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Original Research Article

Effects of brief exposure to loud music on otoacoustic emissions and auditory brainstem responses

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ABSTRACT

Background: Research on noise induced hearing loss pathophysiology has recently focused on synapses rather than outer hair cells, following relevant evidence from animal studies. Findings from human studies, mainly targeting on effect of chronic exposure are controversial. Aim of this study is to investigate the immediate effect of noise exposure to synaptic function with use of ABR and DPOAEs.
Methods: Ten participants with normal hearing levels underwent DP-gram between 1 and 6 KHz and ABR at 90dB and click rates 33/sec and 44/sec before and after exposure to standardized music. Four of them were professional musicians and six were controls. Material for reliable and constant exposure to music was created, consisting of 56 wav files with music with a total duration of 2.5 hours. Files were presented in stable dB SPL levels and absolute control of dB SPL levels to ear phones was ensured. Subjects were asked to listen to music of their preference in maximum for 30 minutes. Patients exceeding 83 dB SPL maximum comfortable levels were eligible for the study.
Results: Statistically significant differences were observed before and after exposure to music for all SNRs from 1.5 to 6 KHz. Changes were similar between musicians and non-musicians. No differences were observed in ABR latencies and amplitudes in any of the waveforms before and after noise exposure.
Conclusions: A reliable technique has been developed for standardized exposure to loud sounds in humans, which can be used in future studies. Exposure to music induced decrease in DPOAE SNRs.

Keywords: Hidden hearing loss, Synaptotpathy, ABR, Music

INTRODUCTION

Hearing loss affects more than 350 million people worldwide and is the most common sensory deficit.[1] Its prevalence has increased during the past 20 years, due to population ageing. Estimated costs related either directly or indirectly with hearing loss, exceed 200 billion euros in Europe.[2] Besides age-related pathology, the most important cause of sensorineural hearing loss (SNHL) is noise exposure[3]. Approximately nine million workers in the USA alone are exposed to time-weighted average (TWA) sound levels of 85 dB(A).[3] Despite the fact, that relevant regulations have been set for decades, noise-induced hearing loss (NIHL) remains the second most common self-reported occupational illness or injury.[4] Occupational noise levels beyond 80 dB(A) significantly increase the risk of hearing, which is related to both exposure duration and intensity.

Although well established as a clinical entity, the pathophysiology of NIHL is still not fully understood. Traditionally, outer hair cells (OHCs) have been considered the primary target of noise induced pathology. Several studies have identified a variety of lesions,

including disruption of the connections between the tectorial membrane and outer hair cell stereocilia, damage of the stereocilia themselves, breaching of the integrity of the reticular lamina or even basilar membrane disruption.[5]

In contrast to the permanent hearing loss caused by chronic noise exposure, the term temporary threshold shift (TTS) describes a non-permanent elevation in hearing thresholds after short periods of noise exposure, which recover within minutes to hours. Continuous or repeated exposures to noise that only induce a TTS, may lead to a permanent threshold shift (PTS).[6] Questions regarding the exact mechanisms, the cochlea elements that are more vulnerable to noise, and whether different types and intensities of noise cause different lesions to cochlea, have not been answered yet.[7]

Kujawa and Liberman presented evidence from mice exposed to 100 dB SPL noise for two hours, questioning the predominant role of OHCs in NIHL patho-physiology.[8] It was observed that even limited exposure to noise, producing fully reversible TTS and not PTS, can induce permanent loss of cochlear synapses. It was estimated, that loss of synapses can reach levels as high as 50% in the presence of normal pure tone audiometry (PTA) thresholds.[9]

It has been clinically observed since the 1940s, that there is a subgroup of patients complaining of auditory symptoms, including difficulty in discrimination, especially in noisy environments, as well as tinnitus and hyperacusis. Schaette and MacAlpline introduced the term hidden hearing loss and presented the hypothesis that cochlear synaptopathy could be the underlying pathology accounting for these symptoms.[10] This hypothesis was further supported by the fact, that high threshold-low spontaneous rate (HT-LSR) auditory fibers, which aid in speech discrimination in loud and noisy environments, are more vulnerable to noise exposure and are lost early in the process. In fifteen out of nineteen animal studies summarized by Hickox et al loss of synapses after noise exposure exceeds 30% in mice after standardized noise exposure.[11] The terms hidden hearing loss (HHL) and Cochlear Synaptopathy (CS) are now used interchangeably, however should be considered highly correlated but not identical, since they refer to the clinical and pathophysiological level respectively. The extent of their causative relationship however remains to be determined.

The primary objective of this study was to identify changes in DPOAEs speech to noise ratio (SNRs) and ABR waveforms immediately following exposure to loud music, provided via earphones in a standardized way. The main hypothesis was that exposure to loud music would induce detectable shifts both to DPOAEs, reflecting changes in OHCs function, as well as to ABR parameters, especially to wave I amplitude, possibly reflecting an acute effect on synaptic function.

METHODS

Study design

This is a within-subjects study looking at the immediate effects of brief exposure to loud music set at maximum comfortable levels on the auditory system in young individuals with normal hearing. Study was conducted between March and December 2018 in the Neurotological Laboratory of the 1st Department of Otolaryngology, University of Athens. Ethics approval was obtained by the local Ethics Committee (No 263/18). All patients signed informed consent after reading a detailed information leaflet.

Participants

Two different groups of participants were recruited in the study. Four of the participants were recruited from the Musician's clinic of the 1st Department of Otolaryngology at the National & Kapodistrian University of Athens. Inclusion criteria included age between 18-45, a history of occupational exposure to music for at least the past five years, and a reported history of an average of at least 15 sessions per month (a session was considered either a live performance, a music rehearsal, or attendance to a live music event). The Non-musician group (NMG) consisted of six participants of the same age range with no history of occupational noise exposure or use of a musical instrument or extensive and habitual exposure to leisure noise due to attendance of music events and clubs. The age limit of 45 was chosen in order to exclude age as a confounder in the analysis. All participants had normal PTA and extended high frequency pure tone audiometry (EHF-PTA) as well as normal tympanometry and distortion product otoacoustic emissions (DPOAEs) to ensure normal middle ear functions and OHC integrity.

Exclusion criteria for both groups were suspected or known diagnosis of inner ear pathology including age-related or noise induced hearing loss, fluctuating hearing loss, Meniere disease or endolymphatic hydrops, evidence of acute or chronic otitis media or otitis externa on examination or a history of middle ear pathology and/or surgery (history of ventilation tubes allowed). Participants with somatosensory or pulsatile tinnitus were also excluded. Finally, participants should be willing to expose themselves to a maximum comfortable level of music (as chosen by each individual subject) for 30 minutes. Patients not willing to undergo this procedure were excluded from the study. Additionally, patients who considered maximum comfortable levels for listening to music without annoyance below 83 dB SPL were also excluded.

Music track creation and sound presentation

When listening to music, levels of sound actually reaching the ear can significantly vary due to different

factors including device used, use of ear phones and their quality and type of music. A set of .wav files were therefore created with modified music (see below) to ensure standardized and constant sound exposure, in terms of intensity. Modifications described below were made by a sound engineer, aiming towards the creation of a set of music tracks with two characteristics (i) reliability of noise output, (ii) constant levels of output for all tracks chosen and for the whole duration of music exposure. This means that participants were exposed to the same constant levels of sound, independent of the tracks they chose to listen. The tracks were presented via earphones connected to the same laptop always under the same conditions, in an audiological booth. Material consisted of 56 modified tracks, with a total duration of more than 2 hours. Participants were asked to determine the maximum comfortable level of sound and following this, to choose music tracks out of the given list for a period of 30 minutes.

The procedure for the development of the modified music tracks material was the following: Song compilation, selection of a 30 sec part from a song and a white noise sample (-10 dB RMS), playback of both samples via a Dell laptop and a headphone set (Audio Technica ATH 40x), recording of the selected samples using a dummy head (with Countryman Isomax II omni inside) at a pro tools DAW (with Metric Halo 2882 sound device). Following these, the samples were initially recorded at the maximum level (100%) of the laptop's output and afterwards using in total eight values (from 100% to 30%) for monitoring the volume reduction at a step of 10%.

For the validation, playback of the recorded samples was conducted via a second pro tools system (Metric Halo ULN 8 sound device), using the same headphone set (Audio Technica ATH 40x). Headphone sets were placed on the dummy head (Countryman Isomax II omni) and routing to the first Pro Tools system preserving the same gain stage, so that samples are recorded at the exact same dB SPL level with the first samples. Terrasonde audio toolbox's microphone was placed at the dummy head to measure in dB SPL scale. Upon completion of the previous procedure, the accurate adjustment of all tracks was performed based on the dB RMS level of the reference tracks (-17dB), using VU meter plug in and pro tools RTAS plug in "Gain". Finally, a listening test was performed for all tracks to validate via the system (2 pro tools systems, dummy head, headphones, audio toolbox) the dB RMS and dB SPL levels simultaneously.

All participants were asked to define the maximum acceptable comfortable level, in which they would feel comfortable to listen to music for 30 minutes. Only participants who chose levels over 83 Db SPL were considered eligible for the study. ABR and OAE recordings were conducted before and immediately after (within 5 minutes) of the exposure to music.

Audiological investigations

Pure tone audiometry (PTA) and extended high frequency pure tone audiometry (EHF-PTA)

PTA and EHF-PTA thresholds were recorded using the Interacoustics Affinity Suite, according to the ASHA guidelines. PTA was conducted at 0.25, 0.5, 1, 2, 3,4, 6 8 kHz and EHF-PTA at 9, 10, 11.2, 12.5 and 14 KHz.

Otoacoustic emissions

All patients underwent distortion product (DPOAE) otoacoustic emissions using the interacoustics titan ABRIS 440 device. Maximum residual noise was set to 30 dB SPL. DPOAEs were recorded in the range between 1 and 6 KHz. DPOAEs were measured at a f2:f1 ratio equal to 1.22. Artefact-free averaging was conducted at each f2 frequency for 20 seconds to allow the noise at each frequency to reach sufficiently low levels. Testing was conducted for 5 minutes per ear across the frequency range and results were continuously displayed and stored in a DP-Gram for further analysis. OAEs were considered present and normal according to normative data.

Auditory brainstem response

ABRs were recorded in a sound proof booth. An amperemeter was used to ensure that resistance in all electrodes was below 3KΩ. Stimuli were 100-μs diotic clicks high-pass filtered at 1.5 kHz and low-passed at 33 Hz and were presented in alternating polarity. Because of the low-pass characteristic of the ER3A inserts, the stimulus delivered to the ear had a restricted bandwidth with a spectral plateau from about 1.5 to 3 kHz. Clicks were presented at two different rates: 33 and 44 clicks/second, at 90dB nHL. In each rate, 4,000 clicks were presented. The two presentation rates were analyzed separately. Annotation of peak and trough of each wave was made manually. The peak and trough of waves were defined as local maxima and minima and amplitude was automatically calculated by the software based on the annotation in milliseconds. Latencies were also calculated based on the annotation of each wave's peak. Amplitudes and latencies for waves I, III and V were extracted.

Statistical analysis

Outcome measures included SNRs for DPOAEs and wave amplitude and latency for ABR. SPSS v.16.0 was used for statistical analysis. Paired t test after assumptions test and alternatively Wilcoxon sign rank test was used to compare outcomes before and after exposure to music. The level of statistical significance was set to 0.05.

RESULTS

In total, 10 patients (20 ears) participated in this study, 5 of which were males and 5 females. Mean age was 34.1 years old (±7.31).

Distortion product otoacoustic emissions

There was a statistically significant decrease in the DPOAE SNR measurements after noise exposure in all 10 participants, with the exception of 1 KHz. Substantial changes were observed in SNRs before and after exposure. SNRs were decreased by 16% in 1 KHz, 25.3% in 1.5 KHz, 18.2% in 2 KHz, 17.4% in 3 KHz, 21.4% in 4 KHz, 22.1% in 5 KHz and 17.9% in 6 KHz. SNRs and p values are summarized in Figure 1.

No differences were identified between left and right ears in terms of DPOAE SNRs in any of the frequencies tested. Moreover, no statistically significant differences were found between musicians and non-musicians, neither before nor after noise exposure, showing that the two groups did not significantly differ in the way they reacted in exposure to music, with the exception of 1 KHz (difference of -4,17 vs. 3.97 respectively, p=0.02).

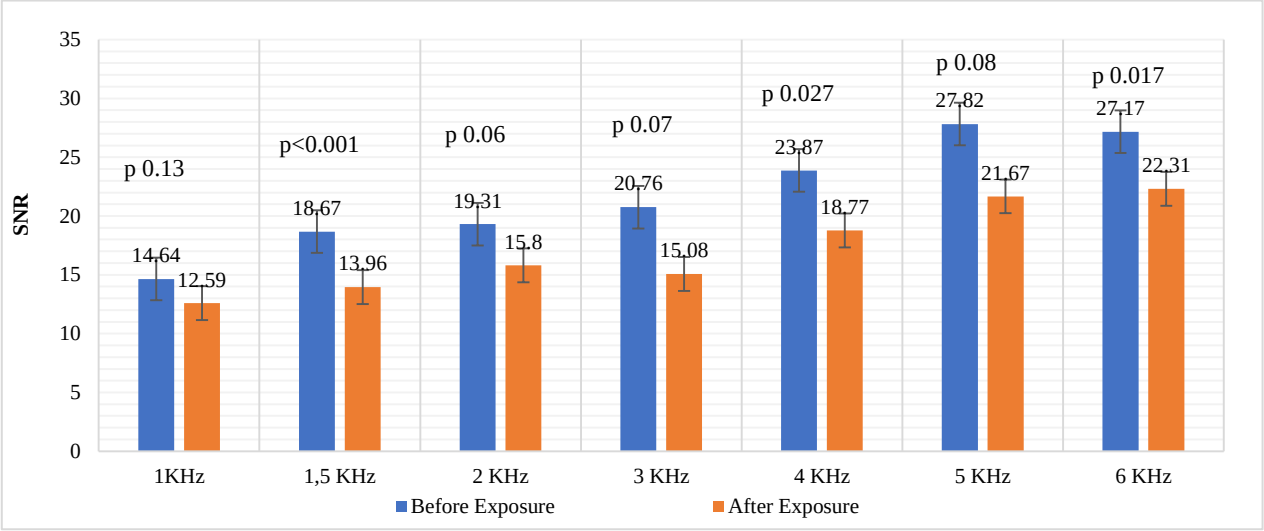

Figure 1: DPOAE before and after noise exposure.

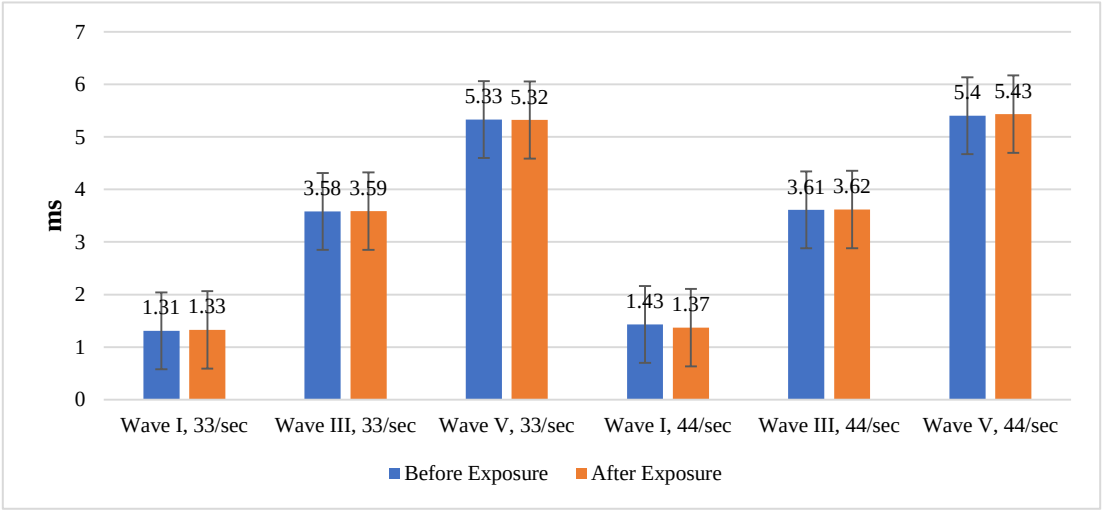

Figure 2: Wave latencies.

ABR wave latencies

No differences were found between left and right ears neither before (baseline) nor after exposure to music, in any of the waveforms, at both presentation rates (33/sec and 44/sec). Additionally, no differences were found between musicians and non-musicians with the exception of in wave I latency at a presentation rate of 44/sec (p=0.036). In all other comparisons, no statistically significant differences were found.

All values of wave latencies before and after exposure to music were not statistically significant for waves I, III and V. Similarly, differences before and after noise

exposure were not found to be statistically significant. Results are summarized in Figure 2.

ABR amplitudes

No differences were observed between left and right ears, both at baseline and after noise exposure. Likewise, no statistically significant differences were observed before and after exposure to music in any wave amplitude in both presentation rates.

Results are presented in Figure 3. However, wave I amplitude was significantly smaller in musicians compared to non-musicians at both 33/sec and 44/sec rates at baseline (p=0.011 and 0.025, respectively). Differences in wave I did not remain significant after exposure (p=0.06 for the 33/sec rate and 0.087 for the 44/sec rate). There were no statistically significant differences in the rest of the wave amplitudes between musicians and non-musicians at both rates.

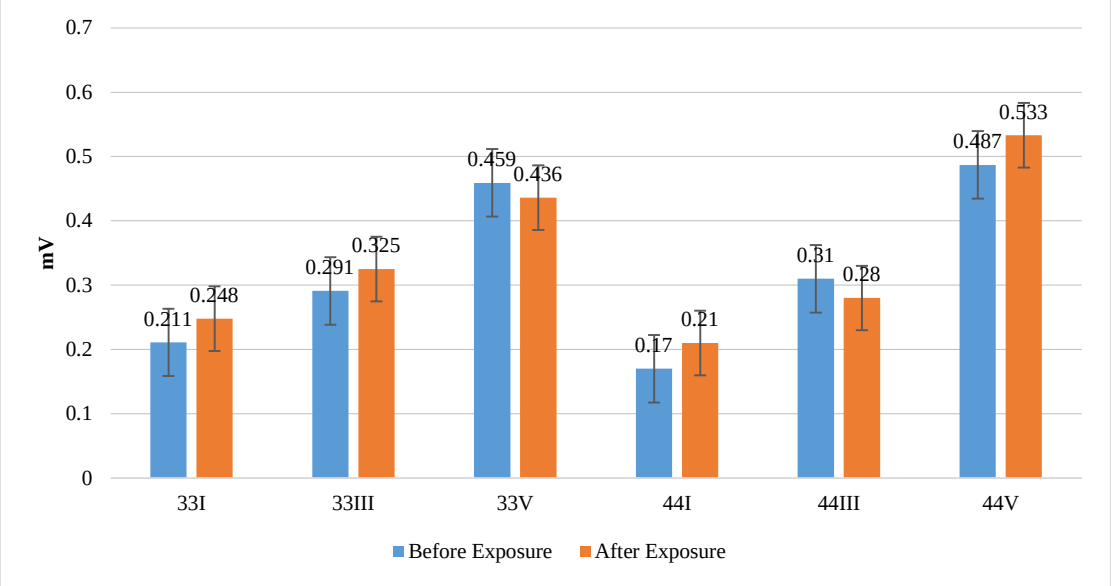

Figure 3: Wave amplitudes.

DISCUSSION

TTS is considered a transient noise related reduction in hearing thresholds, which recover to baseline (pre-exposure) levels. Factors influencing TTS include the type of insult or trauma, the intensity and duration of the insult (single vs repeated and short vs long exposures), and the stimulus type (impulse/impact sound or continuous noise including wide or narrow-band noise).[5] Several cochlea components are involved, including Inner and Outer Hair Cells and especially their stereocilia.

Cochlear synaptopathy has been introduced as a concept by Kujawa and Liberman, based on their findings in mice exposed to loud sounds.[8] Cochlear synapses were identified as the potential primary target for NIHL, since a big proportion of them was lost, even in sound exposure inadequate to induce OHC loss. Initially, they observed that in mice with moderate but Permanent Threshold Shift, OHC loss was much less dramatic compared to loss of synapses.[12] In another study, noise was titrated in level and duration in order to produce a large TTS without OHC loss.[8] Hearing levels were estimated by using OAEs and ABRs and they were found to return to normal. Up to 8 weeks after exposure, it was found that, although OHC were intact, loss of IHC synapses was stable at a level of 40%. Consequently, synaptic loss was suggested as an initial step in the NIHL pathophysiology, and also synapses could be characterized as more vulnerable to noise, compared to OHCs.

These findings were confirmed in other animal studies (mice, guinea pigs, chinchillas and rats). More than forty studies were included in a recent review by Hickox et al, who concluded that loss of ribbon synapses exceeded 50% in majority of the studies with normal DPOAE thresholds at baseline.[11] They also concluded that high threshold low-spontaneous rate fibers, which represent approximately 40% of the synaptic population, are more vulnerable to noise. These fibers have larger dynamic range and reduced susceptibility to excitatory masking by continuous noise stimuli.[13] They are thus considered to be responsible for coding suprathreshold sounds, for fine temporal precision at suprathreshold level, and for discrimination in noise. The latter was the basis for the hypothesis that the existence of auditory symptoms such as difficulties in discrimination in noise and tinnitus in individuals with normal PTA, described as hidden hearing loss, could be explained by underlying cochlear synaptopathy.

Most animal studies examined the consequences of acute exposure to loud sounds, in a range between 92 and 106 dBs with duration of up to 2 hours. Only two studies[14,15] examined the effect of aging to synapses population, which progresses from youth to old age. They observed synpaptic loss throughout the cochlea long before age-related changes in thresholds or hair cell counts. It is obvious that this kind of exposure is not applicable in humans, Therefore, the development of a standardized and replicable tool, allowing constant, stable and measurable in dB SPL exposure to music is a major outcome of this study. Moreover, exposure to maximum comfortable levels for 30 minutes was found to result statistically significant changes in DPOAEs SNRs, suggesting a possible pattern of noise exposure in humans.

The term hidden hearing loss was introduced by Schaette and MacAlpline, in a study comparing electrophysio-logical responses (amplitude of ABR waves I, V and their ratio) in two groups of normal hearing women with and without tinnitus.[9] Statistically significant differences between the two groups were considered as sign of underlying synaptopathy and were correlated to the presence of tinnitus. Since then, the terms HHL and CS have been used interchangeably, however should be considered highly correlated but not identical, since they refer to clinical and pathophysiological level respectively.

In many studies examining cochlea synaptopathy, ABR were used as possible outcome measure. Auditory brainstem responses (ABRs) are well implemented in the every-day clinical practice providing a noninvasive estimation of hearing thresholds. Additionally, ABR waveforms have been correlated with certain anatomical sites along the auditory pathway. More explicitly, wave I captures the synchronous firing of auditory nerve (AN) fibers in the spiral ganglion cells, wave III is considered to express action in the cochlea nucleus and Wave V is thought to be generated by medial superior olive primary cells projecting onto the lateral lemniscus and inferior colliculus. Therefore, synaptic loss was expected to reduce the amplitude of wave I.[16]

In a recent systematic review, findings pro and against the cochlea synaptopathy hypothesis were controversial.[17] Liberman et al identified significant differences in AP/AP ratio in electrocochleography between two groups of young adults with and without history of noise exposure. Bramhall et al also identified difference in ABR wave I amplitude between veterans with and without exposure to gunshots.[18] In another study, 26 young adults with normal PTA were divided based on their frequency of attendance in loud recreational activities.[19] Envelope Following Response, an electrophysiological method not commonly used in clinics, which is part of ABR and reflects sustained neural activity integrated over a population of neural elements. Again, differences were identified between the groups.

Cumulative noise exposure was also used as a factor to divide groups in two additional studies. Stamper and Johnson used a structured questionnaire targeting to evaluate noise exposure during the past 12 months and found reduced wave I amplitude in the group with history of higher exposure.[20] Similarly, Valderrama et al estimated life time exposure to noise, reaching the same conclusion (wave I amplitude reduced in relation to noise).[21]

In contrary, other studies failed to confirm these findings and question the ability to detect or even the existence of synaptopathy. Ridley et al investigated the differences between normal hearing and patients with high frequency PTA notch, concluding that no differences were identified in amplitude.[22] However, the age range was very large (18-64 years old), probably affecting the results. Prendergast et al presented two studies in 126 and 30 participants concluding as well that noise exposure history was not correlated to wave I amplitude, even in the sub groups with more and less nose exposure.[23,24] However, the way that noise history was estimated was based on information retrospectively collected by the patients, potentially leading to memory bias. Moreover, most participants were students with mean age 23, which means that their cumulative noise exposure might not be enough to induce cochlea synaptopathy

Majority of human studies focus on the effect of chronic rather than acute noise exposure. In a recent review of human studies targeting synaptopathy, findings from studies using ABR had controversial results. Most of the studies used history of noise exposure, either for the whole life time or during the past 12 months before measurements.[17] Several studies, concluded that wave I was significantly reduced in groups with increased noise exposure, whereas others did not.[18.21-23,25-27] However, most of these studies suffered from methodological flaws and diversities, including variance in measurement and settings, reduced synaptic vulnerability in humans compared to animals, limited range of exposure thresholds inducing either detectable or clinically apparent synaptopathy, existence of synaptopathy in normal population (and thus inability to detect differences) and confounding effect of co-existing OHC functional insufficiencies.[17]

Otoacoustic emissions

According to the findings of our study, OAEs were reduced in a statistically significant level before and after exposure to music. This finding could be evaluated as a stable trend, indicating that music exposure at a level above 83 dB SPL for 30 minutes is be adequate to induce detectable alterations in the cochlea and more specifically to OHC function. Significance of this finding is twofold: on top of the establishment of the described procedure (exposure to music with standardized and processed music files) it can also be an indicator that musicians and non-musicians seem to react in a similar way in the same

exposure. Possible use of this method in future studies might increase the chances of successful translational research, given that noise exposure patterns largely used in animal studies (exposure to levels of noise exceeding 90 dB, usually for a period of two hours) is not applicable to humans.

ABR waveforms

No differences were observed in wave latencies before and after Wave I amplitude differed significantly between musicians and non musicians for both presentation rates. This finding though is consistent with several studies in the literature and has been correlated with possible existence of cochlea synaptopathy due to chronic noise exposure. No statistically significant differences were found between the amplitudes of wave I before and after noise exposure in the study population. According to the hypothesis of the study, identification of significant reduction could be indicative of acute changes of the synaptic function, in the context of cochlea synaptopathy. Absence of this finding though, does not necessarily exclude the possibility of underlying synaptopathy. One reason for not detectable reduction could be inadequate exposure duration. Participants were exposed for 30 minutes, in contrary to 2 hours which is the most common duration in animal studies. Moreover, exposure levels varied among individuals and this could influence the overall responses.

The question of whether acute effects of noise are both present and detectable remains and further research is needed towards this direction.

CONCLUSION

A newly introduced procedure of stable and fully measurable in dB SPL exposure to music has induced statistically significant reduction of DPOAE SNRs after 30 minutes in maximum comfortable levels. ABR latencies and amplitudes did not differ in a statistically significant level, however further research is needed to investigate possible acute effects of noise.

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