Objective evidence of temporal processing deficits in older adults

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Abstract

The older listener's ability to understand speech in challenging environments may be affected by impaired temporal processing. This review summarizes objective evidence of degraded temporal processing from studies that have used the auditory brainstem response, auditory steady-state response, the envelope- or frequency-following response, cortical auditory-evoked potentials, and neural tracking of continuous speech. Studies have revealed delayed latencies and reduced amplitudes/phase locking in subcortical responses in older vs. younger listeners, in contrast to enhanced amplitudes of cortical responses in older listeners. Reconstruction accuracy of responses to continuous speech (e.g., cortical envelope tracking) shows over-representation in older listeners. Hearing loss is a factor in many of these studies, even though the listeners would be considered to have clinically normal hearing thresholds. Overall, the ability to draw definitive conclusions regarding these studies is limited by the use of multiple stimulus conditions, small sample sizes, and lack of replication. Nevertheless, these objective measures suggest a need to incorporate new clinical measures to provide a more comprehensive assessment of the listener's speech understanding ability, but more work is needed to determine the most efficacious measure for clinical use.

Keywords

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Introduction

Older adults often struggle to understand speech in situations where their younger counterparts can hear with comparative ease. Three main factors are suggested to contribute to age-related declines in speech perception, namely peripheral hearing loss, central auditory processing deficits, and decreased cognitive function (CHABA, 1988). The role of central auditory processing in the deterioration of speech understanding has not been firmly established, however, because hearing loss and reduced cognition may confound performance on measures of speech understanding assessed in adverse listening environments (Humes et al., 2012). Objective measures that tap into the auditory nervous system's ability to accurately represent the speech signal may provide a means of evaluating central auditory processing without requiring active cooperation. Electrophysiologic (EEG) and magnetoencephalographic (MEG) recordings are objective measures that have been used to investigate the processing deficits that may contribute to speech perception impairments in older listeners.

Most studies that evaluate age-related impairments in neural speech processing recruit younger and older participants who have audiometrically normal hearing to reduce the confound of hearing loss. Generally, decreased temporal resolution is associated with aging (Gordon-Salant and Fitzgibbons, 1993; Gordon-Salant et al., 2007; Pichora-Fuller et al., 2007), whereas decreased frequency resolution is associated with hearing loss (Florentine et al., 1980; Phillips et al., 2000). It is difficult, however, to completely eliminate the influence of peripheral deficits, as almost all older individuals have decreased hearing in the extended high-frequency region above 8000 Hz (Davis et al., 1992; Matthews et al., 1997). There is evidence that elevated thresholds in this high-frequency region may contribute to speech-in-noise difficulties (Motlagh Zadeh et al., 2019; Yeend et al., 2019). Furthermore, behavioral and neural findings may be affected by other deficits that are not evident on the audiogram, including a subclinical loss of outer hair cells (Abdala and Dhar, 2012; Fabija ska et al., 2012; Hoben et al., 2017; Uchida et al., 2008), strial dysfunction (Ohlemiller, 2009; Schmiedt et al., 1990; Syka, 2010), broadened auditory filters (Hopkins and Moore, 2011; Patterson et al., 1982), cochlear synaptopathy and decreased numbers of auditory nerve fibers (Makary et al., 2011; Schmiedt et al., 1996; Sergeyenko et al., 2013; Viana et al., 2015; Wu et al., 2019), and loss of neural synchrony (Harris and Dubno, 2017; Marmel et al., 2013).

Many previous studies have revealed age-related temporal processing deficits when using compressed or reverberant speech signals (Fujihira et al., 2017; Gordon-Salant and Fitzgibbons, 1993; Gordon-Salant and Fitzgibbons, 2001; Gordon-Salant et al., 2007; Grose et al., 2009; Helfer and Wilber, 1990; Humes et al., 2007; Jenstad and Souza, 2007; Peelle and Wingfield, 2005; Wingfield et al., 2006), or when speech signals are presented in noise (Decruy et al., 2019; Dubno et al., 1984; Füllgrabe et al., 2015; Goossens et al., 2017; Helfer and Freyman, 2014; Presacco et al., 2016b). This review will consider the evidence from studies that have used EEG and MEG recordings to investigate age-related temporal processing deficits and will examine the possible roles of peripheral deficits in these studies. The sections are divided into five main types of measures: auditory brainstem response (ABR), envelope-following response (EFR)/auditory steady-state response (ASSR),

frequency-following response (FFR), cortical auditory-evoked potential (CAEP), and cortical envelope tracking. Within each section, we separate effects of aging and hearing loss. For the purposes of this review, peripheral disorders will encompass cochlear hair cell loss, cochlear synaptopathy, and auditory nerve dysfunction, and central disorders will encompass deterioration beyond the auditory nerve. The final section will consider how this information might influence management recommendations for older adults who have difficulty understanding speech in noise.

Auditory brainstem response (ABR)

Aging—The ABR is dependent on synchronous firing of populations of neurons that is sufficient to be detected with scalp electrodes. For this reason, disorders that result in disrupted neural synchrony, such as auditory neuropathy spectrum disorder, result in absent or highly degraded ABRs (Kraus et al., 2000; Starr et al., 1996; White-Schwoch et al., 2019). Latency differences on the order of fractions of milliseconds can be clinically significant in the detection of retrocochlear pathology (Chandrasekhar et al., 1995). The peaks of the ABR have sources from the auditory nerve through the inferior colliculus (IC); furthermore, the later peaks have multiple sources because of crossed auditory pathways (Kaga et al., 1997; Kuokkanen et al., 2018; Land et al., 2016; Møller and Jannetta, 1983). Aging may result in a loss of auditory nerve fibers, cochlear synaptopathy (Garrett and Verhulst, 2019; Makary et al., 2011; Schmiedt et al., 1996; Sergeyenko et al., 2013; Viana et al., 2015), and changes in neural coding in the auditory brainstem (Brecht et al., 2017; Parthasarathy et al., 2014; Schatteman et al., 2008; Williamson et al., 2015). Because ABR peak latencies are delayed and amplitudes are reduced by neuronal loss or by changes in synchronous firing patterns, the ABR may provide a sensitive measure of temporal processing in older individuals.

Large cross-sectional studies have shown that ABR latencies increase with age. In a study of 98 normal-hearing listeners ranging in age from 25 to 55 years, the click-evoked Wave V latency increased ~ 0.2 ms and amplitude decreased ~ 0.05 μV (Jerger and Hall, 1980). Similarly, a study with 586 normal-hearing subjects ranging in age from infancy to older adulthood revealed that click-evoked Wave V latencies begin to change at the age of ~ 30 years, increasing from ~ 5.7 to 6.0 ms at > 60 years (Skoe et al., 2015). A similar increase in Wave V latencies was also found in recordings to a 40-ms [da] syllable in the Skoe et al. study.

Aging effects on ABR latency and amplitude may differ depending on stimulus parameters and protocols. Using recordings to a high-level click stimulus of 115 dB peSPL and a tympanic-membrane electrode to maximize detection of Wave I in 11 ONH and YNH listeners, Burkard and Sims (2001) found that ONH listeners had slightly longer Wave I latencies than YNH listeners across stimulation rates that varied from 11 to 75 Hz (obtained using conventional ABR) and from 100 to 500 Hz (obtained using a maximum length sequence procedure). Progressively longer latencies were noted with increasing stimulation rates, with equivalent effects between age groups, suggesting that increasing stimulation rate does not tax the auditory system to a greater degree in ONH than in YNH listeners, at least at this high intensity presentation level. The authors reported smaller Wave I amplitudes and

somewhat smaller Wave V amplitudes in ONH compared to YNH listeners. The authors did not provide standard deviations for the amplitude means, however, and ABR amplitudes are highly variable because of differences in baseline noise levels in awake listeners. Therefore, the amplitude effects do not appear to be as robust as the effect for Wave I latency.

In a follow-up study using the same participants, Burkard and Sims (2002) recorded ABRs to the same click stimulus presented at a constant rate of 25 Hz and compared effects of broad-band noise between YNH and ONH listeners. They found similar age-related increases in Wave I latency as in their 2001 rate study, and they also found that noise had equivalent effects on ABRs in older and younger listeners. Assuming that noise and/or rate desynchronize neural firing to stimulus onsets, as suggested by Don et al. (1977), the results of the Burkard studies suggest that ONH listeners are not affected by neural desynchronization to a greater extent than YNH listeners. Note that the sample sizes in both Burkard studies (2001, 2002) were relatively small (n=11, each group); therefore, the studies may have been underpowered for revealing amplitude differences that tend to be highly variable.

Konrad-Martin et al. (2012) found similar results in a study that recorded ABRs to a 100-μsec broadband click presented at 110 dB peSPL at three stimulation rates (11, 51, and 71 Hz) from a sample of 131 predominantly male veterans (ages 26 to 71). Using regression modeling, they demonstrated that aging decreased amplitudes of all ABR wave peaks (I, III, and V) and increased latencies for peaks I and III. They also found that aging did not exacerbate the effects of rate; in contrast, the effect of increased rate on amplitude was somewhat attenuated in the older participants due to their smaller baseline amplitudes at lower rates.

Despite the presence of audiometrically normal thresholds in the older listeners in these studies, their thresholds, particularly in the high frequencies, were elevated compared to those of the younger listeners. The click-ABR is dominated by the high-frequency components of the stimulus. This high-frequency emphasis arises due to temporal dispersion of the low frequency components on the basilar membrane and due to responses of low-frequency tails of higher frequency nerve fibers (Dau, 2003). Therefore, even small elevations in high-frequency hearing thresholds may lead to delayed latencies (Bauch and Olsen, 1986), although recruitment may minimize latency delays at high intensity levels (Kavanaugh and Beardsley, 1979). An example of the effects of slight high-frequency hearing loss is displayed in Figure 1. In the ONH listener with "normal hearing" (thresholds of 25 and 20 dB HL at 6000 Hz and 8000 Hz, respectively), Wave V latency is delayed compared to the YNH listener and is earlier compared to the OHI listener.

The Burkard and Konrad-Martin studies mentioned above controlled for the effects of hearing through recruitment of individuals who had normal hearing thresholds and through further subdividing the older adults into a group with "better hearing" (Burkard & Sims, 2001, 2002: all thresholds 20 dB; Konrad-Martin et al. (2012): pure-tone average of 2000, 3000, and 4000 Hz < 17.5 dB HL). Although these studies controlled for the effects of hearing threshold, threshold elevations above 4000 Hz exist even for the better-hearing older participants compared to the YNH participants. For example, there is a mean difference of

9.83 dB HL between the YNH participants and the better hearing older participants in Burkard and Sims (2001; Table 1, p.2 and Table 2, p. 3). Furthermore, given that the majority of participants in the Konrad-Martin study were men, sex may have played a role in evaluation of age vs. hearing loss effects. Jerger and Johnson (1988) evaluated interactions of age, sex, and hearing level on the ABR latencies and found that hearing loss effects were more pronounced in their female participants. Therefore, effects of elevated thresholds cannot be ruled out in these human studies.

Hearing loss—The effects of sensorineural hearing loss on the ABR are well documented, including delayed latencies and decreased amplitudes with increases in hearing thresholds; however, Wave V latencies may be normal at high presentation levels due to recruitment in cases of mild-to-moderate hearing loss (Stapells et al., 1985). Slope metrics, calculated from latency or amplitude changes with intensity, may have potential for differentiating between outer hair cell loss and cochlear synaptopathy or auditory nerve degradation (Vasilkov and Verhulst, 2019; Verhulst et al., 2016). However, these metrics are highly variable in individuals with hearing loss, and more work needs to be done before it is possible to tease apart effects that might be related to aging vs. outer hair cell loss (Vasilkov and Verhulst, 2019).

Animal studies—Because environmental causes of hearing loss can be controlled, lab-based animal studies may reduce the confounds of hearing loss in aging studies. Many studies have compared auditory function in the "normal-hearing" CBA/CaJ mouse strain to the C57 strain which exhibits significant progressive age-related sensorineural hearing loss. However, even the CBA mice gradually lose hearing at a rate similar to humans but within a compressed time frame (Williamson et al., 2015). ABR recordings in CBA/CaJ mice show no changes with aging; however, hearing loss in the C57 mice results in prolonged ABR latencies and reduced amplitudes, particularly in the early waves (Hunter and Willott, 1987). Studies in other species have shown that peripheral function contributes to ABR abnormalities. Older rhesus monkeys with reduced outer hair cell function (lower distortion-product otoacoustic emissions) have delayed ABR latencies (Torre and Fowler, 2000) and older rats with a greater loss of cochlear ribbon synapses have lower amplitudes, especially for Wave I (Cai et al., 2018). In contrast to these studies showing only peripheral effects on the ABR, a study comparing ABRs in younger and older Mongolian gerbils found an age-related decrease in ABR amplitudes, even when limiting the comparison to older gerbils who had lower ABR thresholds (Boettcher et al., 1993). In addition, the slopes of amplitude-intensity functions were shallower in the older compared to the younger gerbils and there was no relationship between slopes and hearing thresholds. The results should be viewed with some caution, however, as the thresholds of the better hearing older gerbils were slightly elevated compared to the young gerbils (Boettcher et al., 1993; Fig. 2).

Summary—Across studies, the "aging" effects of decreased amplitude and delayed latency appear to be confounded to some extent by hearing loss, especially at frequencies above 4000 Hz. The normal-hearing requirement in aging studies usually includes frequencies up to 4000 Hz, and almost all older individuals have some degree of threshold elevation in the extended high frequency range. Animal studies have shown that these group differences are

most pronounced for the earlier ABR components which are more likely to be affected by peripheral dysfunction. At this time, it is not possible to disentangle aging effects from hearing loss, but perhaps new metrics can be developed that can differentiate these effects.

Envelope-following response (EFR) and auditory steady-state response (ASSR)

To obtain a better understanding of aging effects on auditory processing, studies have employed transient and amplitude-modulated stimuli, as aging may selectively target neurons that respond to amplitude-modulated stimuli rather than neurons that encode onsets (Palombi and Caspary, 1996; Rabang et al., 2012; Walton et al., 2002). In an aging rat model, Parthasarathy et al. (2014) compared aging effects for ABRs and EFRs and found that the relationships between these measures changed with age, such that correlations that were found between ABR and EFR amplitudes in the younger rodents were not found in the older rodents. These findings suggest that the ABR and EFR provide complimentary information regarding neurophysiological changes with aging, and the authors suggest including both assessments when evaluating age-related changes in auditory function.

EFR/FFR/ASSR studies have employed different approaches to investigate temporal processing deficits in older adults. One approach is to challenge the auditory system by varying stimulus parameters, such as modulation depth, stimulation rates, and stimulus trajectory, or by degrading the signal. Another approach is to use complex stimuli that allow examination of neural representation to specific regions of the stimulus (e.g., consonant transition vs. sustained vowel). These approaches may help to clarify the specific nature of temporal processing deficits and may lead to management strategies that target these specific deficits.

Aging—By varying stimulus parameters, an aging effect may be revealed that is limited to a specific stimulus presentation rate or modulation depth. Caution should be employed with this approach, however, to ensure that these parametric studies are guided by prior hypotheses and are corrected for multiple comparisons.

Presentation rate can be manipulated to better understand the locus of the deficit (Gaskins et al., 2019; Herdman et al., 2002) or to approximate the modulations that occur in natural speech (Goossens et al., 2016; Schoof and Rosen, 2016). For example, Leigh-Paffenroth and Fowler (2006) recorded ASSRs to AM tones with carrier frequencies of 500 and 2000 Hz and modulation rates of 20, 40, and 90 Hz and measured numbers of phase-locked responses in YNH and ONH listeners. They found a significant age × rate interaction, such that the ONH listeners had smaller numbers of phase-locked responses than the YNH listeners to the 90-Hz rate but not to the 20- or 40-Hz rates. Given that responses to the 20-Hz and 40-Hz rates are dominated by cortical generators and responses to the 90-Hz rate by brainstem generators (Herdman et al., 2002), these results suggest that brainstem, but not cortical phase locking, is affected by aging. Because there were hearing threshold differences between the groups, it is possible that there were peripheral hearing loss effects on the brainstem responses that were then compensated in cortex.

Grose et al. (2009) investigated aging effects on ASSR amplitudes at low and high modulation frequencies (32 and 128 Hz, respectively) and at low and high carrier

frequencies (500 and 2000 Hz, respectively). These frequencies were chosen to resolve discrepancies in the literature regarding aging effects on temporal processing that showed no aging effects using relatively low modulation rates of 40 Hz (Boettcher et al., 2001) or 30–50 Hz (Purcell et al., 2004), but the existence of aging effects for a relatively higher rate of 90 Hz (Leigh-Paffenroth and Fowler, 2006). Grose et al. (2009) demonstrated an age-related reduction in amplitude for the 128-Hz modulation frequency at both carrier frequencies, but no aging effects were noted for the 32-Hz modulation frequency. They noted that these results were consistent with psychophysical data showing age-related deficits for high but not low modulation rates (He et al., 2008; Purcell et al., 2004).

Based on previous studies, Gaskins et al. (2019) hypothesized that aging effects would be specific to high frequencies (age × rate interaction) in a study that recorded ASSRs to 300-ms bandlimited pulse trains (1000 Hz bandwidth centered around 4000 Hz) presented at five rates (20, 40, 80, 200, and 400 Hz). In this study, the ASSRs showed lower signal-to-noise ratios (SNRs) in the ONH than in the YNH listeners, but only for the highest rate of 400 Hz (after correcting for multiple comparisons). In addition, ASSR energy decreased with increasing rate, but only in the ONH listeners (Figure 2). Given previous results from the Leigh-Paffenroth and Fowler (2006) and Grose et al. (2009) studies, aging effects might have been expected for the 80-Hz and 200-Hz rates as well. The Gaskins study tested 15 participants in each age group, whereas the Grose et al study tested 10 participants in each group and the Leigh-Paffenroth and Fowler tested 11 participants in each group. Therefore, it is possible that true age differences exist only for the highest rates (> 200 Hz), and it would be beneficial to perform a follow-up study with a larger number of participants to obtain a more accurate assessment of aging effects for high-rate modulation stimuli.

Studies that have examined effects of modulation depth have not found aging differences. McClaskey et al. (2019) examined the effects of aging and modulation depth on ASSRs to AM tones with a carrier frequency of 3000 Hz, a modulation frequency of 80 Hz, and modulation depths of 0, −4, and −8 dB (full, moderate, and shallow modulation, respectively). Based on the results of previous studies (Boettcher et al., 2001; Purcell et al., 2004), they did not expect to find aging differences for the relatively low modulation rate of 80 Hz, but instead they focused on effects of AM depth. Although both younger (n=22) and older (n=35) groups were defined as having normal hearing, there were significant group differences in pure tone thresholds, and the authors controlled for thresholds (average of 2000, 3000, and 4000 Hz) in their statistical analysis. They found that shallower AM depths reduced phase locking and amplitude in both listener groups, and there were no age group differences or interactions between age group and AM depth. The authors suggested that the use of transposed tones rather than sinusoidal AM tones may have elicited higher phase locking and reduced differences between the younger and older listeners. They also suggested that deficits at more peripheral levels may be partially compensated at midbrain and cortical levels, thus reducing or eliminating age differences.

Hearing loss—Effects of hearing loss have also been demonstrated with EFR recordings that vary in rate and/or modulation depth. In recordings of EFRs to white noise modulated at 25% depth and at rates that swept from 20 to 600 Hz, older listeners (n=13) with mild ARHL demonstrated detectable EFRs at a lower maximum frequency (mean = 294 Hz)

compared to the maximum frequency (mean = 494 Hz) in YNH listeners (n=25) (Purcell et al., 2004). Dimitrijevic et al. (2016) recorded EFRs in YNH listeners and two groups of middle-aged and older listeners with hearing loss (O1, 41–62 yrs, pure-tone average (PTA) = 30 dB HL; O2, 67–82 yrs, PTA = 49 dB HL) in two conditions, with 12 listeners in each group. In the first condition, EFRs were recorded to a wideband noise carrier modulated at a rate of 41 Hz. The AM depth was continuously varied from 2% to 100% at a rate of 5%/sec. In the second condition, ASSRs (41 Hz) were recorded to fixed AM depths (100, 75, 50, and 25%). As expected, given the relatively low modulation rate of 41 Hz, the overall response amplitude was equivalent between groups. However, the YNH and O1 listeners exhibited a relatively linear increase in amplitude with greater AM depth, whereas the O2 listeners exhibited a narrower dynamic range with saturation at higher AM depths. In addition, EFR phase was relatively constant in the YNH listeners but decreased as a function of AM depth in the older listeners. The authors performed linear regression modeling to partial out effects of age and hearing loss and determined that the 4000-Hz threshold was the significant predictor of response amplitude. It should be noted that both older groups of participants had some degree of hearing loss, so it is difficult to disentangle the effects of aging from hearing loss in the results.

The best approach to differentiating effects of aging and hearing loss would be to match participants on these factors; however, the etiologies of hearing loss differ between younger and older individuals, and these etiologies may contribute to observed group differences. Nevertheless, when feasible, it is informative to compare temporal processing in groups that have been matched on both age and hearing loss, as was done in Goossens et al. (2019). In this study, the authors recruited normal hearing (NH) and hearing impaired (HI) young (20–30 yrs; 20 NH, 10 HI), middle-aged (50–60 yrs; 20 NH, 14 HI), and older (70–80 yrs; 14 NH, 13 HI) individuals. They recorded ASSRs to octave bands of white noise that were modulated at rates of 4, 20, 40, and 80 Hz to represent cortical, thalamus, thalamus/upper brainstem, and brainstem, respectively (Herdman et al., 2002). Based on previous animal models, they hypothesized that hearing loss would be associated with envelope enhancement, especially when stimuli have been adjusted for audibility, and that this association may differ between age groups. They presented the stimuli to both NH and HI groups at 70 dB SPL, rated as comfortably loud by the NH listeners, and they also presented the stimuli at levels that were individually rated as comfortably loud by the HI listeners. Overall, the young and middle-aged HI listeners' responses showed enhanced encoding of envelope modulations in both subcortical and cortical regions compared to their normal-hearing counterparts, whereas the OHI listeners' responses did not show this enhancement. Further, the enhancement to the cortical rates (4 to 40 Hz) disappeared when stimuli were presented at 70 dB SPL to the HI and the NH listeners, but the enhancement remained for the 80-Hz (brainstem) rate. Overall, it appears that hearing loss leads to exaggerated responses to the envelope, but this effect may be reduced with advanced age (over 70).

The Goossens et al. (2019) study included age as a factor in the analysis, but they did explicitly report the effects of age, and it would have been helpful to compare effects of age vs. effects of hearing loss. As noted, the sample sizes of each group were relatively small, due to the difficulty in recruiting and matching NH and HI subjects by age. There were also a large number of conditions, and therefore, the positive findings of the study may have

resulted from random statistical variation. The authors have nevertheless demonstrated that a comparison of age vs. hearing loss effects is feasible, and it would be important to determine if these results are replicable in another study.

Animal studies—Various animal models have been used to clarify the nature of age-related degradation in AM encoding. Homeostatic changes that occur secondary to a loss of afferent input lead to decreased levels of inhibitory neurotransmission, including lower glycine levels and altered glycine receptors in the cochlear nucleus (Banay-Schwartz et al., 1989; Milbrandt and Caspary, 1995; Willott et al., 1997) and lower Gamma aminobutyric acid (GABA) levels and decreased numbers of GABA receptors in the IC and cortex (Caspary et al., 2013; Gutiérrez et al., 1994; Ling et al., 2005; Milbrandt et al., 1996). Given the role of GABA in shaping IC responses to AM stimuli (Caspary et al., 2002), these changes may underlie age-related deficits in AM encoding.

As noted above, these changes are presumed to result from a loss of input from the periphery, and may therefore reflect a central consequence from hearing loss rather than from aging, per se. To separate the effects of aging and hearing loss, Lai et al. (2017) compared EFRs in younger and older Fischer-344 rats using stimulus levels that were equalized for peripheral and central activation between the two groups. Using this approach, the authors aimed to account for potential synaptopathy and threshold elevations that are expected to occur with aging. To accomplish peripheral equalization, the mean Wave I amplitude obtained in the older animals with a stimulus intensity of 85 dB SPL served as a reference, and the stimulus level needed to obtain an equivalent Wave I amplitude was used in ABR and EFR recordings in the younger rats. To accomplish central equalization, the mean EFR amplitude (at 45-, 128-, and 256-Hz modulation rates) obtained in the older animals with a stimulus intensity of 85 dB SPL served as a reference, and the stimulus level needed to obtain an equivalent EFR amplitude was used in EFR recordings to the same modulation rates at several modulation depths in the younger animals. Using matched ABR I amplitudes, the authors found no age differences for Wave V amplitude; however, EFRs were enhanced at 100% but not at 25% modulation depths. Using matched EFR amplitudes, they found no age differences for either AM depth or AM frequency. Overall, these results demonstrate that apparent age-related differences in ABR amplitude are likely due to peripheral degradation, either in the outer hair cells, cochlear synapses, or auditory nerve fibers. However, the EFR results show that aging may affect enhancement of sustained modulations (EFR) to a greater extent than the effects on onset responses (ABR).

The nature of the peripheral deficit in temporal processing was further explored in a gerbil model (Heeringa et al., 2020). The authors found that coding of the temporal envelope and fine structure in the auditory nerve was equivalent between younger and older gerbils when measured at comparable sensation levels. Auditory nerve spontaneous firing rate, however, was decreased in older vs. younger gerbils, and this decrease was related to the extent of hearing threshold elevation and the decrease in auditory nerve fibers. Based on these findings, the authors concluded that age-related temporal coding deficits arise from deficits central to the auditory nerve associated with age-related decreases in inhibition (Canlon et al., 2010; Caspary et al., 1995), but that these deficits may be a consequence of decreased numbers of auditory nerve fibers.

Summary—Overall, these studies appear to show that 1) aging affects temporal processing of higher-rate modulation frequencies, and 2) aging does not affect processing of AM depth at relatively lower modulation frequencies. Aging effects have also been observed in studies that used very low-rate stimuli (e.g., < 10 Hz) (Decruy et al., 2019; Goossens et al., 2016; Henry et al., 2017; Tlumak et al., 2015), suggesting a possible cortical-cognitive role in aging deficits. For example, Henry et al. (2017) found a greater attention-related enhancement to a 2.8-Hz modulation rate (approximating speech rhythms) in YNH than in ONH listeners. The enhanced neural synchronization in young and middle-aged HI listeners noted in Goossens et al. (2019) is consistent with the results of animal studies (Henry et al., 2014; Parthasarathy et al., 2018; Qiu et al., 2000; Zhong et al., 2014). Enhanced synchronization likely reflects homeostatic mechanisms that compensate for reduced afferent input.

Conclusions regarding these studies are not definitive, however, due to the many stimulus conditions and small numbers of subjects that increase the possibility of a Type I error. Confidence in these results would be increased by studies that replicate these findings. It should also be noted that all ONH listeners across the studies were affected by age-related hearing loss (ARHL) to some degree, at least in the higher frequencies and therefore peripheral hearing loss may be a factor in any potential age-related differences.

Frequency-following response (FFR)

Aging—Older adults' poorer speech perception may reflect temporal jitter or neural noise in the auditory system (Pichora-Fuller et al., 2007; Wang et al., 2011). Temporal jitter may result from loss of neural synchrony (Agmon, 2012; Luo et al., 2018) that would interfere with the auditory system's ability to generate the synchronous firing necessary to produce a precise representation of the auditory stimulus (Plack et al., 2014). A study that evaluated effects of temporal jitter on word-in-noise identification in YNH listeners found that jitter reduced their performance to levels previously found in ONH listeners for the same task using non-jittered words (Pichora-Fuller et al., 2007). Mamo et al. (2016) tested this same idea by recording the FFR to a synthesized speech syllable [da] presented in clean and jittered conditions to 22 YNH and 22 ONH listeners. They found a main effect of age, in that ONH listeners had significantly reduced magnitudes of the fundamental frequency (F_0) and its principle harmonics compared to YNH listeners in response to the clean syllable. However, jitter decreased spectral magnitudes only in the YNH listeners, not in the ONH listeners. They concluded that reduced synchrony in the ONH listeners mimicked neural jitter and therefore the introduction of stimulus jitter did not further decrease response magnitude.

Another approach to evaluating aging effects on temporal processing is to determine the specific components of the speech syllable or word that are affected by aging. Anderson et al. (2012) recorded FFRs in 17 YNH and 17 ONH listeners to a 170-ms synthesized [da] syllable presented in quiet and performed several analyses on the consonant-transition (20–60 ms) and steady-state vowel (60–170 ms) regions. They found that the ONH listeners had prolonged latencies compared to YNH listeners, but only for the onset and transition peaks, and not for the steady-state peaks. The other measures, phase locking, response consistency,

and response amplitudes, showed aging deficits for both the transition and steady-state regions. The authors suggested that the reduced inhibitory neurotransmission may interfere with the ONH listeners' ability to phase lock to the rapidly changing formants in the consonant transition.

Two alternate hypotheses for the Anderson et al. (2012) findings would be that ONH listeners experience a general delay in consistent phase locking that is not specific to changing formants or that high-frequency threshold differences between the two groups impact latencies for the high-frequency components of the stop consonant. Presacco et al. (2015) performed a follow-up study to test these hypotheses and contrasted aging effects on responses to [da] and [a]. Although they found age-related latency delays for [da] and not for [a], they noted that within-group differences between the stimuli were only found in the 15 YNH and not in the 15 ONH listeners. In the YNH listeners, the latencies to [da] were earlier than to [a], as would be expected given the higher-frequency energy content of the [da] that would be encoded earlier on the basilar membrane. These same latency differences between the [da] and [a] were not found in the ONH listeners, presumably because a slight loss of audibility impaired encoding of the high-frequency transients, even though their hearing thresholds were within the normal clinical range. A better approach in the future would be to record responses to syllables that begin with the same formants but vary in the length of the transition prior to the vowel, such as the syllables [ba] and [wa].

Clinard and Cotter (2015) adopted a different approach to determine if reduced inhibitory neurotransmission affects encoding of rapidly changing formants. Instead of presenting speech syllables, they recorded FFRs to tones that linearly increased or decreased in frequencies that mimicked formant changes in speech. The starting or ending frequency was 400 Hz for rising or falling tones, respectively, and the frequency changed at rates of 1333, 3999, and 6667 Hz/sec. They used stimulus-to-response (STR) correlations to assess the fidelity of frequency representation and found lower correlations and spectral energy in 9 ONH compared to 10 YNH listeners (Fig. 3). Of particular significance is the fact that they found aging effects even in the lower frequencies (~400 Hz), in contrast to a previous study that showed aging effects only for the relatively higher frequencies (~1000 Hz) and not for the lower frequencies (~500 Hz) when using static stimuli (Clinard et al., 2010). At this low frequency, it is less likely that peripheral hearing loss plays a role in the age effects. The authors suggested that an age-related reduction in inhibitory neurotransmission may lead to a reduction in phase locking to dynamic frequency sweeps, given the apparent role of GABA and glycinergic inhibition in neural processing of frequency modulations (Covey and Casseday, 1999).

Although the Clinard et al. (2010) study did not find aging effects in FFRs to low-frequency 500-Hz static tones, later studies found reduced phase locking/neural synchrony when using stimuli that contain sustained vowel regions (Anderson et al., 2012; Bidelman et al., 2014; Presacco et al., 2016b; Presacco et al., 2015). In particular, one finding that could not be easily explained by peripheral hearing loss was the abrupt drop in synchrony at ~110 ms in ONH listeners' responses to a sustained 170-ms [a] stimulus (Presacco et al., 2015) (Fig. 4). The authors suggested that an age-related reduction in auditory nerve fibers may limit the auditory system's ability to sustain neural firing, similar to the phenomenon of excessive

stapedial reflex decay that is associated with the presence of vestibular schwannomas on the VIII nerve (Olsen et al., 1975). An alternate explanation for these results would be prolonged neural recovery and fatigue in the older listeners, as suggested by the findings of Walton et al. (1998) who observed that neurons in the IC had delayed recovery times in older compared to younger mice.

Previous behavioral studies have demonstrated an age-related perceptual deficit in the ability to use duration cues, such as vowel duration to cue final voicing or silence duration to cue an affricate vs. fricative (Gordon-Salant et al., 2008; Gordon-Salant et al., 2006). Roque et al. (2019 a,b) investigated the potential neural basis of these deficits by recording FFRs to word contrasts that were identical except for a silence duration cue (DISH-DITCH; 2019b) in 15 YNH, 15 ONH, and 15 OHI listeners and a vowel duration cue (WHEAT-WEED; 2019a) in 30 YNH and 30 ONH listeners. They found that STR correlations were lower in the ONH and OHI listeners for the words with longer duration cues, DITCH and WEED, compared to the YNH listeners. These same group differences were not noted for DISH or WHEAT and they surmised that the older listeners had degraded neural representation of the longer duration cues in DITCH and WEED. The lower STR correlation in DITCH but not DISH may arise from a reduction in the aging auditory system's ability to detect short gaps or silent durations that naturally occur in speech (Walton et al., 1998). The lower STR correlation in WEED but not WHEAT (Fig. 5) may arise from an age-related reduction in the ability to sustain neural firing to the longer vowel, associated with a loss of auditory nerve fibers (Schmiedt et al., 1996) or delayed neural recovery (Walton et al., 1998). Upon close examination of the waveforms, one can see a breakdown in neural synchrony in the latter part of the vowel region in the ONH listeners (Fig. 5). The drop in synchrony was not as abrupt as it was in the Presacco et al. (2015) study, perhaps because the F_0 and formants were not as "steady" for the naturally-produced WEED as they were for the synthesized vowel [a]. We note that few studies have investigated aging deficits in the subcortical representation of naturally-produced words, and it would be important to determine if these results can be replicated in another study.

Hearing loss—While the above-mentioned studies recruited listeners with clinically-normal hearing thresholds, the role of hearing loss cannot be ruled out given the inevitable elevation of high-frequency thresholds in the older listeners. For example, Vander Werff and Burns (2011) found that 18 ONH listeners had reduced amplitudes and delayed latencies to a 40-ms [da] syllable compared to 19 YNH listeners, but after covarying for high-frequency hearing thresholds, aging effects were limited to the onset amplitude and offset latency. Similar combined contributions of age and hearing were found in a study that recorded responses to a 40-ms [da] syllable in NH participants in whom age was a continuous variable (22–77) (Clinard and Tremblay, 2013). Therefore, it is important to consider hearing loss effects on the FFR when evaluating aging effects in participants with audiometrically-normal hearing thresholds.

Based on animal models demonstrating that hearing loss results in enhanced envelope encoding in the auditory nerve/midbrain (Dong et al., 2010; Kale and Heinz, 2010; Zhong et al., 2014), it might be expected that the FFR would also be enhanced with age-related hearing loss. To our knowledge, however, only one study showed larger F_0 magnitudes in 15

OHI listeners compared to 15 age-matched ONH listeners in recordings of responses to a 40-ms [da] syllable (Anderson et al., 2013c). This finding has not been replicated in any subsequent studies. Most studies that have compared FFRs in ONH, OHI, and YNH listeners have either found no differences in response magnitude/phase locking between ONH and OHI listeners (Presacco et al., 2019; Roque et al., 2019b) or reduced magnitudes in OHI listeners (Ananthakrishnan et al., 2016; Hao et al., 2018). The Anderson et al. findings may have been the result of false discovery, but it is also possible that the subsequent studies did not replicate the findings because the ages of the groups were not matched, and it is expected that age would reduce amplitudes. Another explanation for differing findings is that different etiologies of sensorineural hearing loss may produce different responses at suprathreshold levels. For example, an individual's hearing loss may be dominated by cochlear hair cell loss and cochlear synaptopathy to different degrees (Verhulst et al., 2018; Verhulst et al., 2016). Cochlear hair cell loss is associated with broader tuning curve and steeper rate-level functions, two factors that contributed to enhanced envelope coding in chinchillas with moderate-to-severe noise-induced hearing loss (Kale and Heinz, 2010). In contrast, age-related cochlear synapatopathy in CBA mice, resulting in a progressive loss of synapses with intact outer hair cells, is associated with reduced EFR amplitudes (Parthasarathy and Kujawa, 2018). We speculate that a measure that could differentiate between these sources of hearing loss may further the understanding of the effects of hearing loss on temporal processing.

Animal studies—As reviewed above, most animal studies have recorded EFRs to AM tones to evaluate aging effects. Recently, however, Parthasarathy et al. (2019) recorded single-unit and multi-unit responses to a 260-ms speech syllable [ba] in younger and older Fischer-344 rats to better understand the mechanisms contributing to age-related decreases in speech processing in humans. Although ABR Wave I amplitude was reduced for the older vs. younger rats, there was an age-related increase in synchronization to the temporal envelope measured in multi-unit neural activity from the IC. Furthermore, despite reduced synaptic input to IC neurons in local field potential recordings, the population response of the IC showed over-representation of the envelope in older vs. younger animals, suggesting a compensatory central gain mechanism along the auditory pathway. This enhancement of the envelope may be associated with changes in the balance of neural representation of the envelope vs. the temporal fine structure and subsequent reduction in decreased speech discrimination (Guo et al., 2017).

Summary—Aging may reduce synchronization to sustained stimulus components and may degrade processing of duration components of stimuli. Latency delays to the onset and transition regions of the speech stimulus appear to be most affected by subtle degrees of hearing loss. Synchronization can be enhanced, however, given a sufficient degree of cochlear hearing loss, consistent with a compensatory gain mechanism suggested by Parthasarathy et al. (2019).

Cortical auditory-evoked potentials (CAEP)

Aging—The EFR, FFR and ASSR studies reveal evidence of substantial degradation of the neural signal in brainstem and midbrain in individuals with ARHL. Yet, ONH listeners

generally understand speech well in quiet or easy listening situations. The cortex has a remarkable ability to at least partially compensate for a degraded signal, as demonstrated by a gradual restoration of tone-evoked neural firing rates in IC and cortex and near-normal tone detection in mice after ouabain-induced cochlear denervation (Chambers et al., 2016). In this study, however, the compensatory plasticity necessary to generate these responses was not sufficient to restore the timing precision required for encoding speech stimuli. An examination of cortical responses may therefore lead to a better understanding of the limits of compensatory plasticity for understanding speech.

As mentioned previously, the ability to detect short gaps appears to be important for speech understanding, especially in noise. Furthermore, lesion studies in rats have demonstrated that the auditory cortex is necessary for perceptual identification of gaps (Ison et al., 1991; Syka et al., 2002). The gap termination response, a burst of neural firing in auditory cortex at the end of a gap, enables the listener to perceive the gap (Weible et al., 2014). Therefore, cortical responses to gaps in noise may reveal neural mechanisms underlying poor gap detection ability in older listeners. Cortical recordings to gaps embedded in noise reveal the traditional onset response (P1-N1-P2 peaks) to the onset of the noise stimulus and a second smaller onset response to onset of the gap. Lister et al. (2011) recorded CAEPs from 24 ONH listeners to silent gaps embedded between two segments of narrow-band noise. They compared these results to previous data collected in 12 YNH listeners (Lister et al., 2007) and found that the ONH listeners had delayed P2 latencies compared to YNH listeners (significant after correcting for multiple comparisons). They suggested that altered auditory inhibition and slower neural conduction times accounted for the aging differences.

Age-related decreases in processing of gaps in noise may extend to speech stimuli. Tremblay et al. (2003) investigated perceptual and cortical representation of voice-onset-time (VOT), a temporal contrast that enables one to distinguish between voiced and unvoiced consonants in YNH, ONH, and OHI listeners (n=10 per group). They used a synthesized [pa] to [ba] continuum that gradually decreased the VOT in 10-ms steps. They found that the ONH and OHI listeners had more difficulty discriminating the 10-ms VOT contrasts than the YNH listeners. They also recorded CAEPs to the 7-step VOT continuum and found that 1) latency of the N1 and P2 peaks increased with increasing VOT, 2) the effect of VOT on latency was greater in the ONH and OHI listeners than in the YNH listeners, and 3) the latencies were equivalent between the ONH and OHI listeners (Fig. 6). The authors suggested a number of possible neural mechanisms for the peak delays observed in the older listeners, including delayed neural refraction, decreased neural synchrony, and a broader distribution of individual neural firing to the onset of the syllable. They also observed that restoration of audibility through the use of hearing aids may not entirely ameliorate central processing deficits that contribute to decreased perception in older listeners. However, extended use of amplification may improve some aspects of impaired temporal processing that have resulted from a lack of afferent input (Karawani et al., 2018a; Karawani et al., 2018b; Rao et al., 2017).

The Roque et al. (2019a,b) studies cited in the FFR section also recorded CAEPs to words that differed in silent duration (DISH vs. DITCH) and vowel duration (WHEAT vs. WEED). The effects on cortical peaks differed between the studies. In the vowel duration study

(Roque et al., 2019a), which included YNH and ONH listeners, the aging effects were limited to the P1 peak-decreased latency and increased amplitude for WEED and decreased latency for WHEAT in ONH vs YNH listeners. In the silence duration study (Roque et al., 2019b), which included YNH, ONH, and OHI listeners, the only group effect on amplitude was a large N1 amplitude in the OHI listeners compared to either the ONH or YNH listeners. In addition, P2 latency was delayed in the ONH and OHI listeners relative to the YNH listeners. These differing aging effects between the studies may be explained by stimulus and subject differences. The WHEAT/WEED stimuli begin with a glide and the DISH/DITCH stimuli begin with stop consonant. The transient in the stop consonant may generate more synchronous firing than the glide consonant and may be less sensitive to age differences that would be present for the P1 component. Furthermore, the hearing levels and ages were higher in the DISH/DITCH study than in the WHEAT/WEED study. The silence duration study recruited older subjects ≥ 60 yrs of age with hearing levels ≤ 25 dB HL from 125–4000 Hz and no hearing limit at 6000 and 8000 Hz. In an effort to reduce the effects of aging, the subsequent vowel duration study recruited older subjects who were younger than in the previous study (≤ 55 yrs of age) and had better hearing thresholds (≤ 20 dB HL from 125–4000 Hz and ≤ 30 dB HL at 6000 and 8000 Hz). Therefore, the P2 latency age effects in the silence duration study may be due to either age and/or hearing loss effects. The P2 peak is a putative marker of object identification (Näätänen and Winkler, 1999; Ross et al., 2013), and an imprecise stimulus representation associated with age or hearing loss may lead to delays in processing this component. The N1 amplitude effect in the silence duration study was only seen in the OHI listeners, and this group was not recruited in the vowel duration study. Another possible reason for the discrepancies between the two studies is a random statistical variation rather than a true aging difference; this possibility is especially likely given the large range of normal values for cortical peak latencies and amplitudes (Zhang et al., 2009).

Hearing loss—Effects of hearing loss are best evaluated in age-matched groups, such as in the study conducted by Campbell and Sharma (2013). They recorded CAEPs to nonsense syllables in middle-aged listeners (37–68 yrs) using a 128-channel electrode cap. They found that P2 latency was smaller in amplitude and delayed in latency in nine HI listeners compared to eight age-matched NH listeners. The levels of hearing loss in this study were relatively mild; therefore, aging effects on the P2 effect in previous studies (Lister et al., 2011; Roque et al., 2019b) may reflect small but significant differences in high-frequency thresholds. In another study that employed linear mixed-effects modeling, no hearing loss effects were noted for N1 and P2 (Koerner and Zhang, 2018) in 18 listeners that varied in age from 40–71 and had hearing levels from normal to moderate; rather aging effects were noted for N1 and P2. Small sample sizes in these and other studies may limit interpretation and replicability of findings (Billings and Madsen, 2018). Another important factor that may limit reproducibility between studies is the stimulus presentation rate; age-related latency changes in amplitude and latency for CAEP peaks may be present for faster but not for slower rates (Tremblay et al., 2004).

Although the above-mentioned studies found age-related decreases in the representation of the temporal components in speech, even when presented in quiet, the most frequent

complaint voiced by most older adults is difficulty understanding speech in background noise. To address this complaint, studies have investigated aging effects on neural processing of speech in noise. For example, Billings et al. (2015) investigated the effects of SNR and level and compared CAEPs to the syllable [ba] presented at 50, 60, 70, and 80 dB SPL and SNRs of −5, 15, 25, and 35 dB in 15 ONH and 15 OHI listeners and compared these data to previously published data in 15 YNH listeners (Billings et al., 2013). They found robust effects of SNR on almost all peak latencies and amplitudes, but effects of level were limited and specific to the OHI listeners. Age-related latency delays across SNR conditions were most pronounced for the latency of N1, P2, and N2 peaks. Similar to the Tremblay et al. (2003) study, they did not find latency differences between the ONH and OHI listeners, suggesting that an age-related decrease in neural synchrony may be the dominant factor for P2 latency in this study. The authors' initial hypotheses concerning hearing loss and aging effects were not specific to any particular peak amplitude or latency, and therefore the results were exploratory in nature. Given the relatively small sample sizes, and the large number of statistical comparisons, it would be important to confirm these results in another study.

Animal Studies—As noted above, age-related changes in the balance of neural inhibition and excitation may partially account for a reduction in temporal precision (Caspary et al., 2008). These changes extend from dorsal cochlear nucleus (Caspary et al., 2005; Schatteman et al., 2008) to cortex (Cisneros-Franco et al., 2018; Hughes et al., 2010; Overton and Recanzone, 2016; Turner et al., 2005). Recanzone (2018) reviewed the evidence for three potential types of temporal processing deficits associated with decreased inhibition in cortex: 1) a decrease in the number of neurons responsive to temporally modulated stimuli, 2) a change in the neural code that transmits temporal information, and 3) a decrease in the temporal fidelity of the neural response. In rhesus macaques, the number of neurons tuned to temporally modulated stimuli does not differ between younger and older macaques; however, the coding strategy of the aged neurons shifts such that there is an overall increase in firing rate which reduces the dynamic range necessary for encoding the envelope (Overton and Recanzone, 2016; Yin et al., 2011). These studies also demonstrated an age-related reduction in the vector strength of cortical neurons, resulting in decreased response fidelity.

Summary—Combined results from humans and animals suggest that central compensation, associated with changes in the balance of neural excitation and inhibition, may result in increased neural firing and exaggerated cortical amplitudes, even in listeners with clinically normal audiometric thresholds. However, latency delays and decreased temporal precision reduce the fidelity of signal representation, likely contributing to perceptual deficits. We note inconsistency across studies in the effects of age and hearing loss on cortical peak latencies and amplitudes; given the large normal variability in these peak measures, we suggest that investigators conduct studies with larger numbers of subjects based on power analyses that accurately include numbers of conditions and variables.

Cortical envelope tracking

In recent years, there has been increased interest in the use of continuous speech samples to obtain a more ecologically valid measure of neural speech processing (Decruy et al., 2019;

Ding and Simon, 2012; Etard et al., 2019; O'Sullivan et al., 2015; Presacco et al., 2016a; Presacco et al., 2016b; Presacco et al., 2019; Vanthornhout et al., 2018). These studies typically present selections from recorded audiobooks in quiet or in the presence of background noise, such as a single competing talker. The recorded signals are analyzed by using temporal response functions (TRFs) to facilitate mapping of speech features onto the EEG/MEG signal (Brodbeck et al., 2018; Wong et al., 2018) or by reconstructing the speech envelope from the neural signal (Ding et al., 2014; Presacco et al., 2016b; Vanthornhout et al., 2018). The TRF analysis produces a cortical waveform with peaks that mirror those of the CAEP (O'Sullivan et al., 2015), while reconstruction accuracy is assessed by linearly correlating the cortical response envelope with the speech envelope to obtain an 'r' value. A higher 'r' value would indicate higher reconstruction accuracy.

Aging—Using MEG recordings, Presacco et al. (2016b) discovered that ONH listeners (n=15) had higher reconstruction accuracy 'r' values than YNH listeners (n=17). The participants listened to audiobook recordings and were instructed to attend to the male talker and ignore the female talker. The male speaker was presented at ~70 dB SPL while the female speaker's level was varied to produce SNRs of +3, 0, −3, and −6 dB SNR. An aging effect of higher reconstruction values was found across SNR conditions. The authors had hypothesized that higher fidelity (higher reconstruction accuracy) would relate to better speech-in-noise understanding, and they did not find any relationships between behavioral and neural measures. This over-representation is consistent with previous studies that found higher amplitudes to early components (P1m and N1m) in older vs. younger listeners' MEG responses to rapidly occurring speech sounds (Sörös et al., 2009) and to the EEG equivalents of these responses (P1 and N1) (Roque et al., 2019a; Snyder and Alain, 2005; Tremblay et al., 2003). Presacco et al. (2016b) also found that reconstruction accuracy decreased when the temporal integration window was reduced from 500 to 150 ms in the ONH listeners but not in the YNH listeners, consistent with a temporal processing deficit (Fig. 7). Interestingly, FFRs to the speech syllable [da] in these same subjects showed lower response amplitudes in the ONH than in the YNH listeners. The authors suggested that the exaggerated amplitudes seen in cortex may arise from a similar central compensatory mechanism to that described in Chambers et al. (2016). In the Chambers study, the central compensation was not complete in IC, but was exaggerated in cortex. The FFRs to the [da] may contain cortical contributions but are likely dominated by responses from IC (Bidelman, 2018; Coffey et al., 2016; Smith et al., 1975; Tichko and Skoe, 2017; White-Schwoch et al., 2019). Therefore, the combined responses from FFR and MEG suggest that degradation evident in midbrain is over-compensated in cortex.

Although the Presacco et al. (2016) study had hypothesized that the older listeners' responses would show higher reconstruction accuracy, they were nevertheless surprised regarding the magnitude of the effect which was seen across listening conditions. Therefore, it would be important to replicate this finding in another study. Decruy et al. (2019) also demonstrated this over-compensation by recording EEG to continuous speech stimuli at subject-specific SNRs. They included a middle-aged group of NH listeners so that they had a continuous distribution of age from 17 to 82 years (n=54). They replicated the finding of an increase in envelope tracking (reconstruction accuracy) with aging, and they noted that for

both envelope tracking and speech-in-noise performance there was an accelerated trajectory in the ONH listeners. Three explanations may be posited for over-representation of the envelope in ONH listeners: 1) ONH listeners may rely more on the low modulation frequencies that are important for speech understanding to compensate for perceived speech understanding difficulties, leading to enhanced envelope tracking, 2) Given the negative correlation between cognitive measures of response inhibition and reconstruction accuracy found in both the Presacco et al. (2016b) and Decruy et al. (2019) studies, an inefficient use of cognitive resources may be a factor in over-representation of the speech envelope in older listeners, and 3) Reduced neural inhibition noted in animal models (Cisneros-Franco et al., 2018; de Villers-Sidani et al., 2010; Hughes et al., 2010; Syka, 2002) or redundant local processing and reduced connectivity (Peelle et al., 2010) may lead to exaggerated responses. The field would benefit from future studies that can elucidate potential sources of age-related over-representation, perhaps in cross-species models.

Hearing Loss—To test whether audibility was a key factor in the exaggerated cortical responses, Presacco et al. (2019) repeated the experiment in a group of 14 OHI listeners. They found that the responses in the ONH and OHI listeners did not differ and reasoned that the exaggerated amplitudes resulted mainly from aging rather than hearing loss effects. Nevertheless, it was not possible to clearly distinguish the effects of aging and hearing loss in this study, especially as the OHI listeners were older than the ONH listeners, and the stimulus levels were not equated for audibility between the two groups.

To address audibility concerns, Petersen et al. (2017) fit their older listeners (n=27), whose hearing ability ranged from normal to severe hearing loss, with hearing aids that used quasi-linear amplification to limit distorting effects of compression. Audiobook stories were presented through the hearing aids via direct audio input, and envelope tracking was recorded with a 103-electrode EEG system. They found that the OHI listeners had robust envelope tracking to the attended speech signal, but they also had reduced attentional modulation (higher similarities between the attended and unattended speech signals) and benefitted to a lesser extent from higher SNRs than the ONH listeners. Two limitations in the Peterson et al. study were that there was no matching of age between the ONH and OHI listeners and that the quasi-linear amplification would have distorted the signal to some extent and consequently affecting envelope tracking. Decruy et al. (2020) addressed these issues by matching 14 NH and 14 HI listeners on age (ages ranged from 21–82) and by using a linear amplification algorithm to equate for audibility. Using 64-channel EEG recordings to Matrix sentences and to two- to three-minute recordings of an audiobook, they found enhanced envelope tracking to the target talker in HI compared to NH listeners. They suggested that the results reflect enhanced envelope sensitivity in individuals with hearing loss (Moore et al., 1996) or compensatory effort in separating the target from the background speech signal.

The Decruy et al. (2020) study included a range of ages and had relatively small sample sizes to investigate envelope tracking across many conditions (20 total for the HI listeners). Therefore, even though the participants were matched on age, it would be important to examine these effects in a larger sample to ensure that the findings can be replicated. Fuglsang et al. (2020) investigated envelope tracking to simultaneous, spatially separated

attended and unattended speech streams in 22 NH and 22 HI listeners whose ages ranged from 51 to 76 years. The study's purpose was to evaluate the effects of hearing loss on cortical synchronization to the attended and unattended speech streams, and to determine if hearing loss affected attention decoding accuracy. Similar to Decruy et al. (2019), they found enhanced reconstruction accuracy in the HI compared to NH listeners for the attended talker, but there were no group differences for the unattended talker. They evaluated attention decoding accuracy by comparing reconstruction accuracy for the attended vs. unattended speech stream. They did not find an effect of hearing loss on attention decoding accuracy, and it was above chance level in both groups. These results have implications for the future of "neuro-steered" hearing aids. If the object of the listener's attention can be identified with single-trial EEG decoding, the hearing aid algorithm may then selectively amplify that particular signal.

Animal Studies—To our knowledge, no studies have yet been published that investigated aging effects on cortical envelope tracking to continuous speech samples in an animal model. However, results from a study that investigated age-related changes in cortical processing of rapidly-presented sound sequences in rhesus macaques are consistent with the above-listed findings in older humans (Ng and Recanzone, 2018). In this study, a sparse, selective strategy was observed in the auditory cortex of young monkeys; in contrast, increased neural excitability and reduced temporal selectivity was observed in older monkeys. The authors surmised that a loss of cortical inhibition may underlie a change from sparse to more diffuse encoding with increased age.

Summary—A consistent age-related increase in envelope tracking has been revealed across studies. At this time, it is unclear how much of this increase is due to aging or hearing loss. The studies that have investigated hearing loss effects on envelope tracking have differed, likely due to the difficulty in finding a sufficient number of participants who differ in hearing thresholds but are matched in age. However, Fuglsang et al. (2020) found increased envelope tracking in a relatively large number of HI participants compared to age-matched NH participants. Cascading effects of reduced afferent input with aging that is compounded with hearing loss may lead to decreased inhibition and increased neural excitability (Ng and Recanzone, 2018).

Summary and Clinical Implications

Temporal processing deficits have been identified in older listeners across a wide range of neurophysiological measures that assess the auditory system from the auditory nerve to cortex. However, the relative contributions of aging and hearing loss are difficult to tease apart, as high-frequency hearing loss is an inevitable consequence of aging across most species. Therefore, it is not possible to state conclusively that central processing deficits, as observed in objective measures, result from aging effects that are independent of decreased afferent input from the cochlea and the auditory nerve.

Despite the difficulty of separating contributions of hearing vs. aging factors, objective measures of temporal processing may be useful components of the audiological assessment. These measures may reveal impaired neural representation that can be compensated on tests

of clinical speech understanding, through increased effort or engagement of cognitive resources (Kuchinsky et al., 2013; Zekveld et al., 2018). The effort needed to perform well on a clinical measure would not be sustainable in everyday settings without incurring fatigue; therefore, behavioral clinical measures may fall short in estimating functional hearing ability. Even though there may be a need for an objective clinical measure, the most efficacious measures for this purpose has not yet been identified. The three main requirements for a clinical assessment, reliability, sensitivity, and specificity (Roeser et al., 2007), have not yet been achieved in any of the measures discussed in this review. For example, the ABR has high reliability, at least for latency, but its sensitivity for identifying speech understanding deficits has not yet been demonstrated. The FFR also has high reliability and FFR deficits appear to relate to speech perception deficits, especially in noise, but most studies that have investigated the role of the FFR in speech understanding in older listeners have been correlative in nature (Anderson et al., 2011; Hao et al., 2018; McClaskey et al., 2019; Roque et al., 2019b), and it would be important to demonstrate its sensitivity/specificity in specific clinical populations. Finally, there has been a great deal of interest in the clinical utility of envelope tracking, especially as ecologically-valid speech samples are used in the measurement. However, more work needs to be done with this measure to improve the ability to detect the response above neural noise, and to ensure that the measure reflects speech understanding itself, rather than the brain's locking onto specific acoustical attributes of the stimulus.

Although implementation of objective measures into clinical test protocols is not yet routine, these findings have implications for patient counseling regarding management of hearing difficulties (Davis et al., 2016). Patients often complain about having difficulty understanding people who speak rapidly or in a noisy background, and they may wonder why their audiogram shows normal thresholds. They may benefit from communication strategies to improve successful communication, such as maximizing the use of visual cues, positioning oneself to reduce background noise, or asking the speaker to speak more slowly and clearly (Johnson et al., 2018).

For patients with hearing loss, the primary form of management is the provision of devices to improve audibility through the use of hearing aids, or in the case of severe to profound hearing loss, cochlear implants. But these studies demonstrate that audibility alone is not sufficient to achieve speech-in-noise performance comparable to that of younger listeners. For this reason, studies have been conducted to determine the potential benefits of auditory training, but the results have been mixed, especially for commercial programs (Henshaw and Ferguson, 2013). Some studies have demonstrated improvement in speech understanding in both normal-hearing and hearing-impaired listeners (Anderson et al., 2013a; Anderson et al., 2013b; Burk and Humes, 2007; Ferguson and Henshaw, 2015), but generalization to untrained stimuli is generally limited (Karawani et al., 2015). And, a recent randomized-control trial in a large group of veterans failed to show any additional benefit of training over the use of amplification alone (Saunders et al., 2016). More work is needed to determine the most efficacious means of treatment, and it would be important to identify the specific processing deficits that could be targeted for remediation.

An understanding of the specific nature of temporal processing deficits is hindered by inconsistent or conflicting results across studies. These inconsistencies may arise from a number of issues in aging EEG research. It is difficult to recruit older listeners with normal hearing, and for this reason, the study sample sizes are typically small. Furthermore, many of these studies are exploratory, rather than hypothesis-driven, assessing aging effects across a wide range of stimulus conditions or EEG measures, increasing the possibility of a significant finding that is actually a random statistical variation. The field would benefit from rigorous studies with a theoretical framework based on animal models, human perception, and previous human EEG studies.

Conclusion

Neural degradation at early levels of processing and overcompensation for this degradation in cortex may contribute to speech understanding difficulties experienced by older listeners. More work is needed to tease apart degradation associated with cochlear dysfunction versus more central pathologies. New clinically-feasible measures are needed to provide a comprehensive assessment of the listener's speech understanding abilities.

Acknowledgments

Abbreviations:

ABR	Auditory brainstem response
ASSR	Auditory steady-state response
EEG	Electroencephalographic
FFR	Frequency-following response
EFR	Envelope-following response
CAEP	Cortical auditory-evoked potential
MEG	Magnetoencephalographic
YNH	Young normal-hearing
ONH	Older normal-hearing
OHI	Older hearing-impaired
peSPL	Peak-equivalent sound pressure level
VOT	Voice-onset-time
PTA	Pure-tone-average

F_0	Fundamental frequency
ARHL	Age-related hearing loss
SNR	Signal-to-noise ratio
IC	Inferior Colliculus
STR	Stimulus-to-response
TRF	Temporal response function

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Highlights

- Older normal-hearing listeners experience neural degradation at early levels of the auditory system

- Neural degradation in brainstem/midbrain is over-compensated in auditory cortex

- Hearing loss leads to exaggeration of neural responses; but this exaggeration may be diminished with advanced aging
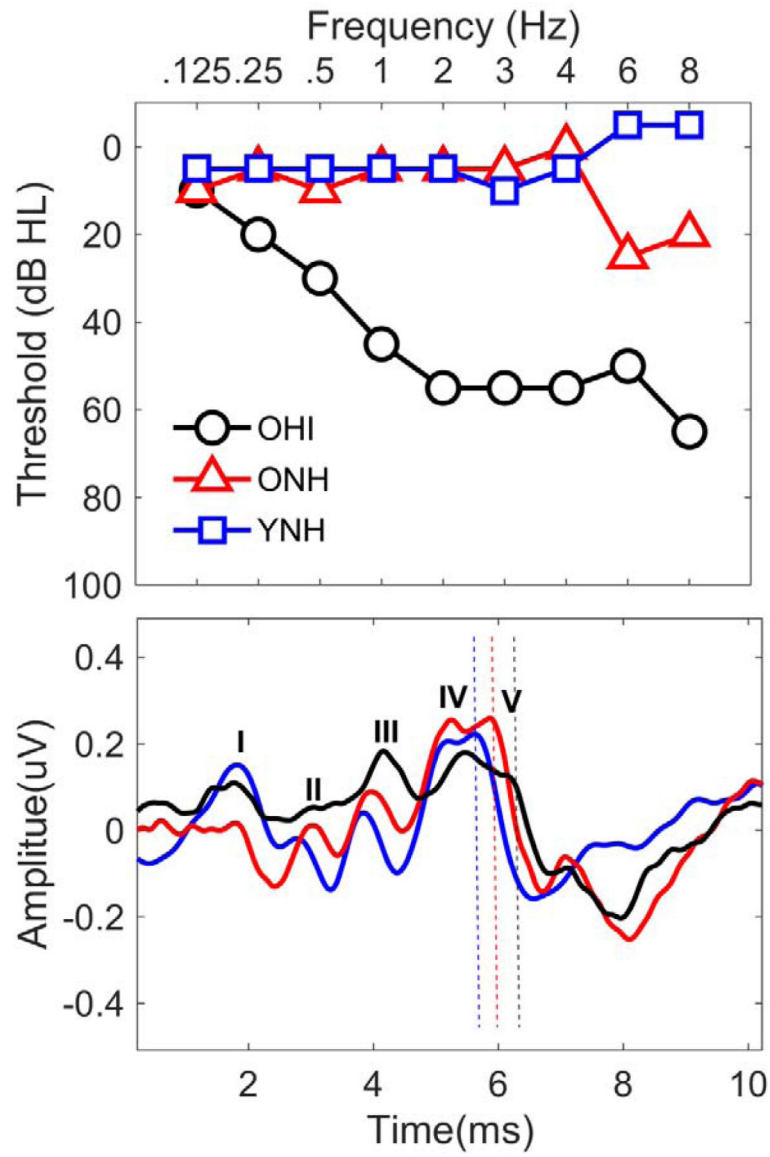
Fig. 1.
Individual audiograms (top panel) and auditory brainstem response waveforms (bottom panel) are displayed for representative young normal-hearing (YNH, blue squares), older normal-hearing (ONH, red triangles), and older hearing-impaired (OHI, black circles) listeners. Even slight high-frequency loss above 4000 Hz is associated with Wave V latency delays.
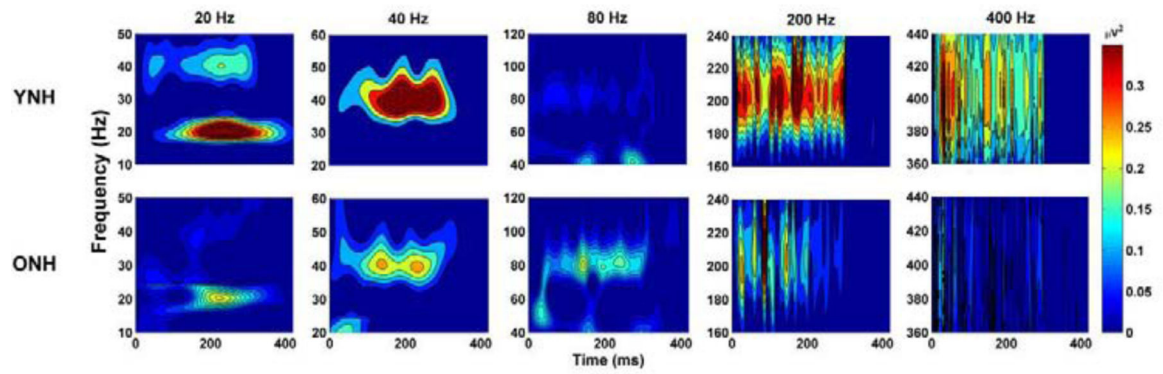
Fig. 2.
Spectral energy corresponding to pulse-train presentation rates shows that responses in younger normal-hearing listeners (YNH, top panel) have higher frequency energy than in older normal-hearing listeners (ONH, bottom panel) that is significant for the 400-Hz rate. Furthermore, a decrease in spectral energy with higher rates is only significant in the ONH listeners. Used with permission from Gaskins et al. (2019).
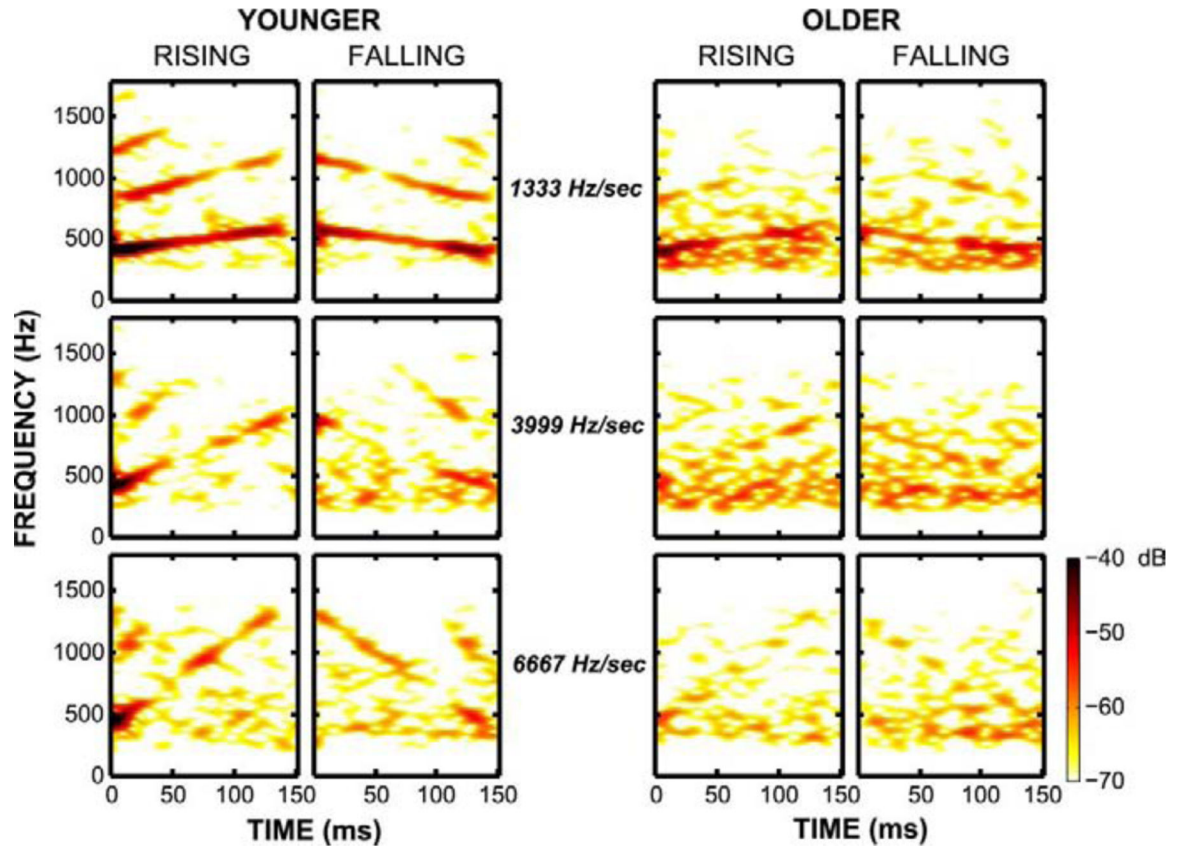
Fig. 3.
FFR spectral amplitudes to dynamic frequency changes are displayed for a representative young normal-hearing listener (left panel) and a representative older normal-hearing listener (right panel). The responses in the younger listener show more robust tracking of dynamic frequency than the responses of the older listener across rates of change. Used with permission from Clinard et al. (2015).
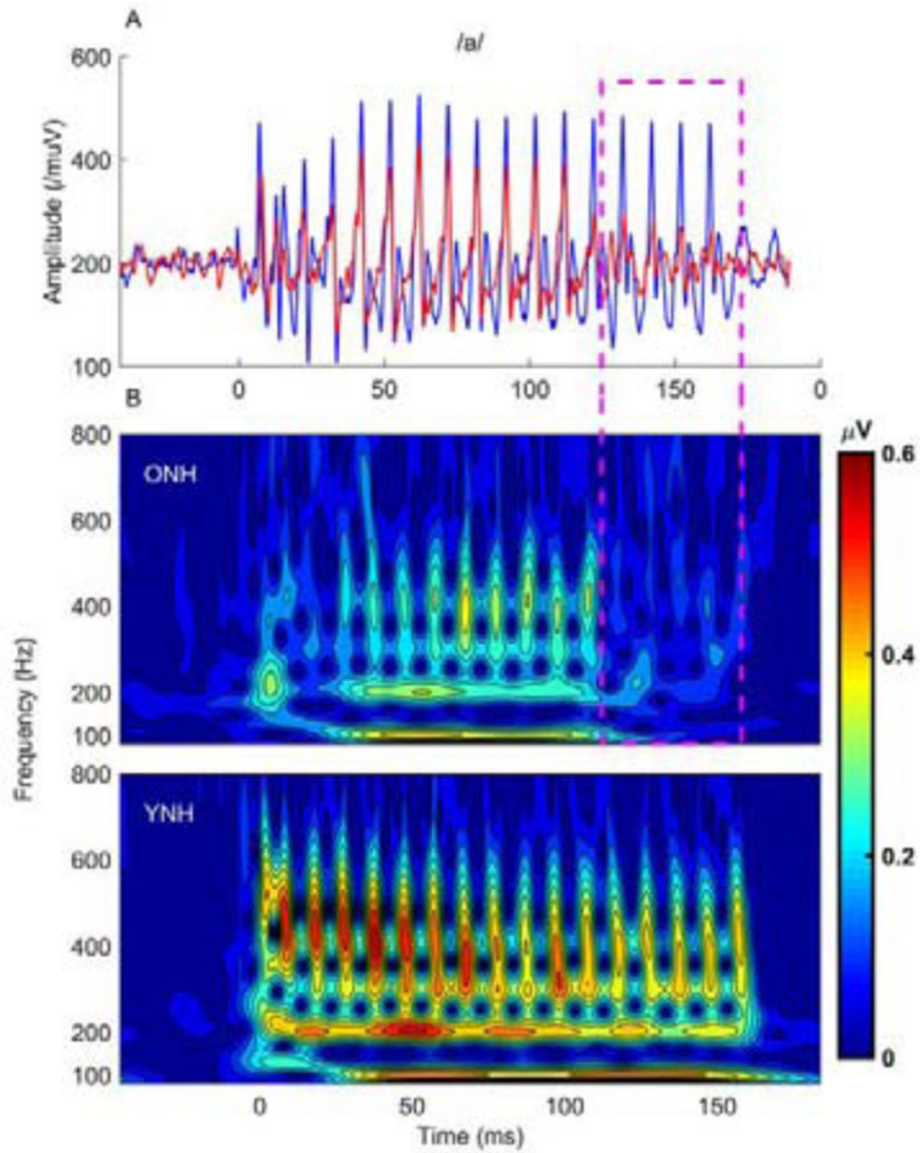
Fig. 4.

An abrupt loss of synchrony in the later response regions to the vowel [a] is noted in older normal-hearing (ONH) listeners' responses in panels A and B. This drop was not seen in the younger normal-hearing (YNH) listeners. The dashed pink rectangle delineates this later region where a dramatic decrease in response amplitude (A) and frequency energy (B) can be seen in the ONH responses. Modified with permission from Presacco et al. (2015).
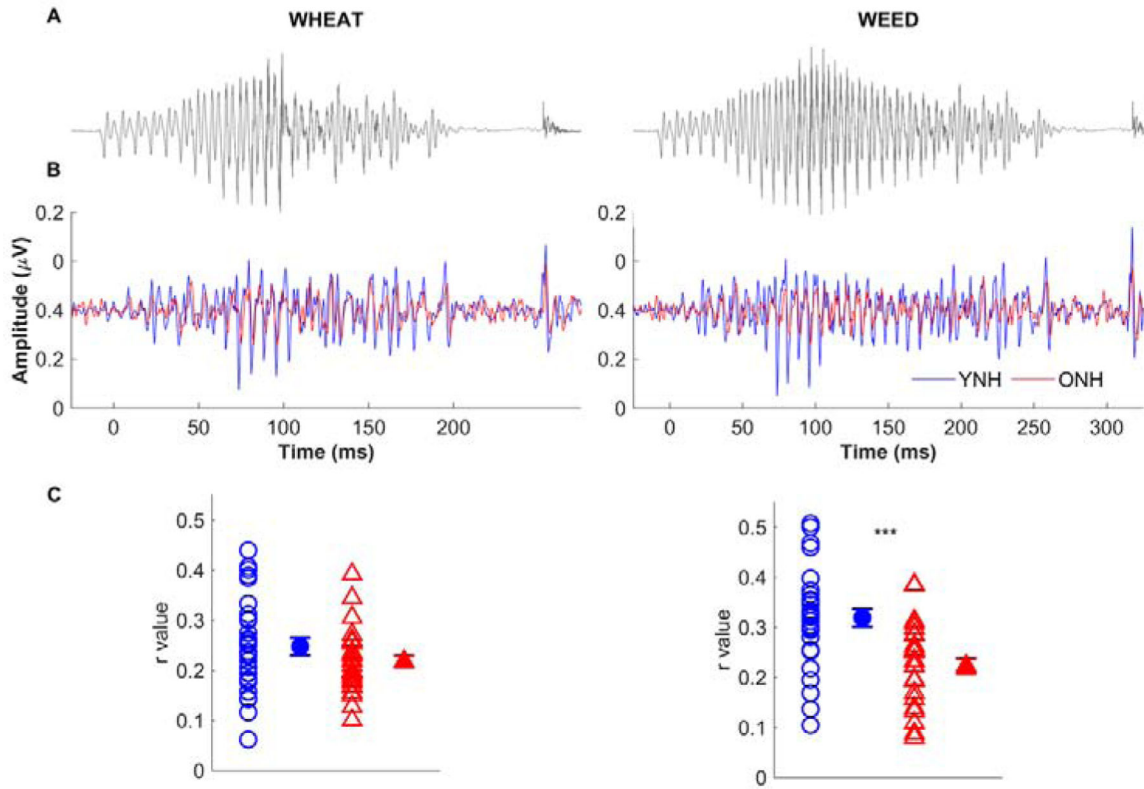
Fig. 5.

Stimulus-to-response correlation 'r' values are lower in ONH than in YNH listeners for the word WEED but not for WHEAT. Panel A shows stimulus waveforms that differ only in vowel duration. Panel B shows response waveforms in young normal-hearing (YNH, blue) and older normal-hearing (ONH, red) listeners. A drop in neural synchrony is apparent in the ONH listeners at approximately 185 ms. Panel C displays individual (open symbols) and mean (closed symbols) for YNH (blue circles) and ONH (red triangles) listeners. Error bars: ± 1 standard error. ***p < 0.001. Modified with permission from Roque et al. (2019a).
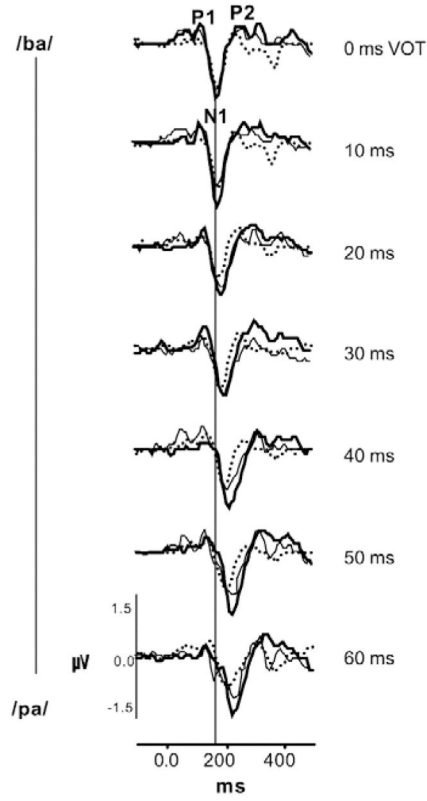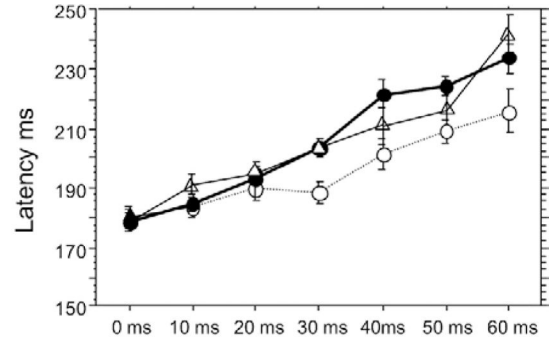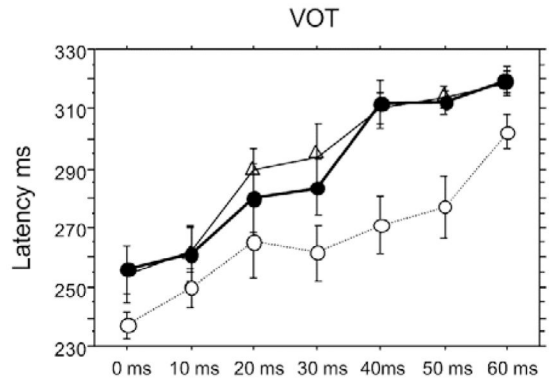
Fig. 6.
Left panel: Group average waveforms show that increasing voice-onset-time (VOT) results in increased latencies of P1, N1, and P2 peaks of the cortical auditory-evoked response. Right panels: N1 and P2 peaks show greater delays with increasing VOTs in older normal-hearing (ONH, filled circles) and older hearing-impaired (OHI, open triangles) listeners compared to young normal-hearing (YNH, open circles) listeners. Error bars: 1 standard error. Used with permission from Tremblay et al. (2003).
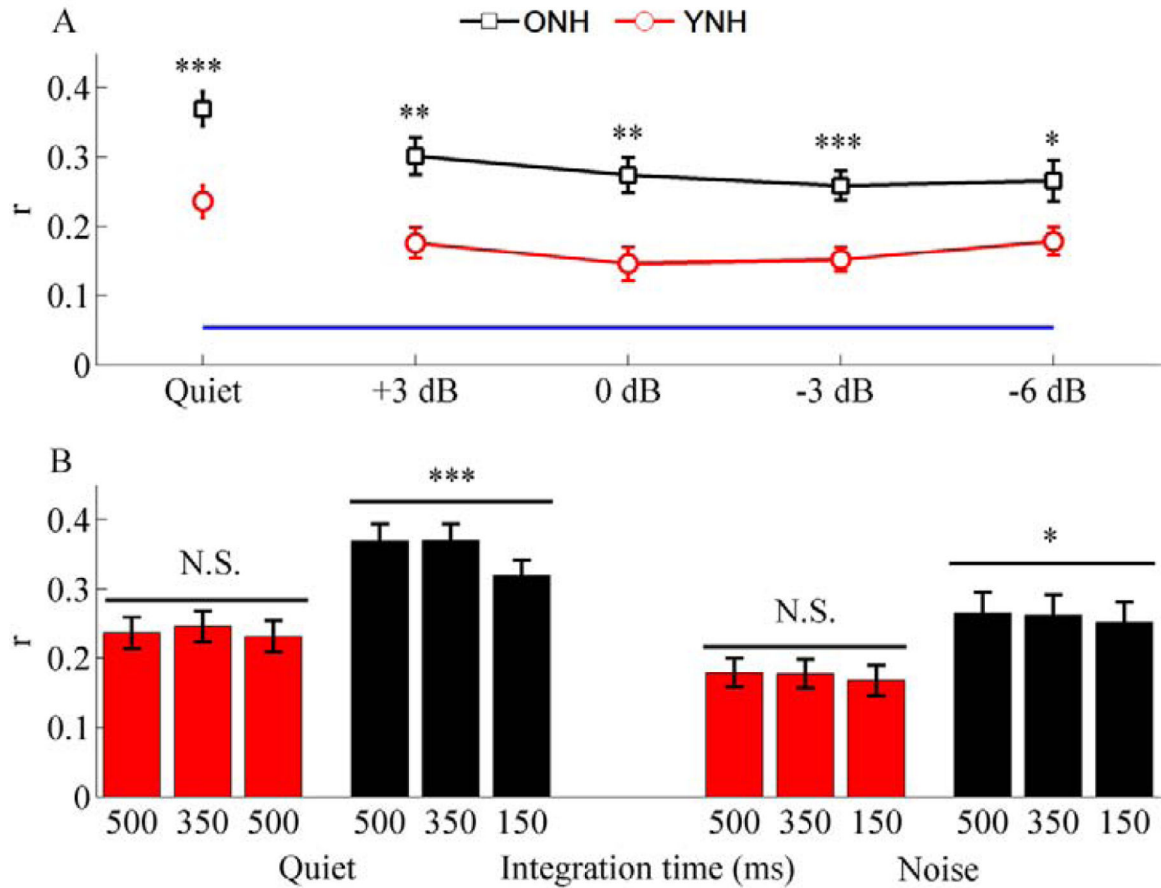
Fig. 7.

Panel A: Older listeners' cortical responses (ONH; black squares) show higher reconstruction accuracies (r values) in quiet and across SNR conditions compared to younger responses (YNH; red circles). Responses of both groups are above the noise floor (blue line). Panel B: ONH responses show a reduction in reconstruction accuracy as the integration time window is narrowed from 500 ms to 150 ms in both quiet and noise conditions, but the YNH responses do not show a similar decrease in reconstruction accuracy. Error bars: ± 1 standard error. *p < 0.05, **p < 0.01, ***p < 0.001, N.S. = not significant. Modified with permission from Presacco et al. (2016a).