Cigarette Smoking and Hearing Loss: Lessons from the Young Adult Periodic Examinations in Israel (YAPEIS) Database

Yehonatan Sharabi MD[1,3], Idit Reshef-Haran MSc[2], Moshe Burstein MD[3] and Arieh Eldad MD[3]

[1]Staff Periodic Examination Center, and [2]Hearing Institute, [3]Israel Defense Forces Medical Corps, Israel

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Abstract

Background: Some studies have indicated a possible link between cigarette smoking and hearing loss.

Objectives: To analyze the association between smoking and hearing loss, other than that induced by noise, and to characterize the type of HL impairment found in smokers.

Methods: We conducted a retrospective cross-sectional study in 13,308 men aged 20–68 (median 34.6 years) who underwent a hearing test as part of a routine periodic examination. For each subject, age, smoking status (current, past or non-smokers) and number of cigarettes per day were noted and a hearing test was performed. The test was performed in a sealed, soundproof room by an experienced audiologist and included pure tone audiometry of 250–8,000 Hz. The audiograms were analyzed and subjects were accordingly divided into two groups: those with HL and at least one of the following impairments in at least one ear: sensorineural, conductive or mixed; and those with no hearing loss (control). Audiograms showing HL typical to noise exposure were excluded.

Results: The prevalence of any type of HL among subjects < 35 years was 4.5%, compared to 10.5% among those > 35 years ($P < 0.0001$). A significantly higher incidence of any type of HL was found in current (11.8%) and past smokers (11.7%) than in non-smokers (8.1%) ($P < 0.0001$). The risk increment of the smoking status for developing HL among subjects under age 35 was 43%, and 17% among those above 35 years. Both mild, flat, sensorineural impairment and conductive impairment were found to be associated particularly with smoking (odds ratio 2.2 and 1.9, respectively).

Conclusions: The incidence of HL unrelated to noise exposure is higher in smokers than in non-smokers, and in young adults the effect is greater.

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Smoking is a widespread addiction among youth (32% in our survey of over 40,000 young adults), and the damage caused by inhaling toxic substances from cigarettes has been widely reported. A vast amount of data points particularly to the connection between smoking and diseases of the cardiovascular system, lungs, malignancy, and the digestive system, but little has been written of the association between smoking and hearing loss

While hearing loss is common among the elderly [1], the young also suffer from hearing impairment, which, according to the impaired spectrum of frequencies, is not induced by noise. The causes probably arise from a combination of genetic and environmental factors. The medical literature describes a relationship between HL and smoking, but these reports are equivocal as some researchers found a correlation [2–5] while others did not [6,7]. Moreover, most of these surveys were performed among the elderly.

The present study aimed to examine the association between smoking and HL and to characterize the type of impairment found in young smokers. An analysis of the relation between noise-induced HL and smoking is beyond the scope of the present study and might confound its objective, therefore, we focused on the relation to HL other than that induced by noise.

HL = hearing loss

Materials and Methods

The study population comprised 13,308 men aged 20–68 (median 34.6 years) who underwent hearing tests during a routine periodic health examination between the years 1995 and 1998. All subjects completed a questionnaire including their age and smoking status. Subjects who were smoking at the time of the examination were regarded as current smokers, and the number of cigarettes per day and duration of smoking (in years) were noted. Subjects who had smoked previously but had ceased at the time of examination were regarded as past smokers, and the date of cessation as well as the number of cigarettes per day smoked at that time were noted. Subjects who had never smoked were regarded as non-smokers.

Hearing tests were performed in a sealed, soundproof room with a calibrated clinical audiometer (GSI-16, USA). An experienced audiologist, who was unaware of the subject's smoking status, conducted the test. The basic hearing test included pure tone audiometry (air conduction hearing threshold and speech reception threshold). The range of frequencies tested was 250–8,000 Hz. In cases where the air conduction threshold in one of these frequencies was >25 dB hearing level, bone conduction threshold and speech discrimination were also examined. Subjects found to have impaired hearing test were then referred for further evaluation and treatment.

An experienced audiologist coded the audiograms according to the type of hearing impairment. Result codes were graded according to severity and, according to the shape of the audiogram, subtyped to low tone loss, high tone loss, flat, sloping or notched. The severity of the HL was determined as follows: 25–35 dB = mild impairment, 40–60 dB = moderate, and >65 dB = severe. For sensorineural it was determined according to the bone conduction threshold at the impaired frequencies, and for conductive or mixed according to the air conduction threshold, primarily at the speech range (500–2,000 Hz). The study population was divided into two groups according to the types of HL. The first group comprised those with HL and at least one of the following criteria in at least one ear:

a) Sensorineural – namely, bone conduction thresholds 25 dBHL in one or more of the following frequencies: low tone (250–1,000 Hz) and high tone losses (2,000–8,000 Hz)

b) Conductive HL – air conduction thresholds 30 dBHL and bone conduction thresholds 20 dBHL in most of the tested frequencies.

Subjects with conductive HL were examined for the presence of cerumen in the ear canal; these subjects were retested after the cerumen was removed. The results of the repeated test were coded and analyzed.

c) Mixed HL (sensorineural and conductive).

The control group comprised subjects with none of the above-mentioned hearing impairments. Those whose audiogram revealed impairment at high frequencies (2,000–8,000 Hz) and/or a typical 4,000 Hz "dip," together with a history of noise exposure – and virtually all of our study population were exposed to noise – were considered as having noise-induced hearing loss [8]. Subjects with noise-induced HL according to the audiogram, and a history of noise exposure, were excluded and regarded as *not* having sustained HL.

Statistical analysis

The relationship between smoking and HL with and without age adjustment was tested by the chi-square and Cochran-Mantel-Haenszel tests. Subjects were divided into two age groups: under the age of 35 and over 35 according to the median age of the study population. A logistic regression model was applied to test the combined influence of age and smoking on hearing impairment. The differences in degree of impairment for each of the three smoking groups (current, past, and non-smokers) were tested by the chi-square test and the frequency of each type of injury was noted.

Results

The mean age of the subjects was 34.5 $\pm$ 3.4 years. The mean age of the group under 35 years was 28.3 $\pm$ 3.3 and those above 35 was 41.2 $\pm$ 3.4. As expected, age was the major determinant for HL, and the prevalence of HL among subjects under age 35 was 4.5% compared to 10.5% among those above 35 ($P < 0.0001$). Table 1 depicts the incidence of HL according to smoking status and age. A significantly higher incidence of HL was found in current and past smokers as compared to non-smokers ($P < 0.0001$). Figure 1 shows the risk increment of smoking status on HL.

Effect of number of cigarettes on HL

The prevalence of HL was significantly higher in smokers of more than 20 cigarettes per day than in those who smoked less than 20, but when this was adjusted for age the difference was not significant.

Type of HL among smokers and non-smokers

The type of hearing loss was analyzed in the study population, and the frequency of each impairment in the various study groups was tested. Conductive impairment was the most frequent finding in all study groups. This finding was characteristic of 6.1% of those who were currently smoking, 6.1% of the group who had smoked in the past, and 3.3% of the non-smokers. The incidence of this type of HL in non-smokers was significantly lower than that of the other two groups [Table 2].

Furthermore, a significant difference in the incidence of mild (bone conduction 25–35 dBHL), flat curve, sensorineural impairment was found among the study groups: 0.7 % in non-smokers vs. 1.5% in current smokers vs. 1.2% in past smokers. Analysis of other subtypes of impairments showed no significant difference between the various study groups.

dBHL = dB hearing level

Table 1. Number of subjects with/without HL according to age group and smoking status

Age (yr)	Current smoker	Past smoker	Non-smoker	Total
All subjects	431/3,639	249/2,117	617/7,552	1297/13,308
%	11.84*	11.76*	8.17	9.7
Subjects = 35	136/1,990	48/757	245/5,116	429/7,863
%	6.83*	6.34	4.79	5.45
Subjects > 35	295/1,649	201/1,360	372/2,436	868/5,445
%	17.89*	14.78	15.27	15.9

* Statistically significant difference compared to non-smokers ($P<0.05$)

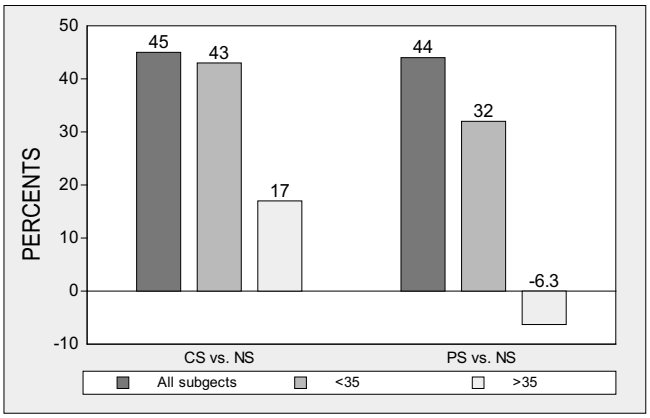

Figure 1. Risk increment of smoking status on hearing loss (CS = current smokers, PS = past smokers, NS = non smokers). *$P < 0.05$

Table 2. Relative risk or odds ratio for type of hearing loss according to smoking status

	Current vs. non-smokers		Past vs. non-smokers		*P*
	Odds ratio	CI (95%)	Odds ratio	CI (95%)	
Conductive impairment	1.85	1.31–2.27	1.85	1.41–2.64	0.0001
Mild, flat, sensorineural impairment (bone conduction 23–25 dBHL)	2.16	1.06–3.26	1.75	0.64–2.87	0.018

Discussion

The above results show that smoking increases the chance of developing HL by 45%, and that the deleterious effect of smoking on HL is more pronounced in young subjects (up to 35 years of age). The most frequent type of hearing impairment in smokers was found to be of mild, flat, sensorineural HL and the conductive type.

Our study is of a large scale but has few limitations: it is

retrospective and the data are based on a computerized code given by the audiologist according to the type of hearing impairment based on the audiograms rather than by analyzing the audiograms themselves. Our study did not aim to reveal the etiology of this phenomenon nor does it shed light on the mechanisms by which cigarette smoking enhances hearing impairment. Theoretically, the literature points to several possible etiologies: for the conductive HL it might be related to a higher prevalence of rhinosinusitis or eustachian tube dysfunction among smokers [9–12], whereas for sensorineural HL it might be due to an oxidative damage caused by toxic substances inhaled with the cigarette smoke [13], or to the acceleration of the arteriosclerotic process. Indeed, certain studies found a correlation between atherosclerotic risk factors other than smoking and hearing injury [14]. A cohort research that tested the relationship between cardiac or cerebral events and HL found that in patients who suffered such an event the frequency of HL was more than threefold higher [15]. In addition, a retrospective study of 699 employees from a low noise environment showed an association between cardiovascular risk factors and HL, especially in young adults [4]. Some studies even indicate that smoking is a risk factor for HL only in the presence of high blood pressure [16]. The smoking effect on HL was also demonstrated in animal studies where cochlear damage was noted after inhalation of cigarette smoke [13].

Reports dealing with the relationship between smoking and HL are sparse. However, results similar to ours were described in a population-based study of 45,541 subjects ranging in age from 48 to 92 years [2]. In this study, HL was defined as a pure tone average greater than 25 dB hearing level in the worse ear. Smoking caused a 69% increase in HL. A survey to estimate the prevalence of HL in adults above age 65 found HL in 54.6%, with a definite statistical correlation to smoking [3]. A case-control study by Itoh and colleagues [17] determined an odds ratio of 2.1 for HL among smokers. On the other hand, a study testing forest workers in Finland found a correlation between HL and low density cholesterol and the use of antihypertensive medications, but not to smoking, systolic or diastolic blood pressure [7]. The fact that no connection was found between HL and smoking was also reported in the population-based Framingham [15] and Baltimore longitudinal studies [6]. There is no clear physiologic parameter to differ between these populations. A possible explanation for this discrepancy may be the different methodologies used in the studies and the fact that they related to different populations (Spanish, Finish and North Americans) at different ages.

A carefully conducted study regarding the relationship between smoking status and age to HL found a correlation between both factors and HL [5]; in addition, they demonstrated a relationship with the number of pack-years. Similarly, Nakanishi et al. [18] showed a dose-dependent relation between HL and number of pack-years of smoking. In our study, the effect of the quantity of cigarettes smoked was found to be associated with HL but it diminished when age was considered, therefore we cannot categorically ascertain a "dose" effect. The damage from cigarettes is accentuated in younger people, thus it may accelerate the effect of aging on HL.

We conclude from our results that the frequency of hearing loss unrelated to noise exposure is higher in smokers than in non-smokers, and that in young adults the effect is greater.

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Correspondence: Dr. Y. Sharabi, National Institutes of Health, CNCS, NINDS, Building 10, Room 6N252, MSC 1620, 10 Center Dr., Bethesda, MD 20892, USA.
Phone: (1-301) 402-2052, Fax: (1-301) 402-0180
email: sharabiy@ninds.nih.gov