BNAM 2018
Baltic-Nordic Acoustics Meeting
15-18 April 2018
Harpa, Reykjavík, Iceland

Evaluation of supra-threshold hearing following an event of recreational acoustic exposure

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Studies with small rodents have exhibited physiological evidence of noise-induced cochlear synaptopathy prior to outer-hair-cell loss following noise-induced large temporary threshold shifts (TTS). The auditory system may thus not fully recover after a TTS. If this noise-induced damage also occurs in humans, this may have consequences for sound processing at supra-threshold levels, especially speech in background noise, and may also challenge current noise regulations. The aim of this study was to investigate if human listeners with normal hearing sensitivity show signs of cochlear synaptopathy after participating in a concert. Young adult listeners with hearing thresholds $\leq$ 20 dB HL between 0.25-8 kHz were recruited and divided into two groups: listeners voluntarily participating in concerts and control listeners with no concert participation during the study. Exposure was assessed with dosimeters in both groups for one event duration. Concert participants were advised to use hearing protectors and exposure levels were determined from actual use. Listeners performed three sessions of audiometry, auditory brainstem response (ABR), and speech in noise measurements. Session 1 was performed within a week up to the concert, session 2 within 24 hours after the concert, and session 3 approximately 4 weeks after the concert. We hypothesized that concert participants would show reduced level-growth of ABR wave I and that wave-I level-growth would be a predictor of speech discrimination score. The data indicate that post-exposure wave-I level-growth was not reduced compared to pre-exposure values, and that neither were speech scores. Therefore, the results might suggest that: a) concert goers do not develop cochlear synaptopathy in response to typical exposure from one event, or b) synaptopathy occurs only for more severe exposure in humans, or c) the utilized measures are not sensitive enough to detect the damage.

1 Introduction

It is well-known that excessive acoustic exposure can cause outer-hair-cell (OHC) damage, which is typically reflected as permanently elevated audiometric hearing thresholds, more commonly known as noise-induced hearing loss (NIHL). Less intense and shorter durations of acoustic exposure can also induce a temporary threshold shift (TTS). TTS have not been considered dangerous to the ear due to the temporary nature of the threshold elevation. However, recent studies with small rodents exhibited physiological evidence of noise-induced cochlear synaptopathy prior to OHC damage following noise-induced large TTS [1]. Their results suggested that exposures causing large TTS also caused immediate and permanent damage to afferent neural synapses of inner hair cells (IHCs). This synaptic loss (cochlear synaptopathy)

seemed to be reflected as reduced neural activity, reflected as lower amplitude of wave I in the post-exposure auditory brainstem response (ABR) in response to high-level stimuli. However, the wave I amplitude in response to low-level stimuli recovered, suggesting the neural damage only affected processing of higher-level, or supra-threshold, stimuli. It was later suggested that acoustic exposures causing large TTS primarily targeted nerve fibres responsible for supra-threshold processing (i.e., low-spontaneous-rate fibres with high thresholds) [2]. This provides a plausible explanation for the noise-induced cochlear synaptopathy selectively affecting processing of higher level stimuli.

If acoustic exposures resulting in TTS in humans are also accompanied by cochlear synaptopathy, this may cause hearing deficits that cannot be revealed with the current audiological screening methods. Today, audiometry is the main clinical measure used to investigate auditory function. Such a measure evaluates a listener's sensitivity to weak stimuli. However, noise-induced auditory damage may affect supra-threshold processing while leaving sensitivity to soft sounds unchanged [1, 2]. For humans, consequences of such damage could be difficulties understanding speech in background noise despite having normal audiometric hearing thresholds, i.e., typically ≤ 20 dB hearing level (HL). This type of auditory deficit has been reported in humans and has been referred to as obscure auditory dysfunction, King-Kopetzky syndrome [3], or more recently as hidden hearing loss [4]. Until now, there has been no physiological explanation for this deficit, but the recent finding of cochlear synaptopathy in animals may pose a potential explanation.

Following, numerous studies have investigated this in humans. As it would not be ethically acceptable to expose human listeners to noise, as done in studies with smaller mammals, the studies with human listeners were performed without exposing to or having knowledge of any physical acoustic exposure. Noise-induced cochlear synaptopathy was investigated using electrophysiological measures (e.g., ABR) and behavioral supra-threshold measures with listeners typically separated into two groups based on their subjective reported history of acoustic exposure, one group being the test group (high risk of cochlear synaptopathy or high-exposure group), and the other being the control group (low-risk or low-exposure group). The results from such studies have so far not been conclusive. While some found evidence suggesting presence of cochlear synaptopathy [5], others did not despite the use of large populations in their studies [6, 7]. Another study [8] also failed to find significant differences across groups but found that, when investigating correlations between electrophysiological and behavioral markers of cochlear synaptopathy, poorer word recognition in noise was associated with a reduction in the level growth of ABR wave I amplitudes. This could suggest a relationship between reduced word-recognition in noise and cochlear synaptopathy [8].

The different findings across studies could potentially be due to the criteria used for separating the listeners into groups. Using a subjectively reported history of exposure as a means of separating listener-groups may not reflect their actual real-life exposure levels. It is difficult to recall and describe acoustic exposure experienced in the past, and one's experience of loudness and understanding of what noise exposure includes may vary greatly across listeners, leading to a lot of uncertainty. In the present study, a design in which acoustic exposure during a single event was objectively quantified was used. The test group consisted of listeners participating in a concert event, while the control group of listeners were in a quiet environment during the measurement period. Electrophysiological and behavioral measures of hearing status thought to be affected by synaptopathy were obtained in both groups prior to and after the event. We hypothesized that listeners in the test group would show a) significantly higher exposure in terms of the A-weighted equivalent sound pressure level (L_{Aeq}), b) a TTS the day after the concert event, c) reduced post-exposure ABR wave I level growth, and d) reduced post-exposure word-recognition scores in noise.

2 Method

2.1 Listeners

Fifteen listeners were recruited for the study. Each listener was given the choice to either participate in a concert event at a pop-rock venue or to avoid environments of excessive acoustic exposure during the study period. The test group consisted of 8 listeners (4 male, 4 female) participating in a concert event. The listeners were offered free earplugs and encouraged to use them during the whole concert. The control group consisted of 7 listeners (3 male, 4 female) not participating in a concert event and instructed to stay in quiet environments. All listeners were young adults aged between 20-32 (mean = 25.1) in the test group and 19-27 (mean = 23.4) in the control group. All had pure-tone thresholds ≤ 20 dB HL from 0.25-6 kHz and ≤ 25 dB HL at 8 kHz. Informed consent was obtained from all listeners.

2.2 Procedure

All listeners participated in 3 test sessions (one pre-exposure session and two post-exposure sessions). The pre-exposure session (Pretest) was carried out within a week before the concert event. This session included screening measures (see

section 2.3), hearing threshold measurements using an alternative forced-choice (AFC) procedure, a test of word recognition score in noise (WRSN), and ABR measurements. During the event (concert event in the test group and quiet event for the control group), all listeners were equipped with a dosimeter for approx. two hours to estimate noise exposure. The first post-exposure session (Posttest1) was completed the day after evaluation of the acoustic exposure. This session included AFC audiometry and ABR measurements. The second post-exposure session (Posttest2) was completed 4-5 weeks after the event. This session included AFC audiometry, the WRSN test, and ABR measurements. Otoscopy and tympanometry were also performed prior to both Post 1 and Post 2 sessions in order to screen for any abnormalities that could affect the results (i.e., cerumen occluding the ear canal or negative middle-ear pressure).

2.3 Screening measures

Otoscopy was performed with an Interacoustics video otoscopy device to ensure a clear ear canal and normal tympanic membrane through visual inspection. Audiometric thresholds (0.25-8 kHz in octave steps, including inter-octaves 3 and 6 kHz, using a 10-down 5-up procedure), the speech recognition threshold (SRT) in quiet and discrimination score (DS) in quiet were measured using a calibrated clinical audiometer (Interacoustics AC40) to ensure audiometric hearing and speech perception in quiet within normal limits. For the SRT measurement, the stimuli were 3 numerals spanning from 0-12 that the listener had to repeat. The DS was measured using wordlists consisting of 25 words at 40 dB HL with stationary speech-shaped noise presented simultaneously in the contralateral ear to avoid cross-hearing. Tympanometry was performed using an AT235 tympanometer to ensure middle-ear volume and pressure within normal limits.

2.4 Acoustic exposure

To assess acoustic exposure during the event, the listeners were equipped with personal sound dosimeters of type Casella dBadge CEL-35X, shoulder-mounted using CEL-6352 Crocodile Clip kits. The dosimeters were calibrated at the beginning of and at mid-term throughout the study using a CEL-120 acoustic calibrator. After each measurement, L_{Aeq} and the C-weighted peak sound pressure level (L_{Cpeak}) were retrieved with the Casella Insight software. The test-group participants were asked to carry the dosimeter on their shoulder from when they entered the concert hall to when they left and to pay attention not to be standing behind a tall person to avoid acoustic shadows at the microphone position. They were offered a set of EAR Soft Neon earplugs providing 36-dB attenuation and were encouraged to use them. The participants in the control group used the dosimeter for a minimum of 1.5 h the evening before the Posttest1 session. Both groups also completed a questionnaire [10] to estimate subjective annual noise exposure ($L_{\mathrm{Aeq}}8760$).

2.5 Alternative-forced-choice audiometry

Threshold sensitivity was evaluated at each session using a 3-AFC procedure in Matlab. The measurement was conducted in a sound-insulated booth using ER2 insert earphones. The task of the listeners was to indicate which of three intervals contained the stimulus (warble tone). Frequencies tested were 0.1, 1, 2, 3, 4, 6, and 8 kHz. A 2-up 4-down tracking procedure was used and threshold was determined across 4 reversals. Each frequency was tested twice and the threshold was taken as the mean between the two measures.

2.6 Word-recognition score in noise

The WRSN test was performed in a sound-insulated booth using the Interacoustics AC40 clinical audiometer with ER3A insert earphones. Lists of 25 words (Dantale I material) [13] were presented monaurally to the listeners with simultaneous constant speech-shaped noise. The WRSN was measured at 3 different signal to noise ratios (10, 5, and 0 dB) with the speech signal kept constant at 70 dB HL. The final score was the percentage of words repeated correctly.

2.7 Auditory brainstem responses

ABRs were recorded using the clinical Interacoustics Eclipse hardware (EP15/EP25) and ER3A insert earphones. Disposable non-invasive inverting electrodes were attached to the mastoids, a non-inverting electrode was placed on the middle of the forehead below the hairline, and the ground electrode between the eyebrows. Electrode impedances < 3 kΩ were ensured. The listeners lay on a bed in a sound-insulated and electrically-shielded both. They were instructed to sleep or relax during the measurement. Click stimuli were presented at 65, 75, 85, and 90 dB nHL with alternating polarity at a rate of 11.1/s. Recordings were made separately for each ear. Efforts were made to keep a residual noise level between 30-40 nV for all measures. ABR wave I-V peak-to-trough amplitudes were selected manually.

3 Results

3.1 Acoustic exposure

Based on *t*-tests, the two groups were found to be exposed to significantly different L_{Aeq} (mean and SD for test group: 97.1 ± 13.5 dBA; mean and SD for control group: 64.5 ± 6.5 dBA; $t = 14.26$, $p < 0.001$). L_{Cpeak} was also significantly higher for the test group than for the control group (test group: 128.0 ± 12.7 dBA; control group: 120.7 ± 10.7 dBA; $t = 2.70$, $p = 0.030$). The large difference in terms of L_{Aeq} is due to the fact that 7 out of 8 listeners in the test group reported not using the provided earplugs during the concert. The adjusted L_{Aeq} for the only listener who did use the earplugs was 64.2 dBA, based on full-time adequate use and on the expected attenuation as reported by the manufac-turer. The questionnaire results revealed that the two groups did not differ in terms of $L_{Aeq}8760$ ($t = 0.82$, $p = 0.434$).

3.2 Alternative-forced-choice audiometry

A slight hearing threshold shift at frequencies above 4 kHz was observed at Posttest1 and Posttest2 compared to Pretest. The Pretest vs Posttest changes were estimated using an arithmetic average of hearing thresholds between 4-8 kHz and the data were analyzed with the reliability-corrected analysis of covariance (ANCOVA) method [14]. In the left ear (Figure 1, left panel), the concert goers (dark grey curves) showed significant threshold increases of approximately 3 dB at Posttest1 ($t = 4.17$, $p = 0.002$) and Posttest2 ($t = 2.95$, $p = 0.002$) vs. Pretest. In the right ear (Figure 1, right panel), a similar significant increase ($t = 2.22$, $p = 0.049$) was only observed at Posttest1.

3.3 Word-recognition score in noise

The percentage of correctly repeated words decreased when lowering the SNR for both groups and in both sessions. At 10 dB SNR, the average scores across both sessions and ears were above 70%; at 5 dB SNR, they ranged from 56.5% to 65%; and at 0 dB SNR, they were below 53%. At 0 dB SNR (Figure 2), there was an average decrease in scores at Posttest2 vs. Pretest in the left ear (left panel), but it was not significant and it was observed in both groups. A reliability-corrected ANCOVA analysis revealed no significant effects of exposure for any of the SNRs.

3.4 Auditory brainstem responses

Figure 3 shows the ABR wave-I amplitude as a function of stimulus level for the two groups and three test sessions. A decrease of ABR wave I level-growth above 85 dB nHL was observed at posttest1 for the test group, but a reliability-corrected ANCOVA model revealed that the effect of the concert exposure on wave I level-growth was not significant. No other significant Pretest vs Posttest effects were observed for the ABR.

4 Discussion

All participants in the test group were exposed to levels above 98 dBA for more than 2 hours, with peak levels above 125 dBC. Such levels are considered dangerous when the duration of the exposure exceeds 15 minutes per day [9]. 5 participants reported feelings of reduced hearing sensitivity and phantom sounds within two hours post-concert, but none reported such sensations the morning after the concert. The levels and durations of the exposures were similar to the one used by Kujawa and Liberman [1] in mice. The groups were found equivalent in terms of past-year exposure ($L_{Aeq}8760$), removing a potential confound as being exposed to noise on a long run can sensibly affect some of the markers of NIND [8, 10]. Despite this match and the rather high levels in the test group, no significant effects of exposure to the concert event were found on WRSN performance and level-growth of ABR wave I amplitudes here.

The only observed effect was an increase in audiometric thresholds of up to 3 dB within 9-15 hours after the concert at frequencies from 4 kHz to 8 kHz. There was no recovery four weeks post-exposure in the left ear, suggesting a small permanent threshold elevation, but greater recovery in the right ear, suggesting a temporary threshold elevation. These differences were small and in agreement with a previous study [11]. The TTS is known to recover in an exponential manner [12] within hours post-exposure. As the measures here were not carried out earlier than 9 hours post-exposure, this could explain why only a maximum 3-dB TTS was observed. In rodents, no signs of synaptopathy were observed for 20 to 30 dB TTS, and only TTS from 40 to 50 dB were associated with NIND [11]. We can assume the TTS minutes after the exposure was larger, however TTS up to 40 dB would presumably require a much louder and longer exposure.

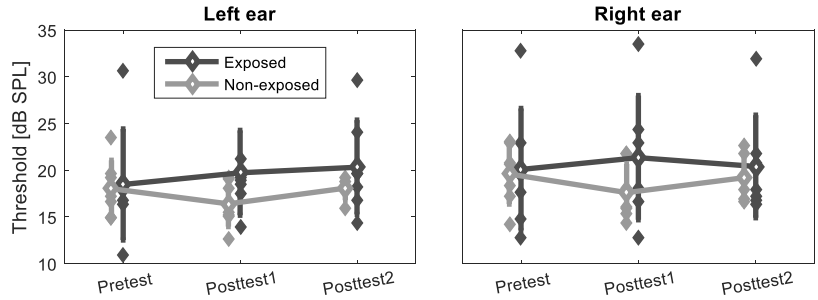

Figure 1: Mean hearing thresholds between 4-8 kHz for the exposed, test group (dark grey curves) and non-exposed, control group (light grey curves) for each session the left ear (left panel) and right ear (right panel). The individual data points and standard deviations are also plotted.

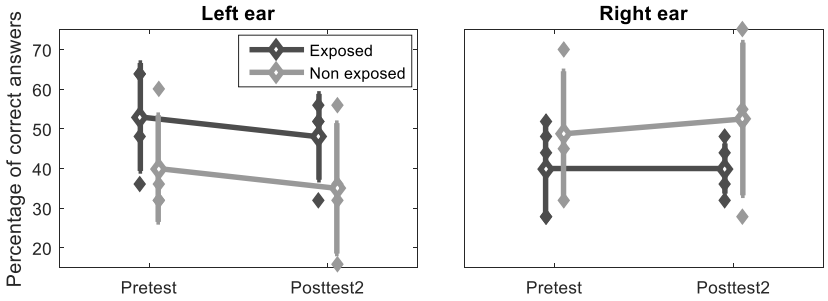

Figure 2: Mean percentages of correctly identified words at Pretest and Posttest2 at 0 dB SNR (exposed test group: dark grey; non-exposed control group: light grey) in both ears (left ear: left panel; right ear: right panel). The individual data and standard deviations are also shown.

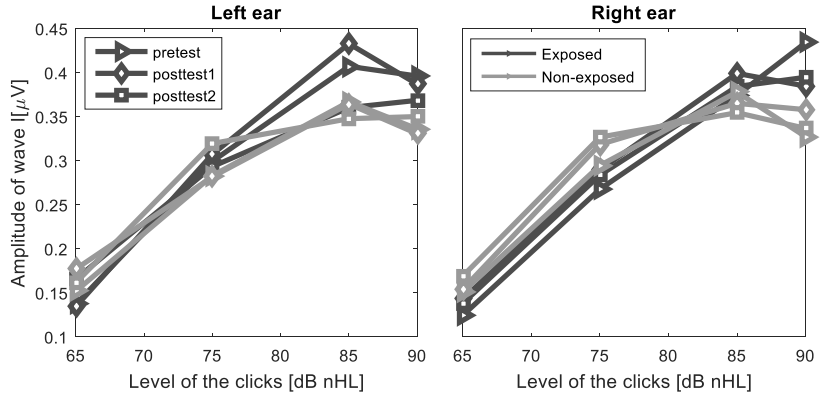

Figure 3: Amplitude of click-evoked ABR wave I as a function of stimulus level for the three sessions (exposed test group: dark grey; non-exposed control group: light grey) in the left ear (left panel) and right ear (right panel).

The lack of significant impact of the concert on WRSN performance 4 weeks post-exposure suggested that the reported acoustic exposure did not cause cochlear synaptopathy to an extent that it would impair speech intelligibility in noise, in agreement with [11] who found no relationship between such exposure and WRSN performance 1 week post-exposure. Also, no significant relationship was found between wave-I level-growth and the concert exposure. The exposure levels and durations may not have been high enough to cause extensive synaptopathy in humans, should the human auditory system be more resilient than that of other species in terms of synaptopathy.

One might see the lack of effect of exposure to one single concert as a very positive finding for those attending live-music performances occasionally. However, the present results do not necessarily mean that it is safe to be exposed to concert noise, as the measures used in the present study may not be sensitive to synaptopathic damage and the small sample size within each group may also have contributed to the lack of observed effects. The AFC audiometry also suggested that the threshold elevation after the concert may have been small but permanent in the left ear. The neural impact of the noise-exposure may be too small to be detected with the techniques and the experimental design used

here. Therefore, longer-term investigations and with optimized study designs are needed. For instance, it could be relevant to perform additional post-exposure measures between events to investigate changes over a longer period, using randomized listeners groups also tested shortly after the event to obtain an accurate TTS estimates.

5 Conclusion

To investigate the effect of concert noise on potential markers of cochlear synaptopathy and hearing status, a group of young adults participating in a concert event and a non-exposed control group were compared in terms of pre- and post-exposure measures of audiometric thresholds, word recognition score in noise, and ABR wave I level-growth. The levels and duration of the exposure of concert participants were similar to the ones used in earlier animal studies. The exposure induced a small hearing threshold shift at frequencies above 4 kHz, which persisted 4 weeks post-exposure in the left ear, but not in the right ear. No relationship was found between the exposure to the concert event and a change in behavioral word recognition scores in noise. Moreover, there was no clear effect of the exposure on the level growth of click-evoked ABR wave I. Overall, no evidence was found that exposure to a two-hour pop-rock concert causes permanent neural damages. Noise-induced synaptopathy may thus occur only for more severe exposures in humans, or the utilized measures may not be sensitive enough to detect such damage.

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