A pilot study of cochlear synaptopathy in military recruits

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ABSTRACT

Speech-in-noise difficulties could be associated with cochlear synaptopathy. In this study, we investigated speech-in-noise and frequency selectivity in young adults exposed to impulse noise.

Ten young military recruits with exposure to firearm noise and 10 non-exposed control subjects were recruited. Subjects presented with normal hearing thresholds and presence of distortion product otoacoustic emissions. The Noise Exposure Structured Interview was used to quantify noise exposure. Speech-in-noise and frequency selectivity tests were carried in the better ear. While speech-in-noise performances were not different between groups, frequency selectivity at 4 kHz was significantly worse in the firearm-exposed group ($p = .005$). A significant correlation was found between noise exposure and frequency selectivity ($p = .015$), but not with speech- in noise performances.

These results suggest that young military recruits with firearm exposure present worse frequency selectivity than peers without noise exposure, despite normal hearing thresholds and the presence of otoacoustic emissions. Impairment of frequency selectivity in presence of normal hearing could reflect hidden damage to inner ear cells or auditory nerve fibers. This procedure could allow the detection of cochlear synaptopathy.

INTRODUCTION

Noise exposure is one of the most predominant cause of hearing loss. In industrialized countries, it is estimated that noise-induced hearing loss may affect 10 to 16% of individuals [1, 2]. While noise exposure in the workplace is commonly known to produce auditory thresholds shifts, recreational and firearms noise exposure can also lead to hearing loss [3].

In the animal model, recent studies have shown that exposure to loud noise (e.g., between 70 and 100 dB SPL) can lead to an injury of presynaptic ribbons in the inner hair cells with a subsequent preferential loss of low-spontaneous-rate auditory fibers (low-SR fibers) [4, 5, 6]. This phenomenon has been referred to as cochlear synaptopathy. It has been demonstrated that damage to these fibers does not affect hearing thresholds and integrity of the outer hair cells [4] since low-SR auditory fibers are not involved in the coding of the amplitude of low level sounds [7, 8]. Suprathreshold deficits (e.g., abnormal speech-in-noise performances, reduced ABR wave I amplitude) have been associated with cochlear synaptopathy [e.g., 9, 10, 11].

Most studies conducted with human participants have investigated recreational noise exposure [e.g., 12, 13, 14). Data from animal studies demonstrate that exposure to impulse noise may cause an injury of presynaptic ribbons, and that this damage might be more focalized than exposure to continuous noise [15, 16].

Suprathreshold auditory performances in humans with impulse noise exposure, suspected to present with signs of cochlear synaptopathy, have not been extensively investigated. Bramhall and colleagues' study of young military veterans showed a reduced Auditory Brainstem Response (ABR) wave I suprathreshold response in participants with higher reported noise exposure [17]. Speech-in-noise and auditory filters were not evaluated.

Therefore, a research gap remains on the association between suprathreshold auditory performances and impulse noise exposure in humans with normal hearing thresholds. The goal of this study was to determine whether participants with firearm noise exposure and participants without noise exposure differed in terms of speech-in-noise performances and auditory filters. We also aimed at measuring any association with lifetime noise exposure.

MATERIALS AND METHODS

The Ethics Committees of the Center for Interdisciplinary Research in Rehabilitation of Greater Montreal (CRIR), the Faculty of Medicine of the University of Montreal and the Royal Military College Saint-Jean (RMCSJ) approved the study protocol. All participants signed a consent form prior to being included in the study.

Participants

Two groups of male participants, aged between 18 and 25 years, were selected. Ten military recruits with exposure to firearms noise and 10 control participants without firearms exposure were recruited. Participants from both groups presented with no family history of hearing loss, and no history of ear surgery, use of ototoxic drugs or neurological disorders. They were required to be of general good health. All participants presented with normal hearing thresholds (equal or better than 20 dB HL) in both ears at all frequencies from 0,25 kHz to 8 kHz. Distortion product otoacoustic emissions were present for all participants from 2 kHz to 8 kHz with a signal/noise ratio (SNR) of at least +3 dB.

Procedures

Military participants (firearm noise-exposed group) were selected at the Military College Saint-Jean and the control group were recruited among students from the University of Montreal. All participants were recruited in the province of Quebec, Canada. Prior to the beginning of the study, participants were asked to sign a consent form and information regarding the study was provided to them. A medical questionnaire was used to exclude participants with family history of hearing loss, and any ear disease history and health condition related to the auditory system.

Participants were scheduled for an assessment session at the School of Speech-Language Pathology and Audiology of the University of Montreal, and at the Military College Saint-Jean. Bilateral otoscopy was carried out in order to exclude participants with abnormalities in the external ear canal and tympanic membrane. Hearing testing was conducted in a single-walled (for sessions at the Military College of Saint-Jean) or double-walled (for sessions at University of Montreal), soundproofed and electric shielded room. The better ear (based on the results of pure-tone audiometry and distortion product otoacoustic emissions tests) was selected for the study.

Lifetime noise exposure

For all participants, lifetime noise exposure was qualified and quantified with the Noise Exposure Structured Interview (NESI) [18]. Participants were asked to list recreational and occupational noise exposure. For each exposure, noise levels were estimated based on communication difficulty (e.g., must raise the volume of his voice to discuss with someone at 1 m distance). The typical duration and frequency of occurrence of exposure were also obtained. To help participants recall previous noise exposure with accuracy, lifespan was segmented based on exposure habits. Firearms noise exposure was quantified for weapons of low-caliber (.22 and .17) and all other hand-held firearms. Information on the usage of hearing protection devices (HPD) was also gathered. For each participant, noise units were obtained for recreational noise exposure ($NESI_{Recreational}$), occupational noise exposure ($NESI_{Occupational}$), firearms noise exposure ($NESI_{Firearms}$) and total noise exposure ($NESI_{Total}$). The noise units are linearly related to the total energy of exposure above 80 dBA. One unit is equivalent to one working year (2080 hours) of exposure to 90 dBA.

Pure-tone audiometry

Air-conduction pure-tone thresholds were obtained bilaterally with an Otometrics Madsen Astera 2 (Taastrup, Denmark) or an Interacoustics AC-40 (Middelfart, Denmark) audiometer. Assessment was carried with Telephonic TDH39p headphones (0,25 – 8 kHz) and Sennheiser HDA200 (9 – 16 kHz). The Hughson-Westlake procedure, described by Carhart and Jerger (1959) [19], was used to obtain hearing thresholds at 0.25, 0.5, 1, 2, 3, 4, 6, 8, 9, 10, 11.2, 12, 14 and 16 kHz. Participants were required to present with normal hearing thresholds in at least one ear between 0.25 and 8 kHz (equal to or better than 20 dB HL).

Distortion product otoacoustic emissions (DPOAEs)

The ILO.v6 DP Echoport ILO 292 was used for the measurements of distortion product otoacoustic emissions (DPOAEs) in both ears. A probe containing two sound sources and a microphone was inserted in the ear of the participant. The equipment was set up with a f2/f1 ratio of 1.22 and presentation levels L1/L2 of 65/55 dB SPL. DPOAEs were measured at 2, 3,

4, 5, 6, 7 and 8 kHz. Selected participants should have exhibited presence of DPOAEs (+3 dB SNR and absolute amplitude > 0 dB SPL) for each of the aforementioned frequencies in at least one ear with hearing thresholds equal or better than 20 dB HL (0.25 – 8 kHz).

Tympanometry

The Interacoustics AT235 tympanometer (Middelfart, Denmark) was used for tympanometry measures. The middle ear compliance and pressure were measured by a 1,500 ms 226 Hz probe tone. Participants were excluded from the study if they were classified with results different than type A in both ears, based on Jerger's classification: middle ear compliance <0.2 cc or middle ear pressure > -150 daPa (decaPascals) [20].

Speech-in-noise

A French speech-in-noise test (the FrMatrix) was used to assess speech recognition performance in noise [21]. The FrMatrix contains a total of fifty words separated in five categories (Noun, verb, number, object and color). Using an adaptative method, participants were asks to repeat 20 random 5-word sentences in the presence of a stationary long-term average speech spectrum noise. The SNR for which participants obtained a speech recognition performance of 20%, 50% and 80% was measured.

Frequency selectivity

Detection thresholds in the presence of masking noise were assessed with the *Masking Threshold* software elaborated by the Audiology Research Laboratory (University of Ottawa, Canada). This software is designed to carry out notch-noise masking experiments, using an adaptative mixed-frequency Bekesy threshold search method. The procedure used was proposed by Hétu & Tran Quoc (1992) [22]. Participants were asked to press a response button when a pure-tone signal was heard through the notch-noise. Masked thresholds were obtained at 1 and 4 kHz. To derive auditory filter shapes and bandwidth, masked thresholds were inputted into roex fitting algorithm, *Shape 1.0* (University of Montreal, Canada). The equivalent rectangular bandwidth (ERB) was calculated for 1 and 4 kHz.

Statistical analysis

Statistical analyses were performed with SPSS V25 (IBM, 2017). Pure-tone threshold averages were calculated for each participant by averaging the hearing thresholds of the better ear from 0.5 to 4 kHz (PTA_4) and from 9 to 16 kHz (PTA_{EHF}). $DPOAE_{mean}$ was calculated by averaging the amplitude in dB SNR of DPOAE in the better ear across all frequencies (2 – 8 kHz).

ANOVA tests were carried out to compare noise-exposed and control groups regarding age, pure-tone averages (PTA_4 and PTA_{EHF}) and $DPOAE_{mean}$. Then, repeated measures ANOVAs with a Greenhouse-Geisser correction were computed for hearing thresholds (0.25 – 16 kHz), DPOAE amplitudes (2 – 8 kHz), speech-in-noise SNR (20%, 50% and 80%) and ERB (1 and 4 kHz).

Finally, Pearson correlations between lifetime noise exposure ($NESI_{Total}$ and $NESI_{Firearms}$) and auditory outcomes were computed.

RESULTS

Participants

Table 1 shows group comparisons for age, audiometric and DPOAE outcomes. While we aimed at selecting participants of similar age between groups, noise-exposed participants were significantly younger (18.7 ± 1.3) than control participants (22.7 ± 2.8) [F (1, 18) = 16.705, p = .001]. Although statistically significant, this age difference is not expected to affect auditory outcomes, and all participants respected the inclusion criteria (to be between 18 and 25 years of age).

As per the inclusion criteria, participants in both groups presented with normal hearing thresholds (equal or better than 20 dB HL) in the better ear for frequencies from 0.25 kHz to 8 kHz and DPOAE amplitudes equal or better than 3 dB SNR at each tested frequency (2 – 8 kHz). Extended high frequencies were also measured from 9 to 16 kHz.

Regarding hearing thresholds (0.25 to 16 kHz), a repeated measures ANOVA with a Greenhouse-Geisser correction showed no interaction between groups and frequencies (F (2.365, 42.572) = .506, p = .637). Additionally, hearing thresholds were not significantly different across tested frequencies (F (2.365, 42.572) = 1.290, p = .288) and for groups (F (1, 18) = .133, p = .720). Therefore, noise-exposed and control-group participants presented with similar hearing thresholds. As for DPOAE amplitudes, no interaction between groups and frequencies was observed (F (2.584, 46.517) = 1.335, p = .275). While a significant difference in frequencies was observed (regardless of groups) (F (2.584, 46.517) = 14.432, p < .001), no significant difference was obtained in DPOAE amplitudes between groups (F (1, 18) = .298, p = .592).

Finally, ANOVAs did not show significant differences between groups regarding PTA_4 (F (1, 18) = 3.102, p = .095), PTA_{EHF} (F (1, 18) = .044, p = .836) and $DPOAE_{mean}$ (F (1, 18) = .134, p = .719).

Table 1: Mean, standard deviation and group comparisons for age, PTA and DPOAE

	Noise-exposed (n=10)		Control (n=10)		p-value
Variable	**Mean**	**SD**	**Mean**	**SD**	
Age (years)	18.70	1.25	22.70	2.83	**.001**
PTA_4 (dB HL)	2.75	2.49	0.50	3.18	.095
PTA_{EHF} (dB HL)	6.25	17.30	4.97	8.65	.836
$DPOAE_{mean}$	7.54	4.24	6.95	2.81	.719

Noise exposure

Figure 1 shows units of total noise exposure ($NESI_{Total}$) and firearms noise exposure ($NESI_{Firearms}$). Total lifetime noise exposure ranged from 0.80 to 19.78. Units of firearms noise exposure were 0 in the control group and ranged from 1.02 to 17.88 in the noise exposed group. Control group presented with significantly lower noise exposure units for $NESI_{Total}$ (F (1, 18) = 10.199, p = .005) and $NESI_{Firearms}$ (F (1, 18) = 12.129, p = .003).

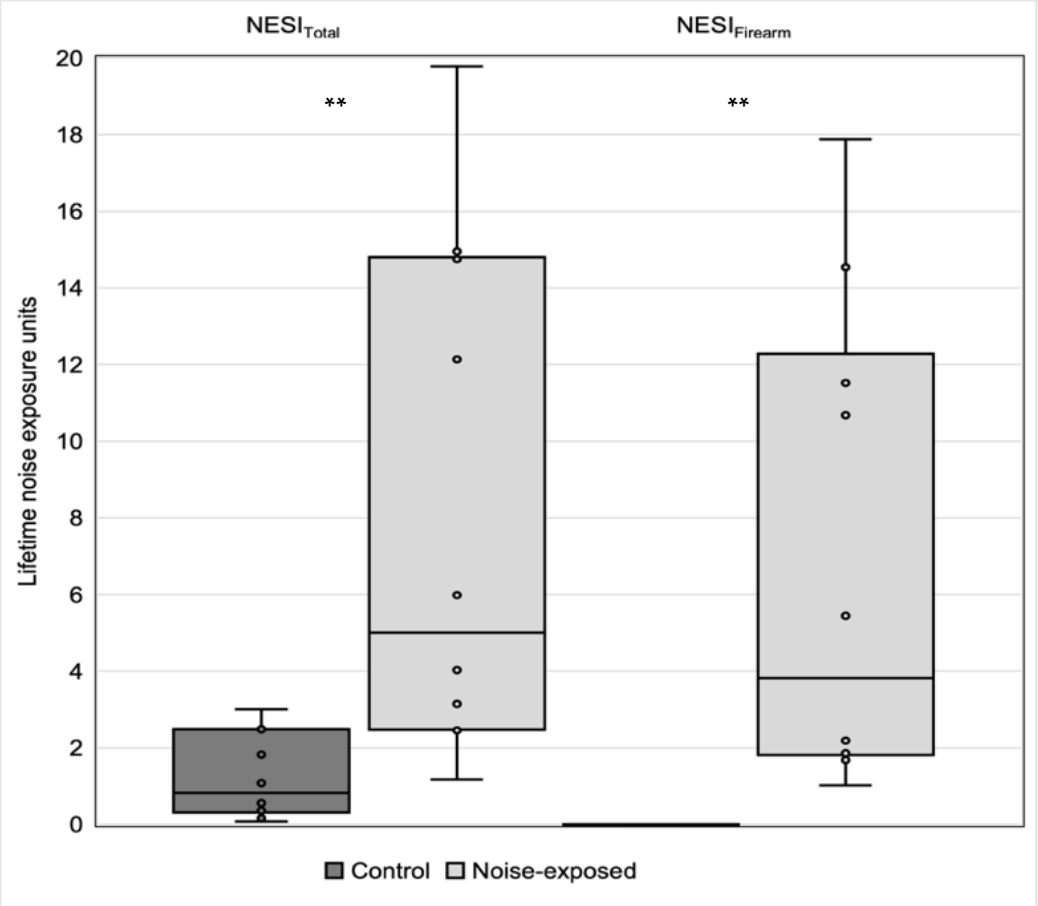
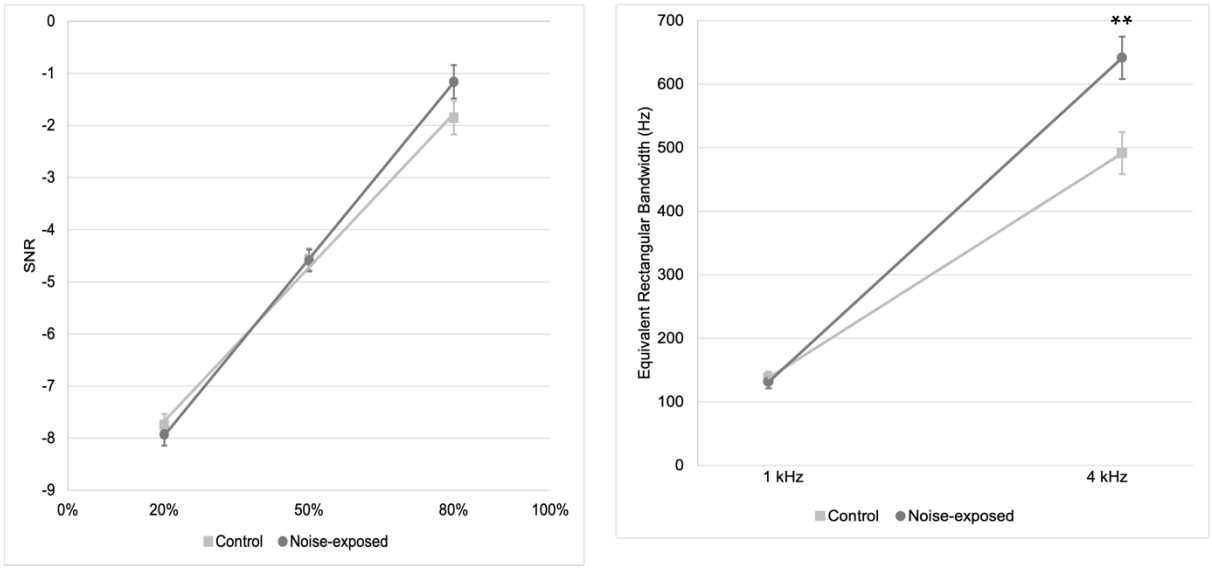


Figure 1: Lifetime noise exposure units for $NESI_{Total}$ and $NESI_{Firearms}$. Error bars represent standard error. ** $p < .01$

Speech-in-noise and equivalent rectangular bandwidth

A repeated measures ANOVA with a Greenhouse-Geisser correction showed no interaction between groups and speech identification scores ($F(1.644, 29.598) = 2.386$, $p = .118$). No significant difference was obtained in SNRs between groups ($F(1, 18) = .386$, $p = .542$). Figure 2, panel A, shows SNRs for each speech identification score. On the other hand, groups and ERB frequency showed a significant interaction ($F(1, 18) = 11.678$, $p = .003$). While ERB was similar between groups at 1 kHz ($F(1, 18) = 0.096$, $p = .761$), the noise-exposed group displayed a larger ERB at 4 kHz ($F(1, 18) = 10.099$, $p = .005$).



Panel A – Speech-in-noise

Panel B – Auditory filters

Figure 2: Panel A: Speech-in-noise SNR for a 20%, 50% and 80% speech identification scores. Panel B: Equivalent rectangular bandwidth for 1 and 4 kHz. Error bars represent standard error. ** $p < 0.01$

Association between noise exposure and auditory outcomes

A Pearson correlation matrix between lifetime noise exposure and pure-tone audiometry, DPOAE, speech-in-noise and auditory filters was obtained for all participants (n=20). No significant correlations between $NESI_{Total}$ and $NESI_{Firearms}$ and most of the aforementioned variables were found (see Table 2). A significant correlation was observed between both units of lifetime noise exposure and equivalent rectangular bandwidth at 4 kHz. A higher unit of noise exposure indicated a larger auditory filter at 4 kHz.

Table 2: Correlation coefficients (Pearson) between lifetime noise exposure and audiometry, DPOAEs, speech-in-noise and auditory filters in all participants (n=20)

	PTA_4	PTA_{EHF}	$DPOAE_{mean}$	FrMatrix 20%	FrMatrix 50%	FrMatrix 80%	ERB 1 kHz	ERB 4 kHz
$NESI_{Total}$	.409	-.172	-.260	.156	.304	.109	-.035	.527*
$NESI_{Firearms}$	.396	-.158	-.228	.101	.211	.158	-.057	.534*

$NESI_{Total}$, total lifetime noise exposure; $NESI_{Firearms}$, firearms noise exposure; PTA_4, pure-tone average at 0.5, 1 and 2 kHz; PTA_{EHF}, Pure-tone average from 9 to 16 kHz; $DPOAE_{mean}$, mean of DPOAE amplitudes from 1 to 8 kHz; FrMatrix, SNR for 20, 50 and 80% speech identification scores; ERB, equivalent rectangular bandwidth at 1 and 4 kHz, * $p < .05$

DISCUSSION

Group comparisons

The aim of our study was to determine whether participants with firearms noise exposure and participants without noise exposure differed in terms of speech-in-noise performances and auditory filters. We also aimed at measuring any association with lifetime noise exposure. All participants were young males, with normal hearing thresholds (0.25 – 8 kHz) and presence of DPOAEs (2 – 8 kHz). The data showed that although noise-exposed participants were slightly (but significantly) younger, they presented with similar hearing thresholds on conventional frequencies and extended high frequencies (0.25 – 16 kHz) than control participants. Both groups also displayed similar DPOAE amplitudes. Lifetime noise exposure ($NESI_{Total}$ and $NESI_{Firearms}$) was significantly higher in noise-exposed participants, which is what we aimed for. It should be noted that although group means were significantly different for $NESI_{Total}$, some participants in both groups might have presented similar units of lifetime noise exposure (e.g., the lowest unit of exposure in the exposed group was 1.17 and the highest in the control group was 3.01). No participants in the control group were exposed to firearms noise.

Speech-in-noise and auditory filters

In this study, we investigated speech-in-noise, which is hypothesized to be affected in individuals with cochlear synaptopathy [e.g., 13, 23]. We did not observe any difference in the SNR necessary to obtain various speech identification performances (i.e., 20%, 50% and 80%) on an adaptative speech-in-noise test. Also, we did not observe a correlation between speech-in-noise performances and lifetime noise exposure, either $NESI_{Total}$ or $NESI_{Firearms}$.

On the other hand, we did observe a significant difference in width of auditory filters between noise-exposed and control participants. Even if the military recruits in our study where significantly younger than their fellow controls, they presented with larger auditory filters at 4 kHz. This difference was not observed at 1 kHz. A higher lifetime noise exposure was also associated with a larger auditory filter. It is known that auditory filters (i.e., the capacity of frequency selectivity) relies primarily on healthy hair cell's function [24]. It has also been shown that perceptual frequency selectivity is typically normal or near normal for audiometric thresholds up to 30–40 dB HL [25]. In our study, the differences in auditory filters cannot be accounted for by a reduction in hearing thresholds or a damage to outer hair cells (OHC) since both hearing thresholds and DPOAE were within normal. Therefore, we can hypothesize that this alteration in auditory filters related to lifetime noise exposure might be related to a form of hidden hearing loss. This observation has been made in the animal model. While OHC survival was a major contributor of filter widths in noise exposed macaque monkeys, frequency selectivity was broader when there was both OHC and ribbon synapse loss [25]. Without histopathological evidence of cochlear or synaptopathy, we cannot prove that a neural deficit explains the observed dysfunction. However, the current results are consistent with the view that suprathreshold auditory performances may arise from inner hair cells and auditory nerve pathology.

Noise-exposed participants in our study only displayed a significant difference in auditory filters at 4 kHz. This result suggest that cochlear synaptopathy in humans might first manifest itself at this specific frequency. A similar pattern of damage has been known for decades regarding hearing loss (i.e., audiometric notching at 4 kHz) [26]. Interestingly, reduced suprathreshold wave I response elicited by toneburst stimuli in veterans was not confined to the 4 kHz region [17]. It was suggested that participants may have initially experienced noise-induced

synaptopathy confined to the 4 kHz region that spread with time to a broader frequency range. In our study, participants were young military recruits with recent firearms noise exposure, which might explain why auditory filters were affected only in the 4 kHz region. To our knowledge no other human study has investigated auditory filters with a similar procedure in normal hearing participants with and without noise exposure, providing little basis for intra-study comparisons. One study suggested that difference between thresholds in quiet and in noise (the latter measured by the TEN test) might be used as a proxy of cochlear synaptopathy (this proxy was called TINR – Thresholds-in-noise residual) [27]. While a correlation between the TINR and some electrophysiological measures (e.g., wave I amplitude ratio) was found, no association was found with lifetime noise exposure.

The question arises has to why no differences were observed in speech-in-noise performances and that these variables were not associated with lifetime noise exposure. Three hypotheses might explain this lack of significant result. First, this study only included 20 participants (10 per group), which might lead to a too small statistical power to be able to identify differences in speech-in-noise performances. Second, speech-in-noise implies many other cognitive or top-down process (e.g., working memory, language abilities) and young adults might be able to compensate for speech-in-noise impairment [28]. Finally, in this study, we investigated suprathreshold auditory performances in young adults, with a short history of firearms usage. The abnormal auditory filters associated with firearms noise exposure might be a precursor of early cochlear synaptopathy, which does not yet manifest with speech-in-noise performances.

CONCLUSION

Based on the results found in this study, we conclude that auditory filters were significantly larger in young normal-hearing individuals with impulse noise exposure from firearms. Furthermore, larger auditory filters were significantly associated with an increase in the quantity of units of noise exposure, as measure by the Noise Exposure Structured Interview. No significant differences were obtained for speech-in-noise performances between individuals with and without noise exposure. The assessment of detection thresholds in the presence of masking noise, unlike a speech-in-noise task, is much less influenced by cognitive factors, which might account for the different results obtained for both tasks. These results are consistent with the view that suprathreshold auditory deficits may arise from inner hair cells and auditory nerve pathology. Therefore, the measure of auditory filters is a non-invasive procedure that can potentially be used to evaluate cochlear synaptopathy in humans.

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REFERENCES

[1] Dobie, R. A. (2008). The burdens of age-related and occupational noise-induced hearing loss in the United States. Ear and hearing, 29(3), 565-577.

[2] Nelson, D. I., Nelson, R. Y., Concha-Barrientos, M., & Fingerhut, M. (2005). The global burden of occupational noise‐induced hearing loss. American journal of industrial medicine, 48(6), 446-458.

[3] Ivory, R., Kane, R., & Diaz, R. C. (2014). Noise-induced hearing loss: a recreational noise perspective. Current opinion in otolaryngology & head and neck surgery, 22(5), 394-398.

[4] Kujawa, S. G., & Liberman, M. C. (2009). Adding insult to injury: Cochlear nerve degeneration after "temporary" noise-induced hearing loss. Journal of Neuroscience, 29(45), 14077-14085.

[5] Furman, A. C., Kujawa, S. G., & Liberman, M. C. (2013). Noise-induced cochlear neuropathy is selective for fibers with low spontaneous rates. Journal of Neurophysiology, 110(3), 577-586.

[6] Fernandez, K. A., Jeffers, P. W., Lall, K., Liberman, M. C., & Kujawa, S. G. (2015). Aging after noise exposure: Acceleration of cochlear synaptopathy in "recovered" ears. Journal of Neuroscience, 35(19), 7509-7520.

[7] Ruggero, M. A. (1992). Physiology and coding of sound in the auditory nerve. In: Popper A.N., Fay R.R. (eds) The Mammalian Auditory Pathway: Neurophysiology. Springer Handbook of Auditory Research, vol 2. Springer, New York, NY.

[8] Bourien, J., Tang, Y., Batrel, C., Huet, A., Lenoir, M., Ladrech, S., Desmadryl, G., Nouvian, R., Puel, J. L., & Wang, J. (2014). Contribution of auditory nerve fibers to compound action potential of the auditory nerve. Journal of Neurophysiology, 112(5), 1025-1039.

[9] Mejia, J., Dillon, H., Van Hoesel, R., Beach, E., Glyde, H., Yeend, I., Beechey, T., Mclelland, M., O'Brien, A., Buchholz, J., Sharma, M., Valderrama, J., & Williams, W. (2015). Loss of speech perception in noise – causes and compensation. Proceedings of the International Symposium on Auditory and Audiological Research, 5, 205-216. Retrieved from https://proceedings.isaar.eu/index.php/isaarproc/article/view/2015-24

[10] Shi, L., Chang, Y., Li, X., Aiken, S., Liu, L., & Wang, J. (2016). Cochlear Synaptopathy and Noise-Induced Hidden Hearing Loss. Neural Plasticity, 2016.

[11] Barbee, C. M., James, J. A., Park, J. H., Smith, E. M., Johnson, C. E., Clifton, S., & Danhauer, J. L. (2018). Effectiveness of auditory measures for detecting hidden hearing loss and/or cochlear synaptopathy: a systematic review. Seminars in hearing, 39(2), 172-209.

[12] Bharadwaj, H. M., Masud, S., Mehraei, G., Verhulst, S., & Shinn-Cunningham, B. G. (2015). Individual differences reveal correlates of hidden hearing deficits. Journal of Neuroscience, 35(5), 2161-2172.

[13] Liberman, M. C., Epstein, M. J., Cleveland, S. S., Wang, H., & Maison, S. F. (2016). Toward a Differential Diagnosis of Hidden Hearing loss in Humans. PLoS One, 11(9), e0162726.

[14] Guest, H., Munro, K. J., Prendergast, G., Millman, R. E., & Plack, C. J. (2018). Impaired speech perception in noise with a normal audiogram: No evidence for cochlear synaptopathy and no relation to lifetime noise exposure. Hearing research, 364, 142- 151.

[15] Hickman, T., Smalt, C., Bobrow, J., Quatieri, T., & Liberman, M. (2018). Blast-induced cochlear synaptopathy in chinchillas. Scientific Reports, 8(1), 1-12.

[16] Harrison, R. T., & Bielefeld, E. C. (2019). Assessing Hidden Hearing Loss After Impulse Noise in a Mouse Model. Noise & health, 21(98), 35.

[17] Bramhall, N. F., Konrad-Martin, D., McMillan, G. P., & Griest, S. E. (2017). Auditory brainstem response altered in humans with noise exposure despite normal outer hair cell function. Ear and hearing, 38(1), e1.

[18] Guest, H., Dewey, R. S., Plack, C. J., Couth, S., Prendergast, G., Bakay, W., & Hall, D. A. (2018). The noise exposure structured interview (NESI): an instrument for the comprehensive estimation of lifetime noise exposure. Trends in hearing, 22, 2331216518803213.

[19] Carhart, R., & Jerger, J. F. (1959). Preferred method for clinical determination of pure-tone thresholds. Journal of Speech & Hearing Disorders, 24 (4), 330-345.

[20] Jerger, J. J. (1970). Clinical experience with impedance audiometry. Archives of Otolaryngology - Head and Neck Surgery, 92(4), 311–324.

[21] Jansen, S., Luts, H., Wagener, K. C., Kollmeier, B., Del Rio, M., Dauman, R., ... & Van Wieringen, A. (2012). Comparison of three types of French speech-in-noise tests: A multi-center study. International Journal of Audiology, 51(3), 164-173.

[22] Hétu, R., & Tran Quoc, H. (1992). Caractérisation des filtres auditifs centrés à 250, 500, 1000, 2000, 3000 et 4000 Hz au moyen d'une procédure de mesure adaptée aux contraintes d'examen clinique. Journal de physique, 2, C1-269 – C261-272.

[23] Shehorn, J., Strelcyk, O., & Zahorik, P. (2020). Associations between speech recognition at high levels, the middle ear muscle reflex and noise exposure in individuals with normal audiograms. Hearing Research, 392, 107982.

[24] Plack, C. J. (2018). The sense of hearing. Chapter 5: Frequency selectivity. Routledge.

[25] Burton, J. A., Mackey, C. A., MacDonald, K. S., Hackett, T. A., & Ramachandran, R. (2020). Changes in audiometric threshold and frequency selectivity correlate with cochlear histopathology in macaque monkeys with permanent noise-induced hearing loss. Hearing research, 398, 108082.

[26] Wilson, R. H., & McArdle, R. (2013). Characteristics of the audiometric 4,000 Hz notch (744,553 veterans) and the 3,000, 4,000, and 6,000 Hz notches (539,932 veterans). Journal of Rehabilitation Research & Development, 50(1).

[27] Ridley, C. L., Kopun, J. G., Neely, S. T., Gorga, M. P., & Rasetshwane, D. M. (2018). Using thresholds in noise to identify hidden hearing loss in humans. Ear and hearing, 39(5), 829.

[28] Schneider, B. A., Daneman, M., & Pichora-Fuller, M. K. (2002). Listening in aging adults: from discourse comprehension to psychoacoustics. Canadian Journal of Experimental Psychology/Revue canadienne de psychologie expérimentale, 56(3), 139.