Research Article

Central Auditory Processing and Cognitive Function in Men with Normal Hearing Exposed to Industrial or Leisure Noises

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Citation: Azizi N, Rahimi V, Tavanai E, Faghihzadeh E, Mohammadkhani G, Najafi S. Central Auditory Processing and Cognitive Function in Men with Normal Hearing Exposed to Industrial or Leisure Noises. Aud Vestib Res. 2026;35(1):74-84.

https://doi.org/10.18502/avr.v35i1.20581

Highlights

- Industrial noise impairs men's auditory and cognitive functions
- Leisure noise from PLD impairs speech perception, memory, and attention
- Industrial noise exposure was correlated with speech-in-noise deficits in men

Article info:
Received: 26 Apr 2025
Revised: 07 Jun 2025
Accepted: 30 Jun 2025

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ABSTRACT

Background and Aim: Noise can lead to temporary or permanent changes in the structure and function of the peripheral and central auditory systems when experienced at high sound pressure levels over time. This study aimed to examine the effects of industrial noise and leisure noise from Personal Listening Devices (PLDs) on central auditory processing and cognitive functions in men with normal hearing.

Methods: In this cross-sectional study, 136 men aged 20–40 were divided into three groups: leisure noise-exposed group due to PLD use (n=45), industrial noise-exposed group (n=46; average Leq<90 dB (A)/8 hours), and control group (n=45, with no history of noise exposure). All participants had normal audiograms. To evaluate central auditory processing, we used the dichotic digits test, the duration pattern sequence test, and the quick speech-in-noise test. Cognitive abilities were assessed using the Rey auditory verbal learning test and the semantic Stroop test.

Results: The industrial noise-exposed group had significantly lower scores in all central auditory and cognitive tests compared to the leisure noise-exposed and non-exposed groups (except for the reaction time and the semantic Stroop test). The leisure noise-exposed group also showed impairments in speech-in-noise perception, short-term memory, and selective attention relative to the non-exposed group. Additionally, a negative correlation was found between noise exposure level and speech-in-noise performance.

Conclusion: Both industrial and PLD noise exposure can impair central auditory and cognitive functions in men with normal hearing, highlighting the need for broader assessments in noise exposure monitoring.

Keywords: Leisure noise exposure; personal listening devices; occupational noise exposure; cognitive ability; central auditory processing disorders; attention

Introduction

Noise, typically defined as "unwanted sound", can have substantial adverse effects on the auditory system, even if the sound is subjectively considered pleasant or desirable when it is above certain thresholds of intensity and duration. Noise exposure is classified into three main categories: environmental noise, occupational noise, and recreational noise [1]. The occupational noise has long been recognized as a significant health risk. The World Health Organization (WHO) attributes approximately 16% of disabling hearing loss to occupational noise exposure [2]. Beyond hearing loss, occupational noise has been linked to impaired cognitive performance, increased workplace errors and accidents, and elevated risks of Alzheimer's disease, social isolation, psychological distress, and cardiovascular diseases [3]. Recreational noise, particularly from Personal Listening Devices (PLDs), is a rising global challenge. WHO reports that 1.1 billion young people are at risk of hearing loss from unsafe listening behaviors, with nearly half of youth in middle- and high-income countries exposed to harmful sound levels via PLDs [4]. Loud music, though subjectively enjoyable, can be addictive and damaging. Prolonged exposure through PLDs is associated with transient threshold shifts, tinnitus, hyperacusis, abnormal pitch perception, and ultimately, permanent hearing loss [5]. Safety standards attempt to mitigate these risks. The NIOSH recommends occupational noise exposure not exceed 85 dB (A) time-weighted average over 8 hours, with exposure duration halved per 3 dB (A) increase. No safety standard fully prevents tinnitus, hyperacusis, or speech-in-noise deficits despite normal audiograms [6]. OSHA extends this limit to 90 dB (A) for 8 hours. For recreational noise, the WHO and the International Telecommunications Union (ITU) in 2019 recommend the WHO-ITU H.870 standard, which suggests that noise levels should remain under 80 dB (A) for a maximum of 40 hours weekly [1]. PLDs can reach sound levels of 101–107 dB (A) at full volume and even moderate volume can be harmful in under an hour [7].

Emerging research reveals that even sound levels at or below 80 dB SPL can lead to physiological damage at multiple levels of the auditory pathway —including the cochlea, midbrain, thalamus, and cortex. Brattico et al. demonstrated that 70–80 dB SPL industrial noise over five years can alter brain hemispheric organization and degrade speech recognition and attention in subjects with clinically normal hearing [8]. Kujala et al. suggested that noise above 80 dB (A) may result in temporary threshold shift as well as synaptic damage at inner hair cells, leading to cochlear synaptopathy, hidden hearing loss, and central auditory processing deficits, which may go undetected by standard audiograms [9]. Normal-hearing individuals exposed to occupational noise often present with deficits in speech-in-noise perception [8], attention [10], temporal processing [11], binaural hearing [12], and memory [13]. These effects are dose-dependent, influenced by both intensity and duration of exposure. Even noise exposure below 80 dB SPL induces central auditory system alterations, including changes in central gain within the midbrain, thalamus, or cortex, and reorganization of tonotopic maps [14], driven by homeostatic plasticity. Animal studies showed that 65 dB SPL noise affects the medial geniculate body and auditory cortex, altering midbrain frequency responses and lateral inhibition [15]. In humans, non-damaging noise intensities (<95 dB A) impair performance on mismatch negativity and central temporal processing tests, indicating deficits in attention and temporal processing [9]. Recreational noise exposure similarly impairs speech-in-noise perception, attention, and memory [16, 17], causing functional deficits masked by normal audiograms [18]. However, Yeend et al. found no direct link between lifetime noise exposure and speech-in-noise perception, suggesting cognitive factors such as attention and working memory primarily determine performance [19]. This implies long-term noise exposure may selectively disrupt higher-order auditory processing, with central deficits occurring independently of peripheral damage [20].

Occupational and recreational noises engage different neurophysiological pathways. Music that is commonly used during PLD usage activates emotion-linked systems, enhancing memory encoding [21] and improving spectral/temporal processing. Occupational noise activates thalamic fear/emotion circuits and the amygdala-HPA axis, triggering stress hormones and defensive responses [22, 4]. This differential neural impact may explain variations in observed central auditory deficits. This study aimed to examine

whether recreational and occupational noise exposures impair central auditory processing and cognitive function, hypothesizing the greater negative impact of occupational noise exposure.

Methods

Participants

This cross-sectional study included 136 male participants divided into three groups: Leisure Noise Exposure (L-NEG, n=45), Industrial Noise Exposure (I-NEG, n=46), and control (n=45). Due to gender-related risk factors, only males were included. Groups had different ages (18–40 years) and hearing levels. Inclusion criteria were: a normal pure-tone hearing threshold (<25 dB HL, 250–8000 Hz), a type A tympanogram, right-handedness, absence of academic/attention deficits, and no history of ototoxic drug or sedative use. Exclusion criteria were absent acoustic reflexes or elevated acoustic reflex thresholds (>95 dB HL). Both noise-exposed groups (L-NEG and I-NEG) had ≥2 years of noise exposure [9]. The I-NEG group included workers from furniture construction and aluminium production industries, exposed to occupational noise levels ≥85 dB (A), involving continuous, impact, and vibration noise. Interviews confirmed inconsistent use of hearing protection. The L-NEG group reported using PLDs for listening to music for ≥22 minutes daily at ≥80% volume for ≥2 years [23]. The total usage time at maximum volume (100%) was ≤1.5 hours. To estimate PLD sound exposure levels, participants reported their most frequently used volume level (percentage of maximum device output). The calculated listening level was determined. As direct measurement was unfeasible. Thus, the regression equation proposed by Portnuff et al. (i.e., 0.6143X+39.395, where X is device volume percentage) was applied to estimate the dB SPL equivalent for earbuds [23]. Finally, equivalent sound pressure levels (Leq (8h) and Leq (24h)) were calculated using the following equation:

$$Leq\ (dB) = 10\log[\frac{1}{T}\sum_{i=1}^{N} ti\ 10^{\frac{LPi}{10}}]$$

Where T denotes the reference time (8 or 24 hours), LPi shows the sound pressure level of exposure, and ti indicates the duration of exposure in hours [24]. In the control group, none of the men were exposed to occupational or recreational noise exposure.

Assessments

First, the case history, including noise exposure information, was recorded, and otoscopic examinations were performed. The Duration Pattern Sequence Test (DPST), Persian version of the Quick Speech in Noise (Q-SIN) test, and the Dichotic Digits Test (DDT) were used for assessment of central auditory processing, and the Rey Auditory Verbal Learning Test (RAVLT) and the semantic Stroop test were used for cognitive assessment. The order of assessments was randomized. Efforts were made to ensure adequate rest intervals between each assessment. In addition, assessments were conducted for workers outside of regular working hours. The immittance tests (Zodiac 901 model, Madsen CO, Denmark) were used to assess the middle ear status and the audiometric tests to determine the hearing thresholds between 250–8000 Hz (Interacoustic AC 33 model, Madsen CO, Denmark) were performed in the acoustic room. The Montreal Cognitive Assessment (MoCA) test was used to rule out cognitive impairment. This test evaluates attention/working memory, executive, episodic memory, language, and visuospatial skills. A cutoff score <25 is considered a referral for diagnosis of mild cognitive impairment [25].

Central auditory processing tests

The DPST takes about 8 minutes to complete and is easier than other temporal processing tests, and is highly sensitive to cortical lesions (85.7% sensitivity, 92% specificity). Each DPST trial presented three 1000 Hz tones, long (500 ms) or short (250 ms), forming a sequence. Six unique duration patterns were used. Thirty trials were administered monaurally to each ear. Participants verbally reported the sequence (e.g., long-short-long). The percentage of correct responses per ear was calculated by multiplying the number of correctly identified sequences by 3.33 [26].

To minimize participant fatigue, an efficient Q-SIN test was used, validated for Iranian adults by Khalili et al. [27]. It was administered at the participant's most comfortable level. Target sentences (male talker) were presented against multi-talker babble background noise. The Signal-To-Noise Ratio (SNR) decreased progressively; the target sentence started clearly audible but became increasingly difficult to understand as the background noise level rose relative to the target speech.

Participants repeated each sentence. The total number of words correctly repeated across the test list was summed. The SNR-loss in dB was calculated using the following equation: SNR-loss=27.5–(total number of correct words) [27].

For the DDT, we utilized monosyllabic Persian digits (1–10, excluding disyllabic 4). Its reliability is higher than 0.84 [28], is unaffected by peripheral hearing loss, and is easy to use. The test comprised 20-digit pairs for a total of 40 test items per ear. Digit pairs on one channel of a Compact Disc (CD) were temporally aligned with pairs on the other channel to create dichotic stimuli. The CD was played using a dual-channel CD player, with channel one directed to the left ear and channel two to the right ear, according to the standard protocol. Participant responses were recorded on a worksheet. The total number of correct responses per ear was multiplied by 2.5 to obtain a percentage score, rounded to the nearest digit [28]. All tests (DPST, Q-SIN, DDT) were conducted in a sound-treated room with stimuli delivered via headphones.

Cognitive processing tests

Cognitive function was assessed using a Semantic Stroop Test software (Ravan Tajhiz Sina Co., Iran). This test has a reliability of 0.71 for accuracy and 0.82 for reaction time [29]. This test was used due to its simplicity, and the software provides us with individuals' data, separately. In this test, neutral and target words were randomly presented with red and green colors on the computer screen. Two keys on the keyboard were marked with red and green color labels (green for the key "?" and red for the key "Z"). It was explained to the participant that he should quickly press the green key for green words and the red key for red ones. The number of correct answers, reaction time, and total time were determined by the software [29].

The RAVLT requires no special equipment, takes a short time, and has a reliability of 0.80. In this test, a list of 15 noun-words was read to the participants. Then, they were asked to recall as many words as they could. The number of words recalled correctly was recorded and determined as a score for the short-term performance. The order of the recalled words was not important [30].

Statistical analysis

Statistical analyses were performed in SPSS v.17. The Kolmogorov-Smirnov test was used to check the normality of data distribution. Based on its results, the Kruskal-Wallis test was used to evaluate the differences in DDT, DPST, RAVLT, Q-SIN, and Stroop test scores (correct answers and reaction time) between three groups. The Mann-Whitney U test was used for pairwise comparisons of the groups. $p<0.05$ was considered statistically significant. The statistical significance levels were adjusted by the Bonferroni correction method for multiple testing, and the adjusted P was reported.

Results

Characteristics of participants

Information of participants in each group, including age, hearing thresholds, intensity levels, and duration of noise exposure, is presented in Table 1. The mean age of individuals in the L-NEG, I-NEG, and control groups was 29.07(3.31), 30.80(5.45), and 29.36(5.45) years, respectively. Statistical analysis revealed no significant differences in age ($p=0.19$) or average hearing thresholds ($p=0.15$) among the groups. The volume percentages of PLDs reported by participants in the L-NEG group are reported in Table 2.

The duration pattern sequence test scores

As shown in Table 3, there was a statistically significant difference in the DPST score among the three groups for both left ear ($H=82.37$, $df=2$, $p<0.001$) and right ear ($H=81.20$, $df=2$, $p<0.001$). Pairwise comparisons revealed that the scores in the I-NEG group were significantly lower than those in the L-NEG group in both left ($p<0.001$) and right ($p<0.001$) ears, and those in the control group in both ears (left: $p<0.001$; right: $p<0.001$). While the difference between the L-NEG and control groups in the right ear was significant ($p<0.05$), the difference in the left ear was not statistically significant ($p>0.05$).

The quick speech in noise test scores

As shown in Table 3, there was a significant difference in the Q-SIN test score (SNR-loss) among the three groups ($H=34.567$, $df=2$, $p<0.001$). Post-hoc analysis showed that the performance of the I-NEG group was significantly poorer than that of the L-NEG ($p=0.018$) and control ($p<0.001$) groups. Also, the L-NEG group

Table 1. Demographic and noise exposure characteristics of the study groups

	Mean(SD) Range		
	I-NEG (n=46)	**L-NEG (n=45)**	**Control (n=45)**
Sound level (dB A)	Leq (8h): 88.80(3.36) 85–95	Leq (8h): 92.62(4.39) dB 88.53–100.82 dB Leq (24h): 80.49(3.89) 89.20–77.19	-
Duration (hour)	6.47(1.24) 4–9	1.77(0.33) 1–2	-
Exposure (dB)	87.79(2.68) 84.42–92.95	85.19(3.67) 81.73–92.73	-
Age (year)	30.80(5.45) 21–40	29.07(3.31) 23–40	29.36(5.45) 19–40
PTAave (0.5, 1, 2, 4 kHz) (dB)	13.15(3.85) 5–20	11.56(4.50) 5–20	11.89(4.03) 5–20
MoCA Score	29.93(0.24) 29–30	29.86(0.40) 28–30	29.91(0.28) 29–30

I-NEG; industrial noise exposure group, L-NEG; leisure noise exposure group, Control; control group, PTAve; pure-tone average, MoCA; Montreal cognitive assessment, Leq; level equivalent

Table 2. Self-reported volume settings and estimated output levels in personal listening devices users

The volume of personal listening devices (%)	**dB eq**	**Frequency of participants(%)**
80	88.539	21(46%)
85	91.6105	7(20%)
90	94.682	12(33%)
100	100.82	5(11%)
Total		45(100%)

eq: equivalents

showed significantly more SNR-loss than the control group (p=0.006).

The dichotic digit test scores

As shown in Table 3, the results of a Kruskal-Wallis test for the DDT score showed a statistically significant difference among the three groups in the left (H=39.62, df=2, p<0.001) and right ears (H=15.66, df=2, p<0.001). Post-hoc analysis revealed that the DDT score of the I-NEG group was significantly lower than that of the L-NEG group for the left and right ears (p<0.001). The I-NEG group had a lower score than the control group in the left (p<0.001) and right (p=0.018) ears. There were no significant differences between the L-NEG and control groups (p>0.05).

The Rey auditory verbal learning test scores

The results in Table 4 indicated a statistically significant difference among the three groups in the

Table 3. Between-group comparisons on measure of central auditory processing

Variable	Groups	Mean(SD)	Median(inter-quartile range)	H	p	Between groups	p
DPST result in right ear (%)	L-NEG	95.77(4.04)	96(96–96)	81.20	<0.001	L-NEG control	0.03*
	I-NEG	83.69(9.60)	83(80–90)			I-NEG control	<0.001*
	Control	98.29(6.67)	100(96–100)			L-NEGI-NEG	<0.001*
DPST result in left ear (%)	L-NEG	96.21(4.58)	96(96–100)	82.37	<0.001	L-NEG control	0.1
	I-NEG	84.20(8.78)	86(76–90)			I-NEG control	<0.001*
	Control	98.44(6.67)	100(96–100)			L-NEG I-NEG	<0.001*
SNR-loss (dB)	L-NEG	0.90(1.38)	0.5(–0.5–0.5)	34.56	<0.001	L-NEG control	<0.01*
	I-NEG	3.04(3.20)	2(0.5–5.75)			I-NEG control	<0.001*
	Control	–0.10(1.22)	–0.5(–1–1)			L-NEG I-NEG	0.018*
DDT result in right ear (%)	L-NEG	83.27(13.21)	88.5(77–92)	15.66	<0.001	L-NEG control	0.87
	I-NEG	71.90(15.77)	75(60–85)			I-NEG control	0.018*
	Control	80.73(9.47)	80(73–88)			L-NEG I-NEG	<0.001*
DDT result in left ear (%)	L-NEG	84.61(14.79)	77.5(62–85)	39.62	<0.001	L-NEG control	0.48
	I-NEG	61.19(14.11)	60(51–70)			I-NEG control	<0.001*
	Control	77.11(9.98)	77.5(68–87)			L-NEG I-NEG	<0.001*

DPST; duration pattern sequence test, L-NEG; leisure noise exposure group, I-NEG; industrial noise exposure group, Control; control group, SNR; signal-to-noise ratio, DDT; dichotic digit test

* Adjusted p-value

RAVLT score (H=78.20, df=2, p<0.001). Post-hoc analysis revealed that the score of the I-NEG group was significantly lower than that of the L-NEG and control groups (p<0.001). Additionally, a significant difference was observed between the L-NEG and control groups (p<0.001).

Semantic Stroop test scores

The percentage of correct answers to the neutral (H=41.14, df=2, p<0.001) and target (H=84.74, df=2, p<0.001) words was significantly different among the three groups. Post-hoc analysis revealed a significant difference between the I-NEG and L-NEG groups for both neutral (p=0.018) and target (p=0.019) words. Also, the percentage for neutral and target words in the I-NEG group was significantly lower than in the control group (p<0.001). The difference was also statistically significant between L-NEG and the control group for neutral and target words (p<0.001). Regarding reaction time, the result of the Kruskal-Wallis test showed no statistically significant difference among the three groups for the neutral (p=0.10) and target (p=0.055) words.

Relationship between exposure level and speech-in-noise perception

Spearmen's correlation test results showed a significant relationship between Leq (8h) and SNR-loss in the I-NEG group (r=0.54, p<0.001) and between Leq (8h) and SNR-loss in the L-NEG group (r=0.34, p<0.05). However, there was no significant correlation between DPST scores and DDT scores with Leq (8h) in both groups (p>0.05).

Discussion

This study aimed to compare central auditory processing and cognitive functions between normal-hearing adults exposed to industrial or leisure noises and those with no noise exposure. The investigation revealed the significant effects of industrial noise exposure

Table 4. Between-group comparisons on measure of cognitive function

Variable	Groups	Mean(SD)	Median (interquartile range)	H	p	Between groups	p
RAVLT result (%)	L-NEG	7.90(1.98)	8(6–9)	87.20	<0.001	L-NEG control	<0.001*
	I-NEG	5.08(1.77)	5(4–6)			I-NEG control	<0.001*
	Control	10.06(1.61)	10(9–11)			L-NEG I-NEG	<0.001*
The correct answer of the neutral words (%)	L-NEG	97.56(2.57)	98(96–100)	41.14	<0.001	L-NEG control	≤0.001*
	I-NEG	93.54(15)	96(95–98)			I-NEG control	<0.001*
	Control	99.56(0.89)	100(99–100)			L-NEG I-NEG	0.018*
The correct answer of the target words (%)	L-NEG	95.18(2.88)	96(95–97)	84.74	<0.001	L-NEG control	<0.001*
	I-NEG	90.70(13)	95(91–95)			I-NEG control	<0.001*
	Control	98.93(1.13)	99(98–100)			L-NEG I-NEG	0.019*
Reaction time to neutral words (ms)	L-NEG	491(63.43)	498(450–528)	4.46	0.10	L-NEG control	-
	I-NEG	533(101.00)	523(457–586)			I-NEG control	-
	Control	505(46.74)	505(475–547)			L-NEG I-NEG	-
Reaction time to target words (ms)	L-NEG	493(59.97)	492(459–539)	5.80	0.055	L-NEG control	-
	I-NEG	540(105.00)	538(456–592)			I-NEG control	-
	Control	510(45.32)	510(479–542)			L-NEG I-NEG	-

RAVLT'; Ray auditory verbal learning test, L-NEG; leisure noise exposure group, Control; Control group, I-NEG; industrial noise exposure group
* Adjusted p-value

on dichotic hearing, auditory temporal processing, speech-in-noise perception, and cognitive domains including selective attention and working memory compared to leisure noise exposure and non-exposure state. Furthermore, the leisure noise exposure leads to significant deficits compared to the non-exposure state in variables such as speech-in-noise perception, auditory temporal processing, selective attention, and memory performance.

Quantitative assessment of speech-in-noise perception showed that the industrial noise-exposed group had significantly higher SNR-loss compared to the leisure noise-exposed and non-exposed groups. This impairment in the I-NEG group aligns with neurobiological evidence that noise exposure preferentially damages the low Spontaneous Firing Rate (low-SFR) of auditory nerve fibers (high-threshold afferents crucial for signal detection in noisy environments). This cochlear neuropathy occurs without elevating conventional pure-tone thresholds, constituting a hidden hearing loss [11]. This pathophysiological mechanism explains why individuals with normal audiograms exhibit low speech-in-noise perception following noise exposure. This finding is consistent with other studies. Eggermont [11] and Wang and Ren [31] showed that noise-induced loss of high-threshold, low-SFR auditory nerve fibers (whose firing dynamics resist saturation in background noise) directly compromises speech-in-noise perception in humans.

The higher score of the leisure noise-exposed

group compared to the industrial noise-exposed group in speech-in-noise perception warrants mechanistic consideration. While comparative studies are limited, fundamental acoustical principles suggest that qualitative differences in sound characteristics (e.g., spectral dynamics, temporal structure, and emotional valence) differentially modulate neural processing. For instance, musical training can improve speech-in-noise perception when presented at a comfortable level that does not cause hearing damage [22]. Dynamic spectral-temporal information is used in music as in spoken language; therefore, it has been suggested that music and language may have a common neural biological processing system. Thus, loud music may have different emotional stress responses than industrial noise.

The higher SNR-loss in the leisure noise exposed group than the non-exposed group is consistent with the results of Ismail et al. [32], who reported that exposure to leisure noise due to excessive PLDs usage cause elevated high frequency audiometry, reduced amplitude of Transient Otoacoustic Emissions (TEOAEs) and decreased speech-in-noise scores despite normal thresholds. Li et al. reported that the fast-speed speech recognition in noise decreased significantly in PLD users compared to non-PLD users [33].

The DPST scores indicated a significant decline in temporal processing in the industrial noise-exposed group compared to the leisure noise-exposed and non-exposed groups. Temporal processing is essential for speech-in-noise perception and relies on the spontaneous firing rate of auditory nerve fibers. Industrial noise exposure has been shown in animal models to disrupt this spontaneous firing rate and damage afferent connections, potentially leading to phase-locking deficits [34]. Additionally, short-term auditory memory and attention —both essential for accurate DPST performance— may also be impaired by industrial noise. These results are consistent with the results of studies showing decreased temporal processing performance in noise-exposed individuals despite having normal hearing [17]. Contrarily, musical training was shown to enhance multiple auditory functions, including improved temporal fine structure processing, spectral resolution, and binaural integration [4]. These effects may explain the relatively preserved temporal processing in the leisure noise-exposed group, as music is often processed within emotionally and cognitively integrative brain systems.

The analysis of the DDT scores indicated the lower scores of the industrial noise-exposed group compared to the leisure noise-exposed and non-exposed groups. Noise can alter neural processing in the midbrain, thalamus, and cerebral cortex even without peripheral hearing loss, resulting in decreased DDT scores. Performance in DDT may be influenced by deficits in auditory processing, attention, and other supramodal processes. These results are in line with the findings of Bhatt and Wang, who reported that dichotic hearing is damaged in people who are exposed to industrial noise, despite having normal thresholds [12]. Musical training can enhance the bilateral processing of stimuli [4]. Therefore, no significant difference was seen in the DDT score between the leisure noise-exposed and non-exposed groups.

Memory performance was significantly impaired in the industrial noise-exposed group more than in the leisure noise exposed group. Noise affects memory by modulating auditory pathways connected to the hippocampus, particularly via lemniscal and non-lemniscal pathways. Functional MRI studies have confirmed these effects, showing reduced hippocampal and increased amygdala activity following chronic noise exposure [13]. In animal studies, it was shown that moderate-intensity noise (80 dB SPL, 2 h/day) can impair the memory, which may result from peroxidative damage, tau hyperphosphorylation, and auditory coding alteration [35]. Also, Liu et al. showed that noise exposure and noise-induced hearing loss, independent of oxidative stress, impaired spatial memory and spatial learning in mice [36]. A human study showed the harmful effects of industrial noise on working memory in people exposed to 85 dB (A) noise [17]. In contrast, music is known to facilitate memory encoding and retrieval. The emotional and contextual components of music can enhance recall and serve as mnemonic devices. However, excessive exposure to loud music may impair specific cognitive abilities such as visuospatial memory, as shown in high-exposure listeners [37].

The semantic Stroop test results indicated lower accuracy in the industrial noise-exposed group compared to the leisure noise-exposed and non-exposed groups, though the difference among the groups in reaction time was not statistically significant. Jafari et al. reported that low-frequency industrial noise impairs both visual and auditory attention, especially at intensities up to 95

dB A [10]. Therefore, it seems that the frequency and content of noise can have a different effect on attention. It was shown that there was a significant difference in the semantic Stroop test accuracy between the industrial noise-exposed and leisure noise-exposed groups. We found no study on the effect of listening to loud music on selective attention to compare the results; however, a study showed that even the level of sound (stimulating or relaxing) can have different or destructive effects on cognitive processes [4]. There is a research gap in this field that should be further investigated. we found a significant relationship between exposure level and speech-in-noise perception, especially in the industrial noise-exposed group. Although a multivariate regression model in a study showed that 23.3% of the variance in the speech-in-noise test was explained by group category and hearing thresholds [38].

Due to limitations in directly measuring PLD use output, we calculated equivalent noise exposure for the leisure noise-exposed group in our study. Developing apps for direct measurement is recommended. Although participants in our study had normal hearing, no high-frequency auditory testing was conducted, warranting future studies to assess the possibility of hidden hearing loss. Furthermore, the male-only cohort limits the generalizability of the results, including the need for future research on both genders.

Conclusion

Exposure to industrial noise can adversely affect temporal processing, speech-in-noise perception, binaural hearing, short-term memory, and selective attention in male workers with normal hearing. Furthermore, the men who frequently use Personal Listening Devices (PLDs) have impaired temporal processing, speech-in-noise perception, selective attention, and memory compared to non-PLD users. These results underscore the necessity of increasing the awareness of men working in noisy environments and regular PLD users. Noise exposure level should be integrated into hearing screening programs and protocols in occupational medicine, particularly for those in industrial settings, to reduce the risk of central auditory processing disorders. Additionally, addressing the potential adverse effects of improper PLD usage should be prioritized in public health programs. Given the existing research gaps, further research is recommended to comprehensively explore the harmful impacts of noise exposure on central auditory processing and cognitive functions.

Ethical Considerations

Compliance with ethical guidelines

This study has been registered with the Code of Ethics IR.TUMS.FNM.REC.1401.168 in the Ethics Committee of Tehran University of Medical Sciences.

Funding

This research did not receive any grant from funding agencies in the public, commercial, or non-profit sectors.

Authors' contributions

NA: Idea, data collection, writing the initial manuscript; VR: Idea, writing the initial draft, reviewing the manuscript and editing, statistical analysis; ET: Idea, reviewing, consulting; EF: Sample size estimation and statistical consultation; GM: Idea and reviewing; SN: Data collection.

Data Availability Statement

Access upon request.

Conflict of interest

The authors declare that they have no competing interests.

Acknowledgments

This research has been supported by Tehran University of Medical Sciences and Health Services grant No 1402-1-103-64462.

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