Hearing Research 371 (2019) 66–74

Contents lists available at ScienceDirect

Hearing Research

journal homepage: www.elsevier.com/locate/heares

Research Paper

Investigating peripheral sources of speech-in-noise variability in listeners with normal audiograms

S.B. Smith [1], J. Krizman, C. Liu, T. White-Schwoch, T. Nicol, N. Kraus*

Northwestern University, Department of Communication Sciences and Disorders, Evanston, IL, USA

ARTICLE INFO

Article history:
Received 5 June 2018
Received in revised form
25 October 2018
Accepted 19 November 2018
Available online 22 November 2018

ABSTRACT

A current initiative in auditory neuroscience research is to better understand why some listeners struggle to perceive speech-in-noise (SIN) despite having normal hearing sensitivity. Various hypotheses regarding the physiologic bases of this disorder have been proposed. Notably, recent work has suggested that the site of lesion underlying SIN deficits in normal hearing listeners may be either in "sub-clinical" outer hair cell damage or synaptopathic degeneration at the inner hair cell-auditory nerve fiber synapse. In this study, we present a retrospective investigation of these peripheral sources and their relationship with SIN performance variability in one of the largest datasets of young normal-hearing listeners presented to date. 194 participants completed detailed case history questionnaires assessing noise exposure, SIN complaints, tinnitus, and hyperacusis. Standard and extended high frequency audiograms, distortion product otoacoustic emissions, click-evoked auditory brainstem responses, and SIN performance measures were also collected. We found that: 1) the prevalence of SIN deficits in normal hearing listeners was 42% when based on subjective report and 8% when based on SIN performance, 2) hearing complaints and hyperacusis were more common in listeners with self-reported noise exposure histories than controls, 3) neither extended high frequency thresholds nor compound action potential amplitudes differed between noise-exposed and control groups, 4) extended high frequency hearing thresholds and compound action potential amplitudes were not predictive of SIN performance. These results suggest an association between noise exposure and hearing complaints in young, normal hearing listeners; however, SIN performance variability is not explained by peripheral auditory function to the extent that these measures capture subtle physiologic differences between participants.

1. Introduction

Audiologists routinely encounter patients with normal audiograms who complain of speech-in-noise (SIN) deficits (Zhao and Stephens, 2007). The etiology of this disorder remains unclear, and there are currently no clinical guidelines regarding its prevention, treatment, or rehabilitation (Jerger, 2011; Pryce and Wainwright, 2008). The existence of this phenotype indicates that one or more auditory processing deficits go undetected by the standard audiometric test battery. Thus, a current focus in auditory research is to better understand the basis of SIN deficits in listeners with normal audiograms and to translate these findings into improved clinical diagnosis and management of afflicted patients.

Various hypotheses regarding the etiology of SIN deficits with normal audiograms have been proposed (Grose et al., 2017; Pienkowski, 2017).[2] Initially, the disorder was considered to be

* Corresponding author.
E-mail address: nkraus@northwestern.edu (N. Kraus).
URL: http://www.brainvolts.northwestern.edu

[1] Current Affiliation: The University of Texas at Austin, Department of Communication Sciences and Disorders, Austin, TX, USA.

[2] SIN deficits with normal hearing sensitivity have also been referred to as King-Kopetzky Syndrome (Hinchcliffe, 1992), Auditory Processing Disorder (Musiek et al., 2018), obscure auditory dysfunction (Saunders and Haggard, 1989), auditory disability with normal hearing (King and Stephens, 1992), idiopathic discriminatory dysfunction (Rappaport et al., 1993), speech-perception-in-noise (SPiN) impairment (Guest et al., 2018), and "hidden hearing loss" when the suspected site of lesion is at the level of the auditory nerve (Liberman et al., 2016; Kujawa and Liberman, 2009).

[3] Previous work has shown sex differences in some parameters of interest to the current study, such as CAP amplitude (e.g., Grinn et al., 2017; Stamper and Johnson, 2015). Notably, these differences emerged when using very intense signals (~120 dB ppeSPL; see Fig. 1D in Grinn et al., 2017). We did not observe sex differences in any parameter in our initial analyses; therefore, sex was not included as an independent variable.

https://doi.org/10.1016/j.heares.2018.11.008
0378-5955/

"nonorganic" and therefore *psychological* in origin (Doerfler and Stewart, 1946; Hinchcliffe, 1992; King and Stephens, 1992). Some studies have reported relationships between anxiety and depression and poor performance on SIN tasks for normal hearing listeners (Pryce et al., 2010; Zhao and Stephens, 2000), whereas others have identified possible contributions from environmental and lifestyle factors (Tremblay et al., 2015). The heterogeneity of these reported relationships makes it difficult to isolate specific psychological or lifestyle characteristics to investigate clinically, particularly in ENT and/or audiologic settings where the evaluation of such parameters falls largely outside the professional scope of practice.

More recent work has focused on *physiological* deficits in auditory processing that are undetectable with the standard audiometric test battery but may be potent enough to cause functional hearing impairment. For example, work in humans indicates that outer hair cell dysfunction can occur without an elevation in standard audiometric thresholds (Badri et al., 2011; Lapsley-Miller et al., 2006; Narula and Mason, 1988). This "sub-clinical" outer hair cell damage is indexed by reduced otoacoustic emissions amplitudes (Hobem et al., 2017; Lapsley-Miller et al., 2006), elevated thresholds in the extended high frequency (EHF) range (>8 kHz), and wider auditory filter bandwidths measured psychophysically within the standard audiometric range (≤8 kHz; Badri et al., 2011; Narula and Mason, 1988; Pick and Evans, 1983). The importance of EHF hearing is further supported by the observation that elevated thresholds in this range are related to poorer SIN performance in reverberant conditions (Besser et al., 2015). It has thus been suggested that EHF thresholds may serve as a "barometer" of cochlear health and should be included in the clinical test battery (e.g., Moore et al., 2017).

Another pathophysiology that has been offered as the cause of SIN deficits with normal audiograms is "cochlear synaptopathy": noise- and/or age-induced synaptic loss between inner hair cells and low-spontaneous rate auditory nerve fibers without systemic hair cell loss (Furman et al., 2013; Hickox et al., 2017; Kujawa and Liberman, 2009). This site of lesion spares hearing threshold sensitivity and hair cell function but reportedly impairs suprathreshold neural encoding of auditory signals (see Carney, 2018 for an alternative interpretation). Thus, the physiologic "profile" of synaptopathy is normal standard audiometric thresholds and otoacoustic emissions with abnormally small auditory nerve compound action potential (CAP) amplitudes evoked by suprathreshold stimuli. This profile has been demonstrated in noise-exposed mice (Fernandez et al., 2015; Kujawa and Liberman, 2009; Wang and Ren, 2012), rats (Lobarinas et al., 2017), and guinea pigs (Furman et al., 2013; Lin et al., 2011 Shi et al., 2013). The existence of cochlear synaptopathy has been confirmed histologically in each of these models as well as rhesus macaques (Hickox et al., 2017; Valero et al., 2017). With the exception of one study, data relating confirmed cases of cochlear synaptopathy with perceptual deficits in animals are currently unavailable. Lobarinas et al. (2017) reported poorer signal-in-noise detection and reduced CAPs in rats with noise-induced cochlear synaptopathy compared to controls; however, the perceptual effects were only observed at low signal-to-noise ratios and did not correlate with degree of CAP reduction. To empirically address the relationship between confirmed cochlear synaptopathy and behavior, more animal studies combining physiologic and perceptual measurements must be undertaken.

Motivated by the aforementioned animal research, several investigators have speculated that human cochlear synaptopathy underlies SIN deficits in listeners with normal audiograms and may also be related to tinnitus and hyperacusis (Kujawa and Liberman, 2009; Lin et al., 2011; Makary et al., 2011; Prendergast et al., 2016, 2017 Schaette and McAlpine, 2011; Spankovich et al., 2018; Stamper and Johnson, 2015a,b). Studies investigating these hypothesized relationships have produced mixed results. For example, Bharadwaj et al. (2015) reported correlations between subcortical neural encoding of amplitude modulations and spatial hearing performance in a small preliminary dataset of young adults. Although these neural responses reflected phase-locked potentials from multiple subcortical auditory nuclei, the authors interpreted their results as being consistent with cochlear synaptopathy, as envelope encoding deficits were also associated with self-reported history of noise exposure (cf. Shaheen et al., 2015). Similarly, Liberman et al. (2016) reported a higher prevalence of hyperacusis, poorer SIN perception, and larger summating potential (SP)/CAP amplitude ratios in a small sample of young listeners with a history of noise exposure compared to controls. It is important to note, however, that the SP/CAP ratio differences were driven by SP amplitude differences, which are mainly *preneural* components related to hair cell activity (Dallos et al., 1982; Eggermont, 2017; Russell and Sellick, 1978) and *not* CAP reductions, as predicted by animal models. This observation, combined with poorer EHF thresholds in the noise exposed group, suggest that group differences may be related to sub-clinical outer hair cell damage and not cochlear synaptopathy, although the relationship between the two remains poorly understood. Multiple studies from different research groups have failed to demonstrate relationships between noise exposure history, SIN deficits, and electrophysiologic measures of synaptopathy (Bramhall et al., 2017; Fulbright et al., 2017; Grinn et al., 2017; Grose et al., 2017; Guest et al., 2018; Prendergast et al., 2017).

Some discrepancies in the human literature may be related to the different tools used to assess noise exposure history, SIN performance, and neurophysiologic function. However, a more significant problem may be that few large-scale studies including a sizeable number of participants with normal audiograms and poor SIN performance have been conducted (Guest et al., 2018). Future large-scale studies should address this issue by *prospectively* recruiting participants with this phenotype (see Grinn et al., 2017), as has been done with a smaller sample by Guest et al. (2018). In the meantime, widespread interest in this problem provides an opportunity for auditory research laboratories and clinics to screen their databases for evidence of peripheral sources of SIN variability in listeners with normal audiograms.

To this end, the present study is a retrospective analysis of a large dataset of young normal-hearing listeners who participated in multiple studies in the Auditory Neuroscience Laboratory at Northwestern University between 2009 and 2017. While these studies had distinct goals, they had a common set of audiological screening tests suitable for addressing the questions posed below. Participants completed case history questionnaires assessing noise exposure, SIN complaints, tinnitus, and hyperacusis. Standard and EHF audiograms, distortion product otoacoustic emissions (DPOAEs), click-evoked auditory brainstem responses, and SIN performance measures were also collected.

The specific goals of this study were to leverage this large dataset to assess:

1. The prevalence of SIN deficits in a group of young adult listeners
2. The association between self-reported noise exposure history and hearing complaints, tinnitus, and hyperacusis
3. The relationship between EHF thresholds and SIN performance
4. The relationship between auditory brainstem response wave I amplitudes and SIN performance

2. Materials and methods

2.1. Participants

The Institutional Review Board of Northwestern University approved the following methods. Participants were selected from the Auditory Neuroscience Laboratory database using the following inclusion criteria: 1) Participant age was between 18 and 30 years, 2) English was the participant's primary language, 3) No significant history of neurologic or otologic disease was reported, 4) Otoscopy revealed normal tympanic membranes and normal standard audiometric thresholds (0.25–8 kHz; $\leq$ 20 dB HL) bilaterally. This screening identified 323 participants. We selected the subset who had completed all measurements described in the Procedures section, resulting in 194 participants (90 females, mean age: 22.56 yr. SD).

2.2. Procedures

2.2.1. Questionnaire

Subjects completed an extensive case history questionnaire prior to or during their first testing visit. Subsections of this questionnaire included: Family History, Medical History, Speech and Language Development, and Education History. The present analysis focused on respondents' answers to medical history questions regarding noise exposure, difficulty hearing in a variety of settings, tinnitus, and hyperacusis.

Participants were asked whether they had experienced excessive noise exposure by indicating "yes" or "no" and, if so, to elaborate on the nature and estimated duration and/or frequency of the exposure. While more sophisticated noise questionnaires exist (see Fullbright et al., 2017; Johnson et al., 2017; Megerson, 2010; Prendergst et al., 2017; Spankovich et al., 2018; Stamper and Johnson, 2015a), some evidence indicates that recall of noise exposures and dosimetry measurements of those exposures are highly correlated (Reeb-Whitaker et al., 2004). Participants' responses to this question were used to divide participants into "noise exposed" and "control" groups.

The following prompt was used to assess each listener's subjective hearing difficulty:

Please indicate all situations in which you have difficulty hearing:

1. On the telephone
2. In a conversation with one person
3. In a group
4. In noisy situations
5. When people talk too fast
6. In large rooms/auditoriums

Affirmative responses were summed and used to quantify subjective hearing difficulty on a scale from 0 to 6. These values were used to calculate mean hearing complaints for noise exposed and control groups.

Participants were asked whether they had chronic tinnitus and, if so, to describe its duration, laterality, quality (ringing, buzzing, roaring, pulsing, or other), and severity on a 0–6 scale. In addition, participants were asked the following question to determine whether they were hyperacusic:

Do you consider yourself to be more sensitive to sound than others (i.e., are you hypersensitive to sound)?

The proportion of subjects reporting tinnitus and/or hyperacusis was evaluated for noise-exposed and control groups, as both are suggested correlates of synaptopathy (Hickox and Liberman, 2014; Kujawa and Liberman, 2015; Plack et al., 2014; Schaette et al., 2014; Schaette and McAlpine, 2011).

2.2.2. Audiograms

Air conduction audiometric thresholds were obtained bilaterally from 0.25 to 14 kHz using a Grason-Stadler GSI 61 audiometer. Thresholds in the standard audiometric range (0.25–8 kHz) were obtained using Etymotic 3A insert earphones, whereas Sennheiser HDA 200 circumaural earphones were used from 10 to 14 kHz. For our statistical analyses, EHF thresholds (10, 12.5, and 14 kHz) were averaged to generate a single number representing extended high frequency sensitivity. All thresholds were quantified using a modified Hughson-Westlake procedure with 10 dB descending and 5 dB ascending step sizes (Carhart and Jerger, 1959). Bone conduction thresholds were obtained in some, but not all, participants and were therefore not analyzed. All testing was conducted in a sound treated room. Although participants were screened for normal hearing bilaterally, only right ear thresholds were assessed for this analysis.

2.2.3. Distortion product otoacoustic emissions (DPOAEs)

DPOAEs were acquired using the Biologic Scout system (Natus Corp., San Carlos, CA). Measurements were made using two primary tones, f_1 and f_2 ($f_2/f_1 = 1.22$; f_2 Level = 55 dB SPL, f_1 Level = 65 dB SPL), presented at discrete frequencies of f2 = 1, 1.5, 2, 3, 4, 6, and 8 kHz. Only right ear DPOAEs were evaluated.

2.2.4. Click-evoked auditory brainstem responses

Auditory brainstem responses (ABRs) were elicited and recorded with a Biologic Navigator AEP system (Natus Corp., San Carlos, CA) using a 100-μs square-wave click (98.5 dB ppeSPL) presented at a rate of 31/sec to the right ear. Responses were collected using the following montage: Cz (+), right earlobe (−), and forehead (GND). A limitation of this montage is that the compound action potential (CAP; synonymous with ABR wave I) tends to be smaller than when recorded using "tip-trodes" or tympanic membrane (TM) electrodes; however, CAP amplitudes are more variable with tip-trodes (Roland et al., 1993, 1995) and TM electrodes may alter the conduction characteristics of the middle ear, particularly at low to mid-frequencies (Smith et al., 2016). Recent evidence indicates that despite being slightly smaller in amplitude, CAPs collected from mastoid electrodes have nearly equivalent test-retest reliability to CAPs collected with tip-trodes (Prendergast et al., 2018). Three click-evoked ABRs consisting of 2000 sweeps were recorded from each subject during his or her test session; these responses were averaged together to create a grand average waveform that was analyzed offline in MATLAB.

Based on previous work suggesting that CAP and SP amplitudes are sensitive to cochlear synaptopathy, we focused our analysis on these early ABR components. CAPs were automatically identified within the 1.45–1.95 ms epoch using MATLAB's *findpeaks* function. SPs were automatically identified as the highest inflection point within the first millisecond after stimulus onset (i.e., the point at which the first derivative of the waveform changed from a positive value to approximately 0). The accuracy of the automated CAP and SP identification was assessed by the first author and adjusted if needed; the author was blind to subject groupings during this process and alterations to automatically picked peaks were required in about 12% of responses. CAP and SP amplitudes were quantified relative to the average pre-stimulus baseline amplitude (−0.8-0 ms), and only response amplitudes exceeding one standard deviation above the pre-stimulus noise floor were considered for analysis. Using this method, CAPs were identified in 189 participants (97%) and SPs were identified in 157 participants (81%). Although SP/CAP ratios have been reported in the literature (Liberman et al., 2016), this metric has poor test-retest reliability

(Prendergast et al., 2018) and obfuscates whether group differences are due to preneural or neural components.

2.2.5. QuickSIN

The QuickSIN is a widely-used non-adaptive test of SIN perception. Participants are asked to repeat sentences they hear diotically from a target speaker in the presence of four-talker babble. The presentation level of the target speaker decreases by 5 dB for each sentence while the background babble is held constant, and the listener receives one point for each target word correctly repeated. The total number of words correct is subtracted from 25.5 to obtain a "signal-to-noise ratio threshold", which reflects the signal-to-noise ratio required for a listener to understand 50% of the target speech. A signal-to-noise ratio threshold $\leq$2 is considered normal performance (see Killion et al., 2004).

2.3. Statistical analyses[3]

The number of hearing complaints reported by control and noise exposed groups were compared using a Mann-Whitney *U* test. Associations between group and self-reported tinnitus and hyperacusis, respectively, were evaluated using chi-squared tests. Repeated measures analysis of variance was used to evaluate differences between groups for audiometric thresholds, DPOAEs, and CAP and SP amplitudes, whereas two-tailed t-tests for unequal variances were used to evaluate QuickSIN scores. Linear regression analyses were used to test if CAP amplitudes or EHF thresholds were respectively predictive of QuickSIN performance. Because relationships between many outcome variables were examined in this study, Bonferroni-adjusted alpha levels of 0.006 per test were used.

3. Results

3.1. Hearing complaints, tinnitus, and hyperacusis

Sixty-three of the 194 participants reported a history of excessive noise exposure. While some participants provided detailed estimates of their noise exposure history (e.g., "drums for 5–6 h/wk for the last 10 years without hearing protection"), others did not elaborate. Thus, further subdividing the noise exposed group by severity was not possible. Eighty-two participants reported one or more situations in which they experienced hearing difficulties; notably, the noise exposed group had significantly more hearing complaints than the control group ($U = 1112$, $p < 0.001$; Fig. 1A). There was no association between noise exposure history and tinnitus ($\chi(1) = 1.32$, $p = 0.25$; Fig. 1B). Hyperacusis was more likely to be reported by noise exposed listeners than by controls ($\chi(1) = 4.21$, $p = 0.046$; Fig. 1C); however, the significance of this relationship did not survive Bonferroni correction.

3.2. Hearing thresholds and DPOAEs

Audiograms and DPOAEs for control and noise exposed groups are shown in Fig. 2. Mean thresholds in the standard audiometric range were highly similar between groups ($F(1,192) = 152.4$, $p = 0.1$; Fig. 2A). Above 10 kHz, mean thresholds were poorer in the noise exposed group than in controls; however, these differences did not reach significance at any frequency.

DPOAEs, a proxy measure of outer hair cell function, did not significantly differ between groups at any frequency ($F(1,189) = 7.02$, $p = 0.09$; Fig. 2B). Similar to audiometric thresholds, however, there was a trend for DPOAEs at the highest test frequencies to be poorer in noise exposed relative to control ears.

3.3. SP and CAP amplitudes

Auditory brainstem response morphology was highly similar between control and noise exposed groups (Fig. 3). SP ($t(93) = -0.13$, $p = 0.90$) and CAP ($t(107) = -0.15$, $p = 0.88$) amplitudes did not significantly differ between groups (Fig. 3, inset).

3.4. SIN performance

QuickSIN performance did not differ between control and noise exposed groups ($t(192) = -0.35$, $p = 0.73$; Fig. 4A). Out of 194 participants, 18 (9%) scored outside the normal performance range (>2 dB SNR) on the QuickSIN. Neither CAP amplitude (($F(1,188) = 3.89$, $p = 0.19$; Fig. 4B) nor mean EHF threshold (($F(1,192) = 2.90$, $p = 0.3$; Fig. 4C) predicted QuickSIN performance based on a linear regression analyses.

4. Discussion

The present study examined peripheral sources of SIN variability in one of the largest reported datasets of young adults with normal audiograms. In the following sections, we contextualize our findings within the current debate regarding listeners with SIN deficits and normal audiograms, discuss limitations of our approach, and suggest avenues for future research.

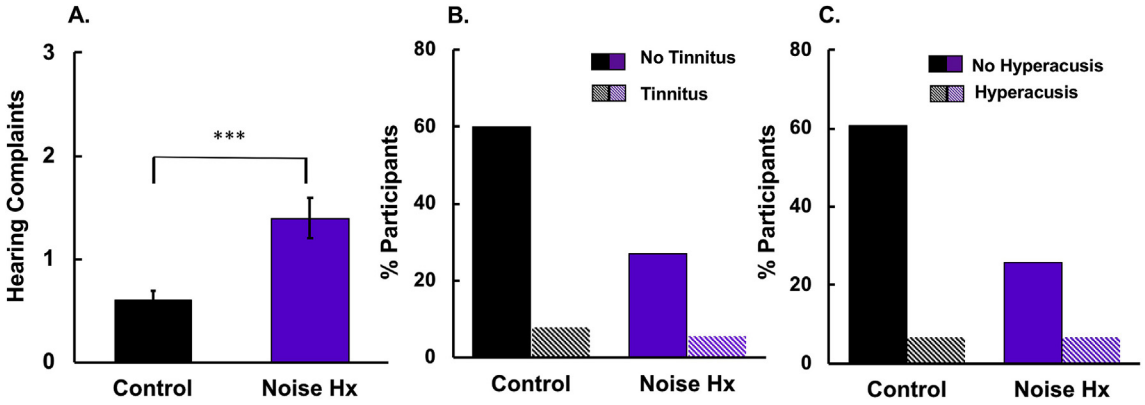

Fig. 1. (A) Participants in the noise exposed group reported on average more situations in which they experienced difficulty hearing than the control group ($p < 0.001$). (B) Tinnitus was reported equally by control and noise exposed groups. (C) Hyperacusis was more likely to be reported by noise exposed participants than controls ($p = 0.046$). Note that the Bonferroni-adjusted alpha levels were 0.006 for each test.

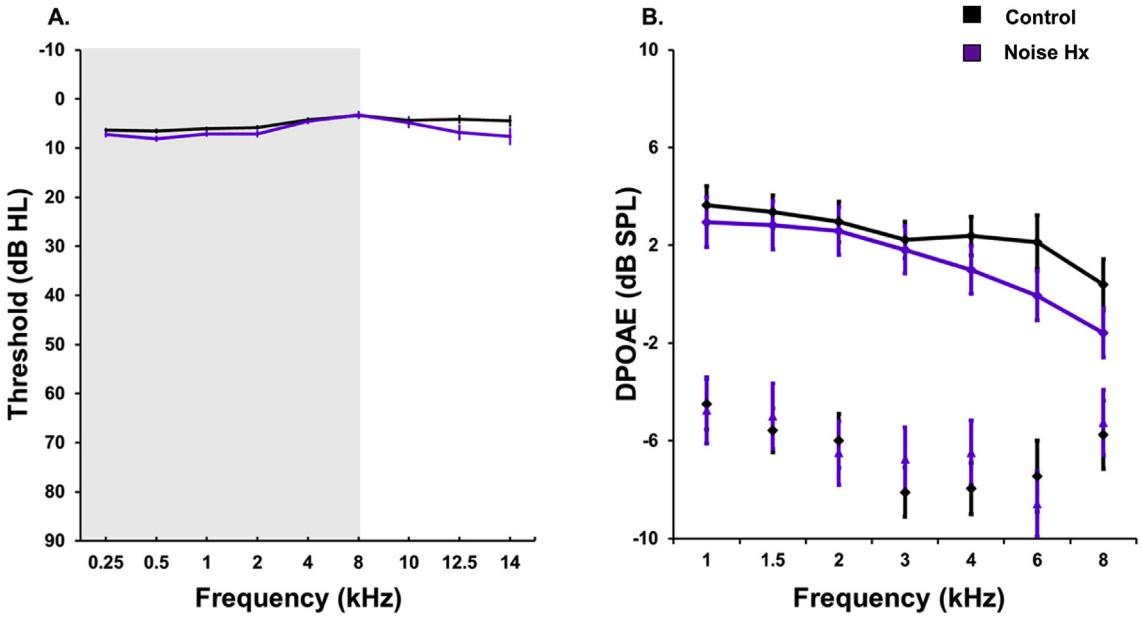

Fig. 2. (A) Mean standard audiometric thresholds (grey shaded area) were highly similar between control (black) and noise exposed (purple) groups. Thresholds began to deviate between groups in the EHF range. (B) DPOAEs levels (connected markers) and noise floors (unconnected markers) are plotted as a function of frequency. DPOAEs did not significantly differ at any test frequency. Above 3 kHz, noise exposed listeners trended toward smaller DPOAEs than controls. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

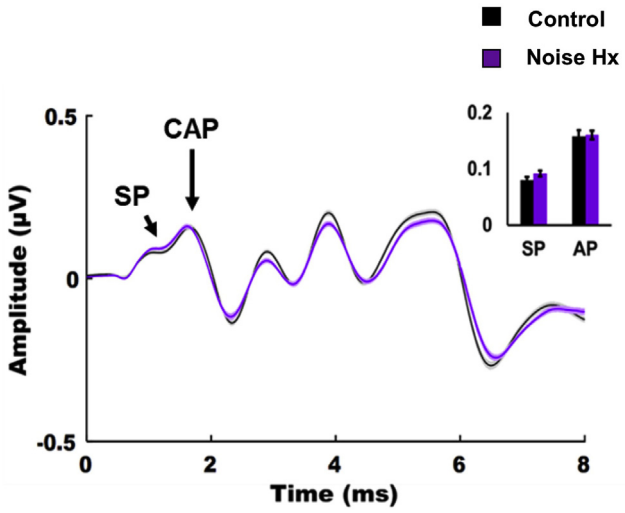

Fig. 3. ABR waveform morphology was highly similar between control (black) and noise exposed (purple) groups (shading = 1 SEM). SP and CAP amplitudes did not differ between the two groups (inset). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

4.1. Phenotype prevalence

The estimated prevalence of SIN deficits in listeners with normal audiograms in our sample depends upon whether self-report or performance-based measurements of SIN ability are assessed. Eighty-two participants (42%) reported one or more situations in which they struggled to hear on the case history questionnaire. In their analysis of the Beaver Dam Offspring Study data, Tremblay et al. (2015) found that approximately 30% of 20–29-year-olds reported similar complaints. This number dropped to 3% when all adults (20–67 years old) were included to estimate population-wide phenotype prevalence. The lower population-wide prevalence may be driven by the fact that older adults complaining of SIN difficulty are more likely to have abnormal audiograms consistent with sensorineural hearing loss.

When a performance-based measurement of SIN ability (the QuickSIN) was used to categorize listeners, only 18 participants (9%) fit the phenotype. This may suggest that the QuickSIN, a tool designed to assess directional microphone and/or FM system candidacy for hearing aid users, is not the best tool for evaluating young listeners. More difficult speech materials, such as single words in noise or time compressed speech in reverberant conditions (e.g., Liberman et al., 2016) may be warranted to assess this demographic; however, these more complex tasks likely make performance dependent on cognitive and/or attentional mechanisms beyond auditory processing. Nevertheless, the suggestion that SIN difficulties are widespread in young listeners with normal audiograms (e.g., Liberman, 2015) may be premature until 1) more epidemiologic data specifically examining this problem are published and 2) a consensus on how to best classify these listeners (i.e., self-report or performance-based metrics) is reached. Perhaps the purest cases of “hidden hearing loss” are those in which sufferers feel impaired enough to seek professional help from ENTs and audiologists. Thus, future studies should recruit directly from clinical settings, where as many as 10% of patients fit the phenotype (Rappaport et al., 1993; Higson et al., 1994). However, our results indicate that listeners' self-reported difficulty may underestimate their true auditory performance. Our findings also reinforce previous reports of a poor concordance between self-reported SIN abilities and performance on clinical measures (Anderson et al., 2013).

4.2. Self-reported noise exposure history, hearing complaints, tinnitus, and hyperacusis

Suggested correlates or sequelae of synaptopathy are difficulty hearing in everyday settings, tinnitus, and hyperacusis (Hickox et al., 2014; Kujawa and Liberman, 2015; Knipper et al., 2013; Plack et al., 2014; Schaette et al., 2014; Schaette and McAlpine,

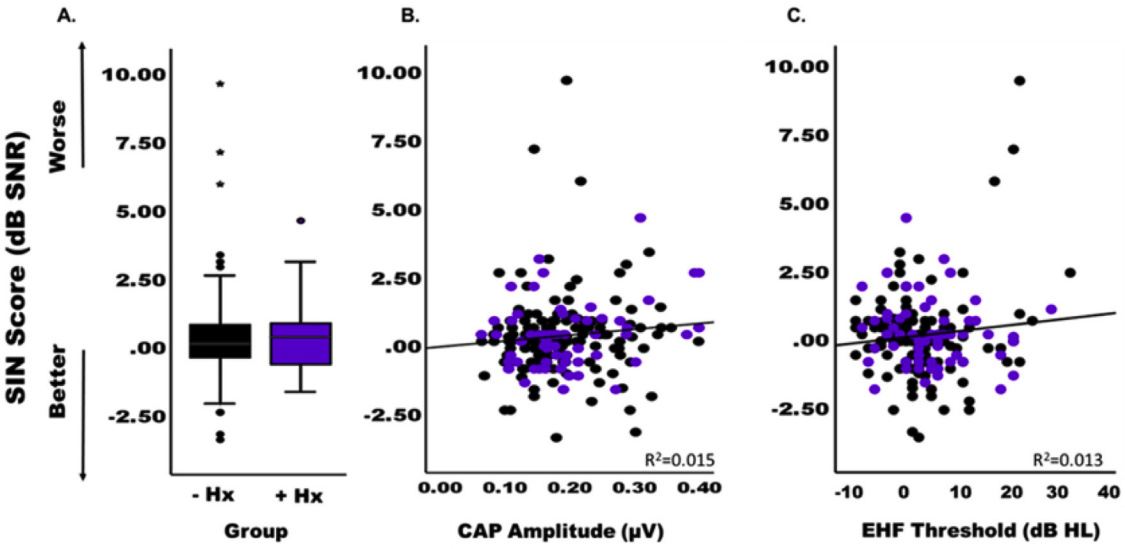

Fig. 4. (A) QuickSIN scores did not differ between control (- Hx) and noise exposed (+Hx) groups. There was not a significant linear relationship between CAP amplitude (B) or mean EHF threshold (C) and QuickSIN score.

2011). Therefore, we were interested in how self-reports of these parameters differed in control versus noise exposed groups. Listeners reporting noise exposure histories were more likely to complain both of SIN deficits and hyperacusis compared to controls; however, the latter relationship did not survive correction for the multiple comparisons made in this study. Tinnitus complaints were equally associated with each group. These findings suggest that, although rudimentary, our questionnaire accurately captured some aspect of noise exposure history and that our grouping of participants was valid. This is further supported by previous work suggesting that recall of task-based noise exposure and dosimetry measurements of those exposures are highly correlated (Reeb-Whitaker et al., 2004).

Development of more sophisticated clinical and research tools to quantify noise exposure history is underway (Johnson et al., 2017; Prendergast et al., 2017). For example, the Noise Exposure Questionnaire (NEQ) has been used by a variety of groups as a method to estimate annual hours of noise exposure (Fullbright et al., 2017; Johnson et al., 2017; Megerson, 2010; Spankovich et al., 2018; Stamper and Johnson, 2015a). Recently, Johnson et al. (2017) presented a distilled version of the NEQ, the 1-Minute Noise Screen, which may be appropriate for identifying "at risk" listeners in clinical and research settings. Prospective studies using dosimetry measurements and/or controlled noise exposures to evaluate the effects of noise on auditory physiology and function in normal hearing listeners are beginning to shed light on the "damage-risk" of synaptopathy in humans (e.g., Grinn et al., 2017; Grose et al., 2017; Skoe and Tufts, 2018). Grose et al. (2017) reported no effect of common recreational noise exposure on perceptual performance in humans and a weak relationship between noise exposure and wave I/V amplitude ratios that would likely not survive statistical correction for multiple comparisons. This suggests that more conservative recommendations for recreational and occupational noise exposure aimed at limiting human cochlear synaptopathy and its hypothesized sequelae may be, at present, premature (see Dobie and Humes, 2017; Murphy and Le Prell, 2017).

4.3. Standard and EHF audiometry

Irrespective of the frequency characteristics of a traumatic sound, the initial signs of noise-induced outer hair cell damage appear in the extreme cochlear base and therefore affect EHF thresholds first (Fried et al., 1976; Liberman and Kiang, 1978; Wang et al., 2002). Work in humans has also revealed that EHF threshold elevation is associated with poorer cochlear tuning in the standard audiometric range (<8 kHz). Broader cochlear tuning results in spectral smearing and may produce more pronounced masking effects in noisy environments (Besser et al., 2015; Narula and Mason, 1988; Pick and Evans, 1983) as well as greater difficulty in real-world tasks, such as SIN perception (Badri et al., 2011). Thus, EHF thresholds may not only be a harbinger of further damage to come with continued noise exposure - they may also index systemic sub-clinical outer hair cell damage that has already occurred.

Audiometric thresholds for our control and noise-exposed groups were highly similar in the standard range and began to diverge in the EHF range (Fig. 2A). Although these differences did not reach significance, others have reported stark group differences in EHF thresholds for control and noise exposed listeners, particularly at the upper frequency limits of a standard clinical audiometer (~16 kHz). In fact, the most consistent finding of studies on the risks of personal music players is EHF threshold elevation (e.g., Le Prell et al., 2013). Like Liberman et al. (2016), we found that mean EHF thresholds were not predictive of speech perception performance (Fig. 4C); however, it is notable that the three worst SIN performers in our dataset had relatively poor mean EHF thresholds. Moore et al. (2017) have advocated for widespread acquisition of EHF thresholds in clinical and research settings, as it provides opportunities to 1) capture variation in hearing sensitivity that standard audiograms do not and 2) counsel patients on occupational and recreational noise exposure risks.

4.4. Cochlear synaptopathy as indexed by SP and CAP amplitudes

Experiments in a variety of animal species have compellingly demonstrated that titrated noise exposure can cause cochlear synaptopathy while sparing both inner and outer hair cells and hearing sensitivity (Fernandez et al., 2015; Furman et al., 2013; Kujawa and Liberman, 2009; Lin et al., 2011; Lobarinas et al., 2017; Shi et al., 2013; Wang and Ren, 2012; Valero et al., 2017; also see Mulders et al., 2018 for a discussion regarding occult inner hair cell damage from noise exposure). Data linking confirmed cases of

synaptopathy to perceptual deficits, however, are currently limited (Lobarinas et al., 2017). Nevertheless, the suggestion that synaptopathy might underlie SIN deficits in individuals with normal audiograms (Kujawa and Liberman, 2009) has motivated many research groups to search for evidence of synaptopathy and its sequelae in humans.

CAP amplitude measurements have been used most often to assess synaptopathy in humans, although other electrophysiologic measures, such as envelope following responses (e.g., Bharadwaj et al., 2015) and cortical responses to interaural phase shifts (Grose et al., 2017), have also been reported. With some exceptions (Liberman et al., 2016; Stamper and Johnson, 2015a,b) most studies evaluating the relationships between *recreational* noise exposure, CAP reductions, and SIN performance have produced null results (Fulbright et al., 2017; Grose et al., 2017; Grinn et al., 2017; Guest et al., 2017, 2018; Prendergast et al., 2017a, 2018; Spankovich et al., 2018). Interestingly, Grose et al. (2017) found modest evidence of human cochlear synaptopathy in listeners with loud music exposure history; however, this relationship would likely not survive statistical correction for multiple comparisons. In addition, loud music exposure did not impact psychophysical or SIN test results. The current state of the literature suggests that synaptopathy may exist in humans, but its perceptual consequences are perhaps overstated.

Here, we examined one of the largest datasets of young normal hearing adults reported in the literature and found that CAP amplitude does not differ between control and noise exposed groups and is not predictive of SIN performance. These results are consistent with the findings of many other research groups. Others' and our results may indicate that either: 1) synaptopathy is not a source of SIN variability in normal hearing listeners or 2) the extent to which synaptopathy can be noninvasively indexed in humans is limited by the available methodology. For example, inter-subject variability in CAP amplitudes is caused by a variety of factors, such as cochlear length (Don et al., 1994) and head size (Nikiforidis et al., 1993), as well as factors related to electrode impedance and montage configuration. These factors may sum to obscure "true" differences in neural responses between test groups if they exist. A limitation of our recording montage is that the inverting electrode was farther from the cochlear source of the SP and CAP compared to other methods (e.g., tip-trode or TM electrode); however, we were able to identify SP in 81% and CAP in 97% of subjects. Future studies of human cochlear synaptopathy may benefit from the use of a stimulus that generates a larger and less variable CAP. For example, broadband and narrowband chirps generate larger CAPs than clicks and tone bursts by compensating for basilar membrane delays and thus synchronizing auditory nerve fiber discharges (Chertoff et al., 2010; Smith et al., 2017). Further, when measured in the presence of high-pass masking, chirp evoked CAPs have been used to estimate auditory nerve fiber survival in animals with punctate laser-induced neural lesions (Earl, 2015; Earl and Chertoff, 2010, 2012, 2013).

4.5. Neural correlates of SIN perception

The frequency following response (FFR) is a neural potential dominated by rostral brainstem and midbrain phase-locking to envelope and fine structure features of a stimulus, such as speech (Chandrasekaran and Kraus, 2010; Krishnan, 2002; Skoe and Kraus, 2010). The FFR has been used extensively to assess neural encoding of speech in quiet and in noise and to explore relationships between physiologic and perceptual processes. Previous work demonstrates a link between the speech FFR and SIN performance across the lifespan. For example, fundamental frequency encoding correlates with SIN performance in children (Anderson et al., 2010; Thompson et al., 2017), young adults (Song et al., 2011), and older adults (Anderson et al., 2011, 2013). Further, speech FFR timing is slower in older adults (60–67 years) with normal hearing (Anderson et al., 2012), a population that nevertheless exhibits SIN difficulties (cf. Anderson et al., 2010). The tight coupling between the speech FFR and perceptual performance suggests that it may be a fruitful tool for evaluating listeners with SIN deficits and normal audiograms. Some researchers have suggested that, although the FFR does not index auditory nerve health directly, it may reflect "downstream" consequences of cochlear synaptopathy (Bharadwaj et al., 2015). Work is underway to assess whether individuals with a history of noise exposure have more degraded speech FFR features and whether these features are at all related to proposed measures of synaptopathy, such as the CAP.

Financial disclosures

This work was supported by the Med-EL Corporation and The Knowles Hearing Center.

Acknowledgments

The authors would like to thank all study participants and former lab members involved in data collection. In addition, the authors thank the Knowles Hearing Center and MED-EL Corporation for their generous support.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heares.2018.11.008.

References

Anderson, S., Skoe, E., Chandrasekaran, B., Kraus, N., 2010. Neural timing is linked to speech perception in noise. J. Neurosci. 30 (14), 4922–4926.
Anderson, S., Parbery-Clark, A., Yi, H.G., Kraus, N., 2011. A neural basis of speech-in-noise perception in older adults. Ear Hear. 32 (6), 750.
Anderson, S., Parbery-Clark, A., White-Schwoch, T., Kraus, N., 2012. Aging affects neural precision of speech encoding. J. Neurosci. 32 (41), 14156–14164.
Anderson, S., Parbery-Clark, A., White-Schwoch, T., Kraus, N., 2013. Auditory brainstem response to complex sounds predicts self-reported speech-in-noise performance. J. Speech Lang. Hear. Res. 56 (1), 31–43.
Badri, R., Siegel, J.H., Wright, B.A., 2011. Auditory filter shapes and high-frequency hearing in adults who have impaired speech in noise performance despite clinically normal audiograms. J. Acoust. Soc. Am. 129 (2), 852–863.
Besser, J., Festen, J.M., Goverts, S.T., Kramer, S.E., Pichora-Fuller, M.K., 2015. Speech-in-speech listening on the LiSN-S test by older adults with good audiograms depends on cognition and hearing acuity at high frequencies. Ear Hear. 36 (1), 24–41.
Bharadwaj, H.M., Masud, S., Mehraei, G., Verhulst, S., Shinn-Cunningham, B.G., 2015. Individual differences reveal correlates of hidden hearing deficits. J. Neurosci. 35 (5), 2161–2172.
Bramhall, N.F., Konrad-Martin, D., McMillan, G.P., Griest, S.E., 2017. Auditory brainstem response altered in humans with noise exposure despite normal outer hair cell function. Ear Hear. 38 (1), e1.
Carhart, R., Jerger, J., 1959. Preferred method for clinical determination of pure-tone thresholds. J. Speech Hear. Disord. 24 (4).
Carney, L.H., 2018. Supra-threshold hearing and fluctuation profiles: implications for sensorineural and hidden hearing loss. Journal of the Association for Research in Otolaryngology 1–22.
Chandrasekaran, B., Kraus, N., 2010. The scalp-recorded brainstem response to speech: neural origins and plasticity. Psychophysiology 47 (2), 236–246.
Chertoff, M., Lichtenhan, J., Willis, M., 2010. Click-and chirp-evoked human compound action potentials. J. Acoust. Soc. Am. 127 (5), 2992–2996.
Dallos, P., Santos-Sacchi, J., Flock, A., 1982. Intracellular recordings from cochlear outer hair cells. Science 218 (4572), 582–584.
Dobie, R.A., Humes, L.E., 2017. Commentary on the regulatory implications of noise-induced cochlear neuropathy. Int. J. Audiol. 56 (Suppl. 1), 74–78.
Doerfler, L., Stewart, K., 1946. Malingering and psychogenic deafness. Journal of Speech Disorders 11 (3), 181–186.
Don, M., Ponton, C.W., Eggermont, J.J., Masuda, A., 1994. Auditory brainstem response (ABR) peak amplitude variability reflects individual differences in cochlear response times. J. Acoust. Soc. Am. 96 (6), 3476–3491.

Earl, B.R., Chertoff, M.E., 2010. Predicting auditory nerve survival using the compound action potential. Ear Hear. 31 (1), 7–21.
Earl, B.R., Chertoff, M.E., 2012. Mapping auditory nerve firing density using high-level compound action potentials and high- pass noise masking. J. Acoust. Soc. Am. 131 (1), 1–16.
Earl, B.R., Chertoff, M.E., 2013. Amplitude of masked compound action potentials indicates location of laser-induced lesions in the auditory periphery. Association for Research in Otolaryngology Abs 36, 440–441.
Earl, B.R., 2015. Using SiNAPS. Audiology 27 (3), 16.
Eggermont, J.J., 2017. Ups and downs in 75 years of electrocochleography. Front. Syst. Neurosci. 11, 2.
Fernandez, K.A., Jeffers, P.W., Lall, K., Liberman, M.C., Kujawa, S.G., 2015. Aging after noise exposure: acceleration of cochlear synaptopathy in “recovered” ears. J. Neurosci. 35 (19), 7509–7520.
Fried, M.P., Dudek, S.E., Bohne, B.A., 1976. Basal turn cochlear lesions following exposure to low frequency noise. In: Transactions of the American Academy of Ophthalmology and Otolaryngology.
Fulbright, A.N., Le Prell, C.G., Griffiths, S.K., Lobarinas, E., 2017, November. Effects of recreational noise on threshold and suprathreshold measures of auditory function. In: Seminars in Hearing, vol. 38. Thieme Medical Publishers, pp. 298–318. No. 04.
Furman, A.C., Kujawa, S.G., Liberman, M.C., 2013. Noise-induced cochlear neuropathy is selective for fibers with low spontaneous rates. J. Neurophysiol. 110 (3), 577–586.
Grinn, S.K., Wiseman, K.B., Baker, J.A., Le Prell, C.G., 2017. Hidden hearing loss? No effect of common recreational noise exposure on cochlear nerve response amplitude in humans. Front. Neurosci. 11, 465.
Grose, J.H., Buss, E., Hall III, J.W., 2017. Loud music exposure and cochlear synaptopathy in young adults: isolated auditory brainstem response effects but no perceptual consequences. Trends in Hearing 21, 2331216517737417.
Guest, H., Munro, K.J., Prendergast, G., Howe, S., Plack, C.J., 2017. Tinnitus with a normal audiogram: relation to noise exposure but no evidence for cochlear synaptopathy. Hear. Res. 344, 265–274.
Guest, H., Munro, K.J., Prendergast, G., Millman, R.E., Plack, C.J., 2018. Impaired speech perception in noise with a normal audiogram: No evidence for cochlear synaptopathy and no relation to lifetime noise exposure. Hear. Res. 364, 142–151.
Hickox, A.E., Liberman, M.C., 2014. Is noise-induced cochlear neuropathy key to the generation of hyperacusis or tinnitus? J. Neurophysiol. 111, 552–564.
Hickox, A.E., Larsen, E., Heinz, M.G., Shinobu, L., Whitton, J.P., 2017. Translational issues in cochlear synaptopathy. Hear. Res. 349, 164–171.
Higson, J.M., Haggard, M.P., Field, D.L., 1994. Validation of parameters for assessing obscure auditory dysfunction—robustness of determinants of OAD status across samples and test methods. Br. J. Audiol. 28 (1), 27–39.
Hinchcliffe, R., 1992. King-Kopetzky syndrome: an auditory stress disorder? J. Audiol. Med. 1, 89–98.
Hoben, R., Easow, G., Pevzner, S., Parker, M.A., 2017. Outer hair cell and auditory nerve function in speech recognition in quiet and in background noise. Front. Neurosci. 11, 157.
Jerger, J., 2011. Why do people without hearing loss complain of hearing difficulties? J. Am. Acad. Audiol. 22, 490–491.
Johnson, T.A., Cooper, S., Stamper, G.C., Chertoff, M., 2017. Noise Exposure Questionnaire: a tool for quantifying annual noise exposure. J. Am. Acad. Audiol. 28 (1), 14–35.
Killion, M.C., Niquette, P.A., Gudmundsen, G.I., Revit, L.J., Banerjee, S., 2004. Development of a quick speech-in-noise test for measuring signal-to-noise ratio loss in normal-hearing and hearing-impaired listeners. J. Acoust. Soc. Am. 116 (4), 2395–2405.
King, K., Stephens, D., 1992. Auditory and psychological factors in ‘auditory disability with normal hearing’. Scand. Audiol. 21 (2), 109–114.
Knipper, M., Van Dijk, P., Nunes, I., Rüttiger, L., Zimmermann, U., 2013. Advances in the neurobiology of hearing disorders: recent developments regarding the basis of tinnitus and hyperacusis. Prog. Neurobiol. 111, 17–33.
Krishnan, A., 2002. Human frequency-following responses: representation of steady-state synthetic vowels. Hear. Res. 166 (1–2), 192–201.
Kujawa, S.G., Liberman, M.C., 2009. Adding insult to injury: cochlear nerve degeneration after “temporary” noise-induced hearing loss. J. Neurosci. 29 (45), 14077–14085.
Kujawa, S.G., Liberman, M.C., 2015. Synaptopathy in the noise-exposed and aging cochlea: primary neural degeneration in acquired sensorineural hearing loss. Hear. Res. 330, 191–199.
Lapsley Miller, J.A., Marshall, L., Heller, L.M., Hughes, L.M., 2006. Low-level otoacoustic emissions may predict susceptibility to noise-induced hearing loss. J. Acoust. Soc. Am. 120 (1), 280–296.
Le Prell, C.G., Spankovich, C., Lobariñas, E., Griffiths, S.K., 2013. Extended high-frequency thresholds in college students: effects of music player use and other recreational noise. J. Am. Acad. Audiol. 24 (8), 725–739.
Liberman, M.C., Kiang, N.Y., 1978. Acoustic trauma in cats: cochlear pathology and auditory-nerve activity. Acta Otolaryngol. 358 (suppl.).
Liberman, M.C., 2015. Hidden hearing loss. Sci. Am. 313 (2), 48–53.
Liberman, M.C., Epstein, M.J., Cleveland, S.S., Wang, H., Maison, S.F., 2016. Toward a differential diagnosis of hidden hearing loss in humans. PLoS One 11 (9), e0162726.
Lin, H.W., Furman, A.C., Kujawa, S.G., Liberman, M.C., 2011. Primary neural degeneration in the Guinea pig cochlea after reversible noise-induced threshold shift. Journal of the Association for Research in Otolaryngology 12 (5), 605–616.
Lobarinas, E., Spankovich, C., Le Prell, C.G., 2017. Evidence of “hidden hearing loss” following noise exposures that produce robust TTS and ABR wave-I amplitude reductions. Hear. Res. 349, 155–163.
Makary, C.A., Shin, J., Kujawa, S.G., Liberman, M.C., Merchant, S.N., 2011. Age-related primary cochlear neuronal degeneration in human temporal bones. Journal of the Association for Research in Otolaryngology 12 (6), 711–717.
Megerson, S.C., 2010. Development of a Screening Tool for Identifying Young People at Risk for Noise-induced Hearing Loss. Doctoral dissertation. University of Kansas.
Moore, D., Hunter, L., Munro, K., 2017. Benefits of extended high-frequency audiometry for everyone. Hear. J. 70 (3), 50–52.
Mulders, W.H., Chin, I.L., Robertson, D., 2018. Persistent hair cell malfunction contributes to hidden hearing loss. Hear. Res. 361, 45–51.
Murphy, W.J., Le Prell, C.G., 2017. In: Making Sound Waves: Selected Papers from the 2016 Annual Conference of the National Hearing Conservation Association.
Musiek, F.E., Chermak, G.D., Bamiou, D.E., Shinn, J., 2018. CAPD: the most common ‘hidden hearing loss’: central auditory processing disorder—and not cochlear synaptopathy—is the most likely source of difficulty understanding speech in noise (despite normal audiograms). The ASHA Leader 23 (3), 6–9.
Narula, A.A., Mason, S.M., 1988. Selective dysacusis—a preliminary report. J. R. Soc. Med. 81 (6), 338–340.
Nikiforidis, G.C., Koutsojannis, C.M., Varakis, J.N., Goumas, P.D., 1993. Reduced variance in the latency and amplitude of the fifth wave of auditory brain stem response after normalization for head size. Ear Hear. 14 (6), 423–428.
Pienkowski, M., 2017. On the etiology of listening difficulties in noise despite clinically normal audiograms. Ear Hear. 38 (2), 135.
Plack, C.J., Barker, D., Prendergast, G., 2014. Perceptual consequences of “hidden” hearing loss. Trends in Hearing 18, 2331216514550621.
Prendergast, G., Tu, W., Guest, H., Millman, R.E., Kluk, K., Couth, S., Munro, K., Plack, C.J., 2018. Supra-threshold auditory brainstem response amplitudes in humans: test-retest reliability, electrode montage and noise exposure. Hear. Res. 364, 38–47.
Prendergast, G., Guest, H., Hall, D., Kluk, D.K., Leger, A., Hickox, A.E., Plack, C.J., 2016. An investigation of hidden hearing loss in young adults with normal hearing. In: Association for Research in Otolaryngology, 39th Midwinter Meeting Abstracts, p. 202.
Prendergast, G., Millman, R.E., Guest, H., Munro, K.J., Kluk, K., Dewey, R.S., Plack, C.J., 2017. Effects of noise exposure on young adults with normal audiograms II: behavioral measures. Hear. Res. 356, 74–86.
Pryce, H., Metcalfe, C., Hall, A., et al., 2010. Illness perceptions and hearing difficulties in King-Kopetzky syndrome: what determines help seeking? Int. J. Audiol. 49, 473–481.
Pick, G.F., Evans, E.F., 1983. Dissociation between frequency resolution and hearing threshold. In: Hearing—physiological Bases and Psychophysics. Springer, Berlin, Heidelberg, pp. 393–399.
Pryce, H., Wainwright, D., 2008. Help-seeking for medically unexplained hearing difficulties: a qualitative study. Int. J. Ther. Rehabil. 15 (8), 343–349.
Rappaport, J., Phillips, D., Gulliver, J., 1993. Disturbed speech intelligibility in noise despite a normal audiogram: a defect in temporal resolution? J. Otolaryngol. 22, 447–453.
Reeb-Whitaker, C.K., Seixas, N.S., Sheppard, L., Neitzel, R., 2004. Accuracy of task recall for epidemiological exposure assessment to construction noise. Occup. Environ. Med. 61 (2), 135–142.
Roland, P.S., Rosenbloom, J., Yellin, W., Meyerhoff, W.L., 1993. Intrasubject test-retest variability in clinical electrocochleography. Laryngoscope 103 (9), 963–966.
Roland, P.S., Yellin, M.W., Meyerhoff, W.L., Frank, T., 1995. Simultaneous comparison between transtympanic and extratympanic electrocochleography. Am. J. Otol. 16 (4), 444–450.
Russell, I.J., Sellick, P.M., 1978. Intracellular studies of hair cells in the mammalian cochlea. J. Physiol. 284 (1), 261–290.
Saunders, G.H., Haggard, M.P., 1989. The clinical assessment of obscure auditory dysfunction - 1. Auditory and psychological factors. Ear Hear. 10, 200–208.
Schaette, R., McAlpine, D., 2011. Tinnitus with a normal audiogram: physiological evidence for hidden hearing loss and computational model. J. Neurosci. 31 (38), 13452–13457.
Schaette, R., 2014. Tinnitus in men, mice (as well as other rodents), and machines. Hear. Res. 311, 63–71.
Shaheen, L.A., Valero, M.D., Liberman, M.C., 2015. Towards a diagnosis of cochlear neuropathy with envelope following responses. Journal of the Association for Research in Otolaryngology 16 (6), 727–745.
Shi, L., Liu, L., He, T., Guo, X., Yu, Z., Yin, S., Wang, J., 2013. Ribbon synapse plasticity in the cochleae of Guinea pigs after noise-induced silent damage. PLoS One 8 (12), e81566.
Skoe, E., Kraus, N., 2010. Auditory brainstem response to complex sounds: a tutorial. Ear Hear. 31 (3), 302.
Skoe, E., Tufts, J., 2018. Evidence of noise-induced subclinical hearing loss using auditory brainstem responses and objective measures of noise exposure in humans. Hear. Res. 361, 80–91.
Smith, S.B., Lichtenhan, J.T., Cone, B., 2016. Behavioral pure tone threshold shifts caused by tympanic membrane electrodes. Ear Hear. 37 (4), e273.
Smith, S.B., Lichtenhan, J.T., Cone, B.K., 2017. Contralateral inhibition of click-and chirp-evoked human compound action potentials. Front. Neurosci. 11, 189.
Song, J.H., Skoe, E., Banai, K., Kraus, N., 2011. Training to improve hearing speech in noise: biological mechanisms. Cerebr. Cortex 22 (5), 1180–1190.

Spankovich, C., Gonzalez, V.B., Su, D., Bishop, C.E., 2018. Self reported hearing difficulty, tinnitus, and normal audiometric thresholds, the National Health and Nutrition Examination Survey 1999–2002. Hear. Res. 358, 30–36.
Stamper, G.C., Johnson, T.A., 2015a. Auditory function in normal-hearing, noise-exposed human ears. Ear Hear. 36 (2), 172.
Stamper, G.C., Johnson, T.A., 2015b. Letter to the Editor: examination of potential sex influences in Stamper, GC & Johnson, TA (2015). Auditory function in normal-hearing, noise-exposed human ears, Ear Hear, 36, 172-184. Ear Hear. 36 (6), 738.
Thompson, E.C., Carr, K.W., White-Schwoch, T., Otto-Meyer, S., Kraus, N., 2017. Individual differences in speech-in-noise perception parallel neural speech processing and attention in preschoolers. Hear. Res. 344, 148–157.
Tremblay, K.L., Pinto, A., Fischer, M.E., Klein, B.E., Klein, R., Levy, S., Cruickshanks, K.J., 2015. Self-reported hearing difficulties among adults with normal audiograms: the Beaver Dam Offspring Study. Ear Hear. 36 (6), e290.
Valero, M.D., Burton, J.A., Hauser, S.N., Hackett, T.A., Ramachandran, R., Liberman, M.C., 2017. Noise-induced cochlear synaptopathy in rhesus monkeys (Macaca mulatta). Hear. Res. 353, 213–223.
Wang, Y., Hirose, K., Liberman, M.C., 2002. Dynamics of noise-induced cellular injury and repair in the mouse cochlea. Journal of the Association for Research in Otolaryngology 3 (3), 248–268.
Wang, Y., Ren, C., 2012. Effects of repeated “benign” noise exposures in young CBA mice: shedding light on age-related hearing loss. Journal of the Association for Research in Otolaryngology 13 (4), 505–515.
Zhao, F., Stephens, D., 2000. Subcategories of patients with King-Kopetzky syndrome. Br. J. Audiol. 34 (4), 241–256.
Zhao, F., Stephens, D., 2007. A critical review of King-Kopetzky syndrome: hearing difficulties, but normal hearing? Audiol. Med. 5 (2), 119–124.