

Effect of simultaneous exposure to occupational noise and cigarette smoke on binaural hearing impairment

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Abstract

In recent years, it has been postulated that cigarette smoking can aggravate noise-induced hearing loss (NIHL). In this study, we aimed to assess the effect of concurrent exposure to cigarette smoke and occupational noise on binaural hearing impairment (BHI). In an analytic study on the workers of a large wagon manufacturing company in 2007, 622 male workers (252 smokers and 370 non-smokers, matched for other variables) participated and their BHI was compared. BHI was significantly higher in smokers than in non-smokers (odds ratio= 5.6, $P < 0.001$, 95% CI =3.4-9.4). Logistic regression confirmed this significant difference as well, and showed a direct relationship between the amount of BHI and pack/years of smoking. Cigarette smoking accompanied by exposure to workplace noise may play a role in causing binaural hearing impairment, so giving up or decreasing the amount of smoking may prevent or at least delay binaural hearing impairment, and eventually reduce its compensation costs.

Keywords: Cigarette smoking, hearing loss, noise, occupational exposure

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Introduction

Noise is one of the most pervasive and important physical hazards at workplaces.^[1] Millions of workers worldwide are exposed to work-place noise.^[2] According to National Institute on Occupational Safety and Health (NIOSH), 14% of working population in the US is exposed to noise higher than 90 dBA in the workplace and this ratio exceeds 25% in such manufacturing plants and industries such as textile, transportation, lumber and wood, and food and kindred.^[3,4] Thus noise-induced hearing loss (NIHL), by early exclusion of skilful and experienced workers from production cycle and high costs of treatment, rehabilitation and compensation, can impose a large social and economic burden on the society.^[5,6] Likewise, NIHL can affect the workers' quality of life and cause problems such as social isolation, depression and an increased risk of accidents.^[7,8] One important issue is that NIHL is frequent, permanent and irreversible, but preventable.^[9]

Long-term exposure to hazardous noise mostly affects hair cells of the organ of Corti resting in inner ear.^[10] Injury of these cells leads to NIHL. The sound frequencies between 3000 and 5000 Hz (especially 4000 Hz) usually cause damage to hair cells, and the damage is gradually spread to adjacent areas sensitive to other frequencies, i.e. 6000 and 8000 Hz.^[11,12]

Recently, Simultaneous exposure to cigarette smoke and occupational noise has been proposed as an important factor which can increase the probability of hearing loss.^[13] Smoking is one of the most common habits in all social classes, including workers.^[14] It has been estimated that more than one milliard persons worldwide smoke.^[15]

By increasing the blood viscosity and decreasing oxygenation, cigarette smoking can impair cochlear blood circulation.^[16] Exposure to cigarette smoke has been propounded as an etiologic factor for cochlear lesions.^[17,18]

In this study, we decided to assess the simultaneous effect of noise and cigarette smoking on workers' hearing impairment. For measuring hearing impairment, we used the formula recommended by American Association of Otolaryngology (AAO).^[19] This method is confirmed by the last edition of "guidelines to the evaluation of permanent impairment" published by American Medical Association.^[20]

The main objective of this study is to examine the hypothesis that cigarette smoking and occupational noise jointly affect hearing impairment, which could be of particular relevance in noise controlling programs in workplaces.

Materials and Methods

We assessed hearing impairment in a large wagon manufacturing factory in the capital of Iran in 2007 by a cross-sectional and descriptive-analytic study.

A team of occupational hygienist working in Health and Safety Executive (HSE) managed noise monitoring. Digital Sound level meter (EXAIR, model 9104) was placed in 25 various stations according to ISO 1999. In this study, after measuring sound level in different parts of the factory, areas with high levels of noise were selected; eventually five areas of the factory with the highest levels of noise were selected as noisy areas. Minimum and maximum noise levels at different stations of these five parts were 88 dBA and 96dBA, respectively (The 8 h time weighted averaged means = 91 dBA).

Our study population ($n = 307$) were male smokers working in these noisy areas. Female workers were not included in this study because of the low noise exposure (almost all women were office workers). After direct interview by an occupational medicine specialist, and considering exclusion criteria (history of ototoxic drug consumption, diabetes mellitus, hyperlipidemia, hypothyroidism, severe or frequent ear infection, chronic middle ear pathology or major ear operations, head injury, exposure to non-occupational noise, such as amplified music, participation in war, and hunting, noise exposure in previous job/jobs, unilateral or conductive hearing loss or any kind of hearing loss with a known etiology except for noise exposure), 55 persons were excluded from the study [Table 1] and 252 male smokers who worked in noisy workplaces comprised the target population (ex-smokers, i.e. those who had quitted smoking were also excluded). Also there were 453 male non-smoker workers in these noisy parts (83 workers were excluded by applying the exclusion criteria).

Inclusion and exclusion criteria for this group were the same as the smokers group.

The excluded cases are shown in Table 1.

Data collection method was also the same. Thus, the only difference between the two groups was cigarette smoking.

All workers participated voluntarily in this study and an informed consent was filled for them (written consent was obtained in Persian).

Hearing threshold was assessed by a qualified audiologist after at least 14 h noise avoidance using Interacoustics Audiometer AD229e (Interacoustics A/S, Assens, Denmark) in an acoustic chamber, meeting the ANSI S3.1–1991 standards.

Table 1: Excluded cases in smoker and non-smoker groups

Parameters	Smoker workers	Non-smoker workers
Diabetes mellitus	11	19
Hyperlipidemia	15	24
Hypothyroidism	3	4
Unilateral hearing loss	9	11
Conductive hearing loss	7	12
Ototoxic drug consumption	4	5
Severe or frequent ear infection	2	3
Major ear operations	2	2
Head trauma	2	3
Total	55	83

Hearing threshold at 500, 1000, 2000, 3000, 4000, and 6000 Hz was measured for air and bone conduction, in both ears.

We assessed binaural hearing impairment according to AAO formula.^[19,20] Measuring binaural hearing impairment (BHI) by this method is as follows:

Hearing impairment in each ear = [(average hearing threshold at 500, 1000, 2000, and 3000 Hz) – 25] × 1.5

Then binaural impairment should be calculated by multiplying the smaller percentage (better ear) by 5, adding this figure to the larger percentage (poorer ear) and dividing the total by 6.^[19,20]

This study was approved by the Ethics Committee of Iran University of Medical Sciences.

The collected data was analyzed using SPSS for Windows software, Version 14.5. The following statistical tests were applied for analysis of the data. T test was applied for testing differences between the case and control groups for quantitative parameters. Chi-square test was applied to examine differences between the case and control groups for categorical parameters. Association of binaural hearing impairment with smoking habit was examined by logistic regression. All tests applied were two-tailed, and a *P* value of 0.05 was considered to be statistically significant.

Results

In all 622 workers, the mean age and duration of exposure to noise was 42.09 years (± 6.8) and 17.8 (± 6.6) years, respectively.

Average binaural hearing impairment in all workers under study was 1.81% (± 5.4). Descriptive statistics of all workers is shown in Table 2.

According to Table 2, average age and duration of exposure to noise in smokers are more than that in non-smokers, but this difference is not clinically important.

In this study, 26.9% of smokers and 6.2% of non-smokers showed binaural hearing impairment (odds ratio = 5.6, 95% CI =3.4 - 9.4, *P* value <0.001). In other words, smokers exposed to noise are 5.6 times more liable to binaural hearing loss than non-smokers.

To more exactly assess the relationship between binaural hearing impairment and smoking and eliminate the confounding effect of age and duration of exposure to noise, we used logistic regression analysis. In this analysis (after eliminating confounding variables) there was still a statistically significant relationship between smoking and hearing impairment. To perform this analysis, we divided the workers into two groups: those suffering from hearing impairment and those without any impairment. Then they were divided into two groups according to their age (≤ 40 years old and more than 40 years old) and their exposure duration to noise (< 20 years and ≥ 20 years), and eventually according to their smoking habit were divided into 3 groups (non-smoker, smoker with less than 15 pack-year smoking, and smoker with ≥ 15 pack-years of smoking). The details of this analysis can be observed in Table 3.

According to Table 3, there is a statistically significant relationship between binaural hearing impairment and cigarette smoking and age and also duration of exposure to noise. This analysis showed that hearing loss odds ratio increases in accordance with increased pack-years of smoking.

Table 2: Descriptive statistics of workers according to smoking

Variable	Smoker		Non-smoker		P value
	Mean (SD ^a)	Range	Mean (SD ^a)	Range	
Age (year)	42.8 (6.07)	21-65	41.5 (7.2)	24-66	< 0.05
Duration ^b (year)	18.7 (6.3)	3-30	17.2 (6.8)	3-31	< 0.05
BHI ^c (percent)	3.9 (7.8)	0-33.7	0.3 (1.7)	0-11.2	<0.001
Smoking (pack-year)	14.6 (11.1)	0.4-64	—	—	—

^aStandard deviation; ^bDuration of exposure to noise; ^cBinaural hearing impairment

Table 3: Relationship BHI and age, duration of exposure to noise, and cigarette smoking by logistic regression analysis

Variable	β	Standard error	Risk ratio (95% CI ^a)	P value
Constant coefficient	-0.1	0.1	—	0.5
Age (year)			1.00	< 0.05
≤ 40 (n=221)	0.9	0.4	2.6 (1.1-6.1)	
> 40 (n=401)				
Duration ^b (year)			1.00	< 0.05
< 20 (n=312)	0.7	0.3	2.1 (1.1-3.8)	
≥ 20 (n=310)				
Smoking (pack-year)			1.00	<0.001
Non-smoker (n=370)	1.9	0.4	7.1 (3.2-15.8)	<0.001
< 15 (n=118)	2.2	0.2	9.6 (5.5-16.8)	
≥ 15 (n=134)				

^aStandard deviation; ^bDuration of exposure to noise; ^cBinaural hearing impairment

Discussion

In our study, among the employees exposed to noise higher than 85 dBA, probability of the incidence binaural hearing impairment in smokers is 5.6 times more than that in non-smokers. Therefore, it can be mentioned that cigarette smoking increases the probability of binaural hearing impairment in a statistically significant manner.

And among smokers, probability of the incidence of binaural hearing impairment increases according to increased pack-years of smoking.

Although there are some studies about the effect of cigarette smoking on occupational hearing loss,^[21-30] we could not find any study on the effect of concurrent exposure to occupational noise and cigarette smoking on binaural hearing impairment based on AAO formula.

Conclusions

It can be concluded that smoking accompanied by occupational exposure to noise may accelerate binaural hearing impairment, which should be confirmed by other studies. If longitudinal studies confirm this finding, we can recommend that giving up smoking or adjusting this habit can delay binaural hearing impairment or decrease its intensity.

We can also recommend that interval between audiometric assessments should be lowered (e.g. biannual instead of annual) to diagnose hearing complications sooner and prevent hearing impairment and eventually reduce the compensation costs due to it.

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