Journal of Iranian Medical Council, Volume 10, Issue no. 1
http://dx.doi.org/10.18502/jimc.v10i1.23280

Original Article

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Investigating the Relationship between Tinnitus and Noise-Induced Neurodegeneration in the Central Auditory Pathway

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Abstract

Background: This study aimed to investigate the relationship between tinnitus and noise-induced neurodegeneration in the central auditory pathway.

Methods: In this study, 46 subjects (23 patients with tinnitus and a history of exposure to loud noise=case group and 23 healthy subjects=control group) participated. The assessment method was the use of Tinnitus Intensity Questionnaire (THI) and General Health Questionnaire-28 (GHQ-28), Pure Tone Audiometry (PTA), Word-In-Noise Perception (WINP) and Dichotic Digit Tests (DDT). The nonparametric Mann-Whitney test and Spearman correlation coefficient were used for data analysis ($p<0.05$).

Results: In both groups, the difference in the mean scores of GHQ-28 ($p=0.037$), WINP ($p=0.001$), and 4000 Hz hearing thresholds ($p=0.014$) were significant. The mean WINP of the case group was significant compared to controls ($p=0.017$). The difference in the mean DDT scores was not significant. In the case group, there was a positive correlation between THI score and duration of exposure to noise, but it was not significant. This non-significant correlation was negative with DDT and WINP scores.

Conclusion: Prolonged exposure to loud noise increases individual stress and reduces mental health. It causes damage to the central auditory pathway and decreases the ability to understand speech in competitive situations. Central auditory processing disorder occurs even when hearing thresholds are normal. In hearing-impaired individuals, who have a longer exposure to noise, the degree of tinnitus annoyance will be more severe. There is no relationship between the degree of central auditory pathway damage and the amount of tinnitus annoyance.

Keywords: Auditory pathways, Perception, Speech, Tinnitus

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Received: 14 Oct 2025
Accepted: 20 Apr 2026

Citation to this article
Keshtegar S, Shariatpanahi E, Doosti-Irani A, Emami SF. Investigating the Relationship between Tinnitus and Noise-Induced Neurodegeneration in the Central Auditory Pathway. *J Iran Med Counc*. 2027;10(1):233-39.

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Introduction

Tinnitus is a common complaint in patients with hearing disorders. According to some studies, 10-15% of the population suffers from tinnitus (1). It may be associated with neurodegeneration of the central auditory pathway that can be caused by prolonged exposure to loud noise. Neurodegeneration caused by noise can be permanent (2). This damage may alter the plasticity of the brain and cause abnormal neural activity associated with tinnitus. Given that the exact pathophysiology of tinnitus is unknown, it is difficult to treat and has a significant impact on patients' quality of life and the development of sleep and attention disorders (3). The mechanisms of hearing damage caused by noise pollution include direct damage to hair cells as well as oxidative stress. Loud noises can put pressure on the structures of the inner ear, leading to damage to hair cells. In addition, they increase the production of free radicals, which damage DNA and cellular proteins. This can ultimately lead to hair cell death and hearing loss (4).

Men are more susceptible to noise-induced hearing loss than women. Heredity, socioeconomic status, race, smoking, high blood pressure, diabetes, and exposure to chemicals may also play a role in the development of this lesion. Exposure to loud noises causes a severe stress response in the brain and damage to the cardiovascular system, leading to inflammation of the blood vessels in these areas and causing serious problems such as heart attack or stroke (5). Noise pollution is known as one of the major environmental problems in urban and industrial societies, and high levels of noise pollution are mainly observed in urban areas with high population density and close to noise sources such as airports, busy roads and factories (2). Considering that Word-In-Noise Perception test (WINP) is a new test that was presented in Persian for the first time in 2023 and examines the function of the cerebral cortex in understanding speech consonants using homotonic monosyllabic words, and before its invention, all Persian language tests that use monosyllabic words only examined speech discrimination or speech recognition, which is function of the brainstem and did not have the ability to examine the auditory brain function. Therefore, this study aimed to investigate the relationship between tinnitus and noise-induced neurodegeneration in the central auditory pathway.

Materials and Methods

Setting

This study was a case-control study conducted from June 2023 to December 2024 in Hamadan University of Medical Sciences. The financial sponsor of this research was Hamadan University of Medical Sciences (registered number:140204202943). The study population included patients referred to Beasat Hospital. The case group included patients aged 15 to 40 years (30.11±7.28) with tinnitus, who had been exposed to noise with an intensity greater than 75 *dBA* for at least 6 months. They included industrial workers employed in the workshops of the city of Hamadan. All the patients were workers in an industrial unit, and before the practical work of this research began, the background noise level in their workplaces had been measured by the occupational health expert of that industrial unit using a sonometer. According to the report, the background noise level in all places of the factory was between 90 and 97 *dB*, which is higher than the hearing safety standard. Besides, exposure to loud noise (75 *dB*) for more than eight hours a day for more than 6 months increases the likelihood of hearing loss (6).

The control group included normal people in the same age group, who had no history of exposure to noise greater than 75 *dBA*. The data collection tools included a demographic questionnaire, patient ear examination, Pure Tone Audiometry (PTA), WINP, Dichotic Digits Test (DDT), Tinnitus Intensity Questionnaire (THI), and GHQ-28 assessments. The samples in this study were selected using convenience sampling. They were 46 people (23=case group and 23=control group).

Inclusion criteria

Tinnitus, age 15 to 40 years [to eliminate the factor of presbycusis or age-related hearing loss, which is usually more likely to occur after the age of 40 (7)], being monolingual (Persian with Iranian dialect), written consent to participate in the study.

Exclusion criteria

Ear diseases and ear infections, history of underlying diseases (rheumatological diseases, vasculitis,

acoustic neuroma, known neurological disorders), history of ear surgeries, use of immunomodulatory drugs including interferons before the start of the study, cortisone, anticonvulsants and sedatives, history of taking ototoxic medications or addiction to tobacco, drugs during the study and patients with mental disorders.

Data collection method

After explaining the research procedure and obtaining written consent from the subjects, they underwent otoscopic examination by an otolaryngologist. In the case group, the severity of tinnitus was assessed based on the THI questionnaire score, and then the GHQ-28 questionnaire was completed to assess the mental health status in the groups. Then, the hearing thresholds of both groups were assessed by an audiologist. In the next step, the WINP test was performed by simultaneously presenting white noise to the test ear. The test materials consisted of 6 lists of 25 monosyllabic words with a consonant/vowel/consonant pattern, each list having a fixed vowel, which was performed using a recorded voice with a signal-to-noise ratio of 5 *dB* (*i.e.*, the intensity of the experimenter's speech was 5 *dB* higher than the intensity of the white noise), and the patient was required to repeat each word he heard. Each correct answer was worth 4 points, resulting in a total score of 100% (7).

Then, the DDT was assessed, which measures the temporal cortex's function for detecting simultaneous sounds and the brainstem's function for recognizing pitch and rhythm of words. This test is performed at the most comfortable listening level. In this test, auditory stimulation is presented to both ears simultaneously. The stimuli are monosyllabic numbers (1-3,5-9). Each stimulus consists of 4 numbers (2 numbers simultaneously to the right ear and 2 numbers to the left ear). During this test, participants are asked to repeat the numbers heard in one ear (test ear) and ignore the opposite ear (non-test) (8).

Data analysis

Stata version 17 software was used to analyze the data. Descriptive information related to qualitative variables was plotted in the form of tables and graphs. Descriptive information related to quantitative variables was displayed in the form of central and dispersion indices. To compare the case and control groups in terms of education, the chi-square test was used, and other quantitative variables (THI, DDT, WINT, and GHQ-28 scores due to non-normal distribution of the data) were compared using the non-parametric Mann-Whitney test. To determine the correlation between quantitative variables, the Spearman correlation coefficient was used. The significance level in all tests was considered less than 0.05.

Research limitations

There were no specific limitation.

Ethical considerations

This study was conducted with the approval of the Ethics Committee of Hamadan University of Medical Sciences (Ethics Committee Protocol Number = 1402.281IR.UMSHA.REC). Verbal and informed consent was obtained from the study participants. The tests and examinations used were routine diagnostic and therapeutic procedures for patients, and no additional costs were imposed on the patients for the study. They were assured that the results of the study would be published in full and anonymously. Not participating in the study did not prevent them from undergoing diagnostic or therapeutic measures. They could withdraw from the study at any stage if they felt dissatisfied.

Results

In the case group, the mean and standard deviation of the duration of exposure to noise was 14.36±6.23 years (range 1.5 to 25). Table 1 shows the frequency of educational level of the participants. The findings indicated that out of 23 patients in the case group, 13 (56%) had hearing loss in the right ear at two frequencies of 4000 and 8000 *Hz* and had hearing thresholds worse than 15 *dB* (7), while 10 patients (44%) had hearing thresholds better than 15 *dB* at the aforementioned frequencies. In the left ear of these patients, 11 (47%) had hearing loss and 12 (53%) had normal hearing. A significant difference was observed between the mean hearing thresholds of the right ear at 4000 *Hz* in the case group compared to the control group (Table 2). The difference in the mean

Table 1. Absolute and relative frequency of education level of the study population

Level of education	Group		p-value
	Control number (percentage)	Case number (percentage)	
Bachelor's and Master's degree	13(56.52)	10(43.48)	0.376*
Diploma	10(43.48)	13(56.52)	
Total	23(100)	23(100)	

* Chi-square test.

Table 2. Mean pure tone audiometric thresholds (PTA) of the right ear of the study population

Frequency (Hz)	Group		p-value
	Control Mean±Standard deviation	Case Mean±Standard deviation	
250-1000	16.40±4.50	18.70±4.61	0.071
2000	9.11±3.48	8.11±1.04	0.948
4000	11.7±0.42	21.18±24.00	0.014*
8000	15.6±0.08	14.15±18.77	0.130

* Nonparametric Mann-Whitney test.

Table 3. Mean pure tone audiometry thresholds (PTA) of the left ear of the study population

Frequency (Hz)	Group		p-value
	Control Mean±Standard deviation	Case Mean±Standard deviation	
250-1000	12.4±0.75	13.4±0.93	0.095
2000	6.8±4.34	6.10±1.52	0.939
4000	9.4±5.6	27.12±23.23	0.005*
8000	10.7±5.78	16.15±4.04	0.093

* Nonparametric Mann-Whitney test.

hearing thresholds of the left ear was also significant (Table 3).

In the right and left ears, the significant difference was observed between the mean WINP scores in the case group compared to the control group (p=0.001). Also, the mean WINP scores in the case group without hearing loss (61.2%) were significantly different (p=0.017) compared to the control group (73.13%). The difference in the mean DDT scores was not significant (p=0.320).

Based on the multivariate logistic regression analysis, significant differences were observed between the means of 4000 *Hz* hearing thresholds of the right ear (p=0.001) and the left ear (p=0.011) and the means of WINP scores of the right ear (p=0.002) and the left ear (p=0.016).

The mean and standard deviation of the GHQ-28 score in the case and control groups were 13.96±9.75 and 8.65±6.41, respectively. A significant difference was observed in the mean GHQ-28 score between the two groups (p=0.037). In other words, patients in the case group had higher stress levels and lower mental health than the control group, and stress was considered a predisposing factor for the occurrence of auditory pathway neurodegeneration.

The mean THI score of the case group without hearing

loss was 2.5, which was significantly different from the mean of the entire case group (2.13). However, the mean THI score of the case group with hearing loss was 20, which confirms the possibility of tinnitus co-occurring with hearing loss. In the case group, the Spearman correlation coefficient between the THI questionnaire score and the duration of sound exposure was positive (r=+0.269), but the correlation was not significant (p=0.215). A non-significant negative correlation of the THI questionnaire score with the DDT and WINP test scores was also obtained.

Discussion

In the present study, the mean PTA of the case group at 4000 *Hz* was significantly higher than that of the control group. In support of the findings, Pienkowski reported that patients exposed to noise may develop hidden hearing loss at frequencies above 8000 *Hz* or below 250 *Hz* (9). Fuente and McPherson stated that noise causes cognitive behavioral disorders, changes in brain neurotransmitter levels, molecular and structural mechanisms, and the more a person is exposed to noise above 75 *dB*, the greater the risk of developing noise-induced hearing loss (10).

Vermiglio showed in a laboratory study on animal models that exposure to loud noise damages the cochlea and the neural structures of the auditory system from the nerve level to the cerebral cortex. In the early stages, a temporary decrease in thresholds occurs, lower neural structures are damaged, and when hearing loss progresses from a temporary decrease in thresholds to a permanent noise-induced hearing loss, higher neural structures and the cerebral cortex are also damaged. In cases of repeated exposure to loud noise, all neural structures will be involved (11).

In this study, the WINP and DDT tests were used to examine the neural processing of the central auditory system (7,8). Since noise exposure can damage the hair cells of the cochlea and reduce the accuracy of signal processing in the auditory brain, it may lead to decreased scores on the WINP and DDT tests (8). The WINP test assesses the ability to understand words in the presence of white noise by detecting speech consonants in the auditory cortex. Since speech perception in the presence of noise is performed by the secondary temporal lobe region with the participation of all brain regions, abnormal results on this test can confirm to some extent the neural damage of the auditory pathway (7). The DDT test also measures the ability to process auditory signals presented simultaneously to both ears. Low scores on this test indicate that the individual has difficulty integrating auditory information received from both ears (8). In the present study, the mean WINP test scores for the right and left ears of the case group were lower than those of the control group, and the differences were significant. However, there was no significant difference in DDT test scores between the two groups. The findings of this study indicated that long-term exposure to loud noise can cause neurodegeneration and central auditory processing impairment, which is associated with a decrease in the ability to understand speech in noise.

Fuente and McPherson investigated the damaging effects of industrial pollutants on the auditory system. Their findings revealed that exposure to loud noise, even in conditions where hearing thresholds were normal, caused some degree of central auditory processing impairment. However, their control group, who had no history of exposure to loud noise, had normal results (10). Wieczerzak *et al* also reported that loud noise disrupts the functional connectivity and interactions of the temporal and prefrontal lobes. Therefore, it impairs learning, memory, and cognitive functions of the brain (12). According to the findings of the study by Liu *et al*, patients with a long history of exposure to loud noise suffered from cognitive impairment, decreased learning ability, and memory impairment (13).

Tepe *et al* also observed in a study of military personnel that loud noises caused neurodegeneration in their study population. In other words, noise pollution causes damage to the central auditory pathway, and exposure to loud noises causes impairment in attention, speech perception in the presence of noise, memory, learning, language, and functional deficits in the auditory cortex (14).

Kohnsal *et al* reported that tinnitus patients with normal hearing thresholds who had a history of exposure to loud noise had deficits in central auditory processing, temporal and spectral resolution functions, frequency discrimination, and speech perception in noise (15). Also, according to the findings of the study conducted by Dewey *et al*, the prevalence of

tinnitus in patients with a history of exposure to loud noise was significantly higher than in non-exposed individuals. They reported that exposure, even in conditions where hearing thresholds are within the normal range, can cause damage to the brainstem and cerebral cortex, thereby providing the basis for tinnitus and central auditory processing disorders (16).

In the current study, in the case group, the Spearman correlation coefficient between the THI score and the duration of exposure to noise was positive, but the correlation was not significant. The findings were consistent with the results of the study by Rubak *et al*. They observed that the adverse consequences of tinnitus increased in industrial workers who had a longer duration of exposure to noise. However, their correlation was not significant (17). Their findings and the present study demonstrated that the likelihood of tinnitus complications rises with increasing duration of exposure to noise, and the lack of significance of the correlations in the two studies means that it cannot be stated with certainty that there is a causal relationship between THI score and duration of exposure to noise. Other factors may also play a role in the occurrence of tinnitus. Despite the lack of statistical significance, these findings are still important because they emphasize that long-term exposure to noise may exacerbate tinnitus complications. This confirms the importance of reducing the duration of noise exposure through job rotation or changing works in acute cases of problems. In the present study, mental health scores in patients with tinnitus were significantly lower than in the control group. Tinnitus is a challenge to quality of life by increasing the risk of hearing loss, ototoxicity, and its psychological effects. Consistent with the findings was a systematic review conducted by Demoen *et al* on health-related quality of life in patients with chronic tinnitus, and their findings showed that chronic distress due to tinnitus had a significant impact on health-related quality of life, and patients with tinnitus have a significantly lower quality of life compared to the group without tinnitus (18).

Conclusion

Prolonged exposure to loud noise increases individual stress and reduces mental health. It also causes damage to the central auditory pathway and reduces the ability to understand speech in competitive situations. Central auditory processing disorders occur even when hearing thresholds are normal, and increased individual stress exacerbates the occurrence of neural damage. In conditions where the duration of sound exposure is longer and hearing loss is present, the degree of tinnitus annoyance will be more severe. Also, there is no relationship between the degree of neural damage to the auditory pathway and the degree of tinnitus annoyance.

Limitations and future directions

The small and non-random sample, which limits the generalizability, was the limitation of the current study. The cross-sectional design, which precludes any causal inference between noise exposure, tinnitus, and neurodegeneration would be better used in future studies.

Acknowledgement

The financial sponsor of this research was Hamadan University of Medical Sciences (ethics code: 1402.281IR.UMSHA.REC). The authors know to thank and appreciate the esteemed participants who cooperated in this research.

Conflict of Interest

The authors declare that they have no competing interests.

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