding and retrieval) conditions. The x-axis is Time (in seconds), and the y-axis is microvolts (*uV*).

	CG	MLD	MOD+	test	*p*
Age	69.1 (3.3)	70.6 (3.9)	73.5 (6.4)	5.57˜	0.06
Sex (F/M)	8 / 6	10 / 11	12 / 11	0.31	0.86
Education (yrs)	16.07 (2.8)	14.9 (3.8)	15.39 (4.0)	0.42	0.66
Better ear PTA	20.3 (2.9)	32.9 (3.6)	50.9 (11.8)	50.09˜	<0.01*
Worse ear PTA	27 (6.4)	39.3 (6.4)	55.2 (12.7)	42.51˜	<0.01*
Self-rated hearing (HHIE-S)	3.7 (8.6)	6.3 (6.5)	11.5 (9.7)	9.53˜	<0.01*
MOCA	25.4 (2)	26.1 (2)	25.6 (2.6)	0.61	0.55

Table 1: Group characteristics (means and standard deviations or medians and interquartile ranges) with results from one-way ANOVA or Kruskal Wallis tests and Chi-square or Fisher's Exact Tests. ~, Kruskal Wallis test. *, For PTA, all groups were significantly different in post-hoc Bonferroni tests. For HHIE-S, MOD+ was significantly different from CG.

	M	SD	Mdn	IQR	Min.	Max.
Shapes Acc.						
CG	80.3	9.8	82.5	12	57	91
MLD	82.7	12.9	88	20	51	96
MOD+	80.4	12.2	82	23	57	94
Shapes RT						
CG	2.0	0.4	1.9	0.4	1.4	2.84
MLD	2.1	0.5	2.0	0.6	1.5	3.50
MOD+	2.0	0.5	2.0	0.7	1.2	2.87
Binding Acc.						
CG	68.4	10.9	68.5	14	50	93

MLD	73.5	13.5	77.0	17	44	94
MOD+	72.0	12.1	74	20	47	96
Binding RT						
CG	2.1	0.5	2.0	0.7	1.5	3.37
MLD	2.3	0.5	2.2	0.8	1.4	3.42
MOD+	2.3	0.6	2.3	0.9	1.2	3.32
Difference Acc.						
CG	-11.9	9.6	-11	16	-29	6
MLD	-9.2	14.6	-9	14	-33	33
MOD+	-8.4	12.8	-12	16	-28	19

Table 2: The mean (M), standard deviation (SD), median (Mdn), interquartile range (IQR), minimum and maximum scores for accuracy and the reaction time (RT) outcomes on the VSTMB task. RTs are in seconds.

	P1	**N1**	**P2**	**P3**	**LPP**
Shapes					
Encoding	MOD+>MLD	MOD+>CG	MLD>CG CG>MOD+ MLD>MOD+		MLD>MOD+
Retrieval	MOD+>MLD		MOD+>CG MOD+>MLD	MOD+>CG MOD+>MLD	MOD+>CG MOD+>MLD
Binding					
Encoding	MOD+>MLD		MLD>CG MLD>MOD+	CG>MLD CG>MOD+	MLD>MOD+
Retrieval	MOD+>MLD		MLD>CG MLD>MOD+		MLD>CG

Table 3: Summary of merged results of ERP analysis demonstrating the group with the greater mean amplitude.

		MLD vs CG	**MOD+ vs CG**	**MOD+ vs MLD**	**MLD vs CG**	**MOD+ vs CG**	**MOD+ vs MLD**
			Shapes			***Binding***	
Encoding							
P1	POR			2.16 (0.04)			2.87 (0.005)^
N1	POR		-2.17 (0.04)				
P2	FCL		-2.13* (0.047)		2.08 (0.046)		
	FCR		-2.8* (0.01)^		2.09 (0.04)		-2.59* (0.02)^
	CPL	2.05* (0.04)		-2.11* (0.04)			
	CPR		-2.36* (0.02)				
P3	CPL					-2.31 (0.02)	
	POL					-2.24 (0.04)	
	POL				-2.18+ (0.03)	-2.29+ (0.03)	
LPP	CPL			-2.47 (0.01)			2.95 (0.004)^
	CPR						3.01 (0.003)^
	POL			-2.21 (0.03)			
Retrieval							
P1	POL						2.48 (0.02)^

	POR		2.63 (0.01)		2.96 (0.003)^
P2	FCL	2.08 (0.04)	2.81 (0.01)		
	CPL		2.19 (0.04)		-2.25* (0.03)^
	CPR		2.37 (0.02)	2.29* (0.03)	-2.75* (0.01)^
P3	FCL	2.71 (0.01)	2.43 (0.02)		
	CPL	2.2 (0.03)			
	CPR	2.05 (0.04)	2.33 (0.02)		
LPP	FCL	2.33 (0.02)^			
	FCR			2.23 (0.03)	
	CPL	2.55 (0.02)			
	CPR	2.92 (0.003)^	3.04 (0.003)^		

Table 4: Permutation *t* values (*p* values). Unless otherwise indicated the ERPs were from the following time windows; P1 (80-140 ms), N1 and P2 (140-250 ms), P3 (250-550 ms), and LPP (550-900 ms). * 80-140ms; # 140-250ms; + 550-900ms; ^ remained significant after adjustment for FDR.

	MLD vs CG		MOD+ vs CG		MOD+ vs MLD	
P1	-0.11 (0.40)	0.18 (0.26)	0.17 (0.08)	0.16 (0.18)	0.14 (0.08)	0.03 (0.69)
N1	0.00 (0.98)	0.21 (0.18)	-0.19 (0.04)*	0.10 (0.41)	-0.21 (0.01)*	0.11 (0.20)
P2	0.21 (<0.001)*	0.06 (0.36)	0.10 (0.09)	-0.03 (0.69)	0.21 (<0.001)*	0.23 (<0.001)*
P3	0.12 (0.04)*	0.09 (0.19)	-0.01 (0.94)	0.09 (0.25)	0.08 (0.19)	0.12 (0.04)*
LPP	0.00 (0.98)	-0.33 (0.001)*	0.05 (0.51)	-0.29 (0.004)*	0.14 (0.09)	0.05 (0.57)

Table 5: Corelations of ERPs with weighted binding score using Spearman's rank correlation coefficient. * Significant correlation ($p < 0.05$).

CRediT authorship contribution statement

David G. Loughrey: Conceptualization, Funding acquisition, Resources, Data curation, Formal analysis, Investigation, Methodology, Project administration, Software, Validation, Visualization, Writing – original draft, Writing – review & editing. **Catherine Jordan:** Project administration, Writing – review & editing. **Agustin Ibanez:** Formal analysis, Writing – review & editing. **Mario A. Parra:** Conceptualization, Formal analysis, Writing – review & editing. **Brian A. Lawlor:** Conceptualization, Investigation, Resources, Methodology, Writing – review & editing. **Richard B. Reilly:** Conceptualization, Supervision, Resources, Funding acquisition, Investigation, Methodology, Formal analysis, Writing – review & editing.

Verification statement

The manuscript has not been previously published and is not under consideration elsewhere. All authors contributed to this work and approved this submission. The authors declare that they have no competing interests.

Highlights

- We explored the neural correlates of VSTM binding in groups with ARHL.
- Greater mean ERP amplitude in ARHL was linked to maintained feature binding.

- There were greater differences during early neural processing and with HL severity.
- Source space analyses revealed greater activity in frontoparietal/temporal regions.
- Our findings suggest novel hypotheses about compensatory neural mechanisms in ARHL.