

Effects of cigarette smoking on the evolution of hearing loss caused by industrial noise

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ABSTRACT

The few studies evaluating the changes caused by cigarette smoking on hearing loss induced by occupational exposure to noise have reached discordant conclusions. The aim of this study is to investigate the interactions between cigarette smoking and occupational exposure to noise as risk factors in the onset and development of hearing loss. The study was performed on a sample of 557 shipyard workers exposed to noise at an Equivalent Level (Leq) of 93 dBA. On the basis of their smoking habits, they were divided into three groups: group (A), non-smokers; group (B), smokers (15-30 cigarettes per day); and group (C), heavy smokers (over 30 cigarettes per day). The study focussed on the audiometric responses of the subjects at the frequencies of 500, 1000, 2000, 3000 and 4000 Hz. The results were then compared using statistical techniques (Internal correlation coefficient, exponential model, ANCOVA, NPC test). Comparison of the audiometric responses showed statistically significant differences between the three groups. Non-parametric analysis, performed using the NPC test, highlighted that the interaction between smoking and exposure to noise has an influence on hearing loss at all frequencies, and particularly at high frequencies (3000-4000 Hz). The data obtained from the examined sample show that smoking and exposure to noise cause an increase in occupational hearing loss and that this is directly related to the number of cigarettes smoked.

Keywords: Smoking; Hearing Loss; Noise; Audiometric Test; Non Parametric Test

1. INTRODUCTION

Hearing loss as a result of exposure to noise is one of the most frequent pathologies found in workers, and is influenced by a number of parameters, such as intensity of the noise, temporal and spectral patterns, duration of the exposure and susceptibility factors. Controlling these parameters may improve the individual response to noise and evolution of the damage.

One of the factors influencing hearing susceptibility which has attracted growing interest in recent years is cigarette smoking, although the role of smoking in abetting the development of sensory neural hearing loss is controversial, as recent literature shows.

Some authors highlight that smokers have a greater risk of hearing loss than non-smokers [1-3], while others find no connection between the onset of sensory neural hearing loss and cigarette smoking [4,5]. Nomura [6] in a review referring to the period 1966-2003, mentions 9 studies which report a positive association between smoking and hearing loss, and 6 which reach completely opposite conclusions. The author, however, concludes by supporting the thesis of a positive association between smoking and hearing loss.

Few studies evaluate the effects of the association between smoking, occupational exposure to noise and hearing loss. Palmer *et al.* [7] hypothesises that exposure to noise in smokers aggravates the degree of sensory neural hearing loss, probably due to the effect that these factors have on the vascular system. In fact, both exposure to noise and smoking cause vasoconstriction in the cochlear vessels with consequent reduced perfusion, increased plasma viscosity and/or possible creation of carboxyhaemoglobin, which aggravate any local hearing loss. Mizoue *et al.* [8] observes that the hearing loss affects only the cells responsible for the hearing response to high frequencies, since their position at the end of the nutritive arteries makes them more vulnerable to ischaemic damage.

Dengerink *et al.* [9], in an experimental study on temporary threshold shifts (TTS) in man, highlights how cigarette smoking modifies the response of hearing to noise exposure, and attributes this effect to carbon monoxide (CO) and nicotine. Ferrite *et al.* (2005) confirm a synergic effect between cigarette smoking, exposure to noise and age on hearing loss caused by industrial noise. Nomura [10], in a cross-sectional study, carried out on 397 Japanese workers exposed to noise, finds a positive association between smoking, exposure to noise and hearing loss, even if this is often masked by atherosclerotic factors.

Jaruchinda [11] asserts that cigarette smoking influences noise injury in helicopter pilots and aircraft mechanics. Uclide asserts that the combined effects of noise and smoking is not interactive but additive. Recently, Pouryaghoub [12] concludes that smoking can accelerate noise induced hearing loss, but more research is needed to understand the underlying mechanism

Other authors reach opposite conclusions. Pyykko *et al.* [13] observe that smoking does not affect the incidence of noise-induced hearing loss in 199 forestry workers. Nakashima *et al.* [14], in a case-control study conducted on 109 subjects, reports that hearing loss is not affected by smoking.

Since the interactions between industrial noise, cigarette smoking and hearing loss are uncertain, this study aims to clarify the relationship between such variables by trying to evaluate the effects of external factors on hearing loss. This information is particularly useful when drawing up prevention programs aimed at protecting workers' hearing. As far as regards smoking, and in the absence of specific studies, the possible effect of the number of cigarettes smoked per day is also taken into consideration.

2. METHODS

The study was carried out on male subjects, who had only ever had one job, as shipwrights at a large shipyard for the construction of high-speed boats in southern Italy. The enrolled sample was composed of the entire workforce of 900 men.

Noise measurement, performed in November 2003 according to European directives 89/391/EEC and 86/188/EEC, showed that all the subjects were exposed to a daily personal exposure level (LEP,d) of 93 ± 2 dBA of equivalent level. The exposure value reported did not display significant variations compared to the controls previously performed by the company every three years from 1992 onwards, as provided for by Italian legislation regarding occupational exposure to noise. This lays down

that the noise risk in working environments and the LEP, d for each individual worker must be assessed.

The subjects enrolled for the study were selected by applying the following exclusion criteria:

- 1) previous exposure to neurotoxic drugs
- 2) frequent use of ototoxic drugs
- 3) metabolism diseases
- 4) haematological or neurological diseases
- 5) acute and chronic ear, nose and throat conditions
- 6) residence since birth in a Council district other than that in which the subject works
- 7) alcoholism
- 8) former smokers, those who had been smoking for under 10 years and subjects whose smoking habits had significantly changed over the years
- 9) hobbies such as hunting, underwater fishing, frequent visits to discos (> once per week)
- 10) work experience with other companies
- 11) length of service < 10 years.

All the subjects normally used individual hearing protection devices supplied by the company (earphones and earplugs).

The sample thus selected was composed of 557 subjects, who were divided into three groups on the basis of their smoking habits. The first was composed of non-smokers since birth (Group A), the second (Group B) of smokers (15-30 cigarettes per day for at least 10 years), and the third (Group C) of heavy smokers (over 30 cigarettes per day for at least 10 years). People smoke less than 14 cigarettes a day have not been taken in consideration because the data is not significant.

The entire sample was given a general medical examination, routine blood tests, otoscopic examination, and a tonal audiometric test after at least 16 hours without exposure to occupational noise.

For the purposes of tonal audiometric assessment, the study took into consideration hearing threshold values at the frequencies of 500, 1000, 2000, 3000 and 4000 Hz.

The study was based on the values recorded by the tonal audiometric traces.

Table 1 shows the sample size, average age and years of service of the subjects, by group:

Table 1. Sample size of the groups and mean values for age and length of service.

Group	Sample size	Age $\pm \sigma$	Length of service $\pm \sigma$
A	215	41.45 \pm 7.81	21.36 \pm 7.57
B	194	40.77 \pm 8.83	20.15 \pm 8.34
C	148	40.12 \pm 8.77	20.25 \pm 8.35
ABC	557	40.98 \pm 8.52	20.77 \pm 8.15

The aim of the analysis was to assess any significant differences, if they exist, between the audiometric responses of the three groups determined not only by their being smokers or not, but also by the quantity of cigarettes smoked daily.

Statistical analysis – To evaluate whether the audiometric responses of the two ears are interdependent, we used an internal correlation coefficient. An exponential model was also used to describe the trend of audiometric responses in function of the risk index. Moreover, to verify whether the trend of the responses may be expressed with a single function, the ANCOVA (Analysis of Covariance) model was adopted. Lastly, we used an NPC (Non-Parametric Combination) test to compare all the audiometric responses of the three groups for the various risk indexes.

Considering that biological age and the duration of exposure influence audiometric response, and consequently may determine modifications in the individual responses at the various frequencies, we used a risk index [14] which made it possible to evaluate the combined action of the aforementioned variables.

The relation which makes it possible to determine the risk index (I_r) is:

$$I_r = 1 - \left(\frac{E_a - A_l}{E_a} \right)$$

where:

A_l represents length of service;

E_a biological age.

each group, the following type of model was used:

$$R_a = a \cdot b^x$$

where $x = (\text{Age of starting work})^{(\text{Risk index})}$.

3. RESULTS

The **Table 2** shows the audiometric responses at different frequencies in the three group. Since we measured the audiometric responses of both ears for each subject, we decided to assess whether there was any close interdependence between them. In order to achieve this we established the internal correlation co-efficients between the distributions of the responses of the two ears, for each group and frequency (**Table 3**).

The internal correlation coefficients all indicate interdependence between the responses of the left and right ear for all frequencies and all groups.

Consequently, analysis may be performed on the responses of either ear, chosen at random, in each subject. The risk index showed the following equation to the describe.

Table 2. Audiometric responses at different frequencies of the three tested groups as mean and DS.

Frequencies (Hz)	Groups		
	A – no smokers	B - smokers	C – heavy smokers
500	10,09 ± 3,094	11,06 ± 2,05	15,78 ± 3,44
1000	11,84 ± 4,015	13,76 ± 3,74	18,58 ± 5,05
2000	21,77 ± 8,039	26,91 ± 5,31	29,32 ± 3,76
3000	33,3 ± 10,11	42,29 ± 8,11	46,32 ± 9,26
4000	36,93 ± 10,35	53,76 ± 8,22	60,47 ± 5,48

Table 3. Values of internal correlation coefficients for each group and five frequencies.

Frequencies (Hz)	Group A	Group B	Group C
500	5,1875	5,340277778	5,779861
1000	5,93125	6,185416667	5,577778
2000	6,185416667	5,691666667	5,857639
3000	6,593055556	6,286805556	6,432639
4000	6,670138889	6,479861111	5,8625

The response trend for each audiometric frequency of the three tested groups were:

$$\begin{aligned}
 &500\text{Hz} \begin{cases} Gr(A) \Rightarrow R_a = 3.0925 \times 1.2661^x & R^2 = 0.8089 \\ Gr(B) \Rightarrow R_a = 3.8716 \times 1.2382^x & R^2 = 0.8419 \\ Gr(C) \Rightarrow R_a = 8.5578 \times 1.1468^x & R^2 = 0.8277 \end{cases} \\
 &1000\text{Hz} \begin{cases} Gr(A) \Rightarrow R_a = 3.0933 \times 1.3063^x & R^2 = 0.8033 \\ Gr(B) \Rightarrow R_a = 6.2183 \times 1.1884^x & R^2 = 0.8255 \\ Gr(C) \Rightarrow R_a = 7.7757 \times 1.2143^x & R^2 = 0.8354 \end{cases} \\
 &2000\text{Hz} \begin{cases} Gr(A) \Rightarrow R_a = 4.8775 \times 1.3459^x & R^2 = 0.8572 \\ Gr(B) \Rightarrow R_a = 14.4558 \times 1.1464^x & R^2 = 0.8037 \\ Gr(C) \Rightarrow R_a = 20.1123 \times 1.0901^x & R^2 = 0.8614 \end{cases} \\
 &3000\text{Hz} \begin{cases} Gr(A) \Rightarrow R_a = 11.0088 \times 1.2481^x & R^2 = 0.8146 \\ Gr(B) \Rightarrow R_a = 22.7629 \times 1.1465^x & R^2 = 0.9180 \\ Gr(C) \Rightarrow R_a = 24.2708 \times 1.1572^x & R^2 = 0.8840 \end{cases} \\
 &4000\text{Hz} \begin{cases} Gr(A) \Rightarrow R_a = 13.3375 \times 1.2264^x & R^2 = 0.8091 \\ Gr(B) \Rightarrow R_a = 32.7116 \times 1.1167^x & R^2 = 0.9298 \\ Gr(C) \Rightarrow R_a = 46.2579 \times 1.0636^x & R^2 = 0.8496 \end{cases}
 \end{aligned}$$

The **Figures 1-5** below show the theoretical trends determined by means of the aforementioned relation, by group, of the audiometric responses at the various frequencies, hypothesising a subject starting work at twenty years (average age of starting work found in each group):

The model proves that I_r increase corresponds to increased hearing loss at each frequency in all tested groups.

The ANCOVA (Analysis of Covariance), applied to

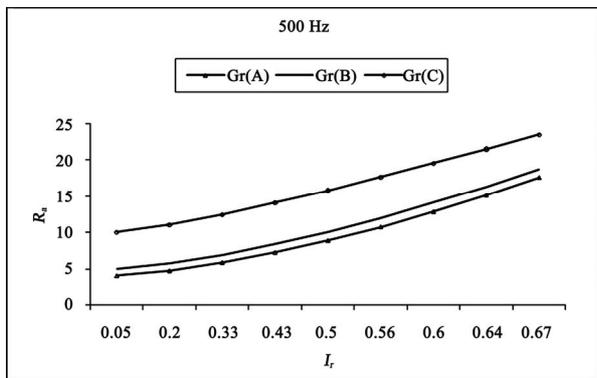


Figure 1. Theoretical trend of audiometric response at the frequency of 500 Hz.

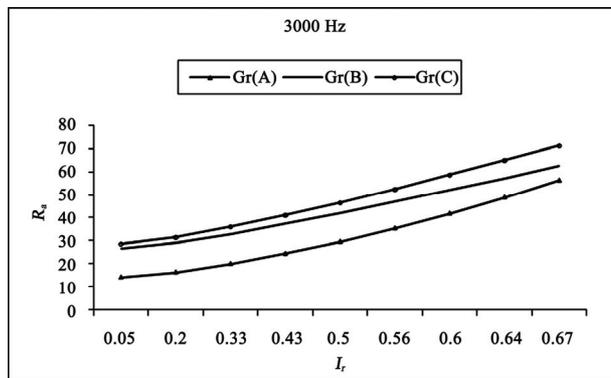


Figure 4. Theoretical trend of audiometric response at the frequency of 3000 Hz.

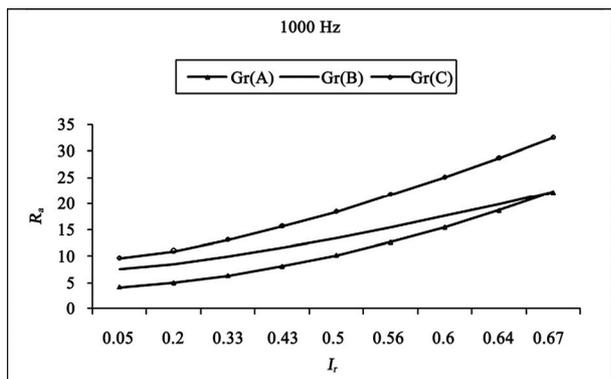


Figure 2. Theoretical trend of audiometric response at the frequency of 1000 Hz.

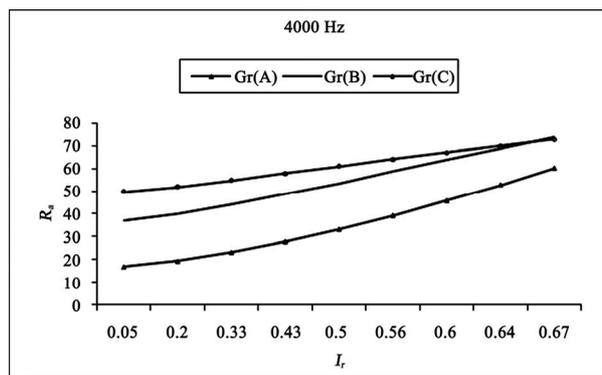


Figure 5. Theoretical trend of audiometric response at the frequency of 4000 Hz.

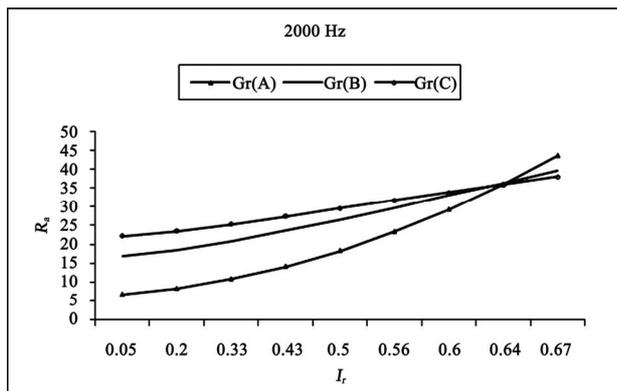


Figure 3. Theoretical trend of audiometric response at the frequency of 2000 Hz.

the audiometric responses of the three groups, demonstrated that the trends of each group, for all the frequencies, may not be described by a single function, as can be seen from the results obtained by means of the parallelism test (Table 4).

The high significance of the p-values indicates that the relation between the variables studied is not sufficiently

Table 4. Results of the parallelism test.

Groups	F	p-value
ABC	877.40.00	0.000
AB	205.74	0.000
AC	12079.35	0.000
BC	16503.14	0.000

represented by one single regression coefficient. Consequently, the three groups had to be considered separately.

The analysis thus far performed allowed us to demonstrate that the three groups, distinguished on the basis of smoking habits, underwent different variations in the temporal evolution of noise-induced hearing loss for all frequencies.

The results obtained led us to analyse the data in greater depth, in order to address a series of questions which naturally sprang to mind:

- 1) does smoking act generically on the audiometric responses, or only at specific frequencies?
- 2) are the audiometric responses equally influenced in the two groups of smokers?
- 3) do age and occupational exposure influence the two

groups compared differently?

For this reason, the I_r for the whole sample was stratified in 10 layers using I_r distribution percentiles.

We stratified I_r relative to all subjects, in 10 layers using I_r distribution percentiles. **Table 5** shows I_r values.

We conducted a comparative analysis for layers and each frequency of R_a , between the three groups. The method used was the Nonparametric test Combination (NPC test), that provides effective solutions for problems of multidimensional hypothesis testing within nonparametric permutations, and is used to test multidimensional hypotheses which are too complex to manage parametrically.

Consequently, we conducted a comparative nonparametric analysis of audiometric responses among the three groups for each layer and all frequencies, using the NPC test. The results obtained, showing that the null hypothesis is not verified for both partials and combined test, prove that relating to cigarettes smoking the three groups are significantly different.

The test results highlighted that the three groups are different for all the frequencies and all the layers ($p = 0.000$).

But are the results attributable to the R_a values for all the frequencies of the three groups, or are just some of these, and only for some frequencies, the cause of the significant differences between non-smokers and more or less heavy smokers?

We thus performed comparisons in pairs of the R_a values between the groups of subjects, by layer and for all the frequencies.

The hypothesis for the comparison between one group (X) and another (Y), homogeneous in terms of age and length of exposure, is as follows:

$$H_{0i(Fr_j)} : \left\{ (R_a Gr(X) \stackrel{d}{=} R_a Gr(Y)) \right\} \text{ with } j = 500Hz, \dots, 4000Hz$$

As far as regards the comparison between non-smokers and smokers, the test showed that the R_a values in the two groups were not differentiated for the frequency of 500 Hz from the fourth layer of the risk index upwards (average length of exposure of 17 years), while for the frequencies 1000 and 2000 Hz from the eighth layer upwards (average length of exposure of 27 years) (**Table 6**).

Table 5. I_r distribution percentiles.

Percentiles	I_r Values	
	Minimum	Maximum
10°	1,519444444	2,231944444
20°	2,240277778	2,634027778
30°	2,670833333	3,002777778
40°	3,009027778	3,279166667
50°	3,289583333	3,472222222
60°	3,545833333	3,684722222
70°	3,69375	3,941666667
80°	3,945833333	4,119444444
90°	4,133333333	4,323611111
100°	4,340277778	4,728472222

Table 6. Results of the comparison between non-smokers and smokers.

Layers	Audiometric Responses					T.C.	
	500 Hz	1000 Hz	2000 Hz	3000 Hz	4000 Hz		
I_{r1}	0.000	0.000	0.000	0.000	0.000	→	0.000
I_{r2}	0.000	0.004	0.000	0.000	0.000	→	0.000
I_{r3}	0.014	0.001	0.000	0.000	0.000	→	0.000
I_{r4}	0,343055556	0.000	0.000	0.000	0.000	→	0.000
I_{r5}	0,3125	0.000	0.000	0.000	0.000	→	0.000
I_{r6}	0,421527778	0.003	0.000	0.000	0.000	→	0.000
I_{r7}	0.069	0.000	0.005	0.000	0.000	→	0.000
I_{r8}	0,236111111	0,119444444	0.098	0.002	0.000	→	0.003
I_{r9}	0,229861111	0.067	0,072917	0.000	0.000	→	0.000
I_{r10}	0,141666667	0,122916667	0,266667	0.020	0.000	→	0.000
	↓	↓	↓	↓	↓		↓
	0.000	0.000	0.000	0.000	0.000		0.000

4. DISCUSSION

The data obtained from the sample under study demonstrated that cigarette smoking interferes with the onset and development of hearing loss caused by industrial noise.

The results of the statistical study applied to the audiometric responses displayed statistically significant differences between non-smokers, smokers and heavy smokers.

This demonstrates that smoking has an influence on noise-induced hearing loss and that this effect is correlated to the quantity of cigarettes smoked daily.

The theoretical model applied for each frequency on the distribution trend of the values of R_a highlighted that, with increased age and length of service, the audiometric responses for all frequencies increase in all three groups, albeit in a differentiated manner.

Furthermore, the ANCOVA (Analysis of Covariance) results show that the trends of the R_a values for the three groups may not be described using a model with the same parameters.

The results of the non-parametric analysis demonstrated that the three groups examined are differentiated, in relation to the audiometric responses for all the frequencies and for each layer.

In particular it was found that the risk factor influences all frequencies, and especially those at the higher end of the spectrum.

These observations are in accordance with the findings of Mizoue *et al.* [8] who, in a study on 4624 steel workers exposed to noise, highlighted an increase in noise-induced damage mainly at high frequencies, in relation to the quantity of cigarettes smoked.

Our result agree with Wild's ones [15] that showed noise-induced hypoacusis increase at 3000-4000 Hz, in a group of long time smokers compared with no smokers with similar occupational history.

Our data show effects at low frequencies, not found by Mizoue *et al.* [8], and this observation is due to the different sensitivity of the statistical techniques used, and in particular in the use of the NPC test.

Regarding the comparison between smokers and heavy smokers, the statistical study, which highlighted significantly different behaviour for all the frequencies, confirmed, as reported by the studies of Nakanishi *et al.* [16] and Mizoue *et al.* [8], that the effects of cigarette smoking have an effect on noise-induced hearing loss which depends on the quantity of cigarettes smoked per day.

The pathogenetic mechanisms involved may be identified as:

1) the ototoxic effect performed by nicotine, which stimulates the nicotine receptors of the acoustic cells as described by Evans [17] and by Blachet *et al.* [18].

2) the mechanisms correlated to ischaemic damage caused by decreased blood flow. In fact, the physiopathology of hearing loss involves a reduction in cochlear blood flow and gaseous exchange, and contemporaneous increased levels of circulating CO caused by chronic exposure to cigarette smoking further reduces the quantity of oxygen to the cochlear cells, thus contributing to sensory neural damage. This hypothesis, suggested by Matschke [19] in acute noise-induced damage, was subsequently confirmed by the studies of Fechter *et al.* [20-22], Rao *et al.* [23] and Shahbaz Hassan *et al.* [24] for chronic noise-induced damage. Rao *et al.* [23], in an experimental study, highlighted that the simultaneous exposure to noise and CO increases noise-induced hearing loss. Fechter *et al.* [22], in an experimental study on laboratory animals, demonstrates that CO increases noise-induced hearing loss. Shahbaz Hassan *et al.* [24] reaches the same conclusions, claiming that CO increases noise-induced hearing loss.

5. CONCLUSIONS

Our data confirm the aforementioned hypotheses also in conditions of chronic exposure.

In conclusion, the observations deriving from the study show that cigarette smoking acts in synergy with exposure to industrial noise on hearing loss, and that this effect is correlated to the amount of cigarettes smoked. Thus, we feel it is necessary to encourage workers occupationally exposed to high levels of noise to limit cigarette smoking.

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