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ORIGINAL RESEARCH

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Extensive hearing loss induced by low-frequency noise exposure

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Abstract

Background: With little attention given to low-frequency traffic noise and our understanding that cochlear function may be highly susceptible to low-frequency noise, there is an urgent need to determine traffic noise-induced hearing loss (NIHL), not only the hearing loss at low frequency but also the possible high-frequency hearing loss.

Methods: The current study aims to investigate the potential for extensive hearing loss induced by exposure to 0.063 kHz octave band noise (OBN), which is an important component of low-frequency traffic noise. The threshold of auditory brainstem response (ABR) was used to evaluate hearing function before and after noise exposure. Chinchillas were randomly assigned into seven different groups. Group 63-3 h/6 h, Group 2 k-3 h/6 h, and group 4 k-3 h/6 h were exposed for either 3 or 6 h to 0.063, 2, and 4 kHz OBN at 90 dB SPL, respectively. The control group was not exposed to noise.

Results: Significant ABR threshold-shifts (TS) were observed at 0.88, 2, 4, and 5.7 kHz in Group 63-6 h, and at 2.8 and 4 kHz in Group 2 k-6 h, and at 5.7 kHz in Group 4 k-6 h. ABR-TS were consistent with outer hair cell (OHC) losses, exposure to 0.063 kHz OBN at 90 dB SPL for 6 h induced large-scale losses of OHC both in low- and high-frequency region.

Conclusions: Exposure to 0.063 kHz low-frequency OBN at 90 dB SPL for 6 h leads to significant hearing loss over an extensive range from low to high frequencies.

KEYWORDS
auditory brainstem response, extensive hearing loss, low-frequency noise

1 | INTRODUCTION

Prolonged exposure to noise of high sound pressure level (SPL) can lead to noise-induced hearing loss (NIHL).[1,2] We are interested in

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low-frequency NIHL, as low-frequency noise (LFN) is becoming increasingly prevalent in urban environments, and this LFN is often of in the high energy range.[3]

LFN is defined to encompass the frequency range of 0.02–0.2 kHz. LFN can be generated by loudspeakers, air conditioning systems, and more prevalently transportation vehicles. The abundance of LFN of high SPL produced by transportation vehicles, such as cars, buses, heavy duty trucks, airplanes, and military vehicles[4,5] leads to a broad risk to individuals to LFN exposure.

The overall SPL of low-frequency urban-traffic noise are correlated with all the frequency components of the noise, the most significant correlations are observed at 0.063 kHz, and the average sound level of 0.063 kHz octave band noise (OBN) from 9 am to 5 pm is about 69.3 dB SPL.[6] However, heavy duty machineries and military automobiles, such as infantry fighting vehicles, can generate LFN at very high SPL, and a major amount of the acoustic energy is below 250 Hz with many of the low-frequency components exceeding 100 dB SPL. The sound levels of 0.063 kHz OBN are usually higher than 120 dB SPL. Military and other transportation personnel are often exposed to these noise levels in their occupation for extended time periods.[5]

Overall, 0.063 kHz OBN is an important noise of concern because of the high SPL of itself, and its significant correlation with the overall SPL of LFN.[4–6] Furthermore, cochlear function may be highly susceptible to this noise. In human, exposure to 0.063 kHz OBN at 84 dB SPL for 8 h can cause temporary and/or permanent decrease in auditory sensitivity. Thus, 0.063 kHz OBN may constitute a significant threat to hearing function.[2] As a result, we believe there is an urgent need to further investigate the hearing loss induced by 0.063 kHz OBN.

Our goal was to investigate not only the potential low-frequency hearing loss induced by 0.063 kHz OBN but also the possible high-frequency hearing damage induced by 0.063 kHz OBN. Our research method was to compare the auditory brainstem response (ABR) threshold-shift (ABR-TS) induced by either low-frequency OBN centered at 0.063 kHz or high-frequency OBN centered at 2 or at 4 kHz, respectively.

2 | MATERIALS AND METHOD

2.1 | Animals

Sixty-three chinchillas, 31 males and 32 females, with the presence of hearing as confirmed by normal Preyer's reflex were used in this study. Animals ranged from 12 to 22 months in age with a mean age of 17 months and weighed between 420 and 630 grams. The current study was approved by the Institute Ethics Committee with the protocol number of 201.

2.2 | Noise exposure

Noise exposure chamber was constructed according to a previous study.[7] OBN was generated by LabVIEW Signal Express (National Instrument) and presented through sound field. The amplification of noise signal and calibration of noise frequency and SPL were according to a previous report.[8]

Chinchillas were randomly assigned into seven groups, each group had nine animals. Group 1 was not exposed to noises, cochlea of Group 1 served as control for histological study. Other groups were exposed to OBN, called exposure groups. Group 63-3 h/6 h, Group 2 k-3 h/6 h, and group 4 k-3 h/6 h were exposed for either 3 or 6 h to 0.063, 2, and 4 kHz OBN at 90 dB SPL, respectively.

2.3 | ABR measurement

For ABR measurements, animals were under light anesthesia (ketamine, 50 mg/kg and xylazine, 15 mg/kg), a supplemental injection of ketamine (25 mg/kg IM) was given if needed. Animal temperature was maintained at 37°C.

Intelligent Hearing System (model name Duet) with a software module of SmartEP (5.51) was used for ABR measurement. Tone bursts were used to evoke ABR, stimulus repetition rate was 30/s. Averaging number of evoked ABR signal was 512, and gain of amplifier was 10,000.

For ABR recording, primary active electrode was placed on animal vertex, reference electrode was placed on the mastoid of tested ear, and ground electrode was placed on the lower back of animal.

For the measurement of ABR-threshold, we used a stimulus of supra threshold level to evoke ABR, then the supra threshold level decreased in 5 dB steps until 5 dB below the minimum stimulus level of visible ABR. Stimulus levels were then increased in 1 dB step to determine the final level of ABR-threshold. ABR-threshold was the lowest stimulus level that produced a detectable and reliable ABR signal which contained all ABR waves.[9]

ABR-thresholds were recorded before and immediately after noise exposure. ABR-thresholds measured before noise exposure were the assigned baseline levels. For each tested frequency the baseline level of ABR-threshold was subtracted from the level after noise exposure, the result was defined as ABR threshold-shift (ABR-TS).[10]

Acute noise exposure can cause both temporary and permanent threshold-shift (TTS and PTS). The extent of TTS recovery is related to the length of time after exposure.[11,12] In each animal, ABR-thresholds were measured at the following nine frequencies, 0.063, 0.088, 0.125, 1, 2, 2.8, 4, 5.7, and 8 kHz. As there are postexposure time differences between the first and subsequent measurements which may alter ABR-thresholds as a result of TTS recovery.

The method adopted to eliminate this potential confounding factor was the even distribution of the postexposure time differences. Each group had nine animals which were randomly designated as A to I. The orders of the nine tested frequencies in each animal were arranged to ensure the time difference between the nine tested frequencies were evenly distributed in the group (Table 1). In this way, if there was any confounding effect of time difference on ABR-threshold, it was controlled and evenly distributed among the groups, and it was not expected to affect the analysis.

TABLE 1 Even distribution of time difference of ABR measurement in a exposure group

	Sequence of ABR measurements of different tested frequencies								
Animal A	1	2	3	4	5	6	7	8	9
Animal B	2	3	4	5	6	7	8	9	1
Animal C	3	4	5	6	7	8	9	1	2
Animal D	4	5	6	7	8	9	1	2	3
Animal E	5	6	7	8	9	1	2	3	4
Animal F	6	7	8	9	1	2	3	4	5
Animal G	7	8	9	1	2	3	4	5	6
Animal H	8	9	1	2	3	4	5	6	7
Animal I	9	1	2	3	4	5	6	7	8
Tested frequency (kHz)	0.063	0.088	0.125	1	2	2.8	4	5.7	8

Note: In an exposure group, nine animals were randomly designated as A to I. In each row from A to I in an individual animal the sequence of ABR measurements of the nine tested frequencies was particularly arranged so that in any single column each frequency was provided the same opportunity to be tested in the order from 1 to 9 (each column). In this way even distribution of post-exposure time difference was achieved.

2.4 | Assessment of sensory cell damage

Silver nitrate staining was used to label the hair cells and stereocilia as described in previously.[13] The measurement of cochlear length and the calculation of outer hair cell (OHC) losses were carried out according to the method reported previously.[14] In brief, cochlea was dissected into small pieces and digital photographs of these pieces were taken with a camera on light microscope, cochlear length was measured from these digital images (Image Pro Plus, Media Cybernetics, CA). The averaged length of the cochlea of our experimental chinchillas was 25 ± 0.8 mm, which was consistent with previous report.[15] The percentage of OHC-losses for each 0.25 mm segment was calculated using a Zeiss Axiovert light microscope with a 40× oil objective len.[14]

2.5 | Statistical analysis

Statistical analyses were performed using IBM SPSS 19. Repeated measures of two-way ANOVA along with a Bonferroni correction of post hoc paired comparisons were used to compare ABR-thresholds and percentage of OHC-losses among control and exposure groups, with a $p < .05$ adopted as an indication of statistical significance.

3 | RESULTS

3.1 | Comparison of ABR-threshold among different exposure hours

The ABR-thresholds for 0.063, or 2, or 4 kHz OBN exposure were plotted as a function of tested frequencies in Figure 1A, or 1B, or 1C, respectively.

Figure 1A (0.063 kHz OBN panel) included the ABR-thresholds of baseline levels, Group 63-3 h, and Group 63-6 h. Figure 1B (2 kHz OBN panel) included the baseline levels, Group 2 k-3 h, and Group 2 k-6 h. Figure 1C (4 kHz OBN panel) included the baseline levels, Group 4 k-3 h, and Group 4 k-6 h. The baseline levels in a single OBN panel were the averaged threshold across the two groups of animals before exposure in the same panel.

In each OBN panel, when exposure time was short (3 h), exposure produced very little, if any, ABR-TS in the 3-h exposure groups in comparison to the baseline levels, $p > .05$. When exposure time was increased to 6 h, significant increases in ABR-thresholds were observed. In 0.063 kHz OBN panel, the levels of ABR-thresholds at 0.88, 2, 4 and 5.7 kHz in Group 63-6 h, were significantly higher than their baseline levels respectively, $p < .05$.

In the 2 kHz OBN panel, the levels of ABR-thresholds at 2.8 and 4 kHz in Group 2 k-6 h, were significantly higher than their baseline levels, $p < .05$. In 4 kHz OBN panel, the level of ABR-threshold at 5.7 kHz in Group 4 k-6 h was significantly higher than its baseline level, or its counterpart in Group 4 k-3 h, respectively, $p < .05$.

3.2 | Comparison of ABR-threshold among 6-h exposure groups

The ABR-thresholds of baseline levels and 6-h exposure groups were plotted as a function of tested frequencies shown in Figure 1D (6-h group panel), it included the ABR-thresholds of baseline levels, Group 63-6 h, Group 2 k-6 h, and Group 4 k-6 h. The baseline levels in this panel were the averaged threshold across the three 6-h groups of animals before exposure in the same panel.

The level of ABR-threshold at 4 kHz in Group 2 k-6 h was considerably higher than that in Group 4 k-6 h, $p < .05$. The ABR-threshold at 5.7 kHz in Group 4 k-6 h was significantly higher than that in Group 2 k-6 h, $p < .05$. The levels of ABR-thresholds at 0.88 and 2 kHz in Group 63-6 h were considerably higher than that in Group 4 k-6 h, $p < .05$. Other comparisons were not statistically significant, $p > .05$.

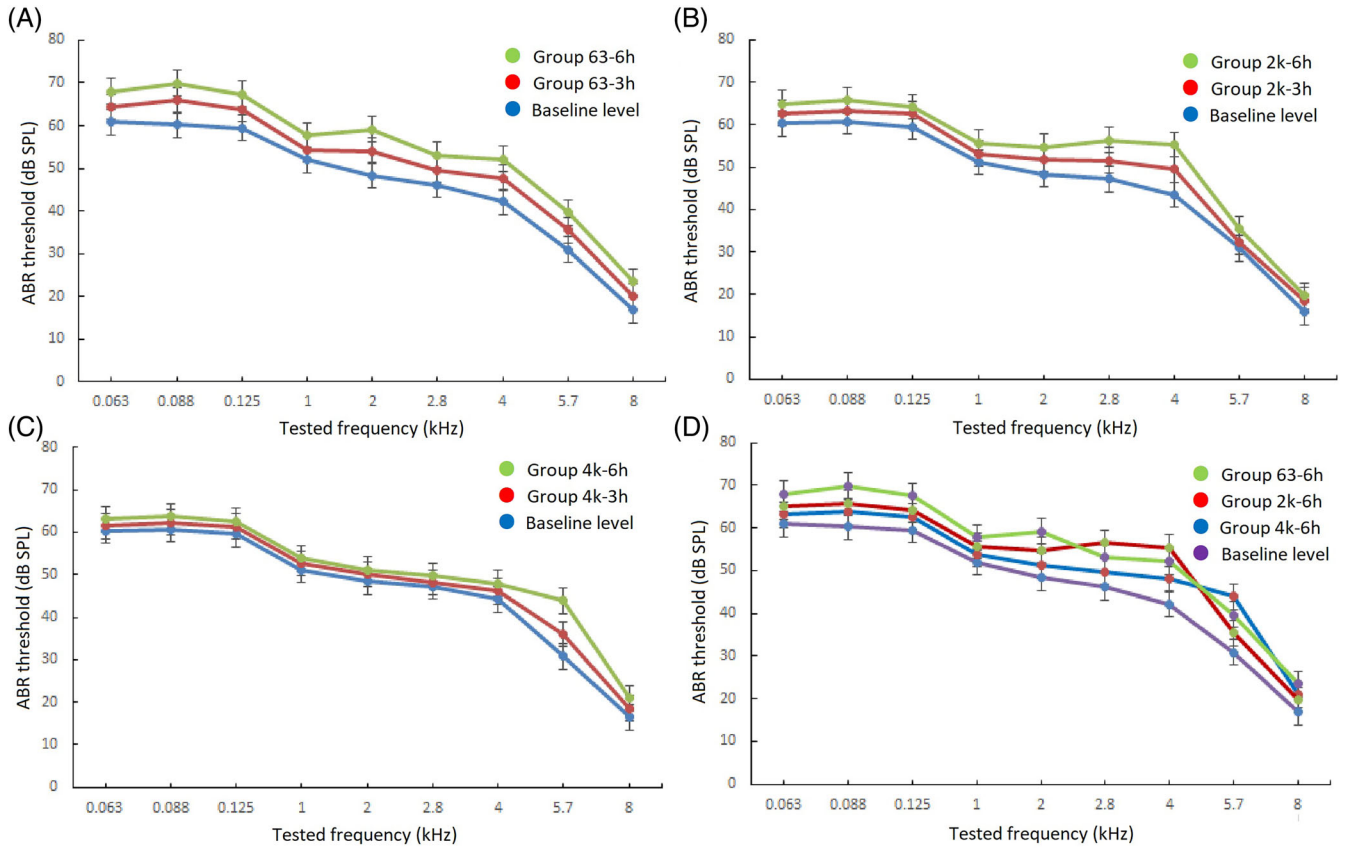

FIGURE 1 ABR-thresholds of baseline-level and exposure groups. (A–C) Indicated 0.063, 2, and 4 kHz OBN exposure, respectively, and each panel included baseline levels, 3-h, and 6-h exposure groups. (D) Included baseline levels, Group 63-6 h, Group 2 k-6 h, and Group 4 k-6 h

Figure 1D reveals additional aspects of ABR-threshold changes. For the baseline levels, when tested frequencies were increased a corresponding decrease in ABR-threshold were observed. In Group 2 k-6 h, the maximal ABR-TS appeared 1 octave above the OBN center frequency at 4 kHz. In Group 4 k-6 h, the maximal ABR-TS was at the upper band limit of the OBN at 5.7 kHz.

In Group 63-6 h, the first significant ABR-TS was at 0.088 kHz, the second and the maximal level of ABR-TS was at 2 kHz, and the third substantial ABR-TS at 4 kHz, plus the fourth remarkable ABR-TS at 5.7 kHz. In addition, the maximal ABR-TS at 2 kHz was 5 octaves above the OBN center frequency; the fourth ABR-TS of the highest frequency at 5.7 kHz was 6.5 octaves above the center frequency.

In summary, exposure to 0.063 kHz OBN could induce not only low-frequency hearing loss at 0.088 kHz but also high-frequency hearing loss at 2, 4, and 5.7 kHz. While exposure to 2 kHz OBN or 4 kHz OBN could only produce high-frequency hearing loss at 4 or 5.7 kHz, respectively.

3.3 | Sensory cell pathogenesis in different cochlear partitions

Based upon the equation of the characteristic location on the basilar membrane (BM) and its characteristic frequency (CF) in chinchilla,[16] and according to the methods reported previously,[14,16,17] a schematic diagram of cochleogram was mapped to estimate position–frequency relationship in our experimental chinchillas. The entire BM was divided into 10 portions at 10% intervals.

Examples of silver nitrate staining in histological control group and Group 63-6 h were shown in Figure 2. Figure 2A showed a representative image from cochlear middle turn of an animal from histological control, the image was taken about the 50% distance section from the apex. Figure 2B–D was images from an individual animal cochlea of Group 63-6 h. 3B was about the 10% distance section from the apex, 2C was from the middle turn of the 60% distance section from the apex, 2D was from the basal turn of the 80% distance section from the apex. As seen in Figure 2, there were minimal OHC-losses in the control in Figure 2A, while some OHC-losses were observed in Figure 2B,D, respectively, and substantial OHC-losses were in Figure 2C.

The percentage of OHC-losses in each portion were calculated and plotted for histological controls and 6-h exposure groups as a function of distance from the apex (%), and a function of CF, which were shown in Figure 3.

The percentage of OHC-losses in all 3-h exposure groups were not significantly higher than that in histological control, $p > .05$. In contrast, the percentage of OHC-losses in Group 63-6 h, Group 2 k-6 h, and Group 4 k-6 h were statistically higher than that observed in histological control, respectively, $p < .05$. There were no statistically

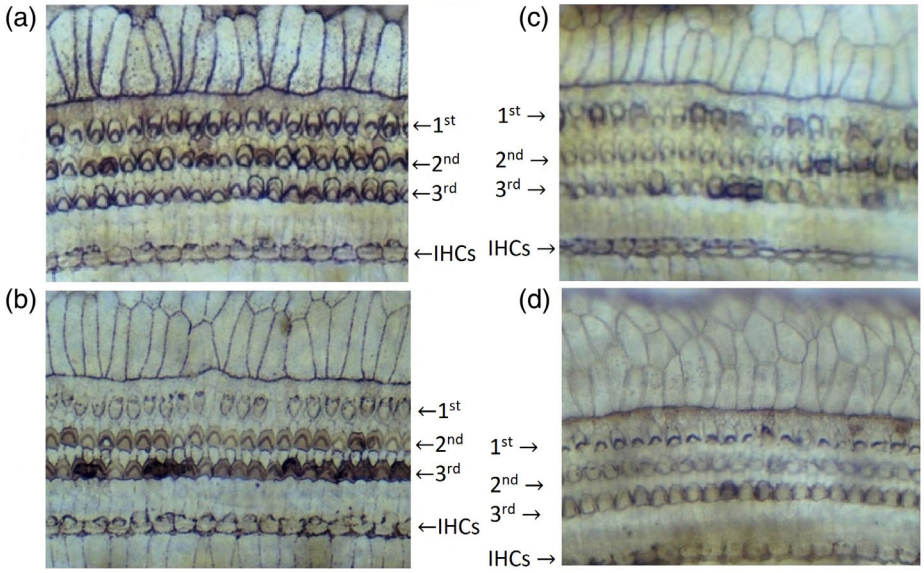

FIGURE 2 Cochlear images. (A) A cochlear section of histological control about 50% distance from the apex. (B–D) from one animal of Group 63-6 h, which were about 10%, 60%, and 80% distance from the apex, respectively

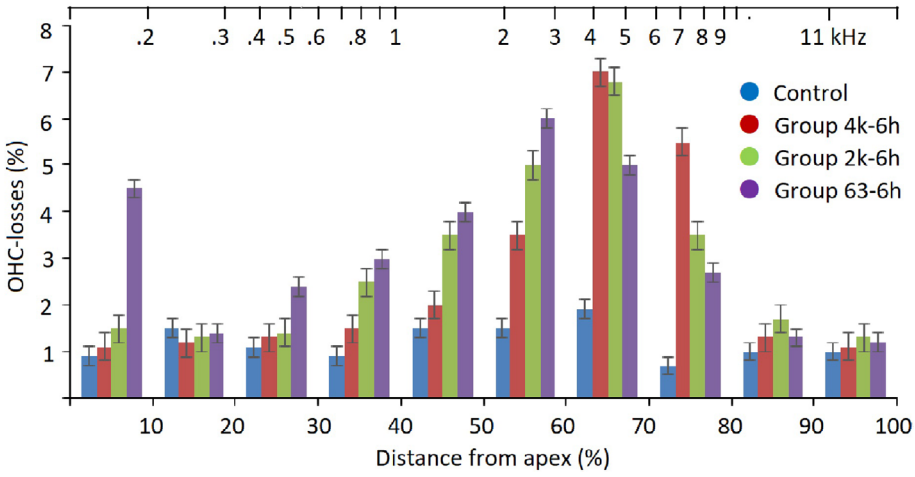

FIGURE 3 OHC-losses in histological control and 6-hour groups. The percentage of OHC-losses in histological control, Group 63-6 h, Group 2 k-6 h, and Group 4 k-6 h, were plotted as a function of distance from the apex (%) and CF

significant differences in the percentage of OHC-losses between Group 63-6 h and Group 2 k-6 h, $p > .05$; or between Group 2 k-6 h and Group 4 k-6 h, $p > .05$. Nonetheless, the percentage of OHC-losses in Group 63-6 h was determined to be statistically higher than that in Group 4 k-6 h, $p < .05$.

As seen in Figure 3, OHC-losses in histological control, were evenly dispersed along the BM, whereas the loss-zones of OHC in Group 2 k-6 h or Group 4 k-6 h were concentrated virtually in the middle turn of BM. The loss-zones in Group 2 k-6 h and Group 4 k-6 h were observed between the 40% to 80% sections or between the 60% to 80% sections from the apex, respectively. With Group 63-6 h there was one considerable loss-zone of OHC in the 10% distance section from the apex, while the major loss-zone was between the 30% and 60% distance sections from the apex. On the side of cochlear basal turn, a substantial loss-zone was also observed between the 60% and 80% distance sections from the apex. Exposure to 0.063 kHz OBN is leading to a more extensive losses of OHC than exposure to either 2 or 4 kHz OBN.

4 | DISCUSSION

The important implication of our research on animal models is that exposure to 0.063 kHz OBN may potentially cause extensive hearing loss in humans at both low and high frequencies. As our data reveals that high-frequency ABR-TS occurred about 5–6.5 octaves above the noise center frequency, 0.063 kHz OBN induced high-frequency hearing loss has the potential to be in the sound frequency of the human voice.[2]

While we observed that 2 or 4 kHz OBN only results in high-frequency hearing loss, the hearing loss induced by exposure to 0.063 kHz OBN was nearly equivalent to the combined effects induced by exposure to both 2 and 4 kHz OBN with respect to the frequency range of ABR-TS from 0.088 to 5.7 kHz and the levels of ABR-TS at 0.088, 2, 4 and 5.7 kHz in Group 63-6 h.

The frequency ranges of ABR-TS of exposure groups were in accordance with histological findings. From a functional perspective, OHC-losses may cause ABR-TS,[18] on the other hand ABR-TS can be

used to estimate the level of functional deficit of hearing loss due to noise induced OHC-losses and nerve fiber inefficiency.[19,20]

Here we observed that for Group 2 k-6 h, the CF of the major loss-zone of OHC ranged about 1–9 kHz. While for Group 4 k-6 h, the CF of the dominant loss-zone ranged approximately from 3 to 9 kHz. These were in contrast to Group 63-6 h where a considerable loss-zone of OHC near the apex was below 0.2 kHz. A large scale of loss-zone in the middle turn was observed between 30% and 80% distance sections from the apex and its CF ranged from around 0.6 to 9 kHz. The breadth and CF range of the loss-zone in the middle turn of Group 63-6 h overlapped the combined breadth and CF range of the loss-zones of Group 2 k-6 h and Group 4 k-6 h. In other word, exposure to 0.063 kHz OBN affects a much larger area of BM than exposure to either 2 or 4.0 kHz OBN.

Our explanation for the extensive ABR-TS of Group 63-6 h is in the following. BM close to cochlear apex is called low-frequency region, because it is wide and flexible, and sensitive to low-frequency stimuli. While BM near cochlear basal turn is called high-frequency area, as it is narrow and stiff, and sensitive to high-frequency stimuli.[21,22]

Low-frequency sound stimulates BM to tune and resonate close to cochlear apex, on the other hand high-frequency sound stimulates BM to tune and resonate near basal turn. However, there is overlapping between low- and high-frequency mechanical tuning of BM.[21,22] Besides stimulating BM close to cochlear apex, low-frequency sound of high SPL may modulate the mechanical tuning of the BM near basal turn, and increase the susceptibility of hair cells of basal turn to low-frequency sound stimulation,[23] leading to the potential of LFN causing extensive injury to hair cells of both close to cochlear apex and near basal turn. But more experiments are needed to validate this hypothesis.

To further investigate the susceptibility of hair cells of basal turn to LFN, fluorescent staining of hair cell nucleus and low-/high-pass filters of ABR signal, will be tried in future to evaluate the effect of LFN on the morphology and function of hair cells of basal turn.

In order to control the possible technician's interpretation bias, the measurements of ABR-threshold and the calculation of the percentage of OHC-losses were carried out by two technicians, respectively. The average of the two technician's results was adopted as the final data.

5 | CONCLUSIONS

The important implication of current study is that exposure to 0.063 kHz low-frequency OBN may lead to more extensive adverse effects on hearing function than high-frequency OBN. As 0.063 kHz OBN is one important component of vehicular noise, prolonged exposure to low-frequency traffic noise of high SPL may constitute a potent threat to hearing function for many individuals. A point that has not been previously reported.

Our research raises concern regarding the damage to hearing function induced by low-frequency noise exposure. Our study adopts a simple experimental paradigm which can be understood and accepted by the public.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

DATA AVAILABILITY STATEMENT

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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