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## Original Article

## Concurrent effect of noise exposure and smoking on extended high-frequency pure-tone thresholds

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## Abstract

**Objective:** Concurrent effect of noise and smoking on hearing loss is a recent concern. In this study, the concurrent effect of noise and smoking on hearing loss in conventional frequencies and frequencies higher than 8 kHz was assessed. **Design:** This was a cross-sectional study on workers exposed to noise who were divided into two groups: smokers and non-smokers. Hearing thresholds were assessed by conventional audiometry, and HFA. Data were analysed using non-parametric tests and Student's t-test. **Study sample:** There were 212 workers. **Results:** Ninety-seven subjects were smokers and 115 individuals were non-smokers. All subjects were exposed to  $92.1 \pm 2.4$  dBA (Leq8h). The highest threshold in conventional and high-frequency audiometry was observed at 6 kHz and 16 kHz, respectively. Hearing threshold at frequencies above 1 kHz was significantly higher in the smokers than non-smokers. There was no correlation between hearing thresholds and pack-years of smoking. **Conclusions:** Concurrent exposure to noise and smoking may be associated with more hearing loss than exposure to noise alone in the conventional and high frequencies. However, other differences between smokers and non-smokers may explain these differences as well.

**Key Words:** Noise; smoking; noise-induced hearing loss; audiometry; high-frequency audiometry

Noise is one of the most pervasive hazardous factors in the workplace. Noise-induced hearing loss (NIHL) as the most common complication of exposure to noise is the second acquired cause of hearing loss after presbycusis and is known as a work-related problem (Schindler & Robinson, 2005; Rabinowitz, 2005). According to National Institute for Occupational Safety and Health (NIOSH), about 5.7 million workers (25% of all US workers) in manufacturing industry are exposed to hazardous noise (Tak et al, 2009). Another report estimates that about 28% of workers are exposed to noise level between approximately 85 and 90 dBA in the European Union (Safety & Work, 2000). Even when the exposure to occupational noise is terminated, exposure to hazardous noise from social sources still exists.

NIHL is typically a bilateral and symmetric sensorineural hearing loss which presents as a V-shaped notch at 3, 4, or 6 kHz, and a recovery at 8 kHz as the first sign (Hong, 2005; Meyer, 2007). When exposure to noise is continued, hearing loss at the aforementioned frequencies may deepen and it may even extend to lower frequencies, i.e. speech frequencies, and impair the worker's normal hearing (Rabinowitz, 2005). There is no acceptable treatment for NIHL, but it is preventable (Schindler & Robinson, 2005).

The Occupational Safety and Health Administration (OSHA) requires that a hearing conservation program (HCP) begins in the workplace when noise exposure exceeds 85 dBA, and a main aspect of HCP is serial screening of hearing thresholds by audiometry (Dell & Holmes, 2012). Workers' screening by periodic audiometric evaluations help detect early changes in hearing status before the development of clinical hearing loss (Daniell et al, 2002). Generally, audiometry is performed at conventional frequencies (0.25–8 kHz). Frequencies higher than 8 kHz have also been introduced as probably more sensitive frequencies to noise, so threshold change in high frequencies may precede hearing loss in conventional audiometric thresholds (Sá et al, 2007). Some studies have proposed extended high-frequency audiometry (HFA) for hearing screening in occupational evaluations (Mehrparvar et al, 2011, 2014; Porto et al, 2004; Singh et al, 2009).

Smoking is associated with many adverse effects such as some malignancies, atherosclerosis, and cardiovascular diseases. There is some evidence that smoking may cause hearing loss (Paschoal & Azevedo, 2009; Ohgami et al, 2011; Oliveira & Lima, 2009; Nakanishi et al, 2000). Some researchers have proposed that hearing loss is directly associated with pack-years of smoking (Foda et al, 2008),

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Abbreviations	
HCP	Hearing conservation program
HFA	High-frequency audiometry
NIHL	Noise-induced hearing loss
NIOSH	National Institute for Occupational Safety and Health
OSHA	Occupational Safety and Health Administration
PTA	Pure-tone audiometry

although there is controversy about the effects of smoking on hearing. Several studies have not shown an association between smoking and hearing loss (Karlsmose et al, 2000; Lindgren et al, 2008; Brant et al, 1996; Starck et al, 1999).

Nakanishi et al showed that smoking mostly affects 4 kHz (Nakanishi et al, 2000). Paschoal and Azevedo found that specific HFA frequencies (12.5 and 14 kHz) are most sensitive to the effects of smoking (Paschoal & Azevedo, 2009). Oliveira and Lima (2009) found as well that high frequencies of audiometry are more sensitive to smoking than low frequencies (Oliveira & Lima, 2009). But Starck et al found that smoking without the presence of other risk factors could not increase the risk of sensorineural hearing loss (Starck et al, 1999).

Concurrent effect of noise and smoking on hearing is a concern as well. Some studies have shown that smoking and noise may have additive or multiplicative effects on hearing threshold in standard audiometric frequencies (Foda et al, 2008; Mizoue et al, 2003; Pouryaghoub et al, 2007). In a recent study, Tao and colleagues (2013) found that smoking can aggravate the effect of noise on hearing only in workers with more than 10 years of exposure to noise (Tao et al, 2013). Ferrite et al also found a combined effect for smoking and noise on hearing loss among women (Ferrite et al, 2013).

It has been proposed that the underlying mechanisms of the effect of smoking on hearing include hypoxia due to vascular changes, capillary vasoconstriction, increased blood viscosity (Tao et al, 2013), antioxidative mechanisms (Cruickshanks et al, 1998), and the direct ototoxicity of nicotine (Sung et al, 2013). Furthermore, it has been reported that smoking and hypoxia affects high frequencies of hearing sooner than low frequencies (Fechter et al, 1987; Oliveira & Lima, 2009).

Considering HFA as a more sensitive test for evaluation of the effect of noise on hearing and higher sensitivity of high-tone frequencies (higher than 8 kHz) to smoking and hypoxia, concurrent effects of smoking and noise on these frequencies is also important. Therefore in this study, we aimed to assess the concurrent effect of noise and smoking on high-frequency pure-tone thresholds among workers exposed to noise.

**Method**

In a cross-sectional study from 1 March 2012 to 30 September 2012, we assessed the concurrent effect of occupational noise exposure and smoking on hearing threshold in standard and extended high frequencies in workers exposed to industrial noise higher than 85 dBA.

*Study population and risk variables*

Subjects were smokers randomly selected from different units of the tile industry who were exposed to continuous noise higher than 85 dBA. Controls were non-smokers from the same industry with similar noise exposure patterns (Table 1). All workers worked in

**Table 1.** Detailed data of noise measurement.

	Subjects		Frequency analysis, mean(SD), kHz											
	Smoker	Non-smoker	<i>L<sub>Aeq8h</sub></i>	0.03125	0.0625	0.125	0.125	0.125	0.5	1	2	4	8	16
Spray drying	15	20	94.9	73.1 (0.6)	80.6 (0.5)	83.3 (0.6)	85.7 (1.5)	86.3 (1.2)	82.1 (1.7)	88.3 (1.8)	86.2 (1.6)	92.1 (2.3)	92.1 (2.3)	78.2 (2.1)
Forming	18	17	94.1	80.2 (0.2)	81.3 (0.3)	83.7 (0.5)	81.6 (1.2)	85.2 (1.8)	79.3 (0.9)	91.5 (1.6)	79.3 (1.3)	86.2 (2.7)	86.2 (2.7)	80.0 (1.8)
Drying	17	20	92.8	77.2 (1.2)	76.5 (1.6)	74.5 (2.4)	82.2 (1.6)	82.8 (1.5)	70.2 (0.2)	90.1 (0.8)	82.1 (1.8)	84.3 (0.9)	84.3 (0.9)	75.9 (1.2)
Glazing	15	20	92.1	61.2 (0.7)	69.5 (1.6)	82.26 (1.5)	82.3 (2.6)	85.3 (2.2)	78.3 (1.3)	81.8 (1.1)	80.6 (1.3)	91.2 (2.1)	91.2 (2.1)	76.8 (1.5)
Firing	14	19	90.9	82.6 (1.8)	81.3 (2.8)	81.23 (1.2)	82.5 (1.6)	82.2 (1.5)	80.5 (2.1)	82.6 (2.3)	79.2 (1.2)	89.4 (1.6)	89.4 (1.6)	62.3 (1.1)
Packing and loading	18	19	89.4	79.5 (2.9)	80.0 (1.8)	74.2 (1.8)	77.3 (0.6)	79.2 (1.3)	81.4 (1.2)	80.0 (0.7)	80.5 (1.6)	87.6 (0.9)	87.6 (0.9)	70.2 (3.9)

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eight-hour shifts. None of the workers used hearing protection devices regularly.

Those with conductive hearing loss, diabetes mellitus, hyperlipidemia, hypothyroidism, hypertension, history of exposure to ototoxic substances or ototoxic drugs, history of participation in military missions, history of head trauma, congenital hearing loss, unilateral hearing loss, and ex-smokers were excluded from the study.

All subjects were males under 50 years old. Female workers were not enrolled in this study because there were few female workers exposed to hazardous noise. Twenty-three subjects were excluded from the study due to aforementioned criteria (16 subjects in the case, and seven subjects in the control group).

Data about smoking were obtained from each participant's self report at the time of health evaluations. Questions about smoking habit included the experience of smoking as non-smoker, ex-smoker, and smoker. Ex-smokers were not enrolled in the study and smokers were assessed by pack-years of smoking (i.e. the number of cigarettes smoked per day divided by 20 multiplied by years of smoking). In this study smokers were those who smoked at least 1 pack-year. We considered hearing loss in audiometry as more than 20 dB decrease in hearing threshold at each frequency (Schindler & Robinson, 2005).

#### Occupational noise measures

A preliminary survey showed that sources can be considered as a plane source (due to evenly distribution of sources in the units) with almost non-fluctuating pattern (fluctuation was less than 5 dB). There was also no significant change in production rate or other influential parameters on noise level in different working days. There was no impact or impulse noise. Therefore, measurements were performed randomly in a selected workday in a working week. The noise in each unit of the factory (i.e. spray drying, forming, drying, glazing, firing, packing and loading) was measured and the mean exposure of the subjects according to their workstation was calculated. Eight hour equivalent continuous noise level in A-weighted network (LAeq8h) was measured by a calibrated sound level meter (Casella CEL-383, UK) at a height of 160 cm. Production units were virtually divided into 5 × 5 metre quadratic squares and measurements were performed at the center of each square. Measuring time was around 3 to 5 minutes in each square and the sound level meter was read at a time interval of 60 seconds over the measurement duration, and the arithmetic mean was calculated. Octave band analysis was carried out as well to find the noise spectrum (octave levels are reported without A-weighting). This analysis in various work areas showed that noise was in the frequency range between 0.03125 to 16 kHz, with high sound levels in 2, 4, and 8 kHz. Table 1 shows detailed data about noise measurement.

#### Hearing threshold examination

At first, otoscopic examination was performed for all subjects by an occupational medicine resident. Then standard pure-tone audiometry (PTA) was performed for all subjects (considering at least 16 hours abstinence from exposure to noise) with a clinical audiometer (device: AC 40, Interacoustic, Denmark; headphone: TDH-39) at 0.250, 0.5, 1, 2, 3, 4, 6, and 8 kHz frequencies. Extended HFA was performed for each participant (device: same audiometer, headphone: Koss, R/80) at 10, 12, 14, and 16 kHz frequencies. Both tests were done in an acoustic chamber meeting ANSI 2004 criteria (ANSI, 2004).

Thresholds were measured in the range of -10 to 100 dBHL. Air conduction thresholds were established first in the right ear, then the left ear for all subjects across different frequencies. For threshold detection, descending and ascending techniques were used. Thresholds were defined as the lowest intensity at which the individual detected the sound. Evaluation of hearing thresholds was duplicated in each subject to ensure repeatability. For bone conduction, bone oscillators were placed on the mastoid bone and the same procedure was performed.

#### Statistical analysis

Data were analysed by SPSS software (ver. 19). An independent samples T-test was used to compare means in variables with normal distribution. A Kolmogorov-Smirnov test was used to test normality of data and showed that audiometric data are not normally distributed, so non-parametric tests were used for data analysis (i.e. Spearman's correlation test and Mann-Whitney U test).

#### Ethics statement

The study was approved by the ethics committee of Shahid Sadoughi University of Medical Sciences (# 2428). An informed consent was obtained from all participants.

## Results

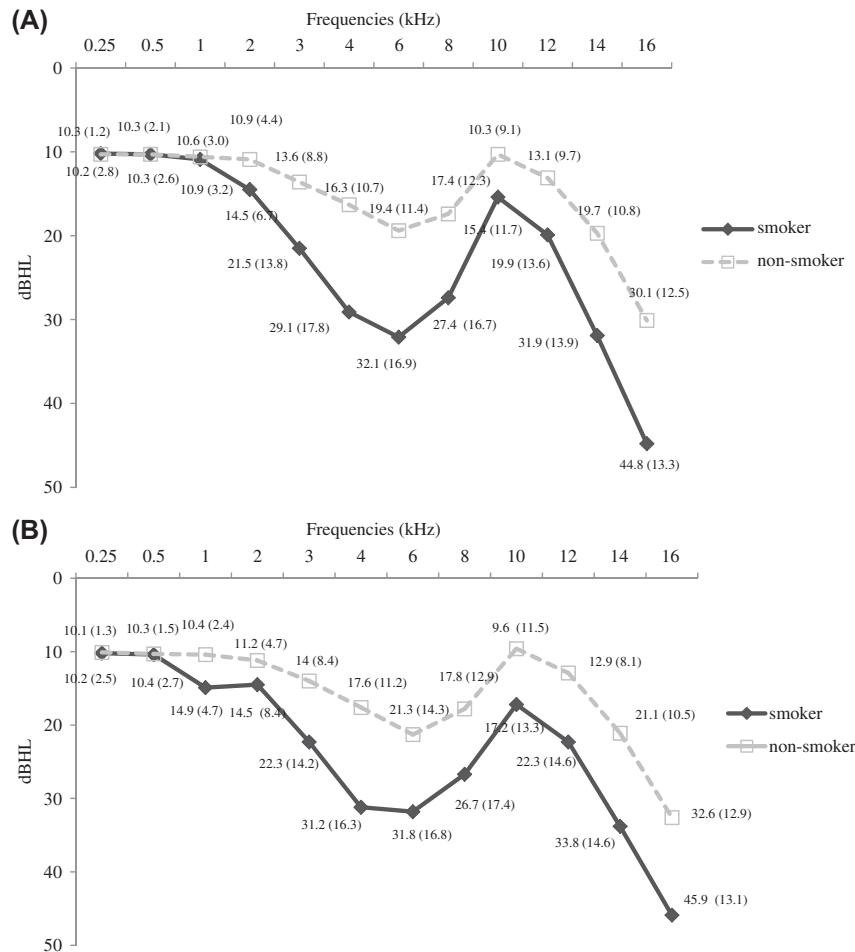
In total 212 tile and ceramic workers exposed to hazardous noise (higher than 85 dB) were evaluated. Ninety-seven subjects were smokers (group of smokers) and 115 individuals were non-smokers (control group). Mean age and work experience of the subjects was  $36.89 \pm 6.70$  years (range: 21–50 years), and  $12.00 \pm 6.17$  years (range: 3–30 years). Subjects in the smokers group smoked  $6.7 \pm 2.4$  pack-years (range: 1–35 pack-years). The mean exposure to noise in all subjects was  $92.1 \pm 2.4$  dBA (Leq8h). The highest level of noise was found in spray drying (94.9 dBA), and the lowest noise level was observed in loading (84.9 dBA). Workers had been selected from different parts of the factory with different number of workers in two groups, so the mean exposure of subjects in two groups showed a negligible difference which was not practically and statistically significant. Therefore, the groups were comparable in terms of noise exposure. Table 2 shows descriptive statistics of all subjects in two groups.

The highest threshold in conventional and high-frequency audiometry was observed at 6 kHz and 16 kHz, respectively. Figure 1 shows the mean hearing thresholds at different frequencies in the smokers and non-smokers groups.

**Table 2.** Comparison of demographic data between case and control groups.

Variables	Smoking status	Mean	SD*	t	df*	P-value
Age (year)	Smoker	37.87	7.59	1.93	210	0.057
	Non-smoker	36.06	5.75			
Sound level (dBA)	Smoker	92.0	2.5	-0.463	210	0.643
	Non-smoker	92.1	2.4			
Work period (year)	Smoker	12.68	6.79	1.45	210	0.145
	Non-smoker	11.42	5.56			

\*SD: Standard deviation, df: degree of freedom.



**Figure 1.** Comparison of mean (SD) of hearing thresholds at different frequencies in right (A) and left (B) ears.

Mann-Whitney U test showed that hearing threshold was significantly higher in the group of smokers at frequencies higher than 1 kHz. Table 3 compares median of hearing threshold at different frequencies between two groups.

The smokers were also divided into heavy smokers with 10 pack-years of smoking or more ( $n = 24$ ) and light smokers ( $n = 72$ ), and the comparisons failed to show a significant difference between two groups regarding hearing threshold at all frequencies. There was not a significant correlation between hearing threshold at conventional and high frequencies and pack-years of smoking which was confirmed by scatterplots as well. Figure 2 shows the scatterplot of pack-years of smoking and hearing thresholds in 12, 14, and 16 kHz in both groups after excluding one outlier with 35 pack-years of smoking ( $n = 96$ ).

## Discussion

Noise affects sensory hair cells in the cochlea by mechanical, metabolic and vascular mechanisms as well as production of oxygen radicals. Smoking may affect hearing by such mechanisms as hypoxia, oxygen radicals production, vasoconstriction or direct nicotine toxicity (Tao et al, 2013; Cruickshanks et al, 1998; Sung et al, 2013), some of which are the possible mechanisms of the effect of noise on hearing. There is still controversy about the effect of smoking and the concurrent effect of smoking and noise on hearing.

In conventional audiometry which is routinely performed as a part of HCP, 3, 4, and 6 kHz are the frequencies most commonly affected by noise and probably due to smoking (Nakanishi et al, 2000; Mizoue et al, 2003). Higher hearing thresholds in HFA following exposure to noise is also important; this may be explained by the cochlear anatomy, and vascularization of the cochlear base (Paschoal & Azevedo, 2009; Zimmerman et al, 2006). There is controversy about the effect of smoking on HFA: some studies have shown that smoking affects HFA (Paschoal & Azevedo, 2009; Ohgami et al, 2011; Oliveira & Lima, 2009); but the results of the Starck et al study are against this assumption (Starck et al, 1999).

In the current study, we found that high frequency thresholds especially 16 kHz were more severely affected due to exposure to noise and cigarette smoking. Other studies have also shown that the thresholds of frequencies higher than 8 kHz are more sensitive to noise than conventional thresholds, i.e. 8 kHz and lower (Mehrparvar et al, 2011; Porto et al, 2004; Singh et al, 2009; Lopes et al, 2009; Türkkahraman et al, 2003). To the best of our knowledge the concurrent effect of noise and smoking on high frequencies of audiometry ( $\geq 10$  kHz) has not been yet studied.

We found that both smokers and non-smokers suffered from hearing loss. The hearing thresholds were highest at 16 kHz, and then at 14 kHz and 6 kHz in both groups. In their study, Ottoni and colleagues (2012) found the frequencies of 16 kHz and 6 kHz to be the most sensitive to effects of noise in four different industries.

**Table 3.** Comparison of hearing thresholds between smokers and non-smokers using Mann-Whitney U test.

Audiometric frequencies (kHz)	Smoking status	Right ear				Left ear			
		Median	Mean rank	Mann-Whitney U	P value	Median	Mean rank	Mann-Whitney U	P value
0.250	Smoker	10	106.16	5545.0	0.89	10	105.12	5443.5	0.54
	Non-smoker	10	106.78						
0.5	Smoker	10	107.39	5491.0	0.71	10	107.85	5446.5	0.57
	Non-smoker	10	105.75						
1	Smoker	10	112.30	5015.0	0.06	10	109.51	5285.5	0.27
	Non-smoker	10	101.61						
2	Smoker	15	128.29	3463.5	<0.001	10	121.55	4117.5	<0.001
	Non-smoker	10	88.12						
3	Smoker	15	135.99	3215.0	<0.001	20	131.91	3112.5	<0.001
	Non-smoker	10	85.96						
4	Smoker	25	135.99	2717.0	<0.001	30	138.98	2426.5	<0.001
	Non-smoker	10	81.63						
6	Smoker	30	135.79	2736.5	<0.001	30	134.13	2897.5	<0.001
	Non-smoker	15	81.80						
8	Smoker	25	133.51	2958.0	<0.001	20	129.14	3381.0	<0.001
	Non-smoker	15	83.72						
1	Smoker	10	133.73	2936.5	<0.001	10	136.58	2660.0	<0.001
	Non-smoker	10	83.53						
12	Smoker	15	132.21	3083.5	<0.001	15	135.14	2799.5	<0.001
	Non-smoker	10	84.81						
14	Smoker	30	136.83	2635.5	<0.001	30	136.22	2694.5	<0.001
	Non-smoker	15	80.92						
16	Smoker	45	139.66	2361.0	<0.001	45	136.79	2639.5	<0.001
	Non-smoker	25	78.53						

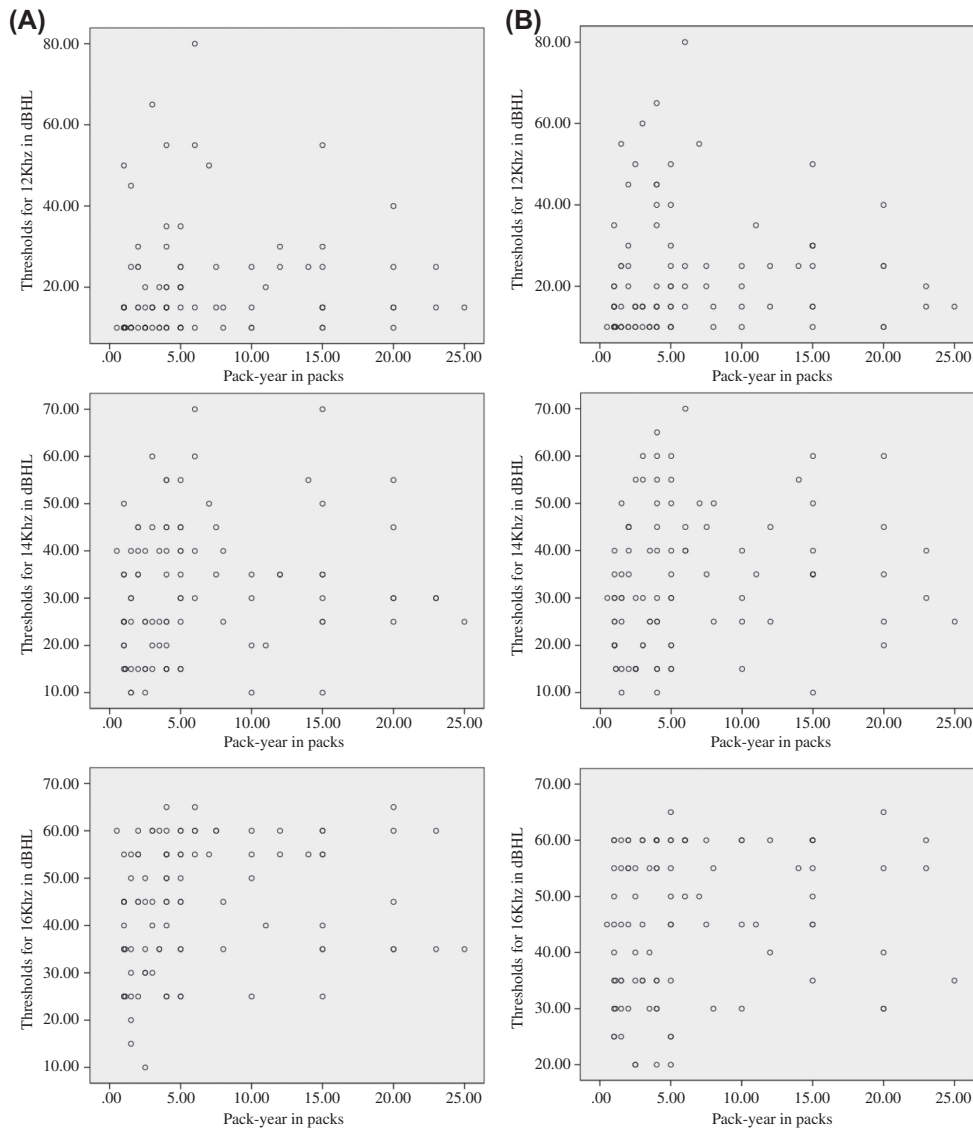
SEM: standard error of mean, SD: standard deviation.

Most studies have found that smoking and noise affect 4 kHz more severely than other conventional frequencies (Mizoue et al, 2003; Uchida et al, 2005; Mohammadi et al, 2010), but in this study we found 6 kHz as the most sensitive frequency to noise and smoking in conventional audiometry. We used TDH-39 headphone for conventional audiometry which is subject to technical errors in frequencies higher than 4 kHz, so it is possible that this difference is due to this issue (ANSI, 2004; Schlauch & Carney, 2011). The difference can also be attributed to different spectra of noise in different workplaces. Ottoni et al found a significant difference among four industries in terms of noise spectrum (Ottoni et al, 2012). In the present study, peak frequency was at 8 kHz, which did not coincide with audiometric configuration in this industry. Ottoni et al similarly could not find a clear association between audiometric configuration and noise spectrum (Ottoni et al, 2012).

In the current study, the hearing threshold in conventional audiometry was higher in smokers than non-smokers at 2, 3, 4, 6, and 8 kHz, consistent with some previous studies (Mohammadi et al, 2010; Foda et al, 2008; Wild et al, 2005). This difference was not observed at lower frequencies (i.e. 0.25, 0.5, and 1 kHz). We found that hearing thresholds at 10, 12, 14, and 16 kHz were significantly higher in smokers than non-smokers; although these results should be interpreted cautiously. We did not have detailed information about wearing hearing conservation devices by the workers; we found only that the use of these devices was completely irregular. Some of the workers probably had exposure to noise in their previous or second jobs and their social activities (such as motor cycling) or their military service without mentioning in the interview due to forgetfulness or secondary gain. Both of these factors are important confounding factors in terms of the effect of noise on hearing.

In the current study the dose-response relationship of smoking and hearing loss was also important. Foda et al (2008) have found a positive correlation between hearing loss and pack/years of smoking, but the current study failed to show a significant correlation between pack/years of smoking and hearing thresholds at conventional and high frequencies. The categorization of the smokers into heavy and light smokers also did not show a difference between two groups in terms of mean hearing thresholds. A positive correlation or a dose-response relationship between smoking and hearing loss could not be obtained with confidence from the results of this study.

According to the results of the current study, smoking may aggravate the effect of workplace noise on hearing thresholds in both conventional and high-frequency audiometry. The effect on conventional audiometry confirms the results of most previous studies (Foda et al, 2008; Mizoue et al, 2003; Pouryaghoub et al, 2007). The exacerbating effect of smoking on hearing loss due to noise in HFA is a new issue. This concurrent effect, especially on high frequencies, can be explained by several mechanisms. Smoking and noise both induce vascular changes, which is more prominent in high frequencies due to cochlear anatomy, and vascularization of the cochlear base (Starck et al, 1999; Tao et al, 2013; Chen, 2002; Paschoal & Azevedo, 2009). Release of some chemicals from cigarette smoke which are ototoxic may explain the hearing loss as well (Morata et al, 1993; Mehrparvar et al, 2013). So according to the results of the current study, due to higher sensitivity of HFA to the concurrent effect of noise and smoking together, it can be recommended to use HFA for hearing screening in occupational settings, especially for those workers who are smokers. It is also important to pay attention to the factors other than smoking which may affect hearing and have exacerbating or additive effects with noise.



**Figure 2.** Scatterplot of pack-years of smoking and hearing threshold in right (A) and left (B) ears.

In this study the concurrent exposure to noise and cigarette on hearing was evaluated by HFA in addition to conventional audiometry. We compared hearing thresholds among two similar groups regarding exposure to noise, age, and work experience.

This study had some limitations as well. This was a cross-sectional study, so it suffers from the intrinsic limitations of these kinds of studies, and finding a causal relationship was not possible; our clinical audiometer was not able to assess frequencies higher than 16 kHz; we could not assess how the workers used hearing conservation devices in detail; the number of females was few in this study, so we excluded the females, therefore our results could not be used for females; we used a headphone (TDH-39) for measurement of hearing thresholds which is subject to technical errors and we could not prepare another headphone.

## Conclusions

The results of this study showed that conventional and high frequency hearing thresholds in workers with exposure to noise and

smoking are higher than those with exposure to noise alone. A causal effect could not be concluded from the results of this study and other differences between the smokers and non-smokers may also explain the differences observed in this study. Further research is needed to establish if a concurrent effect from smoking and noise exposure are associated with elevated high frequency hearing thresholds or if other factors can explain this difference.

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Author contribution: A.H. Mehrparvar (design of the study, data analysis, manuscript preparation, and final review); S.J. Mirmohammadi (design of the study, and final review); S.H. Hashemi (design of the study, data gathering, and manuscript preparation); M.H. Davari (data gathering and data analysis); Mehrdad Mostaghaci (data analysis and manuscript preparation); A. Mollasadeghi (design of the study and data gathering); Z. Zare (data gathering and data analysis).

**Declaration of interest:** The authors report no conflicts of interest.

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