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Research Paper

Differential effects of hearing impairment and age on electrophysiological and behavioral measures of speech in noise

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ABSTRACT

Understanding speech in background noise is difficult for many listeners with and without hearing impairment (HI). This study investigated the effects of HI on speech discrimination and recognition measures as well as speech-evoked cortical N1-P2 and MMN auditory event-related potentials (AERPs) in background noise. We aimed to determine which AERP components can predict the effects of HI on speech perception in noise across adult listeners with and without HI. The data were collected from 18 participants with hearing thresholds ranging from within normal limits to bilateral moderate-to-severe sensorineural hearing loss. Linear mixed effects models were employed to examine how hearing impairment, age, stimulus type, and SNR listening condition affected neural and behavioral responses and what AERP components were correlated with effects of HI on speech-in-noise perception across participants. Significant effects of age were found on the N1-P2 but not on MMN, and significant effects of HI were observed on the MMN and behavioral measures. The results suggest that neural responses reflecting later cognitive processing of stimulus discrimination may be more susceptible to the effects of HI on the processing of speech in noise than earlier components that signal the sensory encoding of acoustic stimulus features. Objective AERP responses were also potential neural predictors of speech perception in noise across participants with and without HI, which has implications for the use of AERPs as a potential clinical tool for assessing speech perception in noise.

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1. Introduction

Listeners with hearing impairment (HI) are known to show a wide range of performance on speech-in-noise tasks. Differences in factors such as HI severity and etiology, spectrotemporal resolution, or age-related changes in working memory and cognitive processing may contribute to this variability in performance (Besser et al., 2015; Buss et al., 2004; Dubno et al., 1984; Jin and Nelson, 2010; Lorenzi et al., 2006; Ruggles et al., 2012). One approach to understanding the impact of age-related hearing loss is to obtain non-invasive electrophysiological measures to determine how the timing and magnitude of the objective neural responses to speech along the auditory pathway may account for some of the behavioral variability across individuals in noise. Previous studies have well established that the presence of background noise can impact auditory event-related potentials (AERPs) to speech as well as non-speech stimuli (Bidelman et al., 2014; Billings et al., 2009; Koerner and Zhang, 2015; Kozou et al., 2005; Maamor and Billings, 2017; Muller-Gass et al., 2001; Whiting et al., 1998). Furthermore, noise-induced changes in AERPs have been shown to be correlated with changes in the ability to perceive speech in background noise (Anderson et al., 2013b; Anderson et al., 2011; Bennett et al., 2012; Billings et al., 2013; Koerner et al., 2016; Song et al., 2011). However, most neurophysiological data were from adults with normal hearing or with careful control of subject variables such as age and hearing sensitivity. As very few have systematically examined listeners with various degrees of hearing loss, it remains unclear whether cortical electrophysiological measures can be a reliable predictor of speech perception performance in noise across individuals with HI.

Abbreviations: AERPs, auditory event-related potentials; CV, consonant-vowel; PTA^2, two-frequency pure-tone average; LME, linear mixed-effects; ITPC, inter-trial phase coherence; MMN, mismatch negativity

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The present ERP study aimed to examine the roles of age and hearing impairment in the neural coding of speech sounds in terms of brain-behavior correlates to better understand the neural mechanisms underlying the highly variable performance of speech-in-noise perception in this clinical population. In previous neurophysiological work (Martin et al., 1997; Martin and Stapells, 2005; Martin et al., 1999), noise-masking conditions were administered to normal hearing (NH) listeners in order to have a better control of subject characteristics. Behaviorally, reduced audibility from noise masking led to poorer discrimination and longer reaction times for detecting a /ba/-/da/ contrast, and the effects of reduced audibility were largest when noise masking encompassed the 1–2 kHz frequency range, which contains formant transition cues that are important for accurate discrimination of the /ba/-/da/ stimuli. These studies also documented latency increases and amplitude decreases in the N1, MMN, N2, and P3 AERPs in response to a /ba/-/da/ stimulus contrast after simulating reduced audibility caused by different degrees and configurations of hearing loss. These findings are similar to those from previous work that examined the effects of noise masking on AERPs (Bennett et al., 2012; Billings et al., 2009, 2013; Koerner et al., 2016, 2017; Kozou et al., 2005; Anderson et al., 2013a). However, results showed that there was an important differential effect of noise on these AERP responses, such that the N1 response was present as long as stimuli were audible, while later AERP components such as the MMN and P3 responses were present only if stimuli were discriminable (Martin et al., 1997, 1999; Martin and Stapells, 2005). Thus, while each AERP component appeared to be sensitive to the effects of reduced audibility on speech perception, the later cortical responses tended to be more susceptible to the effects of noise masking.

Several studies have revealed HI-related deficits in the cortical encoding of speech stimuli in quiet using various AERP components (Oates et al., 2002; Polen, 1984; Wall et al., 1991). For example, in an attempt to examine the effects of different degrees of HI on AERPs and behavioral measures of speech discrimination, Oates et al. (2002) found that the presence of HI tended to cause latency increases and amplitude decreases in N1, MMN, N2, and P3 responses to speech stimuli in quiet, which became more pronounced with greater decreases in hearing sensitivity. These results suggest that reduced audibility from HI tends to manifest as increases in AERP latencies and decreases in AERP amplitudes in quiet. However, more recent studies have also shown that the combination of increased age and hearing thresholds may actually enhance N1 and P2 responses in quiet or in high SNR listening conditions to both speech and non-speech stimuli (Alain et al., 2014; Bidelman et al., 2014; Harkrider et al., 2006; Tremblay et al., 2003). For instance, while assessing the effects of age-related HI on the P1, N1, and P2 responses to a voice-onset-time /ba/-/pa/ continuum in quiet, Tremblay et al. (2003) showed that older participants with HI had significantly larger N1 amplitudes in response to more voiceless stimulus representations compared to both younger and older participants with HI, which the authors attributed to deficits in central inhibition. Differences in results across these studies may be attributed to dissimilarities in experimental methodology, including speech stimuli, presentation levels, and SNRs, as well as variability in participant groups. A large barrier to examining individuals with HI is the inherent lack of homogeneity across participants, which may cause difficulty in the interpretation of results. For example, even if participants are grouped based on audiometric hearing thresholds, there may be differences in the etiology and length of hearing impairment as well as suprathreshold auditory processing or cognitive abilities, which could greatly impact the neural coding and perception of speech. Furthermore, it is difficult to control for other confounds such as the effects of aging on auditory processing as well as unknown effects due to other co-occurring health conditions.

Despite differences in results across these previous studies, results suggest that reduced audibility from noise-masking or HI has a differential effect on the neural coding of speech along the auditory pathway. Similar to results from earlier noise-masking studies using NH participants (Martin et al., 1997, 1999; Martin and Stapells, 2005), Oates et al. (2002) revealed that the effects of HI on AERPs in quiet appeared to be larger for later AERP components, such as the P3 response, compared to the earlier N1 and MMN responses (Oates et al., 2002). Bidelman et al. (2014) also revealed a potential differential effect of age-related HI on subcortical and cortical neural responses to a /u/-/a/ vowel continuum in quiet in which stimuli differed by the first fundamental frequency. Results showed that the subcortical frequency following response (FFR) was weaker in older participants with HI compared to younger NH participants while the cortical N1 and P2 responses were stronger in older participants with HI compared to younger NH participants. This work provides evidence that the presence of HI may be impacting certain stages of auditory processing differently than others.

Much less is known about how HI affects the cortical encoding of speech in background noise. Billings et al. (2015) represents some of the first work to examine cortical AERPs in response to speech stimuli in noise in participants with HI. Although there was a significant effect of HI on behavioral measures of speech perception, their results showed that HI did not significantly alter cortical speech-evoked P1-N1-P2 responses in noise when a group of older HI participants were compared to a group of older NH participants. In order to further examine the effects of HI on different levels of cortical processing in response to speech in noise, the current study examined N1-P2 and MMN responses in adult listeners with and without HI. Cortical event-related potential components can be divided into exogenous, sensory potentials, and endogenous, cognitive potentials (Picton et al., 2000). The N1-P2 complex is an obligatory response that is thought to reflect the sensory encoding of audible, acoustic information at the auditory cortex. On the other hand, the mismatch negativity (MMN) reflects pre-attentive perceptual and cognitive processing mechanisms responsible for sensory discrimination. This difference is evident in results from Martin and colleagues (Martin et al., 1997, 1999; Martin and Stapells, 2005), who showed that the N1 response was present as long as speech stimuli were audible, regardless of whether stimuli were discriminable, while the MMN response became absent as soon as participants were no longer able to discriminate speech stimuli. Similarly, research examining categorical perception using voice-onset time (VOT) contrasts has shown that while the N1 response is influenced by acoustic characteristics of the stimulus, it does not represent a neural correlate of VOT perception (Sharma et al., 2000). In contrast, the strength of the MMN response better reflected VOT discrimination, such that MMN responses to distinct across-category stimuli were larger and more robust than MMN responses to within-category stimulus pairs (Sharma and Dorman, 1999). Together, the N1-P2 and MMN AERP components can provide information about the effects of various factors, such as the presence of HI or background noise, on different levels of cortical processing underlying auditory and speech perception. To our knowledge, no previous studies have directly compared the systematic effects of HI on these cortical responses to speech in noise.

When paired with behavioral speech perception tests, AERPs can be used to examine neural correlates of behavior and to better understand mechanisms underlying impaired performance in background noise. Billings et al. (2015) showed that passive N1 and P2 responses could predict the effects of HI on sentence-level

perception in background noise. Less is known about whether the passive MMN response is a neural correlate of hearing-related changes in speech perception in noise. Since the MMN is recorded in the absence of attention or any overt behavioral response, it is considered a feasible clinical tool for assessing or predicting the effects of different factors, such as the presence of hearing loss of background noise, on behavior in a number of populations, including children or adults who are not able to make consistent or reliable behavioral responses. However, it is well known that the MMN has high inter- and intra-subject variability (Kurtzberg et al., 1995; Lang et al., 1995; Martin et al., 1999, 2008; Näätänen et al., 2007, 1995; Stapells, 2002), which greatly limits its use in assessing performance at the individual level. Recent findings suggest that a measure of cross-trial cortical oscillatory activity associated with the MMN response may represent a more robust measure of neural processing than MMN latency or amplitude (Koerner et al., 2016). Traditional AERP waveform averaging focuses on capturing synchronous neural activity that is time- and phase-locked to an auditory stimulus but does not allow for an examination of ongoing EEG oscillatory activity underlying AERP components, as any "random" trial-by-trial activity is canceled out during the averaging process. It is possible that trial-by-trial latency jitter induced by factors such as HI or background noise is linked to reduced AERP amplitudes and delayed AERP latencies in the averaging process. Therefore, not only are averaged AERP components distorted by this trial-by-trial variation, but they are also unable to reflect useful information from this underlying oscillatory activity. The present study was designed to determine whether the MMN and its associated event-related cortical oscillations can predict the effects of HI on speech-in-noise perception.

The current work extended a double oddball paradigm used in our previous studies on NH listeners (Koerner et al., 2016, 2017) to examine the effects of HI on AERPs and event-related cortical oscillations in response to two speech contrasts in background noise. Behavioral tests of phoneme discrimination and sentence recognition were included to examine potential brain-behavior relationships. It was expected that participants with poorer hearing thresholds would have lower performance on behavioral tasks, prolonged AERP latencies, as well as reduced AERP amplitudes and cortical oscillatory activity. However, it was expected that HI would have a differential effect on N1-P2 and MMN responses, such that sensory processing of acoustic speech cues would be less impacted by HI compared to later mechanisms that reflect auditory change discrimination. This study also aimed to examine the effects of several other factors on the neural coding and perception of speech in noise, including differences in signal-to-noise ratio, stimuli, and age. Because background noise influences audibility, it was predicted that a change in SNR would also have a significant impact on neural and behavioral responses to speech. It was also predicted that, consistent with previous work, neural and behavioral responses to the consonant change would be weaker than that to the vowel change. In addition, it was predicted that there would be no effects of age on behavioral or neural responses in our group of adult participants. Finally, it was expected that objective N1-P2 and MMN responses as well as their associated event-related cortical oscillations would be significantly predictive of phoneme- and sentence-level behavioral performance across listening conditions and participants with various degrees of hearing sensitivity.

2. Materials and methods

2.1. Participants

The participants in this study were 18 right-handed, native speakers of American English (7 males, 11 females) between 40 and 71 years old ($M = 62.11$, $SD = 8.27$) with hearing sensitivity ranging from within-normal-limits to moderate-to-severe sensorineural hearing loss (Fig. 1, Table 1, Supplementary Fig. 6), as determined by a standard audiological assessment for pure tones from 0.25 to 8 kHz. Participants were excluded from the study if audiometric evaluation revealed any conductive hearing loss, as defined as an air-bone gap greater than or equal to 15 dB, or any asymmetries that were greater than 20 dB at two or more frequencies. Participants had no history of speech, language, or cognitive difficulty. The Human Research Protection Program at the University of Minnesota approved the research protocol and all participants provided informed consent prior to beginning the study.

In order to assess the effects of HI on the neural coding and perception of speech in noise, a two-frequency pure-tone average (PTA^2) was calculated for each participant based on the average of hearing thresholds at 1 and 2 kHz in the better ear. Similar measures have been used in previous studies that examined the effects of reduced audibility or HI on neural responses to CV speech syllables (Oates et al., 2002); however, instead of using this measure to group participants into categories that quantify the degree of hearing loss (i.e. "mild" or "severe"), the current study used PTA^2 as a continuous variable. This method quantifies variability across participants that would normally be grouped together based on degree of hearing loss, which may allow for a better examination of how variability in hearing thresholds across participants impacts the neural coding of speech.

2.2. Stimuli

A passive double-oddball paradigm was developed using the consonant-vowel (CV) syllables, /ba/, /da/, and /bu/, to elicit AERP responses. Detailed methodology regarding the creation of these stimuli has been described previously (Koerner et al., 2016). Each syllable had a duration of 170 ms and had a steady-state fundamental frequency of 100 Hz and a steady F4 at 3300 Hz. Formant transitions were generated in the first 50 ms of each syllable by the HLsyn software (Sensimetrics Corp., USA). The CV syllable /ba/ had onset frequencies at 328 Hz, 1071 Hz, and 2298 Hz for F1, F2, and F3. For /da/, the F1, F2, and F3 onset frequencies were 362 Hz, 1832 Hz, and 2540 Hz. For the CV syllable /bu/, the formant onset frequencies

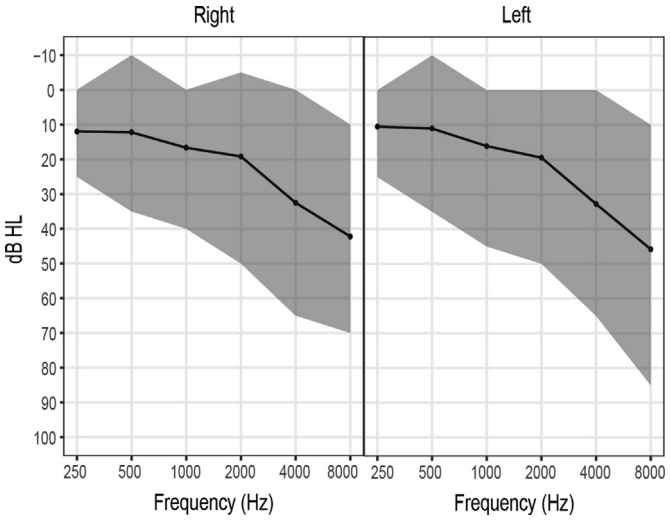

Fig. 1. Mean pure-tone hearing thresholds (from 0.25 to 8 kHz) for the right and left ears of each participant displayed as a solid black line. The shaded envelope depicts maximum and minimum pure-tone hearing thresholds for the group of 18 participants.

Table 1
Sex, age, and PTA^2 values for each participant (n = 18). Means and standard deviations for the distribution of age and PTA^2 are provided.

Sex	Age (years)	PTA^2 (better ear)
F	50	0
F	58	7.5
F	60	5
F	61	15
F	65	42.5
F	71	42.5
M	40	2.5
M	50	2.5
M	65	15
M	62	40
M	69	32.5
F	65	5
F	60	12.5
F	67	20
M	68	0
M	69	12.5
F	69	22.5
F	69	15
M (SD)	62.11 (8.27)	16.25 (14.41)

were for F1, F2, and F3 were 230 Hz, 900 Hz, and 2480 Hz. The vowel portions (50–170 ms) of the /ba/ and /da/ syllables had steady-state center F1, F2, and F3 frequencies of 674 Hz, 1140 Hz, and 2350 Hz while the vowel portion of /bu/ had the steady-state center F1, F2, and F3 frequencies of 320 Hz, 860 Hz, and 2620 Hz.

The double oddball paradigm was used to compare responses to a consonant change with weaker and transient acoustic cues to that of a more salient and stable vowel change. This paradigm allows for a within-participant control condition, as responses to the two deviant stimuli that were recorded within the same testing session can be compared. These CV syllables were also used in an active listening condition to obtain phoneme-change detection sensitivity and reaction time. Participants were also presented with IEEE sentences (IEEE, 1969) to obtain sentence-level recognition scores.

Speech and noise stimuli for the EEG test sessions were presented using EEvoke software (ANT Inc., Netherlands) and Goldwave (Goldwave, Inc., 2015) while all behavioral test materials were presented using MATLAB. All CV and sentence-level speech stimuli were presented in speech-shaped background noise that was created using the long-term speech spectrum of the CV syllables and the IEEE sentence corpus, respectively. The root mean square (rms) value for each speech and noise stimulus was normalized so that speech stimuli for all AERP and behavioral measures were always presented at 70 dB SPL in two listening conditions: speech-shaped background noise at a 0 dB signal-to-noise ratio (SNR) and at a −3 dB SNR. These SNRs were chosen to systematically reduce audibility across a group of NH and HI participants. Articulation Index represents an estimation of audibility, which can be impacted by reduced hearing thresholds as well as by varying signal and noise levels. A measure of AI was used to ensure that at a presentation level of 70 dB SPL, only 20–40% (AI values of 0.2–0.4) of the speech signal would be audible for participants with hearing thresholds ranging from within normal limits to mild sloping to severe sensorineural hearing losses. An AI of around 0.3 has previously been shown to be a point at which listeners show wide variability in their abilities to perceive speech in noise (Nelson et al., 2012). Therefore, AI values of 0.2–0.4 were chosen to ensure that participants had a range of performance on behavioral speech recognition tasks without reaching ceiling or floor performance. All stimuli were presented via two sound field speakers (M-audio BX8a) located at 45° to the left and right of the participant at a distance of approximately 1 m.

2.3. Procedure

Testing was completed in an electrically and acoustically treated booth (ETS-Lindgren Acoustic Systems) in Zhang Lab at the University of Minnesota. The reported N1-P2 and MMN AERP data are from a passive EEG recording session and behavioral responses are from an active phoneme-change detection task. Details regarding stimulus presentation have been reported previously (Koerner et al., 2016). Stimulus presentation order was pseudo-randomized in a double-oddball paradigm so that no blocks began with a deviant stimulus and so that two deviants were never presented in succession. The double-oddball paradigm contained two speech contrasts: a vowel change (from /ba/ to /bu/) and a consonant change (from /ba/ to /da/). The standard /ba/ stimulus had a probability of occurrence of 0.75 and a total of 832 trials while the two deviant stimuli, /bu/ and /da/, each had a probability of occurrence of 0.125 and a total of 104 trials for each stimulus. The SNR listening conditions were counter-balanced across participants to avoid potential order effects. Each condition consisted of 10 blocks with a 5 s inter-block interval. The interstimulus interval (ISI) was randomized from trial to trial within the range of 600–700 ms. Both deviant stimuli were presented alone as standard stimuli in 4 separate alternating blocks of 30 repetitions each, for a total of 120 repetitions of each stimulus. These “standard” presentations of /bu/ and /da/ elicited the N1-P2 responses and were subtracted from the deviant stimuli in the double-oddball paradigm to obtain MMN responses. This resulted in an “identity MMN” for each deviant stimulus, which avoids acoustic confounds between the standard and deviant stimuli in interpreting the MMN data (Kraus et al., 1992; Zhang et al., 2009; Pulvermuller and Shtyrov, 2006). During the passive recording session, participants were instructed to relax, minimize excessive movements or eye blinks and stay awake while ignoring the auditory stimuli played through the speakers by focusing on a muted movie of their choice with subtitles.

Behavioral responses for syllable detection were recorded in a separate discrimination task using the same stimuli and presentation levels as in the double-oddball paradigm described above. The participants were asked to focus their attention on detecting phoneme-changes and to make button-press responses using a desktop keyboard whenever they heard a consonant or vowel change. Sentence recognition scores were also recorded in response to randomized lists of IEEE sentences. Participants were instructed to type word by word, as best as they could, the sentences that they heard.

2.4. Data analysis

2.4.1. AERP measures

Continuous EEG data were recorded using the Advanced Neuro Technology EEG System (Advanced Source Analysis version 4.7) and a 64-channel Ag AgCl electrode WaveGuard cap with a REFA-72 amplifier (TMS International BV) (bandwidth = 0.016–200 Hz, sampling rate = 512 Hz). The average impedance of electrodes was below 5 kOhms. ERP waveform analysis was completed offline in BESA (Version 6.0, MEGIS Software GmbH, Germany). The offline EEG data were bandpassed at 0.5–40 Hz. The ERP epoch length consisted of a 100 ms prestimulus baseline and a 700 ms post-stimulus interval. Automatic artifact rejection criteria were set at ± 50 μV. The N1, P2, and MMN responses were analyzed with a common average reference at the Cz electrode. Grand average waveforms in each noise condition were used to define analysis windows for measuring N1, P2, and MMN peak latency, which were confirmed by visual inspection of individual AERP waveforms. N1 and P2 peak latency, relative to the pre-stimulus interval, was measured within a time window of 80–180 ms and 180–300 ms for

the 0 dB SNR condition, respectively, and 100–200 ms and 200–350 ms for the −3 dB SNR condition. Similarly, MMN latency was assessed within the time window of 100–300 ms for the 0 dB SNR condition and 150–350 ms for the −3 dB SNR condition. Mean amplitude for the N1 and P2 responses were calculated using an averaging window of 10 ms centered at the peak, while MMN mean amplitude quantification used an averaging window of 20 ms centered at peak.

Trial-by-trial phase locking associated with the N1-P2 responses was calculated in delta (0.5–4 Hz), theta (4–8 Hz), and alpha (8–12 Hz) frequency bands using the inter-trial phase coherence (ITPC) measure from the EEGLAB software (Delorme and Makeig, 2004). Previous studies have shown that the trial-by-trial synchronization of neural activity in delta, theta, and alpha frequency bands reflects auditory processing and the generation of the N1-P2 response (Edwards et al., 2009; Koerner and Zhang, 2015). Inter-trial phase coherence estimates EEG trial-by-trial mean normalized phase as a function of time and frequency. These values range from 0, which indicates no synchronization across trials, to 1 which indicates perfect synchronization across trials. Inter-trial phase coherence data was averaged across each frequency band at electrode Cz and averaged peak ITPC values in time windows corresponding to the N1 and P2 components were extracted for each frequency band, listening condition, and participant. Induced power, known also as induced event-related spectral perturbation (ERSP), was also calculated in delta, theta, and alpha frequency bands at time points corresponding to the N1 and P2 responses at electrode Cz. The ITPC measure described above represents evoked oscillatory activity, which is phase-locked to the stimulus (Bidelman, 2015; Shahin et al., 2009). In contrast, induced oscillatory activity is not phase-locked to the stimulus. Trial-by-trial induced power was calculated in the current study by subtracting the evoked response from the EEG response on each trial. In other words, this measure allows for an estimation of oscillatory power that cannot be explained by the power of the averaged event-related potential response (David et al., 2006). In order to estimate spectral characteristics of the MMN response, logarithmic spectral power in the theta band was extracted using the subtracted MMN waveform at electrode Cz over the entire analysis epoch, including the pre-stimulus baseline from −100 to 700 ms, using the EEGLAB software (Delorme and Makeig, 2004). It has been shown that modulation of theta power is linked with cognitive memory processes and likely contributes to the generation of the MMN response during auditory processing (Fuentemilla et al., 2008; Hsiao et al., 2009; Ko et al., 2012; Koerner et al., 2016). Each spectral calculation used a modified short-term Fourier Transform (STFT) with Hanning window tapering that was implemented in EEGLAB (Koerner and Zhang, 2015), which is recommended for the analysis of low frequency activities. The modified STFT method used overlapping sliding windows that are adapted to the target frequency bins to overcome limitations due to the use of fixed windows in conventional analysis. Zero-padding was applied to short epochs with insufficient sample points for Fourier transform. The frequency range for calculating ITPC and spectral power was 0.5–40 Hz with a step interval of 0.5 Hz.

All statistical analyses were completed in R (R Core Team, 2014). Linear mixed-effects (LME) models were created, with $\alpha = 0.05$, to examine the statistical significance of each fixed effect, including stimulus type (/da/ or /bu/), SNR condition (0 or −3 dB SNR), age, and HI (PTA^2), on each AERP measure using a "by-subject" intercept as a random effect.

2.4.2. Behavioral measures

In the phoneme discrimination test, sensitivity (d') and reaction time for the detection of consonant and vowel changes in the double-oddball paradigm were obtained from the button-press responses recorded during each noise condition. Linear mixed-effects regression models were used to determine statistical significance of each fixed effect, including stimulus type (/da/ or/ bu/), SNR condition (0 or −3 dB SNR), age, and HI (PTA^2), on behavioral discrimination accuracy and reaction time across all participants using a "by-subject" random intercept.

Participants completed two lists of IEEE sentences (IEEE, 1969) spoken by a female and two lists spoken by a male for each listening condition. Each list consists of 10 low context sentences with 5 key words in each sentence. Therefore, each participant was presented with 4 sentence lists for 200 key words total per SNR listening condition. Word-by-word responses were automatically scored by MATLAB and all incorrect responses were checked by a researcher to ensure that spelling mistakes did not result in an incorrect response. An additional LME model was carried out to examine the significance of SNR listening condition (0 or −3 dB SNR), age, and HI (PTA^2) on sentence recognition across participants.

2.4.3. Brain-behavior relationships

Linear mixed-effects (LME) models were developed in R (R Core Team, 2014) and the *nlme* package (Pinheiro et al., 2016) to determine whether objective AERPs and measures of event-related cortical activity were predictive of behavioral speech perception at the syllable- and sentence-levels (Koerner and Zhang, 2017). Data transformations included an arcsine transform on percent correct behavioral sentence recognition data as well as re-scaling AERP latency and behavioral reaction time values to make their scales comparable to other variables. Participants were treated as a random effect using a "by-subject" random intercept in each LME model while speech stimulus (/bu/or/da/), SNR condition (0 or −3 dB SNR), age, and PTA^2 were included as blocking variables to account for repeated measure and inherent differences within and across participants. AERP latency, amplitude, ITPC, induced ERSP, or spectral power values were added as fixed effects to predict percent correct phoneme detection, reaction time, and sentence recognition scores across participants. Separate models were developed for neural responses to the consonant and vowel change for prediction of sentence-level performance. The significance of each fixed effect in predicting each behavioral outcome measure was assessed with a $\alpha = 0.05$.

3. Results

Statistical analysis revealed that HI had a significant effect on behavioral speech tasks (Table 3) as well as the MMN response, but did not significantly impact N1 or P2 AERPs (Table 2). In contrast, results showed that age had a significant effect on N1 and P2 AERPs but not the MMN response (Table 2). Results also showed significant effects of stimulus (/bu/ vs. /da/) and SNR listening condition (0 vs. −3 dB SNR) across AERP and behavioral responses (Tables 2–3, Figs. 2–5). Linear mixed-effects regression analysis showed that AERPs represent neural correlates of phoneme-and sentence-level performance across participants (Tables 4–6).

3.1. Brain measures

Results from the LME models showed that there was a significant effect of stimulus on N1 latency ($F(1,52) = 21.29$, $p < 0.001$) as well as ITPC associated with the N1 response in delta ($F(1,52) = 7.23$, $p = 0.010$), theta ($F(1,52) = 11.51$, $p = 0.010$), and alpha ($F(1,52) = 7.64$, $p = 0.008$) frequency bands across listening conditions. There was also a significant effect of stimulus on P2 latency ($F(1,52) = 4.48$, $p = 0.039$) as well as ITPC associated with the P2 response in the theta ($F(1,52) = 8.93$, $p = 0.004$) and alpha

Table 2
F-statistics of the effects of stimulus (/da/ vs. /bu/), SNR listening condition (−3 vs. 0 dB SNR), age, and PTA^2 across participants for N1, P2, and MMN measures.

N1	Effect of Stimulus	Effect of SNR Condition	Effect of Age	Effect of PTA^2
Latency	21.29**	8.27**	3.10	0.78
Amplitude	3.40	7.65**	0.15	3.98
Delta ITPC	7.23**	10.82**	7.43*	0.62
Theta ITPC	11.51**	12.20**	2.37	0.11
Alpha ITPC	7.64**	7.80**	2.23	0.13
Induced Delta ERSP	0.40	1.42	0.29	0.21
Induced Theta ERSP	0.11	1.22	1.21	0.05
Induced Alpha ERSP	0.28	0.53	0.97	0.02
P2				
Latency	4.48*	25.85***	6.94*	0.02
Amplitude	3.75	1.00	2.27	2.11
Delta ITPC	2.38	1.20	1.87	0.00
Theta ITPC	8.93**	2.66	5.43*	0.11
Alpha ITPC	7.94**	1.49	4.17	0.17
Induced Delta ERSP	0.43	0.10	0.40	3.87
Induced Theta ERSP	0.10	0.08	0.00	0.94
Induced Alpha ERSP	0.01	0.51	0.01	0.36
MMN				
Latency	9.93*	10.43**	0.04	1.47
Amplitude	2.59	1.32	0.73	4.68*
Theta Power	3.34	0.02	0.63	5.97*

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$.

Table 3
F-statistics of the effects of stimulus (/da/ vs. /bu/), SNR listening condition (−3 vs. 0 dB SNR), age, and PTA^2 across behavioral speech-in-noise measures.

Behavioral Measure	Effect of Stimulus	Effect of SNR Condition	Effect of Age	Effect of PTA^2
Phoneme Discrimination Accuracy (d′)	1339.95***	2.39	0.29	12.00**
Phoneme Discrimination Reaction Time (ms)	12.12**	0.12	1.58	0.65
Sentence Recognition (% correct)	–	135.68***	3.18	3.29

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$.

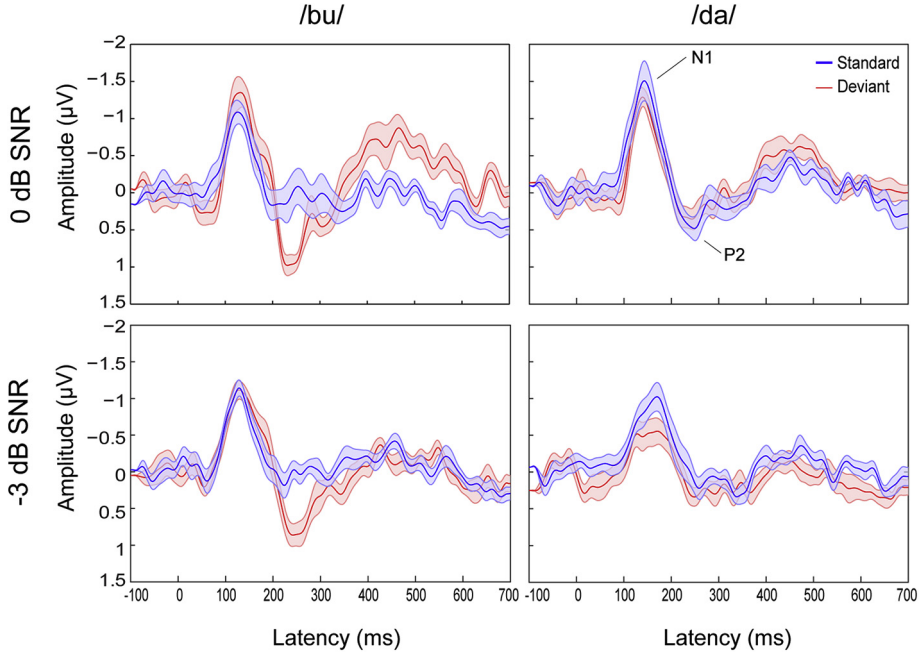

Fig. 2. Grand average ERP waveforms in response to standard (blue) and deviant (red) presentations of /bu/ (left column) and /da/ (right column) speech stimuli in 0 dB SNR (top row) and −3 dB SNR (bottom row) listening conditions. Note that these waveforms are averaged across all participants. Data used for analysis in the current study included N1-P2 responses from the standard (blue) presentations of /bu/ and /da/ across listening conditions as well as MMN difference waves which were computed by subtracting the neural response to the standard stimuli (blue) from that of the deviant stimuli (red) for /bu/ and /da/ across listening conditions. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

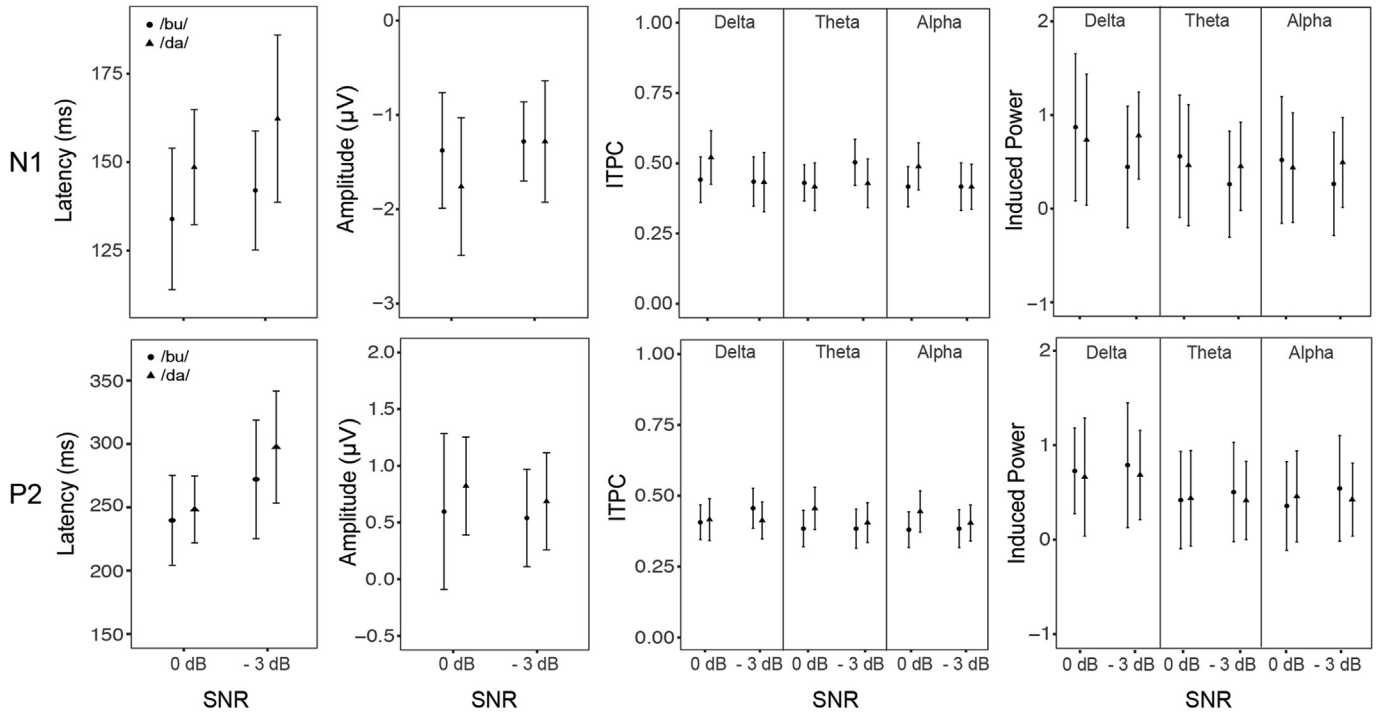

Fig. 3. Means and standard deviations (error bars) for N1 and P2 latency, amplitude, ITPC, and induced power measures in response to /bu/ (circle) and /da/ (triangle) stimuli in 0 and -3 dB listening conditions across all participants.

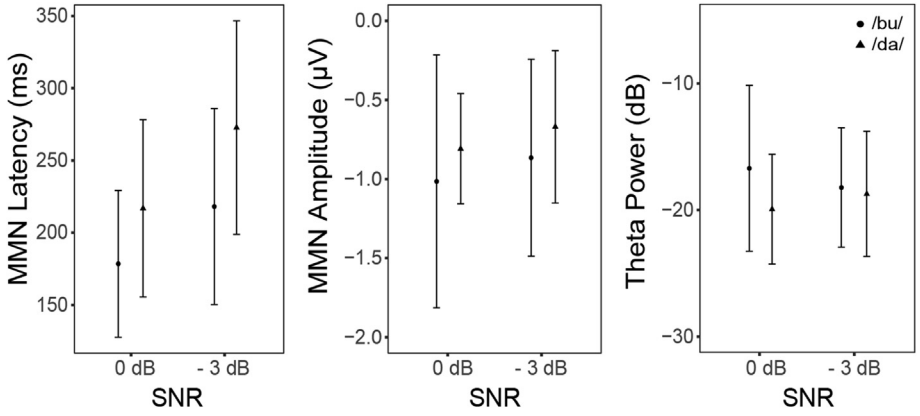

Fig. 4. Means and standard deviations (error bars) for MMN latency, amplitude, and theta power measures in response to /bu/ (circle) and /da/ (triangle) stimuli in 0 and -3 dB listening conditions across all participants.

($F(1,52) = 7.94$, $p = 0.007$) frequency bands across listening conditions. N1 and P2 latencies were significantly delayed in response to the consonant change compared to the vowel change across listening conditions. In contrast, trial-by-trial phase locking in frequency bands associated with the N1 and P2 responses was stronger in response to the consonant change compared to the vowel change. Post-hoc analysis revealed that this trend was driven by a significant difference between ITPC values in the 0 dB SNR listening condition for delta ($t(33) = -2.68$, $p = 0.011$), theta ($t(32) = -2.98$, $p = 0.005$), and alpha ($t(33) = -2.78$, $p = 0.009$) frequency bands associated with the N1 response and theta ($t(33) = -3.07$, $p = 0.004$) and alpha ($t(33) = -2.81$, $p = 0.008$) bands associated with the P2 response.

The LME models also revealed that there was a significant effect of SNR listening condition on N1 latency ($F(1,52) = 8.27$, $p = 0.006$), N1 amplitude ($F(1,52) = 7.65$, $p = 0.008$) as well as ITPC associated with the N1 response in the delta ($F(1,52) = 10.82$, $p = 0.002$), theta ($F(1,52) = 12.20$, $p = 0.001$), and alpha ($F(1,52) = 7.80$, $p = 0.007$) frequency bands across stimuli. The N1 amplitude as well as ITPC across frequency bands was smaller in the 0 dB SNR condition compared to the more difficult −3 dB SNR condition. There was also a significant effect of SNR condition on P2 latency ($F(1,52) = 25.85$, $p < 0.001$), such that latency was longer in response to speech stimuli in the −3 dB SNR condition compared with the 0 dB SNR condition.

Finally, the LME models showed that there was a significant effect of age on ITPC in the delta band associated with the N1 response ($F(1,15) = 7.43$, $p = 0.016$) as well as an effect of age on P2 latency ($F(1,15) = 6.94$, $p = 0.019$) and ITPC in the theta band associated with the P2 response ($F(1,15) = 5.43$, $p = 0.034$) across stimuli and listening conditions. Younger participants tended to have shorter P2 latencies and higher ITPC in the N1 delta band and P2 theta band compared to older participants.

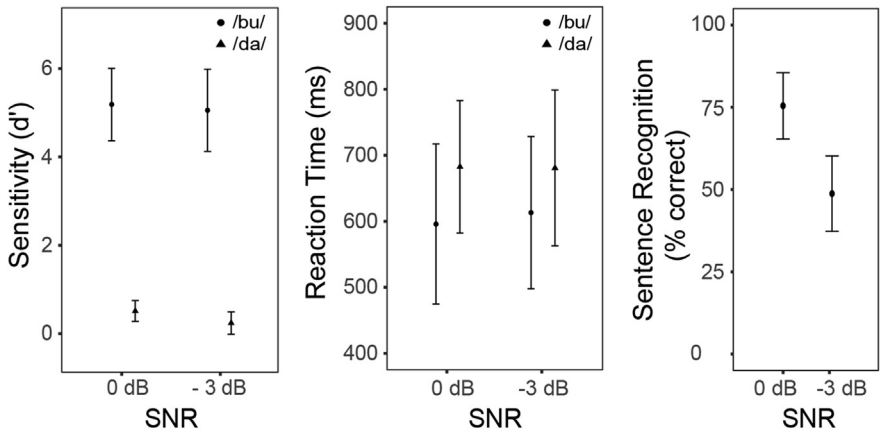

Fig. 5. Means and standard deviations (error bars) for phoneme change detection sensitivity (d') and phoneme change detection reaction time (ms) in response to /bu/ (circle) and /da/ (triangle) stimuli in 0 and -3 dB listening conditions across all participants and percent correct sentence recognition scores in 0 and -3 dB listening conditions across all participants.

Table 4
F-statistics (F) and regression coefficients (β) for fixed effects (N1 latency, amplitude, ITPC, and induced ERSP in delta, theta, and alpha frequency bands) in response to the vowel change (/bu/) and consonant change (/da/) for predicting speech perception across participants.

N1	Percent Correct Phoneme Detection		Phoneme Detection Reaction Time		Percent Correct Sentence Recognition (/bu/)		Percent Correct Sentence Recognition (/da/)	
	F	β	F	β	F	β	F	β
Latency	0.19	0.34	0.87	0.84	1.61	−0.12	2.19	0.09
Amplitude	0.16	0.06	0.44	−0.17	2.27	0.05	0.75	0.05
Delta ITPC	0.18	−1.65	0.06	0.49	3.32	0.05	3.53	0.26
Theta ITPC	0.67	0.04	0.03	−6.79	0.00	−0.12	0.17	0.62
Alpha ITPC	0.15	2.28	1.28	6.87	2.04	0.69	0.02	−0.29
Induced Delta ERSP	2.95	0.08	0.25	0.09	0.17	0.13	0.17	−0.07
Induced Theta ERSP	0.80	−0.29	0.17	0.19	1.63	−0.33	0.05	0.23
Induced Alpha ERSP	0.01	−0.04	0.11	−0.27	3.13	0.17	1.76	−0.16

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$.

Table 5
F-statistics (F) and regression coefficients (β) for fixed effects (P2 latency, amplitude, ITPC, and induced ERSP in delta, theta, and alpha frequency bands) in response to the vowel change (/bu/) and consonant change (/da/) for predicting speech perception across participants.

P2	Percent Correct Phoneme Detection		Phoneme Detection Reaction Time		Percent Correct Sentence Recognition (/bu/)		Percent Correct Sentence Recognition (/da/)	
	F	β	F	β	F	β	F	β
Latency	0.26	0.11	2.20	0.52	0.42	0.02	6.72*	−0.07
Amplitude	1.42	0.10	1.11	−0.24	0.35	−0.33	2.69	0.00
Delta ITPC	2.97	4.97	2.04	1.61	1.91	1.67	5.15*	0.12
Theta ITPC	2.51	−8.06	0.34	15.03	3.67	−1.35	2.28	0.13
Alpha ITPC	2.25	5.16	2.53	−14.31	0.27	0.33	0.56	0.72
Induced Delta ERSP	0.56	0.22	3.65	0.37	0.62	0.09	0.07	0.07
Induced Theta ERSP	2.26	−0.05	3.42	−0.99	2.74	−0.29	0.97	−0.05
Induced Alpha ERSP	0.49	−0.38	0.00	−0.06	2.34	0.17	0.03	−0.03

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$.

The linear-mixed effects regression models revealed that there was a significant effect stimulus ($F(1,52) = 9.93$, $p = 0.003$) and listening condition ($F(1,52) = 10.43$, $p = 0.002$) on MMN latency. In particular, MMN latency was longer in response to the consonant change compared to the vowel change across conditions and was also longer in the −3 dB SNR condition compared to the 0 dB SNR condition across stimuli. The LME models also revealed a significant effect of hearing loss, as measured by PTA^2, on MMN amplitude ($F(1,15) = 4.68$, $p = 0.047$) and power in the theta band ($F(1,15) = 5.97$, $p = 0.027$). Results showed that MMN amplitude and theta power were significantly weaker with poorer PTA^2 values across listening conditions and stimuli.

Table 6
F-statistics (F) and regression coefficients (β) for fixed effects (MMN latency, amplitude, and theta power) in response to the vowel change (/bu/) and consonant change (/da/) for predicting speech perception across participants.

MMN	Phoneme Detection Sensitivity		Phoneme Detection Reaction Time		Percent Correct Sentence Recognition (/bu/)		Percent Correct Sentence Recognition (/da/)	
	F	β	F	β	F	β	F	β
Latency	0.10	−0.07	2.26	0.08	4.10	−0.00	0.82	−0.00
Amplitude	4.82*	−0.25	2.86	0.26	0.72	−0.03	0.41	0.03
Theta Power	1.61	−0.02	14.53***	−0.09	1.10	−0.00	0.00	−0.00

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$.

3.2. Behavioral measures

Results from the LME models showed that there was a significant effect of stimulus (/bu/ vs. /da/) on phoneme change-detection sensitivity ($F(1,52) = 1339.95$, $p < 0.001$) and reaction time ($F(1,52) = 12.12$, $p = 0.001$), such that the ability to accurately detect the consonant change was drastically poorer than the ability to detect the vowel change across listening conditions. Linear mixed-effects regression analysis also revealed a significant effect of SNR listening condition on percent correct sentence recognition ($F(1,15) = 135.68$, $p < 0.001$), such that performance was poorer in the −3 dB SNR listening condition compared to the 0 dB SNR listening condition across participants. There was also a significant effect of hearing impairment on phoneme-change detection sensitivity ($F(1,52) = 12.00$, $p = 0.003$), such that those with better hearing thresholds had higher behavioral performance scores compared to those with poorer hearing sensitivity.

3.3. Brain-behavior relationships

The linear mixed-effects regression models revealed that ITPC in the delta frequency band associated with the P2 ($F(1,9) = 5.15$, $p = 0.049$) response to /da/ as well as P2 latency in response to /da/ ($F(1,9) = 6.72$, $p = 0.029$) were significant predictors of sentence-level perception across listening conditions. Results showed that decreased delta ITPC or increased P2 latency resulted in decreased sentence recognition performance. Additionally, the LME regression analysis showed that MMN amplitude was a significant predictor of phoneme change-detection sensitivity ($F(1,49) = 4.82$, $p = 0.033$) and that theta power was a significant predictor of phoneme change-detection reaction time ($F(1,49) = 14.53$, $p < 0.001$). Results showed that as MMN amplitude decreased, behavioral phoneme detection sensitivity tended to decrease and as spectral power in the theta band decreased, behavioral reaction time for phoneme detection increased. An examination of residual plots revealed that all residuals were normally distributed without any trend toward heteroscedastic variance for each model.

4. Discussion

This work primarily aimed to examine the effects of age and HI on the N1-P2 and MMN responses to a consonant versus a vowel change. In addition, this study was designed to determine whether AERPs could predict the effects of HI on phoneme- and sentence-level speech perception in noise.

4.1. Effects of HI on the neural coding and perception of speech in noise

Our results showed that the presence of hearing loss did not have a significant impact on speech-evoked N1-P2 responses. This is consistent with results from Billings et al. (2015), which showed no significant effect of HI on N1-P2 responses to speech across various presentation levels and SNRs. However, the current results are not fully consistent with previous studies that have shown enhanced N1-P2 responses in older participants with HI when participants were tested in quiet listening conditions or conditions with more low-level noise (Alain et al., 2014; Bidelman et al., 2014; Harkrider et al., 2006; Tremblay et al., 2003). It has been suggested that these enhanced neural responses may be a result of greater release from neural inhibition caused by hearing impairment (Alain et al., 2014; Bidelman et al., 2014; Billings et al., 2015; Harkrider et al., 2006; Tremblay et al., 2003). Disagreement between previous studies and the current results may be due to differences in experimental methodology, such that under certain quiet listening conditions or noise conditions with better SNRs, greater release from neural inhibition occurs and enhances N1-P2 responses in those with poorer hearing sensitivity. Studies examining the subcortical encoding of speech using the frequency following response (FFR) have also revealed significant effects of hearing impairment on the neural coding of speech in quiet listening conditions (Anathakrishnan et al., 2016; Bidelman et al., 2014) and in background noise (Anderson et al., 2013a). Anderson et al. (2013a) examined the effects of hearing loss on the FFR to the speech stimulus /da/ in background noise presented at a 10 dB SNR and showed that while HI participants had similar encoding of temporal fine structure information in noise compared to NH participants, HI participants had stronger responses to temporal envelope cues than NH participants in noise. It would be reasonable to expect that HI-related effects on the subcortical FFR might also appear in the later cortical N1-P2 response. However, to our knowledge, no studies have examined the subcortical encoding of speech in noise at more difficult SNRs similar to those used in the current study. As mentioned above, these differences in experimental stimuli and listening conditions may influence the effects of HI on the passive neural coding of speech.

This work adds to our current body of knowledge by revealing how HI impacts the encoding of speech in noise at different stages of cortical processing. While HI did not have a significant effect on N1-P2 AERPs or MMN latency in response to speech in noise, our results showed that it did significantly impact MMN amplitude and theta power as well as CV syllable discrimination across listening conditions. These results may suggest that later cortical mechanisms that are involved in perceptual and cognitive stimulus discrimination processes may be more sensitive to detrimental effects of HI on the neural coding of speech in noise. This pattern of results is consistent with those from earlier studies which showed that reduced audibility from simulated HI (Martin et al., 1997, 1999; Martin and Stapells, 2005) and the presence of actual HI (Oates et al., 2002) tended to have a differential effect on AERP components. Several possible explanations support our findings regarding the differential effect of HI on the N1, P2, and MMN AERPs, including the presence of cognitive processing deficits occurring in addition to reduced audibility from HI. In addition to neural generators in the auditory cortex, frontal areas have also been implicated in the neural generation of the MMN and have been associated with an involuntary orienting response to stimulus change (Bidelman and Dexter, 2015; Giard et al., 1990; Näätänen et al., 2007; Yago et al., 2001). Since the MMN is thought to index this attentional switch to a change in auditory stimuli, it is possible that our results reflect that this involuntary attention trigger underlying the MMN is less sensitive in these individuals with HI. In other words, factors associated with the presence of HI may also be impacting more top-down central processing mechanisms, such as attention, the effects of which would be most evident in the MMN response compared to earlier AERP components. Another possible explanation for the differential effect of HI on AERPs seen in the current study involves potential differences in temporal integration and the formation of sensory percepts across participants with varying degrees of hearing sensitivity. The MMN reflects the violation of a predictable sensory memory trace of repeating, “standard” stimuli by an occasional “deviant” stimulus and is therefore dependent on the accurate formation of memory traces of preceding “standard” stimuli (Winkler and Näätänen, 1992; Näätänen, 1995; Yabe et al., 1998). Studies have previously shown that the MMN is sensitive to deficits related to the formation of a memory trace and abnormal temporal integration in clinical populations (Grau et al., 2001; Kujala et al., 2000; Ahveninen et al., 1999). It is possible that differences in the MMN across participants with greater degrees of hearing loss are related to the faster decay or improper formation of a sensory memory trace. Even in the absence of HI, AERPs have been shown to be sensitive to mild cognitive impairment (Baldeweg et al., 2004; Bidelman et al., 2017; Golob et al., 2007). Possible cognitive deficits in auditory attention or memory that occur in addition to reduced audibility from HI may also be responsible for the pattern of results seen in our behavioral sentence recognition task. Unlike results from Billings et al. (2015), the current study showed that the effects of HI on sentence recognition approached but did not reach statistical significance. Given that there was a wide range in sentence recognition scores across participants and listening conditions, it is possible that additional factors beyond reduced audibility from HI, including potential cognitive effects related to impaired sensory memory or attention that would also impact the MMN response, were affecting higher-level sentence-level recognition in noise for the stimulus presentation level and SNRs used in this study.

Although Oates et al. (2002) only recorded AERPs in quiet, our AERP findings showed consistent patterns with HI having a

significant effect on MMN amplitude, but not MMN latency in response to speech. A possible explanation is that the presence of HI can induce large variability in AERP amplitude while HI related effects on AERP latency are more predictable and stable. It is possible that the presence of hearing loss exacerbates trial-by-trial latency jitter such that during the averaging process, amplitudes are more affected in the final averaged AERP waveform (Koerner & Zhang, 2015). This potential issue further highlights the use of time-frequency analysis measures to extract and examine cortical oscillatory activity in frequency bands of interest that are typically ignored by traditional AERP averaging techniques.

4.2. Effects of stimuli and SNR listening conditions on the neural coding and perception of speech in noise

Our results showed that the neural coding of the consonant change was weaker than that of the vowel change for N1 latency and MMN latency, which is consistent with results from the behavioral phoneme detection task. These results are consistent with previous neural and behavioral findings which suggest that weaker, more aperiodic consonants are impacted more in background noise than stable, periodic vowel sounds (Cunningham et al., 2002; Russo et al., 2004; Shetake et al., 2011; Song et al., 2011). However, our current results also revealed weaker trial-by-trial neural synchrony in response to the vowel change compared to the consonant change across participants, primarily in the 0 dB SNR listening condition. These unexpected results in the more favorable SNR listening condition may be understood by examining acoustic differences between the two stimuli. F1, F2, and F3 formant transitions as well as steady F1, F2, and F3 center frequencies for the vowel portion of the CV syllables were higher in frequency for /da/ compared to /bu/. It is possible that trial-by-trial neural phase locking associated with N1 and P2 is stronger in response to higher compared to lower frequency stimuli, but that this higher cortical synchrony does not translate to shorter neural processing times for the CV syllable /da/. Future studies are needed to examine the potential differential effects of stimuli on the neural coding of speech contrasts in noise to determine how stimulus presentation level, SNR, or noise type may impact responses across participant populations.

4.3. Effects of age on the neural coding and perception of speech in noise

Although this study attempted to control for potential effects of age by only using a group of adult participants, it was not possible to adequately match participants by age across a wide range of hearing sensitivities. It has been shown that aging may impact a range of cognitive and auditory processes that are important for speech perception (Gallun et al., 2012; Ruggles et al., 2011). Indeed, our analysis showed that age was significantly correlated with PTA^2 in the current group of participants ($r = 0.52$, $p = 0.001$), such that older participants tended to have poorer hearing sensitivity. Even though age and PTA^2 were correlated, age was included as a factor in our LME models to ensure that any potential effects of HI on the neural coding and perception of speech were not driven by underlying variables associated differences in age. Our data is consistent with Billings et al. (2015), showing that while there was no effect of HI on N1 and P2 responses, age had a significant impact on N1 and P2 responses to speech in noise when a group of older NH participants were compared to a group of younger NH participants. This trend was unexpected given the narrow age ranged used in the current study. A possible explanation for these results is given by Billings et al. (2015), which speculated that impaired neural synchrony due to central aging effects may impact obligatory N1-P2 responses to speech in noise. However, our data revealed that differences in age did not have a significant impact on the MMN response, which may suggest that variability in the pre-attentive auditory change discrimination in noise may not be driven by aging effects, but may be more related to differences in hearing abilities. Furthermore, our results did not show a significant effect of age on sentence perception across listening conditions used in this study. Since this study only used a group of adult listeners with varying degrees of hearing loss, future studies could systematically examine how age may modulate the effects of HI on neural and behavioral measures of speech perception in noise.

4.4. Neural correlates of the effects of HI on speech-in-noise perception

Our results show that both cortical P2 and MMN responses may be predictors of speech perception in noise in participants with HI. These results are consistent with those from Billings et al. (2015), who showed that N1 and P2 responses are predictive of speech perception in noise across participants with HI. However, although previous studies have examined whether the MMN response is able to predict speech perception in noise in NH individuals (Koerner et al., 2016), no studies have examined whether the MMN is predictive of the effects of HI on speech perception in noise. Therefore, our data added to the extant literature in showing that the MMN here on individuals with HI represented a potential neural correlate of speech perception in background noise. There is also novel evidence that measures of trial-by-trial neural synchrony and spectral power associated with these AERP components are potential indicators of behavioral performance across participants with and without HI. While it has been established that the sensory, perceptual MMN response represents an index of auditory change detection, the high inter- and intra-subject variability typically seen in this AERP represents a tremendous barrier to using this passive cortical response for clinical assessment of speech perception difficulties (Kurtzberg et al., 1995; Lang et al., 1995; Martin et al., 1999, 2008; Näätänen et al., 2007; Näätänen, 1995; Stapells, 2002). In addition, although the passive MMN depends on sensory and cognitive processing mechanisms for pre-attentive change discrimination, it may not fully reflect additional central processing mechanisms necessary for active discrimination tasks. Despite this limitation, our results suggest that the MMN may be a useful measure for exploring the effects of HI and background noise on speech perception, and that spectral power analysis may represent an additional, complementary tool to examine mechanisms how various factors impact cognitive processes related to the perception of speech in noise.

4.5. Novelty, limitations, and future directions

Previous work that examined the effects of HI on the neural coding of speech in noise showed that HI did not have a significant effect on N1 and P2 responses to speech in noise (Billings et al., 2015). The current work replicated these findings and further revealed that, for the stimuli and listening conditions tested in this study, hearing sensitivity may have a differential effect on perceptual and cognitive processes for recognizing speech in the presence of background noise. More specifically, results suggest that variations in hearing sensitivity do not have the same effect on the early sensory encoding of acoustic cues in noise as the later cognitive processing of speech discrimination in background noise. These results provide valuable information regarding the effects of HI on the encoding of speech-in-noise at different stages of cortical processing and suggest that the MMN may be more sensitive measure to track hearing-related changes in the neural coding of

speech in background noise. Future research should compare these AERP responses to speech in noise in individuals who receive amplification from hearing aids, as this would allow for an examination of whether greater accessibility to speech cues alters the differential effect of HI on neural responses that reflect sensory and cognitive processing mechanisms. These findings could influence rehabilitation strategies for individuals who have difficulty communicating in noise, as solutions targeting top-down cognitive processing mechanisms may result in improved neural coding and perception of speech in noise compared to those that focus on improving the bottom-up neural processing of acoustic cues.

Previous studies that examined the neural coding and perception of speech in noise have determined that AERPs represent potential neural correlates of the effects of background noise on speech perception (Bennett et al., 2012; Billings et al., 2013; Koerner et al., 2016, 2017). However, less is known about whether these neural responses can track variability in speech-in-noise perception caused by HI. Although previous studies have established significant brain-behavior links between the passive MMN response and the ability to perceive speech in noise in NH listeners (Bidelman and Dexter, 2015; Oates et al., 2002; Koerner et al., 2016; Kozou et al., 2005; Zhang et al., 2009), this is the first study to explore these relationships in HI listeners. Furthermore, our results showed that time-frequency analysis techniques represent additional tools that can be used to assess speech processing across individuals, which has both theoretical and practical implications.

Our analysis revealed that trial-by-trial induced oscillatory power associated with the N1-P2 response was not affected by changes in stimulus (/da/ vs. /bu/), SNR listening condition (−3 vs. 0 dB SNR), participant age, or PTA^2 (Table 2, Fig. 3). In addition, this measure did not appear to be predictive of any of the behavioral tasks examined in this study across participants (Tables 4–5). In other words, unlike the ITPC, which reflects evoked oscillatory activity (Bidelman, 2015; Shahin et al., 2009), trial-by-trial induced oscillatory activity does not appear to be related to changes in neural activity due to differences in speech stimulus (/da/ vs. /bu/), SNR (−3 vs. 0 dB SNR), or participant variables such as age or HI, and does not appear to be underlying variability in speech perception across participants. A lack of change in trial-by-trial power with a concomitant change in trial-by-trial phase locking (ITPC) across experimental factors is thought to support the idea that stimulus-evoked phase alignment of oscillatory activity at least partially contributes to the neural generation of AERPs (Makeig et al., 2004), which is consistent with the current pattern of results and those of previous studies (Fuentemilla et al., 2006; Koerner and Zhang, 2015; Koerner et al., 2017). Therefore, these results may provide additional evidence supporting the idea that stimulus-related phase alignment of cortical oscillations plays an important role in the neural generation of N1 and P2 responses.

The current work is the first to treat HI as a gradient variable among listeners in examining the effects of HI on the cortical encoding of speech in noise. This method may allow for more precise examination of how slight differences in hearing thresholds impacts the neural coding of speech, as participants are not grouped together simply based on ranges of hearing sensitivity. For instance, previous studies often grouped participants with a PTA^2 less than 25 dB HL in a "normal hearing" group and those with a PTA^2 between 25 and 49 dB HL in a "mild hearing loss" group (Oates et al., 2002). To ensure that the pattern of our results was not driven by the four participants who had PTA^2 values greater than 25 dB HL, we also repeated our analysis of the effects of hearing loss on each MMN measure using a categorical grouping method similar to previous studies. The results showed that there was actually no significant effect of "hearing loss group" on our MMN measures when our four participants with PTA^2 values greater than 25 dB HL were compared against the remaining participants with PTA^2 values less than 25 dB HL (latency: $F(1,15) = 1.40$, $p = 0.25$; amplitude: $F(1,15) = 1.87$, $p = 0.19$; theta power: $F(1,15) = 2.81$, $p = 0.11$). These results suggest that our participants with PTA^2 values greater than 25 dB HL were not driving the pattern of results, but that the variability in MMN measures across all participants who varied in PTA^2 values was able to better identify effects of hearing sensitivity on neural responses, even when hearing sensitivity was considered normal as measured by the standard pure-tone audiogram. However, it should be noted that the disparity in sample sizes between our "normal hearing" and "hearing impaired" participants in this categorical grouping is a limitation which makes it difficult to make a strong claim.

Limitations in our study need to be acknowledged. As stated previously, it is difficult to control for factors that likely vary across participants with HI. The current study estimated HI using a two-frequency PTA (PTA^2, Oates et al., 2002; Korczak et al., 2005), which represents only one attempt to estimate a participant's degree of peripheral hearing impairment. There is a growing body of evidence showing that a standard pure-tone audiogram is not able to accurately predict difficulties perceiving speech perception in complex auditory environments (Killion & Niquette, 2000; Kujawa & Liberman, 2009). Estimating the effect of HI using pure-tone hearing thresholds may not be sensitive to other factors that impact auditory perception above and beyond the effects of reduced audibility, such as reduced suprathreshold spectrotemporal processing abilities, deficits in cognitive processing, or impaired central auditory processing abilities. As discussed above, it is possible that the differential effects of HI on AERPs observed in this study are related to factors beyond reduced audibility as measured by PTA^2, including cognitive factors such as reduced sensory memory or attention (Füllgrabe et al., 2015; Moore et al., 2014). Therefore, attempting to examine and control for the effects of these other factors may help to improve our understanding of how HI and potentially co-occurring cognitive deficits impact these neural measures and how AERPs relate to behavioral performance in background noise across participants in this clinical population.

An additional limitation of the current study is the use of a single, fixed presentation level across participants with varying degrees of HI, which resulted in stimulus presentation at different sensation levels for each participant. In other words, stimuli were presented closer to hearing thresholds for participants with greater degrees of hearing loss. However, previous studies (Billings et al., 2009) and our own data here show that such signal level differences in our experimental setup do not significantly impact the N1-P2 responses. Thus, our findings on the later-occurring MMN response are likely not attributable to differences in sensation levels across participants. Due to time constraints and considerations on fatigue-related EEG artifacts, testing sessions using multiple presentation levels and listening conditions were not completed for each participant. Future work could include systematic control of the presentation levels set above a defined speech or pure-tone hearing threshold. Recent work has suggested that there may be a differential effect of noise type on AERPs across participants with and without HI (Maamor and Billings, 2017). Therefore, future work should additionally explore the effects of stimulus factors and SNRs on AERPs and their relationship with behavioral performance in this clinical population. Inclusion of a quiet listening condition would also allow for a more comprehensive examination of the effects of HI on the neural coding of speech in noise, as the AERPs elicited in background noise could be compared to "baseline" responses in a quiet listening condition for each participant. The role of attention is another important factor to consider as it has been shown to affect not only AERP responses but

also how consonants and vowels differentially contribute to speech intelligibility (Koerner et al., 2017). Larger sample sizes and more diverse participant populations are also needed to determine the reliability of these measures. Despite these limitations, the current findings contribute new evidence that AERPs may be useful in assessing and predicting performance in clinical populations who have difficulty communicating in complex listening environments.

5. Conclusion

This study was designed to determine how hearing impairment impacts N1-P2 and MMN AERPs and their associated event-related cortical oscillations in response to speech in noise. Furthermore, this work aimed to determine whether these neural measures can predict phoneme- and sentence-level perception in background noise across participants with and without HI. Consistent with our predictions, HI had a significant impact on the MMN response, but not on the sensory N1-P2 complex. Results were also consistent with our hypothesis that these objective neural responses represented potential predictors of speech-in-noise perception across listening conditions. These findings have important clinical implications regarding the use of electrophysiological measures in assessing and predicting speech perception in clinical populations with hearing impairment.

Financial disclosures/conflicts of interest

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heares.2018.10.009.

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