The variability in potential biomarkers for cochlear synaptopathy after recreational noise exposure

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ABSTRACT

Objectives. Speech-in-noise tests and suprathreshold auditory evoked potentials are promising biomarkers to diagnose cochlear synaptopathy (CS) in humans. This study investigated whether these biomarkers changed after recreational noise exposure. **Design.** The baseline auditory status of 19 normal hearing young adults was analyzed using questionnaires, pure-tone audiometry, speech audiometry and auditory evoked potentials. Nineteen subjects attended a music festival and completed the same tests again at day 1, day 3 and day 5 after the music festival. **Results.** No significant relations were found between lifetime noise-exposure history and the hearing tests. Changes in biomarkers from the first session to the follow-up sessions were non-significant, except for speech audiometry, that showed a significant training effect (performance improvement). **Conclusions.** Despite the individual variability in pre-festival biomarkers, we did not observe changes related to the noise-exposure dose caused by the attended event. This can indicate the absence of noise-exposure-driven cochlear synaptopathy in the study cohort, or reflect that biomarkers were not sensitive enough to detect mild CS. Future research should include a more diverse study cohort, dosimetry and results from test-retest reliability studies to provide more insight into the relationship between recreational noise exposure and cochlear synaptopathy.

KEYWORDS

Cochlear synaptopathy, hidden hearing loss, recreational noise exposure, speech in noise, envelope following response, auditory brainstem response, auditory evoked potentials

INTRODUCTION

Noise exposure in young adults mainly takes place during leisure time activities such as watching movies or plays, visiting nightclubs, attending music festivals or concerts, and listening to personal music players. In this population, concerts and music festivals are the loudest activities (Degeest, Clays, et al., 2017; Keppler, Dhooge, et al., 2015b; Petrescu, 2008; Smith et al., 2000). Sound level measurements during such events confirm that noise levels can exceed 100 dBA (Derebery et al., 2012; Mercier et al., 2003; Ryberg, 2009; Yassi et al., 1993). Despite the potential risk of noise-induced hearing loss (NIHL), the majority of young adults do not wear hearing protection devices (HPDs) when attending concerts (73.8%; (Degeest, Keppler, et al., 2017) or performances at music festivals (86%; (Mercier et al., 2003). After attending these music venues, 36-86% of young adults experience hearing related symptoms (HRS) such as temporary hearing loss, dullness or tinnitus (Keppler, Dhooge, et al., 2015b; Mercier et al., 2003; Smith et al., 2000).

The reduction of hearing sensitivity followingnoise exposure has extensively been investigated in animal and human studies (Bramhall et al., 2019). The incidence of NIHL depends on many factors, amongst others the noise characteristics: the intensity, frequency and duration of the exposure (Clark, 1991). NIHL can cause permanent threshold shifts (PTS) due to mechanical damage of the cochlear hair cells or supporting cells and metabolic changes that cause cell degeneration and cell death (Wang et al., 2002; Yamane et al., 1995). The reduction of hearing sensitivity after noise exposure can also be reversible. Mechanical damage of outer-hair-cell (OHC) stereocilia, reversible changes in supporting cells, or a swelling of auditory-nerve-fiber (ANF) terminals (Wang et al., 2002) can result in a temporary threshold shift (TTS) which recovers within minutes or hours (Emmerich et al., 2002; Trzaskowski et al., 2014) to weeks (Le Prell et al., 2012; Ryan et al., 2016). Research investigating the pathophysiology of TTS in rodents

66 indicates that excessive glutamate release excitotoxicity is the source of ANF terminal swelling
67 following noise exposure (Hakuba et al., 2000; Puel et al., 1998). Recent studies have shown that
68 TTS may not be as benign as previously thought, since it can be accompanied by permanent
69 deficits at the synapse level where type-I ANF terminals connect to the inner hair cells (IHCs).
70 Over the past years, this synapse loss has been named cochlear synaptopathy (CS) and has been
71 linked to noise exposure and aging in mice (Furman et al., 2013; Kujawa & Liberman, 2009;
72 Sergeyenko et al., 2013).

73 A single IHC contains 10 to 30 presynaptic ribbon-like structures surrounded with glutamate
74 vesicles. Each ribbon connects with a postsynaptic glutamate receptor patch at the ANF terminal
75 side (Liberman et al., 1990). ANFs with low and medium spontaneous firing rates have higher
76 thresholds and connect at the modiolar side of the IHC. They can be distinguished from ANFs
77 with high spontaneous firing rates and lower thresholds, that connect to the pillar side of the
78 IHC (Liberman, 1982). This kind of synapse is unique for the cochlea and retina, and allows fast
79 synaptic vesicle fusion with glutamate release, and fast recovery. Besides encoding a wide
80 dynamic range of sound intensities, the synaptic efficiency enables phase-locking to the
81 stimulus, i.e. action potentials follow the temporal precision of the stimulus fine structure and
82 its envelope. The phase-locked spikes are further encoded in the subcortical and cortical
83 pathway and are important for suprathreshold listening tasks, e.g., speech intelligibility and
84 sound localization (Henry et al., 2016; Safieddine et al., 2012).

85 Damage to the IHC synapses due to noise exposure, as seen in CS, can occur immediately after
86 noise exposure and can precede a slow degeneration of spiral ganglion cells (Lin et al., 2011).
87 Synapses that connect to ANFs with low spontaneous rates were shown to be most vulnerable,
88 whereas ANFs with higher spontaneous rates are more robust to noise damage (Furman et al.,

2013). This susceptibility of low-spontaneous-rate ANFs, combined with the distribution pattern wherein multiple ANFs couple to a single IHC, could be the reason why auditory thresholds recover or remain unaffected by permanent synaptic damage. CS could hence be a possible explanation for the pathophysiology of hidden hearing loss, where normal auditory thresholds are accompanied by suprathreshold hearing deficits such as decreased speech intelligibility in noise (Bharadwaj et al., 2014; Smith et al., 2019). Furthermore, it has been argued that this pathology may play a role in the origin of symptoms such as tinnitus or hyperacusis (Liberman & Kujawa, 2017; Schaette & McAlpine, 2011).

Animal studies and computational auditory modeling studies have attempted to define biomarkers for diagnosing CS noninvasively and have shown the potential of auditory evoked potentials (AEPs). More specifically, the auditory brainstem response (ABR) and envelope following response (EFR) are promising tests, given that ABR- and EFR-based derived metrics relate directly to the number of histologically-verified synapse counts in animal models (Kujawa & Liberman, 2009; Möhrle et al., 2016; Parthasarathy & Kujawa, 2018; Shaheen et al., 2015). Suprathreshold ABRs are generated by the synchronous firing of ANFs and brainstem neurons in response to the rapid onset of a brief stimulus (Hall, 2007). In rodents with CS, a reduced amplitude of wave I or reduced wave I/V amplitude ratio was observed, which captures the decreased integrity of the peripheral auditory nerve (Furman et al., 2013; Kujawa & Liberman, 2009, 2015; Lin et al., 2011). The EFR is elicited by continuous, amplitude-modulated signals and the strength of the response to the stimulus envelope modulation frequency reflects temporal coding precision in the subcortical neurons (Bidelman et al., 2015; Purcell et al., 2004). Animals or models with CS showed reduced EFR-strength, which reflects a deficit in temporal coding fidelity of the auditory system (Bharadwaj et al., 2014; Parthasarathy et al., 2018; Shaheen et

al., 2015; Vasilkov & Verhulst, 2019; Verhulst et al., 2018), even when hearing sensitivity recovered or remained normal.

Relating these AEP biomarkers to CS in living humans is far more difficult. Comorbidity with OHC loss and inter-subject variability of AEP measures in humans complicate the use of these methods. Moreover, CS diagnosis through direct synapse counts is not possible in living humans and only few human temporal bone studies have been conducted in relation to CS (Makary et al., 2011; Viana et al., 2015). Therefore, other methods have to be used to evaluate how AEP markers could relate to CS in living humans. Some studies were able to relate ABR and EFR strength to noise-exposure history (Bramhall et al., 2017; Grose et al., 2017; Paul et al., 2017; Skoe & Tufts, 2018), aging (Garrett & Verhulst, 2019; Konrad-Martin et al., 2012; Vasilkov & Verhulst, 2019) or tinnitus (Bramhall et al., 2018; Schaette & McAlpine, 2011), but other studies could not confirm these findings (Fulbright et al., 2017; Guest et al., 2018; Prendergast, Guest, et al., 2017; Spankovich, Le Prell, et al., 2017). Longitudinal studies in humans, where AEP-measurements are used to evaluate auditory-nerve integrity before and after noise exposure, are scarce. One study reported unchanged compound action potential (AP) amplitudes in electrocochleography, the analogue of ABR wave I amplitude, in relation to recreational noise exposure (Grinn et al., 2017).

This study investigates the relationship between recreational noise exposure and markers of CS and OHC damage to understand whether this exposure results in TTS, PTS, CS and/or speech in noise deficits. By adopting a test battery that includes pure tone audiometry (PTA) at conventional and extended high frequencies (EHFs), speech audiometry tests in quiet (SPiQ) and in noise (SPiN) and AEP measurements, we hypothesize that speech intelligibility and AEP measurements may deteriorate due to noise exposure, even if PTA is not affected or recovered

shortly after noise exposure. Multiple speech audiometry conditions, ABR and EFR stimuli were used to study the relation of outcome measures to the individual noise-exposure history (Part I) and to analyze the variability in these measures before and in a period of five days after attending a music festival (Part II).

METHODS

PARTICIPANTS

Young adults aged 18 to 25 years who planned to attend a music festival during the summer of 2019 were recruited for this study. Volunteers completed a recruiting questionnaire, which was used to exclude subjects with known hearing pathologies, history of ear surgery or tinnitus. Study participation consisted of four measurement sessions: one baseline measurement session (session 1) between 1 to 3 days before the start of the festival and 3 follow-up sessions: 1 day, 3 days and 5 days after the end of the music festival. Further on, these follow-up sessions will respectively be referred to as session 2, session 3 and session 4. Subjects were asked not to expose themselves to noise other than that of the festival during the follow-up period and not to use party drugs. Participants were free to use hearing protection.

Twenty subjects participated in the first session, 8 males and 12 females (mean age 21.5 years ± 2.24 SD; standard deviation). Of those subjects, the best ear was chosen as the test ear based on tympanometry and PTA. This was the right ear for 7 subjects (4 males) and the left ear for 13 subjects (4 males). One male subject (right testing ear) dropped out of the study for the last measurement session, resulting in 19 participating subjects for the follow-up analysis in Part II, with mean age of 21.6 years (SD 2.27). At every test session, subjects completed a test battery consisting of questionnaires, PTA, SPiQ and SPiN tests, and AEP measurements. The test protocol

157 had a duration of maximum 3 hours per session (including breaks and information) and tests
158 were conducted in the same order for every subject and at every session.

159 This study was approved by the ethical committee of the Ghent University Hospital (Belgium)
160 and was performed following the statement of ethical principles of the Declaration of Helsinki.
161 All subjects were informed about the testing procedures and signed an informed consent.
162 Subjects received a financial compensation for their participation.

163 QUESTIONNAIRES

164 The questionnaire of the baseline session consisted of four parts and was completed online or
165 during the first session. The first part consisted of sociodemographic questions, questions
166 regarding present HRS, illness, recent drug use and noise exposure. Finally, the general use of
167 hearing protection devices and the type of hearing protection was queried.

168 The second part consisted of questions regarding noise-exposure history based on a Dutch
169 translation of Jokitulppo et al. (2006) by Keppler, Dhooge, et al. (2015b). A list of relevant,
170 noisy recreational activities was presented. First, subjects estimated the frequency of noise
171 exposure caused by each activity and the duration of each exposure in hours. Second, the
172 exposure level (Laeq) was estimated using a relative scale based on communication effort,
173 ranging from 60 dBA (sound level of a normal conversation) to 100 dBA (sound level which
174 makes communication impossible). The use of HPD was asked and converted in the equation
175 as a Laeq-reduction of 10 dB. Finally, for each activity, the total number of years that this
176 activity was present in their lives on the one hand, and for the last three years on the other,
177 was asked. Cumulative lifetime-noise-exposure history (Laeq,life) was calculated using the
178 formula of Jokitulppo et al. (2006). An analogous calculation was made for recent noise-

179 exposure history (Laeq,recent), which represents cumulative noise exposure over the past
180 three years.

181 The third and fourth part respectively consisted of the Youth Attitude to Noise Scale (YANS),
182 (Widen et al., 2006) and Beliefs about Hearing Protection and Hearing Loss (BAHPHL),
183 (Svensson et al., 2004) as modified by Keppler et al. (Keppler, Dhooge, Degeest, et al., 2015;
184 Keppler, Dhooge, et al., 2015a; Keppler & Dhooge, 2010). Both questionnaires use a five-
185 degree Likert Scale ranging from "totally disagree" to "totally agree" to determine the
186 attitudes and beliefs towards noise, hearing protection and hearing loss. A higher score
187 reflects more positive attitudes towards noise, less concerns about hearing loss and less belief
188 of benefit of HPDs.

189 At sessions 2, 3 and 4 a questionnaire that evaluates HRS, recent drug and alcohol
190 consumption, and between-session noise exposure was completed. The first follow-up session
191 contained additional questions regarding the festival duration and noise exposure. For each
192 level of communication effort, the subject was asked to estimate a percentage of exposure
193 time and whether HPDs were used in this listening situation to determine the festival noise
194 exposure (Laeq, festival) based on a 40-hour-week noise exposure calculation (Jokitulppo et
195 al., 2006; Keppler, Dhooge, et al., 2015b).

196 OTOSCOPY AND TYMPANOMETRY

197 Otoscopic inspection of the ear canal and tympanic membrane was performed at the baseline
198 session. All test ears had normal otoscopic results.

199 Middle-ear admittance was bilaterally measured at the baseline session, using a GSI TympStar
200 (Grason-Stadler) tympanometer with a 226 Hz, 85 dB sound pressure level (SPL) probe tone.
201 All test ear tympanograms had an ear canal volume between 0.6 and 2.0 mmho, a static

acoustic admittance level within a range of 0.3 to 1.7 mmho and a middle-ear pressure between -100 and 100 daPa, and were defined as normal type-A tympanograms.

PURE-TONE AUDIOMETRY

PTA thresholds were measured while subjects were seated in a double-walled sound-attenuating measurement booth. At the baseline session, both ears were tested to select the test ear. Air-conduction thresholds were measured at conventional octave frequencies 0.125, 0.250, 0.500, 1, 2, 4 and 8 kHz and half-octave frequencies 3 and 6 kHz, and at EHFs of 10, 12.5, 14 and 16 kHz using the modified Hughson-Westlake procedure. An Equinox Interacoustics audiometer was used and stimuli were transmitted using Interacoustics TDH-39 headphones and Sennheiser HAD-200 headphones for conventional frequencies and EHF, respectively. If no threshold could be obtained at the maximum output level, the threshold was considered as this maximum stimulus level for data analysis. The ear with better thresholds on conventional frequencies was chosen as a test ear, provided that these thresholds were all 20 dB HL or better and that this ear had a normal otoscopic evaluation and a type A tympanogram. Since air-conduction thresholds on conventional frequencies never exceeded 20 dB HL and type-A tympanograms showed normal middle-ear admittance in all test ears, bone conduction measurements were not performed.

SPEECH INTELLIGIBILITY IN QUIET AND IN NOISE

At each session, SPiQ and SPiN tests were performed at the test ear using the Flemish Matrix sentence test (Luts et al., 2014) in Apex 3 software (Francart et al., 2008). Sentences were presented in a relatively quiet room using a laptop connected to a Fireface UCX soundcard (RME) and HDA-300 (Sennheiser) headphones.

At each session, five randomly chosen experimental test lists were presented in five differently filtered conditions, presented in a random order. By filtering speech stimuli, different auditory mechanisms can be targeted. Low-pass (LP) filtered speech understanding is thought to mainly rely on temporal fine-structure cues, whereas high-pass (HP) filtered speech would mostly rely on envelope coding of the auditory system (Garrett et al., 2020). Therefore, the five conditions consisted of two conditions in quiet, where speech was filtered with a zero-phase 256th-order FIR LP filter or with a zero-phase 1024th-order FIR HP filter with cutoff values of 1500 and 1650 Hz, respectively. Three conditions were presented in a speech-shaped noise with a fixed level of 70 dB SPL: a broadband condition (BB) where no filtering was applied, a LP and a HP condition, where speech as well as noise signals were filtered using the same filter cutoff values as for LP and HP condition in quiet, respectively. Due to the filtering, the SPL levels of 0 dB SNR LP and HP conditions were 70 and 53 dB SPL, respectively. During the first session, two training lists were presented in the BB-in-noise condition to minimize the known learning effect of this test (Kollmeier et al., 2015).

For all test lists, subjects were asked to repeat the five-word sentences in a closed, forced-choice setting (10 options for each word were given). Speech was presented in an adaptive procedure using a staircase paradigm to determine the speech-reception threshold (SRT) with a minimal step size of 0.1 dB. The mean signal level or mean SNR of the six last reversals was used as the SRT for the SPiQ and SPiN-tests, respectively. Lower SRT-values reflect better speech intelligibility.

AEP MEASUREMENTS

At each session, AEP measurements were performed with the Universal Smart Box (Intelligent Hearing Systems) using SEPCAM software. Subjects were seated in a comfortable armchair in

247 a quiet room and were watching a muted video during the recordings. The skin preparation
248 was performed using NuPrep gel and Ambu Neuroline snap electrodes were placed on vertex,
249 nasion and bilateral earlobes. The electrode impedances never exceeded 3 kΩ. For one
250 subject, a mastoid electrode configuration was used due to an immovable earring. All stimuli
251 were presented using ER-2 probes (Etymotic Research). Both ears were covered with earmuffs
252 to minimize noise interaction and all inessential electrical devices were turned off during the
253 measurements. Subjects were instructed to lean their head back and to relax as much as
254 possible.

255 ABRs were collected using 5000 alternating polarity sweeps of six stimuli types: broadband
256 80-µs click stimuli and narrowband 500-ms toneburst (TB) stimuli. Slow click rates of 11 Hz as
257 well as fast click rates of 120 Hz were both presented at levels of 80 dB and 100 dB peak
258 equivalent SPL (peSPL). Faster click rates cause minimal neural recovery time and have earlier
259 been proposed for neurodiagnostic purposes (Valderrama et al., 2012). TBs with center
260 frequencies of 1 and 4 kHz, which provide more frequency specific information, were
261 presented at 70 dB SPL at a rate of 20 Hz. The raw signals were stored and processed offline
262 in Matlab R2018b using the custom-made "sepcam2mat" function. Recorded ABR traces were
263 bandpass filtered between 100 and 1500 Hz with an 800th order FIR filter "filtfilt" Matlab
264 function to remove the filter delay. Twenty millisecond epochs were extracted relative to the
265 stimulus onset and baseline correction was applied by subtracting the mean-value of each
266 epoch. Two hundred epochs, equal number of each polarity, with the highest peak-to-trough
267 values were rejected and the remaining 4800 epochs were averaged (Keshishzadeh, Garrett,
268 & Verhulst, 2020). Mean ABR level series, starting from 100 dB-peSPL and down, were plotted
269 to visually identify the peaks based on the mean latency intervals by Table 8-1 in Picton (2011).
270 Accordingly, ABR wave I, III and V peaks and latencies were identified manually and were

confirmed by visual inspection of a second audiologist. **Figure 1** shows exemplar ABRs to 80 and 100 dB peSPL, 11-Hz clicks. Identified peaks are specified with O (80 dB peSPL ABR) and X (100 dB peSPL ABR). The wave I/V amplitude ratio was defined as the ratio of peak-to-baseline amplitudes of wave I and wave V, respectively.

EFRs were evoked using 1000 sweeps of two stimulus types. Both stimuli consisted of a 4-kHz amplitude-modulated tone and are distinguished by their modulation waveform. Specifically, sinusoidal amplitude modulation (SAM) was applied in the first stimulus and rectangular amplitude modulation (RAM) in the second (25% duty cycle). The latter was adopted from Vasilkov et al. (2021), as this stimulus was found to yield strong EFRs which are sensitive to detect CS in auditory model simulations. Both stimuli had a modulation frequency of 110Hz, a modulation depth of 100%, and were presented at 70dB SPL. Stimuli had a duration of 500 ms and were presented at a rate of 2 Hz.

The EFR processing was performed offline in Matlab R2018b. First, recorded EFRs were bandpass filtered using the same bandpass FIR filter as applied to ABRs, but with low and high cutoff frequencies of 50 Hz and 5000 Hz, respectively. Then, 400-ms epochs were extracted from the 100 to 500-ms time-interval, relative to the stimulus onset and 20 epochs with the highest peak-to-trough values were rejected. The remaining 800 epochs were averaged and the corresponding magnitude spectrum was constructed using the fast Fourier Transform (FFT). Additionally, a bootstrapping approach was adopted in the frequency domain to estimate the noise-floor and variability of the EFR. Exemplar SAM and RAM EFR spectra and corresponding noise-floors are shown in **Figure 2**A and C, respectively. For detailed explanation of the bootstrapping procedure see Keshishzadeh, Garrett and Verhulst (2020). EFR strength was defined as the summation of the signal-to-noise spectral magnitude at

294 fundamental frequency and the following three harmonics, i.e. 110, 220, 330 and 440 Hz, if
295 they were above the noise-floor (Vasilkov et al., 2021). Noise-floor corrected SAM and RAM
296 EFRs are shown in **Figure 2**B and D. Arrows represent the EFR magnitudes at modulation
297 frequency (110 Hz) and existent spectral peaks at corresponding harmonics.

298 DATA ANALYSIS

299 Statistical data analysis was performed in two stages using IBM SPSS Statistics 25. Descriptive
300 parameters were calculated and tests of normality – i.e. Shapiro-Wilk test, histograms, Q-Q
301 plots, and box and whisker plots - were applied.

302 First, the baseline session was analyzed. Hearing-test results of audiometry, speech
303 audiometry SRT, EFR strength, ABR-amplitudes and -latencies of waves I, III and V and I/V
304 amplitude ratio were analyzed in relation to lifetime noise-exposure history. Although
305 Laeq,life was normally distributed as defined by descriptive parameters and the Shapiro-Wilk
306 test, Spearman's ranked-order correlation coefficients (r_s) were reported as outliers and/or
307 not-normally distributed test parameters were present in the data for one or more
308 audiometric frequencies on the one hand, and for one or more test conditions of speech
309 audiometry, EFR or ABR on the other. Two-tailed significance level was reached for
310 correlations when $p<0.05$, suggesting a monotonic and significant relationship between
311 Laeq,life and the considered hearing-test parameter.

312 Second, intersession variability was analyzed. As there were outliers and the data was not
313 normally distributed for some sessions or test conditions, the assumptions for using repeated
314 measures analysis of variance (ANOVA) were not met and a non-parametric related-samples
315 Friedman's two-way ANOVA by ranks was conducted to determine statistically significant
316 changes in all hearing test parameters over sessions. If significance level ($p<0.05$) was reached,

post-hoc analysis of least-square means was performed for intersession differences between session 1 on the one hand and session 2, 3 and 4 on the other. The two-tailed significance level was reached if $p<0.017$ when applying a Bonferroni correction for multiple comparisons.

For all testing results, the shift from the first to the second session was calculated. Non-parametric Man-Whitney-U tests were conducted to compare those shifts between groups of subjects that reported HRS after the musical event and those who did not. Significance level was reached when $p<0.05$, suggesting a significant difference between those two groups.

RESULTS PART I

QUESTIONNAIRES

Frequencies of Laeq,life and Laeq,recent are shown in **Figure 3**. Mean Laeq,life was 70.17 dBA (SD 7.83, range 56.46 – 90.16) when corrected for HPD-use. Attending a discotheque or dance party were reported as the loudest activity (m 79.5 dBA, SD 8.87, range 60.0 – 90.0), followed by attending a concert or a festival (m 74.2 dBA, SD 5.07, range 70.0 – 80.0). All but one subject had attended such events before they participated in this study. Noise-exposure history for the last three years, as defined by Laeq,recent, had a mean of 66.29 dBA (SD 7.92, range 54.41 – 84.94). Pearson correlation showed a strong positive, statistically significant correlation between Laeq,recent and Laeq,life ($r(20)=0.973$, $p<0.001$). Mean total YANS-score was 2.81 (SD 0.67, range 1.26 – 4.05) and mean total BAHPHL-score was 2.42 (SD 0.47, range 1.50 – 3.38). Twelve subjects (60%) reported commonly wearing HPDs when exposed to loud noises, five subjects (25%) used customized ear plugs.

BASELINE HEARING STATUS

Distribution of hearing thresholds for conventional frequencies and EHF of the test ear are shown in **Figure 4**. The mean pure-tone average of 3, 4, 6 and 8 kHz (PTA3-8kHz) was 4.88dB

HL (SD 2.75, median 5.00, range -1.25 – 8.75) and the mean pure-tone average of 10, 12.5, 14 and 16 kHz (PTA10-16kHz) was 5.94 (SD 9.59, median 2.50, range -10.00 – 25.00). Neither PTA3-8kHz nor PTA10-16kHz significantly correlated to lifetime noise-exposure history (p>0.05). Spearman's correlation coefficients ranged from -0.28 to 0.09.

BASELINE SPEECH AUDIOMETRY

Figure 5A and B represent the SRT-values of SPiQ and SPiN respectively. No monotonic relation was found between SPiQ and Laeq,life, as Spearman's correlation coefficients were 0.24 (p=0.30) for the LP filtered speech and -0.20 (p=0.41) for the HP filtered speech, and both were statistically insignificant. Spearman's correlations of SPiN to Laeq,life showed weak positive correlation coefficients, ranging from 0.02 to 0.44 with the highest correlation coefficient for the LP condition. However, none of the correlation coefficients were statistically significant (p>0.05).

BASELINE AEP-MEASUREMENTS

ABR latencies and amplitudes of waves I, III and V in the baseline session are given in Table 1. As expected from literature, latencies of all waves increased with lowering the stimulus level (i.e. 100dB peSPL vs 80dB peSPL), faster stimulus presentation (i.e. 120 Hz vs 11 Hz) or narrower stimulus-spectrum (i.e. TB vs click stimuli; (Picton, 2011)). A statistically significant relation with Laeq,life was found for the ABR wave V latency using a 11Hz 100dB click stimulus (rs=0.66, p=0.002). No other significant relations of ABR latency with Laeq,life were found. Spearman's correlation coefficients ranged from 0.02 (p=0.92) to 0.20 (p=0.40) for wave I, from 0.01 (p=0.99) to 0.37 (p=0.11) for wave III and from 0.02 (p=0.93) to 0.37 (p=0.11) for wave V latencies.

Second, wave I, III and V ABR amplitudes showed no statistically significant monotonic relation to Laeq,life. For wave I, Spearman's correlation coefficients of the different stimulus types ranged from -0.16 (p=0.51) to 0.14 (p=0.56), for wave III from -0.34 (p=0.15) to 0.27 (p=0.25) and for wave V from -0.25 (p=0.29) to 0.003 (p=0.99).

Finally, no significant correlation was found between the ABR I/V amplitude ratio and Laeq,life in all conditions. Spearman's correlation coefficients ranged from -0.41 (p=0.87) to 0.32 (p=0.18) for the various ABR stimuli.

EFR strength in relation to Laeq,life is shown in **Figure 6**. For all but one subject, higher EFR-strength was found using the RAM (mean 0.085, SD 0.029, median 0.088, range 0.007 – 0.136) compared to the SAM-stimulus (mean 0.030, SD 0.010, median 0.028, range 0.012 – 0.056), as was predicted by auditory models (Vasilkov et al., 2021). A negative, but weak and statistically non-significant Spearman's correlation coefficient was found in relation to Laeq,life for SAM-stimulus (r_s=-0.34, p=0.15) as well as for RAM-stimulus (r_s=-0.17, p=0.49).

RESULTS PART II

QUESTIONNAIRE

The mean duration of the music festival attendance was 2.68 days (SD 1.29, range 1 - 5), where subjects self-reported to be exposed to music 8.9 hours a day (SD = 2.39, range 5 - 12). Estimation of noise-exposure levels during the festival, HPD-attenuation taken into account, resulted in an average Laeq,w,festival of 76.2 dBA (SD 7.82, range 59.35 – 88.19).

Thirteen subjects (68.4%) reported to have worn HPDs during the music festivals and only 7 participants (36.8%) had worn them 50% of the time during noise exposure. Eight subjects (42.1%) experienced HRS after the noise exposure: six subjects (31.6%) experienced dullness after the music event, 2 persons (10.5%) experienced a subjective hearing loss and 3 persons

386 (15.8%) reported tinnitus. Dullness and tinnitus were experienced both unilaterally or
387 bilaterally and disappeared after a maximum of 24 hours after the noise exposure, whereas a
388 subjective hearing loss was experienced in both ears and disappeared after 30 minutes. Only
389 one of the test subjects that had worn HPDs more than 50% of the noise exposure time
390 reported HRS. Baseline testing results of PTA, SPiQ and SPiN, and EFR did not differ
391 significantly between subjects who reported HRS after the event, and subjects who did not.
392 ABR results only showed between-group differences in the rate of 120 Hz, 100 dB click
393 condition only. For this condition, the group with HRS had a significantly (p=0.01) smaller P3
394 latency (m 5.30 ms, range 4.60 – 5.50) than the group without HRS (m 5.70 ms, range 5.20 –
395 6.10). Second, wave-I amplitudes were significantly (p=0.005) higher in the group with HRS (m
396 0.13 µV, range 0.02 – 0.22) than the group without (m 0.06 µV, range 0.03 – 0.09). Finally, a
397 significantly higher I/V amplitude ratio was found in the HRS group (m 0.40, range 0.27 – 1.55)
398 compared to the non-HRS group (m 0.19, range 0.09 – 0.49).

399 LONGITUDINAL ANALYSIS HEARING STATUS

400 In the second session, i.e. one day after noise exposure, a TTS was only found in one subject
401 (0.05%). Specifically, this person had an average threshold elevation of 10 dB or more on 2, 3
402 and 4 kHz, as described by Occupational Safety and Health Administration standards (OSHA,
403 1974). For this subject, an average elevation of 11.7 dB was found in session 2 compared to
404 session 1, which recovered to values below 10 dB in sessions 3 and 4. A residual shift of 6.7dB
405 from session 1 was present in the fourth session. This subject experienced bilaterally tinnitus
406 (24h) but reported no subjective hearing loss nor dullness. All other subjects had an average
407 threshold shift from session 1 to session 2 at 2, 3 and 4 kHz ranging from -3.3 to 5.0 dB. At
408 single conventional audiogram frequencies, a threshold elevation of 10 dB or more was found
409 in eight subjects (42.1%) during the second session. At extended high frequencies, threshold

shifts of 10 dB or higher were found in nine subjects (47.3%) between session 1 and session 2. All frequencies included, a threshold shift of 10 dB or more was found in 15 subjects (78.9%).

Pure tone audiometry and χ^2 results are displayed in Table 2. Significant intersession differences were found for auditory thresholds of 500 Hz (p=0.033) and 10 kHz (p=0.005). However, post-hoc analysis with Bonferroni adjustment showed no significant threshold shift from the first session to a follow-up session (p>0.017). PTAs revealed statistically insignificant between-session differences (p>0.05). Even though subjects who experienced HRS showed a median positive shift, i.e. higher values, in PTA3-8kHz (3.13 dB HL, range -2.50 – 10.00) and PTA10-16kHz (3. 75 dB HL, range -3.75 – 6.25) in the second session and subjects without HRS did not (respectively 0.00 dB HL, range -3.75 – 3.75 and 0.00 dB HL, range -6.25 – 11.2), between-group differences were not significant (p=0.18 and 0.44, respectively).

LONGITUDINAL ANALYSIS SPEECH AUDIOMETRY

Figure 7A displays the results of the SPiQ test. SPiQ showed slightly increased SRT-values from session 1 to session 2 for both LP- and HP-filtered speech conditions. Friedman's ANOVA showed statistically significant intersession differences only for the HP filtered condition ($\chi^2(3)=29.46$, $p<0.001$). Although post-hoc analysis with Bonferroni-correction showed statistically insignificant changes between session 1 and session 2, a significant improvement (p=0.008) in speech intelligibility from session 1 (med =22.67, IQR 8.04) to session 4 (med=20.78, IQR 5.28) was found.

The same trend of improved speech intelligibility over sessions was found for SPiN, as shown in **Figure 7**B. Friedman's ANOVA showed statistically significant intersession differences for the BB as well as the LP condition ($\chi^2(3)=29.72$, $p<0.001$) and the HP condition ($\chi^2(3)=27.97$, $p<0.001$). Post-hoc pairwise comparisons with Bonferroni-correction showed a significant

improvement of SNR from session 1 to 4 for all conditions (BB $p=0.002$, LP and HP $p<0.001$) and a significant improvement from session 1 to 3 for the LP condition ($p=0.006$). However, no statistical significant differences between session 1 and session 2 were found for all test conditions.

Between-group comparisons of persons with or without HRS, did not show significant differences in SRT-shift from session1 to session 2 for both SPiQ (p range 0.11 – 0.41) and SPiN (p range 0.06 – 0.77).

LONGITUDINAL ANALYSIS AEP-MEASUREMENTS

Table 3 represents all results and χ^2-values of AEP-measurements. ABR-latencies showed statistically insignificant changes between sessions for any wave or stimulus type. ABR wave III showed significant intersession differences only for the 11-Hz 100-dB peSPL stimulus ($\chi^2(3)=7.93$, $p=0.48$) and wave V changed significantly over time for the 11-Hz 80-dB peSPL stimulus ($\chi^2(3)=12.92$, $p=0.005$), but post-hoc comparisons with Bonferroni correction showed no significant differences between the first session and sessions 2, 3 or 4 ($p>0.017$). Amplitude ratios I/V did not reveal any significant intersession changes for the considered stimulus types ($p>0.05$).

EFR strength of RAM stimuli was consistently higher than that of SAM-stimuli. Even though median EFR strength showed a slight reduction from the first to the second session for both SAM and RAM stimuli, EFR strength did not significantly change across session ($p>0.05$).

ABR-shifts from session 1 to session 2, compared between groups who did or did not experience HRS, respectively, can be found in Table 4. Shifts in ABR latencies, amplitudes and I/V amplitude showed some diffuse significant group differences for certain stimuli, e.g. ABR wave I latency shift for 11-Hz 80-dB click stimulus ($p=0.02$), wave-III latency ($p=0.04$), ABR

wave-I (p=0.04) and wave-V (p=0.02) amplitude shift for TB 4-kHz stimulus, and I/V-amplitude ratio for the 120-Hz 100-dB stimulus (p=0.03). However, no consistent trends across different stimulus types were observed.

EFR-strength showed a negative shift, i.e. an EFR-strength reduction from session 1 to session 2 for both stimulus types within the group that reported HRS (SAM: med = -0.004 range = -0.03 - 0.02; RAM: med = -0.007; range = -0.06 – 0.05). In the group without HRS, a negative shift was only found with the SAM stimulus (med = -0.006; range = -0.01 – 0.1), but not for the RAM stimulus (med = 0.008; range = -0.06 – 0.03). No significant group differences were found between groups with or without HRS in EFR-strength-shifts, neither for SAM (U=49.00, p=0.71) nor for RAM-stimuli (U=52.00, p=0.88).

DISCUSSION

PART I

The first aim of this study consisted of a baseline evaluation of hearing status in relation to the noise-exposure history of 19 young, normal-hearing adults. Our inclusion criteria, specifically normal auditory thresholds and the absence of HRS, eliminated ears with OHC-dysfunction as much as possible.

As other studies failed to find a relation between young adults' hearing thresholds or distortion product otoacoustic emissions (DPOAEs) and their lifetime noise-exposure history (Degeest, Clays, et al., 2017; Keppler, Dhooge, et al., 2015b; Wei et al., 2017; Williams et al., 2015), no substantial OHC-loss due to noise exposure was expected in this population. Indeed, PTA10-16kHz was never worse than 25 dB HL and showed no significant correlation with lifetime noise-exposure history, indicating relatively intact functioning of the OHCs. The

assumption of a normal functioning cochlear amplifier facilitates interpretation of the other hearing test results in the context of hidden hearing loss or CS.

CS has been thought to influence suprathreshold hearing function and to affect speech understanding, especially in difficult listening situations (Kujawa & Liberman, 2009; Lin et al., 2011). Therefore, it could be hypothesized that SPiQ- or SPiN-metrics would relate to lifetime noise-exposure history if CS was present. However, no relation was found in this study population. This supports the results of the majority of studies investigating speech intelligibility in relation to noise-exposure history of normal-hearing persons, where no relation was found either (Couth et al., 2020; Fulbright et al., 2017; Grinn et al., 2017; Guest et al., 2018; Prendergast, Millman, et al., 2017; Yeend et al., 2017). In contrast, some studies have reported a link between noise-exposure history and speech intelligibility (Hope et al., 2013; Kumar et al., 2012; Liberman et al., 2016). The inclusion of subjects that were regularly exposed to occupational noise or a music education could have contributed to a higher noise-exposure dose in these latter studies and increased the risk of CS in the tested population. However, OHC damage could not be ruled out in all these studies since, respectively, audiometric thresholds were only measured up to 4kHz (Hope et al., 2013), or speech intelligibility deterioration was accompanied with audiometric loss in EHFs (Liberman et al., 2016). A recent study by Prendergast et al. (2019) found a link between noise-exposure history and speech intelligibility albeit showing a better performance in subjects with higher lifetime noise-exposure history at levels above 85 dBA. As many of their highly-exposed subjects worked in the music industry, listening strategies and attention training on complex soundscapes are proposed by the authors to explain this difference.

The baseline AEP-measurements in the present study did not show significant correlations to lifetime noise-exposure history. First, ABR wave-I, -III and -V amplitudes and the ABR I/V amplitude ratio were not significantly correlated to noise-exposure history. These observations corroborate results from a number of human studies that did not show a clear link between ABR wave-I amplitudes and recreational noise exposure (Couth et al., 2020; Fulbright et al., 2017; Grinn et al., 2017; Liberman et al., 2016; Prendergast et al., 2018; Ridley et al., 2018; Skoe & Tufts, 2018; Spankovich, Le Prell, et al., 2017). However, two studies performed by Bramhall et al. (2017) and Stamper and Johnson (2015a) revealed significant wave-I amplitude reductions in subjects with higher noise-exposure history, although for the latter study this was only true for female subjects (Stamper & Johnson, 2015b). Furthermore, Valderrama et al. (2018) reported reduced wave-I amplitudes in relation to recreational and occupational noise exposure, but this relation was not significant after removal of outliers. Amplitudes of wave III have less commonly been analyzed and have not shown significant relations to noise exposure (Ridley et al., 2018; Skoe & Tufts, 2018). Furthermore, wave-V amplitudes did not show a relation to recreational noise exposure in literature (Prendergast et al., 2018; Ridley et al., 2018; Skoe & Tufts, 2018; Spankovich, Bishop, et al., 2017) and neither did I/V amplitude ratios (Prendergast, Guest, et al., 2017; Spankovich, Bishop, et al., 2017). One study did report a reduction in I/V ratios in subjects that frequently attended music concerts (Grose et al., 2019). On the contrary, a recent study by Couth et al. (2020) showed higher I/V-amplitude ratios in musicians compared to non-musicians. Even though noise-exposure history was similar between both groups in that study and noise-exposure history did not show a significant relation to I/V amplitude, the highly-exposed subjects showed OHC-dysfunction.

523 Second, regarding ABR latencies, no relation to noise-exposure history was found in the
524 present study. This is in line with several studies, where ABR wave-I latencies (Couth et al.,
525 2020; Spankovich, Bishop, et al., 2017) or wave-V latencies (Spankovich, Bishop, et al., 2017)
526 did not relate to recreational noise exposure. On the contrary, Skoe and Tufts (2018) did find
527 a delay in waves I, III and V for subjects with a higher noise-exposure based on one-week
528 dosimetry measurements. The more highly-exposed subjects in this study were mostly
529 students who participated in music ensembles on campus. A delay in wave V latency with
530 increasing noise exposure was also found by Couth et al. (2020), but only for males, and by
531 Prendergast, Guest, et al. (2017) where significance disappeared after correcting for age.

532 Finally, EFR strength did not correlate to lifetime noise-exposure history in the present study.
533 This is in line with other studies in normal-hearing subjects (Grose et al., 2017) and in normal-
534 hearing subjects with tinnitus (Guest et al., 2017). Prendergast, Guest, et al. (2017) found a
535 significant negative correlation between EFR strength and noise exposure. However, this was
536 only true for males and after controlling for age, this correlation was no longer significant. A
537 study by the same lab could not find noise exposure as a predictor of EFR- strength
538 (Prendergast et al., 2019). In contrast, animal studies (Möhrle et al., 2016; Parthasarathy and
539 Kujawa, 2018; Shaheen et al., 2015) and those that used computational modelling of the
540 auditory periphery or human data have shown the possible value of EFRs in diagnosing CS
541 (Bharadwaj et al., 2015; Garrett & Verhulst, 2019; Keshishzadeh, Garrett, Vasilkov, et al., 2020;
542 Paul et al., 2017; Vasilkov et al., 2021).

543 As described above, studies trying to demonstrate the presence of CS in humans by relating
544 possible biomarkers to noise-exposure history, e.g. SPiN, ABR-amplitudes or amplitude ratios,
545 ABR-latencies or EFR strength, report contradictory results (see Bramhall et al., 2019 for

review). The disagreement between those studies can be caused by multiple reasons. First, study population characteristics such as age vary across studies. Age-related CS could be dominant over noise-induced CS and therefore hiding the effects of noise-exposure history. The use of different age ranges between studies implies that in some study cohorts mostly occupational noise exposure was studied (Hope et al., 2013; Kumar et al., 2012; Prendergast et al., 2019; Valderrama et al., 2018), whereas other studies focused on recreational noise exposure. Second, methodological differences could lead to different conclusions. Based on (the absence of) EHF audiometry- or OAE-results, OHC-deficits cannot be ruled out in some study cohorts (Liberman et al., 2016). Furthermore, methods to calculate or measure noise-exposure history differ between studies. As objective measurements of noise exposure such as dosimetry is hard to be collected during a lifetime period, subjective and retrospective questionnaires or interviews are used to estimate noise-exposure history. Beach et al. (2012) stated that realistic loudness estimations can be made through/via questionnaires about recent noise exposures when compared to dosimetry metrics. However, questionnaires mostly do not take into account frequency components of noise or acoustic traumas caused by impulse noises. Other factors such as blood type (Doğru et al., 2003), nutrition (Kopke et al., 2005), fitness (Kolkhorst et al., 1998), smoking (Ferrite & Santana, 2005; Pouryaghoub et al., 2007), alcohol use (Kraaijenga et al., 2018; Upile et al., 2007) and drug use (Church et al., 2013) have been suggested to influence susceptibility to NIHL, but have not been taken into account in studies.

PART II

The second part of this study evaluated longitudinal biomarker changes before and after attending a music festival.

569 Eight subjects (42.1%) experienced HRS after the music festival, comparable to Mercier et al.
570 (2003), where 36% experienced HRS after attending a music festival. It has to be noted that
571 the majority of subjects wore HPDs, at least for some time during the concerts. However, of
572 those subjects who reported using HPDs, only a few wore them consistently. Only one of the
573 subjects who wore HPDs over 50% of the time during noise exposure experienced HRS, which
574 is in line with findings that HPDs reduce HRS (Kraaijenga et al., 2018) or reduce TTS (Ramakers
575 et al., 2016). Baseline testing results of subjects that reported HRS after the event, did not
576 significantly differ from those who did not, suggesting that cross-sectional use of the present
577 test battery does not predict the risk of developing symptoms such as tinnitus after noise
578 exposure.

579 A TTS as defined by OSHA-standards (OSHA, 1974) was only found in one subject one day after
580 the music festival. No significant group changes in auditory thresholds were found for any of
581 the tested frequencies. The difficulty of detecting a TTS in non-laboratory situations such as
582 attending concerts or nightclubs was earlier described (Le Prell et al., 2012). The experienced
583 noise-exposure is known to vary considerably between subjects attending such events, as
584 noise intensity levels vary depending on distance relative to the stage and the music genre
585 (Opperman et al., 2006). Furthermore, it is difficult to distinguish a small TTS from test-retest
586 variability (Le Prell et al., 2012; Schlauch & Carney, 2012), which is amongst others dependent
587 on participant experience and motivation (Schlauch & Carney, 2012). The observations in the
588 above studies contrast findings from other studies that did not observe a TTS in the majority
589 of subjects after attending concerts, festivals or music venues (Emmerich et al., 2002; Le Prell,
590 2019; Opperman et al., 2006; Ramakers et al., 2016). These studies evaluated hearing
591 thresholds immediately after noise exposure, whereas the greatest auditory-threshold
592 recovery was found to occur within two-to-four hours after the noise exposure (Emmerich et

593 al., 2002; Grinn et al., 2017; Le Prell et al., 2012). Therefore the possibility of a recovered TTS
594 in the present study population should be considered, especially in those subjects that
595 experienced HRS (Masterson et al., 2016; Schmuzigert et al., 2006).

596 SPiQ and SPiN tests showed no significant differences one day after noise exposure compared
597 to the baseline session. This finding is in contrast with the study of Grinn et al. (2017), where
598 the noise dose of a noise exposure event has been related to a decrease in speech in noise
599 intelligibility. However, this study showed a significant improvement in SRT-values for session
600 4 compared to session 1 for each condition, which could be related to a learning effect. Two
601 training lists were used as suggested in literature concerning the Flemish Matrix speech
602 material to overcome the largest training effect. However, smaller and non-significant
603 improvement in SRT-values are described even after the presentation of two training lists
604 (Kollmeier et al., 2015). As a consequence of this training effect, possible deteriorations in
605 speech intelligibility shortly after noise exposure could remain undetected.

606 No significant changes could be found between the first session and sessions 2, 3 or 4 for AEP-
607 measurements, as determined by analysis of ABR amplitudes and latencies of wave I, III and V
608 and ABR I/V amplitude ratio and EFR-amplitudes. Grinn et al. (2017), who measured ABR wave
609 I amplitudes by conducting electrocochleography, did not find amplitude reductions after
610 attending a noisy event.

611 DETECTING AND MONITORING CS IN NORMAL HEARING SUBJECTS

612 In the present study, no relation could be found between noise-exposure history and the
613 possible biomarkers of CS, nor did those biomarkers change over sessions surrounding a music
614 event in normal hearing young adults. Baseline testing results and shifts from session 1 to
615 session 2 did not differ between subjects that experienced HRS after the music event, and

616 those who did not. Thus, no strong conclusions can be made regarding the presence of noise-
617 induced CS in relation to noise-exposure history nor regarding the appearance of CS shortly
618 after visiting a music festival in this cohort of young normal-hearing subjects.

619 Possibly, the noise-exposure history and recreational noise exposure dose (76.2 dBA) in the
620 present study were not sufficiently high to cause CS. Permanent consequences of recreational
621 noise on hearing has been doubted before, as reviewed by Carter et al. (2014). This theory is
622 reflected by the lack of TTS in all but one subjects at one day after the festival. In noise-
623 exposed animals, smaller TTSs of 20 – 30 dB, measured at one day after a single noise
624 exposure, were shown not to result in CS (Fernandez et al., 2015; Jensen et al., 2015) whereas
625 TTSs up to 50 dB were reported for noise-exposed animals with substantial CS (Furman et al.,
626 2013; Kujawa & Liberman, 2009; Lin et al., 2011). It may hence be that the young normal-
627 hearing listeners we considered did not suffer from CS.

628 Second, it is possible that the biomarkers for CS are not sensitive or specific enough to detect
629 or to monitor CS in normal-hearing young adults. The interpretation of speech in noise
630 audiometry is complicated by a possible training effect. Confounding factors in AEP-
631 measurements include amongst others intersubject variability due to head size and sex
632 (Mitchell et al., 1989) and intrasubject variability due to different degrees of awakeness (part
633 II, Pethe, Begall et al., 1996).

634 In order to distinguish between the absence of CS in the study cohort and a lack of specificity
635 in the CS biomarkers, future studies may provide more insight. First, it would be interesting to
636 further investigate intersubject variability and test-retest reliability of the biomarkers of CS in
637 a cohort of normal-hearing subjects. Second, the use of dosimetry, where noise doses can be
638 measured for specific noise events, logging of HPD-use during a music event, and hearing

process evaluation immediately after the event will provide the needed information regarding recent noise exposure doses, HPD-use and temporary versus long-term effects on hearing. Third, as AEP-measurements can be conducted with different stimulus parameters and rates, their sensitivity to detect CS can be further investigated by further comparing their results within the same study cohorts (Vasilkov et al., 2020; Wilson et al., 2020). Finally, larger test groups will allow us to compare different possibly confounding factors such as the use of HPD and different levels of noise-exposure history.

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Figures

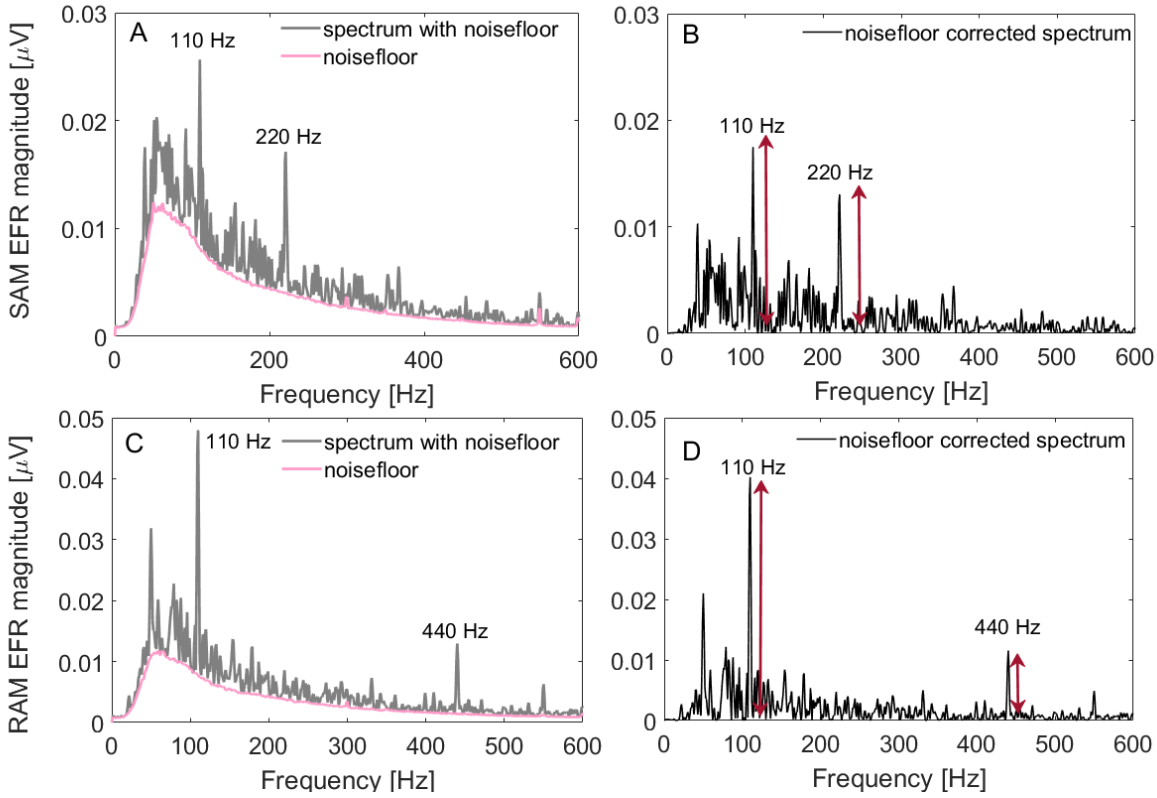

***Figure 1**: Exemplary ABR waveforms to 80 dB peSPL (dark grey) and 100 dB peSPL (blue), 11 Hz clicks. Respective ABR wave I, III and V peaks are specified by O and X for 80 and 100 dB peSPL click stimuli, respectively.*

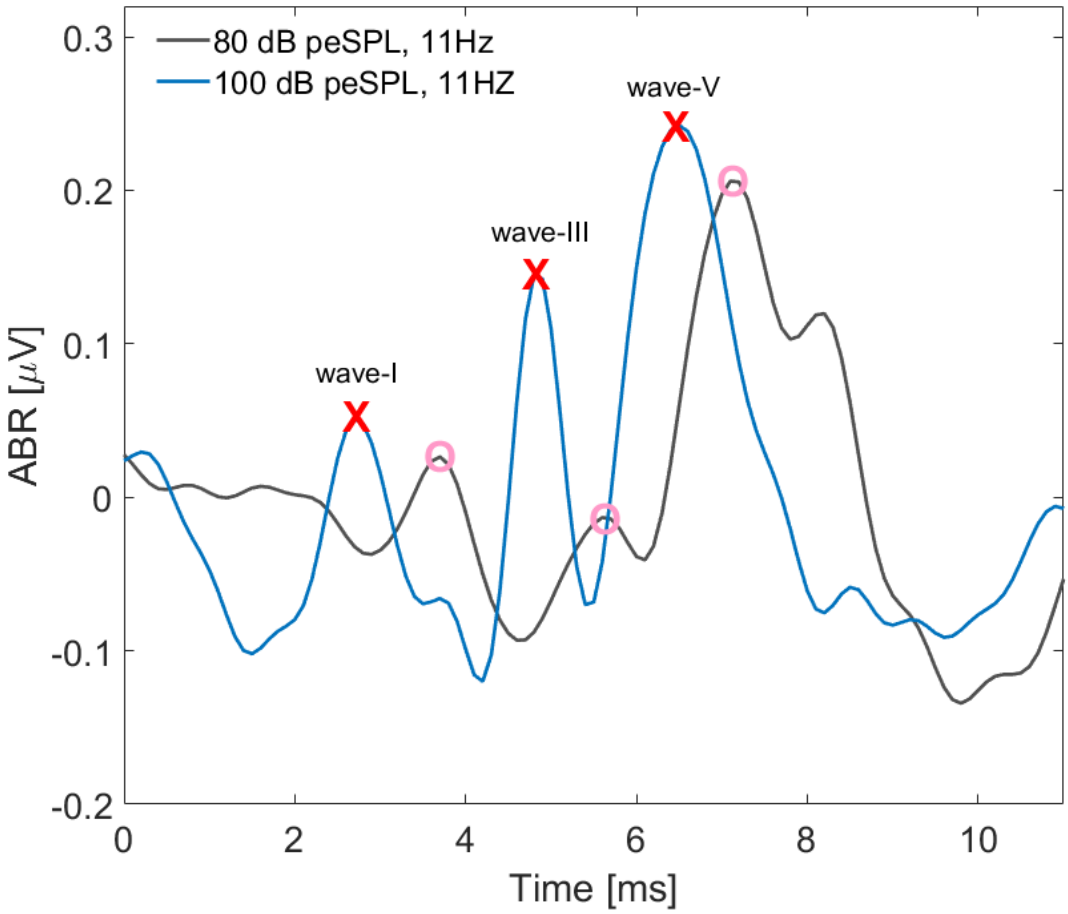

Figure 2: *Exemplary EFR spectra. (A) SAM EFR spectrum (grey) and estimated noise-floor (pink). (B) Noise-floor corrected SAM EFR after bootstrapping. (C) RAM EFR spectrum (grey) and estimated noise-floor (pink). (D) Noise-floor corrected RAM EFR after bootstrapping. . Arrows in panels (B) and (D) show the magnitudes of EFR at modulation frequency (110 Hz) and harmonics.*

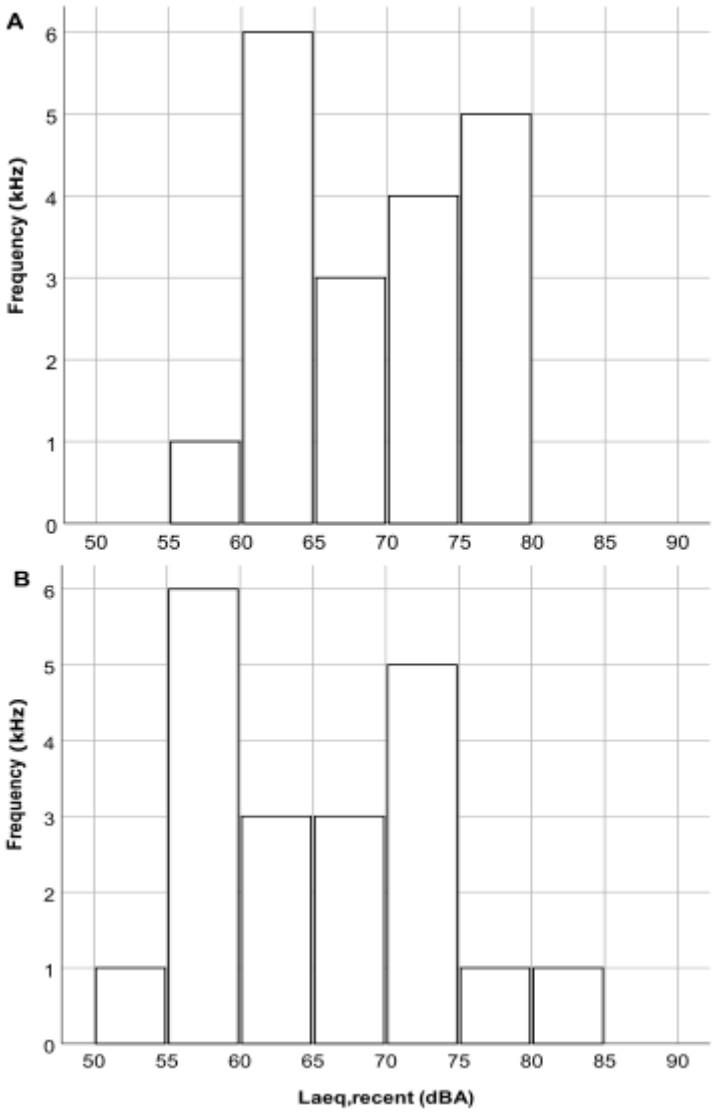

Figure 3*: Frequency distribution of Laeq,life (A) and Laeq,recent (B).*

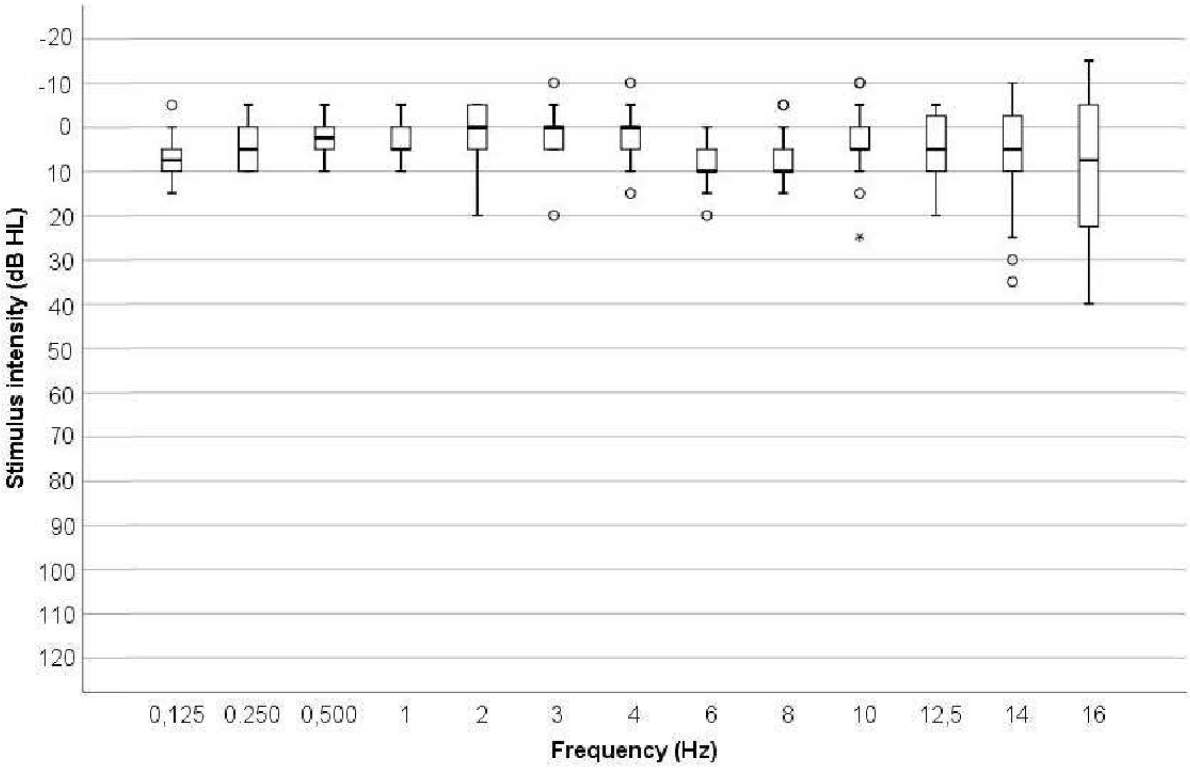

***Figure 4**: Boxplots of baseline audiometric thresholds.*

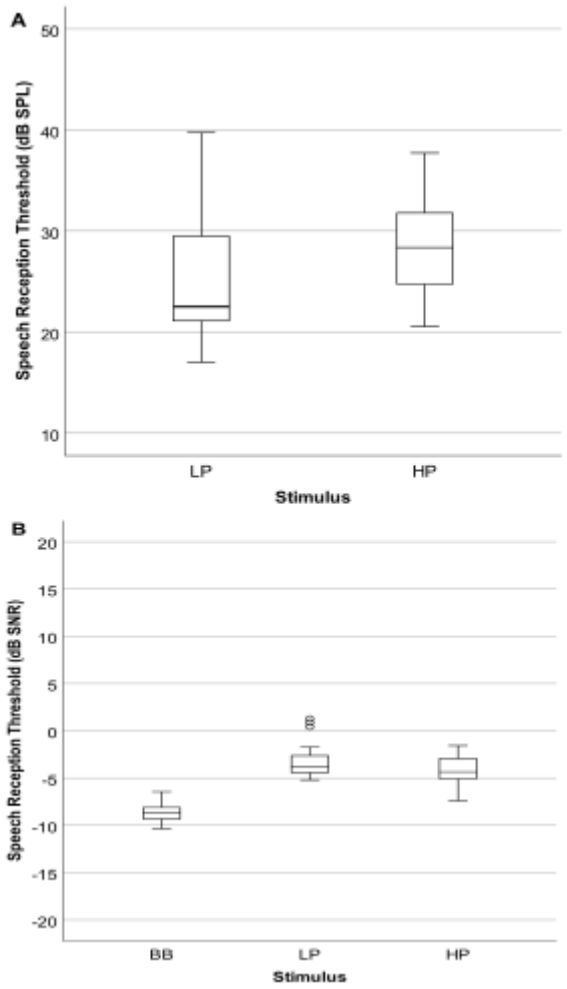

Figure 5: *Boxplot of SPiQ (A) and SPiN (B) results at baseline session.*

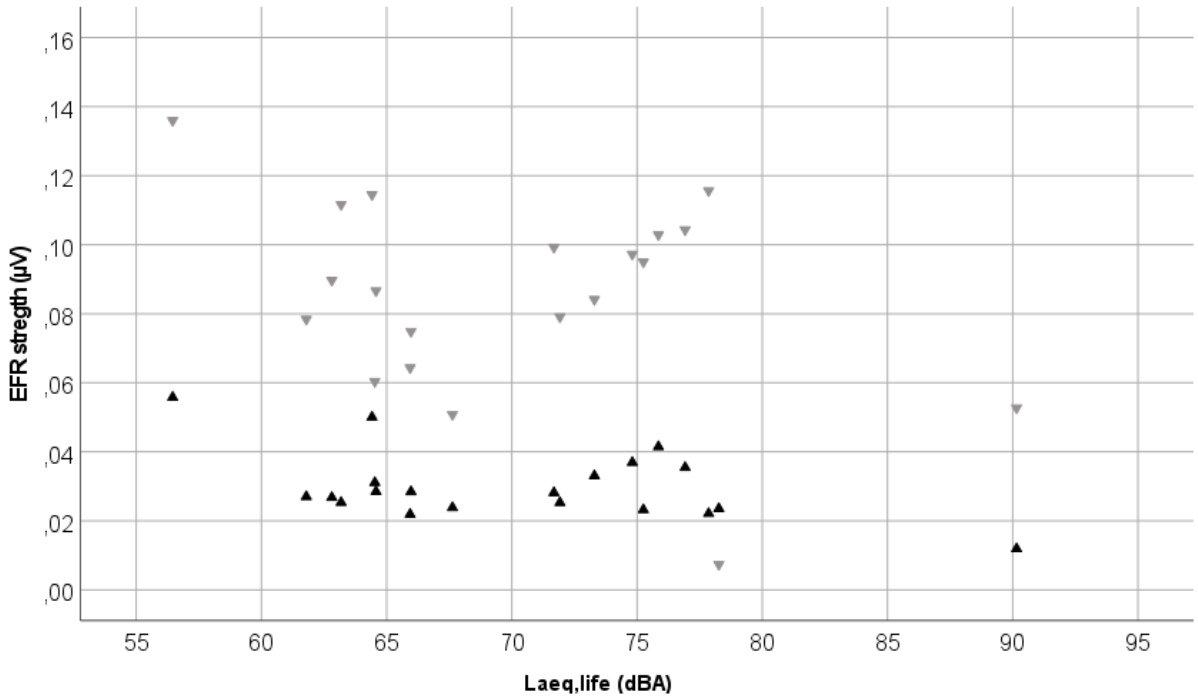

Figure 6: *Scatterplot of EFR strength in relation to Laeq,life for SAM-stimuli (black triangles) and RAM-stimuli (grey triangles).*

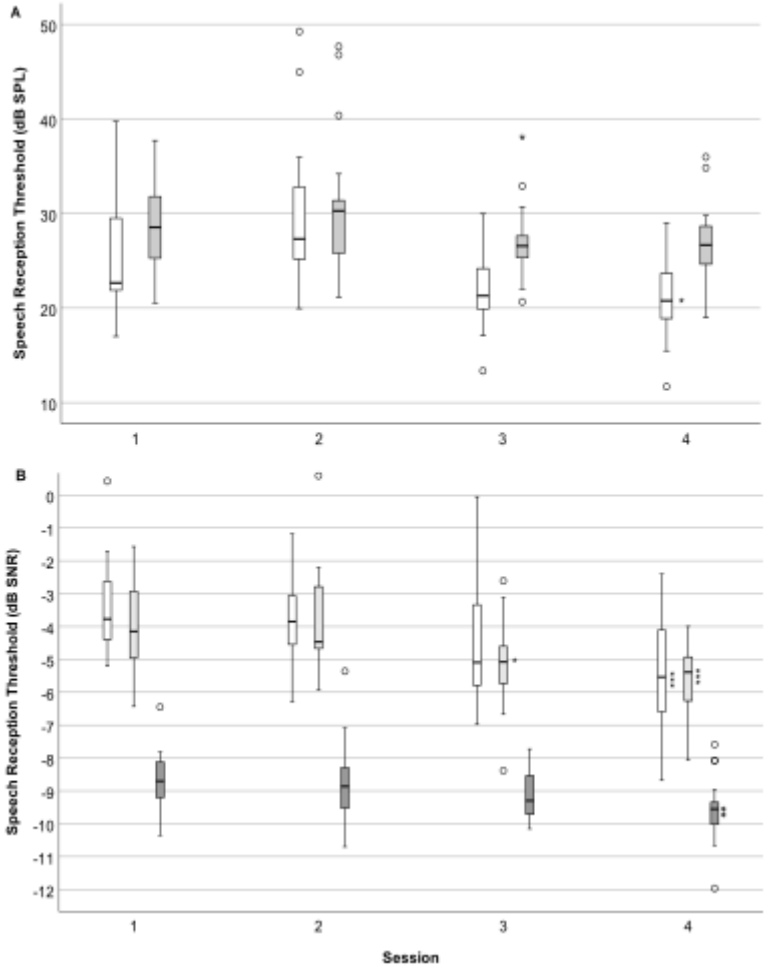

Figure 7: *Boxplots of speech audiometry in quiet (A) and in noise (B) for all conditions and all sessions. Dark grey boxplots represent SRTs for BB stimuli, light grey and white boxplots represent SRTs for HP and LP conditions, respectively. Intersession differences, compared to session 1, were significant if p<0.017 and are highlighted with one, two or three asterisks (resp. p<0.017, p<0.003 and p<0.0003). Both SPiN and SPIQ showed no significant differences between sessions 1 and 2.*

1116 **Tables**

Table 1: ABR data for ABR- and EFR responses of all stimulus types. Mean, SD, median and IQR are presented for ABR latencies and amplitudes of waves I, III and V and I/V amplitude ratio and EFR-strength.

stimulus			11Hz 100dB$_{peSPL}$ click	11Hz 80dB$_{peSPL}$ click	120Hz 100dB$_{peSPL}$ click	120Hz 80dB$_{peSPL}$ click	TB$_{4kHz}$	TB$_{1\ kHz}$
latencies	I	mean	2.67	3.32	3.14	3.70	3.96	4.36
		SD	0.19	0.27	0.39	0.27	0.44	0.53
		median	2.70	3.35	3.10	3.70	3.90	4.45
		IQR	0.20	0.27	0.40	0.20	0.75	0.67
	III	mean	4.94	5.46	5.47	6.01	6.15	6.48
		SD	0.23	0.31	0.33	0.33	0.38	0.70
		median	4.90	5.50	5.45	6.00	6.05	6.50
		IQR	0.20	0.35	0.40	0.27	0.65	0.75
	V	mean	6.73	7.18	7.32	7.90	7.94	5.57
		SD	0.38	0.43	0.38	0.43	0.35	0.43
		median	6.70	7.25	7.30	8.00	7.90	8.60
		IQR	0.47	0.35	0.47	0.37	0.40	0.45
amplitudes	I	mean	0.15	0.08	0.09	0.05	0.02	0.02
		SD	0.10	0.06	0.06	0.04	0.04	0.05
		median	0.17	0.07	0.08	0.04	0.03	0.02
		IQR	0.14	0.11	0.08	0.09	0.05	0.06
	III	mean	0.07	0.03	0.13	0.15	0.02	-0.01
		SD	0.07	0.07	0.10	0.09	0.06	0.07
		median	0.05	0.03	0.11	0.14	0.01	-0.01
		IQR	0.13	0.10	0.13	0.15	0.05	0.10
	V	mean	0.31	0.27	0.27	0.23	0.13	0.10
		SD	0.10	0.11	0.11	0.08	0.08	0.07
		median	0.30	0.27	0.26	0.22	0.13	0.09
		IQR	0.12	0.10	0.13	0.08	0.09	0.10
amplitude ratios	I/V	mean	0.47	0.34	0.39	0.22	-0.96	0.44
		SD	0.30	0.31	0.34	0.37	3.80	1.14
		median	0.44	0.30	0.31	0.21	0.15	0.17
		IQR	0.46	0.38	0.26	0.42	0.37	0.55
stimulus			SAM			RAM		
EFR-strength		mean	0.029			0.085		
		SD	0.010			0.029		
		median	0.028			0.088		
		IQR	0.011			0.037		

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*Table 2: χ2-values of related-samples Friedman's ANOVA for PTA. Significant results are highlighted with *(p<0.05), **(p<0.01) or ***(p<0.001). Median test values and IQRs are shown for each test session.*

			Session 1 (pre)		Session 2 (1st post)		Session 3 (2nd post)		Session 4 (3d post)	
	Frequency	χ^2	med	IQR	med	IQR	med	IQR	med	IQR
Auditory thresholds (dB_{HL})	125Hz	$\chi^2(3)=5.56$	5	5	10	10	5	10	5	10
	250Hz	$\chi^2(3)=3.47$	5	10	5	5	5	10	5	10
	500Hz	$\chi^2(3)=8.73^{*}$	0	5	5	10	0	5	0	5
	1kHz	$\chi^2(3)=4.73$	5	5	0	5	0	5	0	10
	2kHz	$\chi^2(3)=0.26$	0	10	0	10	0	10	0	10
	3kHz	$\chi^2(3)=0.22$	0	5	0	5	0	5	0	10
	4kHz	$\chi^2(3)=2.83$	0	5	0	5	0	5	0	10
	6kHz	$\chi^2(3)=2.03$	10	5	10	15	10	15	10	10
	8kHz	$\chi^2(3)=6.33$	10	5	10	10	5	10	10	10
	10kHz	$\chi^2(3)=12.69^{**}$	5	5	5	10	5	10	5	10
	12kHz	$\chi^2(3)=4.88$	5	15	10	20	5	15	5	15
	14kHz	$\chi^2(3)=4.76$	5	15	5	10	0	15	0	5
	16kHz	$\chi^2(3)=7.39$	10	30	10	20	10	25	5	25
	$PTA_{3\text{-}8kHz}$	$\chi^2(3)= 3.84$	5	5	5	8.75	3.75	5	5	8.75
	$PTA_{10\text{-}16kHz}$	$\chi^2(3)=6.97$	2.5	12.5	7.5	13.75	6.25	10	5	7.5

*Table 3: χ^2-values of related-samples Friedman's ANOVA for AEP measurements. Significant results are highlighted with *(p<0.05), **(p<0.01) or ***(p<0.001). Median test values and IQRs are shown for each test session.*

			Session 1 (pre)		Session 2 (1st post)		Session 3 (2nd post)		Session 4 (3d post)	
	Frequency	χ^2	med	IQR	med	IQR	med	IQR	med	IQR
ABR latency wave I (ms)	11Hz 100dB$_{peSPL}$	$\chi^2(3)=6.78$	2.70	0.20	2.70	0.10	2.70	0.10	2.70	0.20
	11Hz 80 dB$_{peSPL}$	$\chi^2(3)=0.88$	3.40	0.30	3.30	0.20	3.30	0.40	3.30	0.20
	120Hz 100 dB$_{peSPL}$	$\chi^2(3)=3.55$	3.10	0.40	3.20	0.40	3.00	0.80	3.20	0.50
	120Hz 80 dB$_{peSPL}$	$\chi^2(3)=0.88$	3.70	0.20	3.70	1.50	3.70	0.20	3.70	0.50
	TB$_{4kHz}$	$\chi^2(3)=2.27$	3.90	0.80	4.00	0.90	4.30	0.90	3.90	0.60
	TB$_{1kHz}$	$\chi^2(3)=0.62$	4.50	0.70	4.30	0.90	4.50	0.70	4.40	0.50
ABR latency wave III (ms)	11Hz 100dB$_{peSPL}$	$\chi^2(3)=3.16$	4.90	0.20	4.90	0.20	4.90	0.20	4.90	0.20
	11Hz 80 dB$_{peSPL}$	$\chi^2(3)=3.85$	5.50	0.30	5.70	0.30	5.50	0.30	5.50	0.30
	120Hz 100 dB$_{peSPL}$	$\chi^2(3)=1.72$	5.50	0.40	5.40	0.40	5.40	0.40	5.40	0.30
	120Hz 80 dB$_{peSPL}$	$\chi^2(3)=0.36$	6.00	0.30	6.10	0.30	6.00	0.30	6.10	0.50
	TB$_{4kHz}$	$\chi^2(3)=1.42$	6.00	0.55	6.20	0.65	6.20	0.45	6.20	0.70
	TB$_{1kHz}$	$\chi^2(3)=6.95$	6.50	0.70	6.80	0.40	6.60	0.50	6.50	0.40
ABR latency wave V (ms)	11Hz 100dB$_{peSPL}$	$\chi^2(3)=3.12$	6.70	0.50	6.70	0.30	6.70	0.30	6.70	0.30
	11Hz 80 dB$_{peSPL}$	$\chi^2(3)=1.76$	7.30	0.40	7.20	0.40	7.20	0.30	7.20	0.30
	120Hz 100 dB$_{peSPL}$	$\chi^2(3)=2.17$	7.30	0.50	7.40	0.20	7.30	0.30	7.40	0.40
	120Hz 80 dB$_{peSPL}$	$\chi^2(3)=4.11$	8.00	0.40	8.00	0.40	7.90	0.50	8.00	0.40
	TB$_{4kHz}$	$\chi^2(3)=0.14$	8.00	0.45	8.00	0.50	7.90	0.55	7.90	0.30
	TB$_{1kHz}$	$\chi^2(3)=1.06$	8.60	0.40	8.60	0.40	8.60	0.80	8.70	0.70
ABR amplitude wave I (µV)	11Hz 100dB$_{peSPL}$	$\chi^2(3)=1.61$	0.178	0.152	0.135	0.161	0.134	0.163	0.137	0.197
	11Hz 80 dB$_{peSPL}$	$\chi^2(3)=3.88$	0.068	0.11	0.061	0.12	0.087	0.15	0.091	0.14
	120Hz 100 dB$_{peSPL}$	$\chi^2(3)=1.11$	0.081	0.080	0.096	0.072	0.096	0.102	0.092	0.070
	120Hz 80 dB$_{peSPL}$	$\chi^2(3)=0.28$	0.042	0.096	0.075	0.096	0.046	0.088	0.070	0.125
	TB$_{4kHz}$	$\chi^2(3)=0.95$	0.022	0.060	0.027	0.036	0.023	0.068	0.008	0.044
	TB$_{1kHz}$	$\chi^2(3)=4.71$	0.013	0.065	0.006	0.077	0.029	0.062	0.015	0.061

ABR amplitude wave III (µV)	11Hz 100dB$_{peSPL}$	$\chi^2(3)=7.93^{*}$ p=0.48	0.044	0.137	0.057	0.130	0.073	0.100	0.103	0.112
	11Hz 80 dB$_{peSPL}$	$\chi^2(3)=2.43$	0.022	0.103	0.028	0.101	0.034	0.088	0.024	0.107
	120Hz 100 dB$_{peSPL}$	$\chi^2(3)=1.11$	0.114	0.122	0.096	0.079	0.133	0.072	0.112	0.081
	120Hz 80 dB$_{peSPL}$	$\chi^2(3)=3.44$	0.139	0.155	0.104	0.064	0.100	0.103	0.108	0.104
	TB$_{4kHz}$	$\chi^2(3)=7.52$	0.009	0.062	0.024	0.079	0.058	0.126	0.026	0.044
	TB$_{1kHz}$	$\chi^2(3)=3.82$	-0.020	0.101	-0.026	0.068	-0.001	0.076	-0.002	0.101
ABR amplitude wave V (µV)	11Hz 100dB$_{peSPL}$	$\chi^2(3)=6.35$	0.303	0.103	0.340	0.757	0.303	0.107	0.304	0.092
	11Hz 80 dB$_{peSPL}$	$\chi^2(3)=12.92^{**}$ p=0.005	0.269	0.108	0.287	0.113	0.254	0.122	0.279	0.092
	120Hz 100 dB$_{peSPL}$	$\chi^2(3)=3.82$	0.274	0.144	0.269	0.137	0.267	0.111	0.260	0.126
	120Hz 80 dB$_{peSPL}$	$\chi^2(3)=3.38$	0.222	0.085	0.248	0.152	0.242	0.136	0.231	0.079
	TB$_{4kHz}$	$\chi^2(3)=2.18$	0.111	0.113	0.129	0.070	0.130	0.078	0.134	0.091
	TB$_{1kHz}$	$\chi^2(3)=1.80$	0.091	0.103	0.100	0.066	0.062	0.083	0.095	0.059
ABR I/V amplitude ratio	11Hz 100dB$_{peSPL}$	$\chi^2(3)=1.67$	0.43	0.47	0.39	0.51	0.41	0.46	0.41	0.48
	11Hz 80 dB$_{peSPL}$	$\chi^2(3)=4.71$	0.29	0.39	0.23	0.37	0.33	0.62	0.27	0.59
	120Hz 100 dB$_{peSPL}$	$\chi^2(3)=1.04$	0.29	0.25	0.31	0.47	0.39	0.44	0.32	0.21
	120Hz 80 dB$_{peSPL}$	$\chi^2(3)=0.54$	0.21	0.43	0.24	0.47	0.23	0.37	0.29	0.45
	TB$_{4kHz}$	$\chi^2(3)=2.15$	0.05	0.38	0.22	0.47	0.16	0.59	0.05	0.29
	TB$_{1kHz}$	$\chi^2(3)=0.51$	0.15	0.49	0.05	0.85	0.24	0.86	0.02	0.73
EFR strength (µV)	SAM	$\chi^2(3)=1.13$	0.028	0.011	0.024	0.010	0.027	0.013	0.032	0.014
	RAM	$\chi^2(3)=2.87$	0.088	0.037	0.080	0.059	0.087	0.040	0.098	0.054

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*Table 4: U-values and significance levels of Man Whitney U test for AEP measurements. Median test shifts from session 1 to session 2, minima and maxima are shown for subjects that have experienced Hearing Related Symptoms (HRS) and those who did not. Significant between-group differences are highlighted with *(p<0.05), **(p<0.01) or ***(p<0.001).*

		no HRS (n=11)			HRS (n=9)			Man-Whitney U	
	Stimulus	Median	Minimum	Maximum	Median	Minimum	Maximum	U	p
latency wave I	11 Hz 80 dB	-0,1000	-0,20	0,10	0,0500	-0,10	0,40	78,00	0,03 *
	11 Hz 100 dB	-0,1000	-0,20	0,20	0,0000	0,00	0,60	79,50	0,02 *
	120 Hz 80 dB	0,0000	-0,30	0,20	0,1500	-0,70	1,20	66,00	0,23
	120 Hz 100 dB	0,1000	-0,30	1,20	-0,0500	-0,70	0,20	27,50	0,10
	TB 1 kHz	0,0000	-1,10	1,40	-0,1000	-0,60	1,00	49,00	1,00
	TB 4 kHz	-0,2000	-1,00	1,90	-0,1500	-1,00	0,80	39,50	0,66
Latency wave III	11 Hz 80 dB	0,1000	-0,10	0,80	0,1500	-0,10	1,00	50,50	0,94
	11 Hz 100 dB	0,0000	-0,80	0,10	0,0000	-0,10	0,10	57,50	0,55
	120 Hz 80 dB	0,0000	-0,30	1,20	-0,0500	-0,60	0,20	35,00	0,30
	120 Hz 100 dB	0,0000	-0,80	0,20	0,0500	-0,20	0,70	72,00	0,10
	TB 1 kHz	0,1000	-0,70	1,50	0,2000	-1,20	0,70	48,00	0,94
	TB 4 kHz	0,2000	-0,40	1,30	0,0000	-0,70	0,40	16,50	0,02 *
Latency wave V	11 Hz 80 dB	-0,1000	-0,20	0,10	0,0000	-0,10	1,40	73,00	0,08
	11 Hz 100 dB	0,0000	-0,10	0,40	0,0500	-1,30	0,20	53,00	0,82
	120 Hz 80 dB	0,0000	-0,10	0,40	-0,0500	-0,50	1,10	46,50	0,82
	120 Hz 100 dB	-0,1000	-0,20	0,70	0,1000	-0,10	1,10	73,00	0,08
	TB 1 kHz	0,1000	-0,40	1,40	-0,0500	-0,60	0,10	29,00	0,13
	TB 4 kHz	0,0500	-0,50	2,00	-0,1000	-0,20	0,40	32,00	0,32
Amplitude wave I	11 Hz 80 dB	-0,0076	-0,12	0,07	0,0096	-0,05	0,11	52,00	0,88
	11 Hz 100 dB	0,0076	-0,15	0,13	0,0338	-0,20	0,20	56,00	0,66
	120 Hz 80 dB	0,0189	-0,08	0,17	-0,0042	-0,18	0,17	45,00	0,77
	120 Hz 100 dB	0,0184	-0,08	0,10	-0,0214	-0,13	0,08	31,00	0,18
	TB 1 kHz	-0,0115	-0,08	0,24	-0,0592	-0,12	0,23	35,00	0,30
	TB 4 kHz	-0,0154	-0,07	0,04	0,0580	-0,05	0,16	71,00	0,04 *
Amplitude wave III	11 Hz 80 dB	0,0210	-0,04	0,13	0,0103	-0,10	0,05	32,00	0,20
	11 Hz 100 dB	-0,0424	-0,13	0,14	-0,0266	-0,13	0,16	55,00	0,71

	120 Hz 80 dB	-0,0125	-0,46	0,13	-0,0298	-0,28	0,14	55,00	1,00
	120 Hz 100 dB	-0,0029	-0,29	0,10	-0,0330	-0,24	0,10	47,00	0,88
	TB 1 kHz	-0,0264	-0,17	0,08	0,0318	-0,04	0,24	71,00	0,11
	TB 4 kHz	-0,0186	-0,15	0,10	0,0316	-0,08	0,12	53,00	0,55
Amplitude wave V	11 Hz 80 dB	0,0108	-0,09	0,27	0,0202	-0,15	0,30	50,00	1,00
	11 Hz 100 dB	0,0590	-0,15	0,15	0,0108	-0,04	0,23	55,00	0,71
	120 Hz 80 dB	0,0071	-0,15	0,19	0,1152	-0,15	0,22	61,00	0,41
	120 Hz 100 dB	0,0085	-0,30	0,15	0,0240	-0,09	0,27	65,00	0,26
	TB 1 kHz	0,0114	-0,08	0,06	-0,0039	-0,08	0,03	45,00	0,77
	TB 4 kHz	0,0426	-0,10	0,15	-0,0429	-0,16	0,04	17,00	0,02 *
I/V amplitude ratio	11 Hz 80 dB	-0,0364	-0,67	0,40	-0,0373	-1,01	0,42	48,00	0,78
	11 Hz 100 dB	0,0817	-0,54	0,45	0,1142	-0,95	0,39	50,00	0,66
	120 Hz 80 dB	0,0847	-0,55	1,00	-0,0123	-0,95	0,25	31,00	0,31
	120 Hz 100 dB	0,0926	-0,36	8,57	-0,0568	-0,77	0,05	17,00	0,03 *
	TB 1 kHz	-0,1842	-4,04	63,34	-0,3445	-1,16	3,85	35,00	0,49
	TB 4 kHz	0,0700	-0,45	16,34	0,5031	-0,21	13,93	50,00	0,41