Environmental noise in Beirut, smoking and age are combined risk factors for hearing impairment

E. El Zir,[1] S. Mansour,[2] P. Salameh[3] and R. Chahine[2]

الضجيج البيئي في بيروت، والتدخين، عاملان أساسيان متداخلان يؤديان إلى فقدان أسرع للسمع مع تقدم العمر

إيلي الزير، صلاح منصور، باسكال سلامة، رامز شاهين

الخلاصة: تم بحث تأثير الضجيج البيئي والتدخين على فقدان السمع لدى 440 شخصاً من قاطني بيروت ممن تتراوح أعمارهم بين 21 و50 سنة. وتم تقسيم المشاركين إلى أربع مجموعات: غير المدخنين؛ والمدخنين الذين يعيشون في مناطق صاخبة (70 – 90 ديسي بل)؛ وغير المدخنين والمدخنين الذين يعيشون في مناطق هادئة (45 – 55 ديسي بل). فَوُجِدَ أن التدخين يصاحبه فقدان للسمع عند مستوى 8000 هيرتز وفقاً لتحليل المتغيرات الثنائية التفاوت والمتعددة التفاوت. كما ظهر تفاعل مترابط في الترددات العالية (غالباً عند 8000 هيرتز) بين التدخين، والضوضاء بعد سن الأربعين. أما في المجموعات العمرية من 21 إلى 39، فلم يلاحظ تأثير ضائر يُعتَدّ به إحصائياً للتدخين ولا للضجيج البيئي على السمع في الترددات المنخفضة.

ABSTRACT Effect of smoking and environmental noise on hearing impairment was investigated in 440 people aged 21–50 years living in Beirut. Participants were divided into 4 groups: non-smokers and smokers living in noisy areas (70–90 dBA) and non-smokers and smokers living in quiet areas (45–55 dBA). Smoking was associated with hearing loss at 8000 Hz, in both bivariate and multivariate analysis. An additive interaction at high frequencies (mostly at 8000 Hz) between smoking and noise appeared after age 40 years. At age 21–39 years, neither smoking nor environmental noise had a significant adverse effect on hearing capacity at low frequencies.

Le bruit ambiant à Beyrouth, le tabagisme et l'âge : des facteurs de risque combinés de déficience auditive

RÉSUMÉ Les effets du tabagisme et du bruit ambiant sur la déficience auditive ont fait l'objet d'une étude chez 440 sujets âgés de 21 à 50 ans vivant à Beyrouth. Les participants ont été divisés en quatre groupes : non-fumeurs et fumeurs vivant dans des quartiers bruyants (70-90 dBA) et non-fumeurs et fumeurs vivant dans des quartiers calmes (45-55 dBA). Le tabagisme était associé à une perte auditive à 8000 Hz, dans l'analyse bivariée comme dans l'analyse multivariée. Une interaction additive aux hautes fréquences (généralement à 8000 Hz) entre le tabagisme et le bruit apparaissait après 40 ans. Entre 21 et 39 ans, ni le tabagisme ni le bruit ambiant n'avait d'effet néfaste significatif sur les capacités auditives aux basses fréquences.

[1]Department of Audiology, El Arz Hospital, Zalka and Haykal Hospital, Tripoli, Lebanon.
[2]Faculty of Medical Sciences; [3]Faculty of Pharmacy, Lebanese University, Beirut, Lebanon (Correspondence to R. Chahine: rchahine@lb.refer.org).
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Introduction

Prolonged or high intensity sound can injure the cell structures of the inner ear, and subsequently cause hearing threshold loss (HL). This can be temporary or can lead to permanent hearing impairment. Slight HL is often unnoticeable, but over time the losses add up and progress to hearing impairments that interfere with daily life. Symptoms of HL vary and may include tinnitus (ringing tone sensation) and muffled or distorted sounds [*1*].

While HL among elderly individuals is common, mostly due to presbyacousis as a normal process of ageing, HL among young people is less common and more frequently caused by a combination of genetic and environmental factors [*2,3*]. One risk factor for noise-induced HL could be smoking. Smoking is a widespread addiction among young people and the damage caused by inhaling toxic substances from cigarettes has been widely reported, particularly regarding the connection between smoking and diseases of the cardiovascular system and lungs, and malignancy. The few reports regarding the relationship between smoking and HL remain equivocal. An association between current smoking and HL among older adults has been reported from Japan [*4*]. Data also indicate that current smokers are 1.7 times more likely to have HL than nonsmokers and that nonsmoking participants who lived with a smoker were almost twice as likely to have hearing loss as those who did not [*5*]. This suggests that exposure to environmental tobacco smoke may also be associated with HL. Other available suggest showed that hyperlipidaemia and smoking, but not smoking alone, showed a significant difference regarding HL compared to a control group [*6*].

In a study on the effect of smoking and noise on hearing, smoking in the absence of other risk factors did not increase the risk for sensory neural HL, but smoking in combination with elevated blood pressure and Raynaud's phenomenon put workers at higher risk for HL than any of these factors alone [*7*]. A study investigating hearing problems in a sample of 3000 elderly Mexican Americans concluded that prevention of hearing problems, common in this population, may be done at many levels: control of hypertension, amelioration of arthritis and decreasing consumption of alcohol and cigarettes [*8*]. Although Noorhassim and Rampal [9] reported a multiplicative association between occupational noise, age and smoking, a Japanese team reported that smoking was not associated with low-frequency hearing loss [*10*].

Smoking may be a risk factor for high-frequency HL, and its combined effect on hearing may compound the effects of exposure to occupational noise. Several studies have reported an association [*11–14*] while others have found no such association [*15,16*].

Most of these surveys were performed in specific places, and mainly targeted elderly people subjected to occupational, non-environmental noise. Environmental noise (also known as community noise or residential noise) is defined as noise emitted from all sources except that of the workplace. The main sources of environmental noise are traffic, industry, construction, public works and the neighbourhood. The open air electricity generators in Lebanon as well as the frequent use of car horns by drivers present a significant source of environmental noise pollution across the country, more so in the heavily populated capital, Beirut.

The present study aims to determine the association between smoking and environmental (non-occupational) noise in Beirut on HL in young people.

Methodology

Environmental noise exposure assessment

Outdoor noise was the parameter used to assess community noise at 8 different points in residential areas of Beirut. Noisy areas were selected at 4 major crossroads in Greater Beirut, 2 in the east and 2 in the west. Similarly, 4 quiet areas were selected near non-commercial roads, 2 in the east and 2 in the west. Measurement of community noise was performed with the sound level meter (Radio Shack, model 33-2050). The average energy equivalent sound level for 8 hours in a residential area (LAeq, 8 h) was measured 4 times at 15 minutes intervals between 08.00 and 04.00 on a crossroads in the residential areas under study during 1 week every month of 2004. We used the World Health Organization (WHO) guideline values for evaluating the measured noise levels [*17,18*]. We considered the effect of the combination of noise events which is related to the combined sound energy of those events.

The sum of the total energy over a certain period of time gives a level equivalent to the average sound energy over that period (LAeq, T) [*17,18*]. The A-weighting filter is most commonly used in noise measurements because it weighs lower sound frequencies as less important than mid frequencies, and higher frequencies as more important. As recommended in the WHO guidelines, LAeq, T, used to measure continuing sounds, such as road traffic noise, is a parameter accepted worldwide. Scientists measure the levels of different sounds with a unit called the A-weighted decibel (dBA). The A-weighting reflects how people respond to sound. In a typical community, noise starts to make people highly annoyed when the sound level outside their home is around 55 dBA.

In this study, noisy areas were defined as the places where noise frequency was ≥ 65 dBA, and quiet areas as places where noise frequency was < 65 dBA.

Participants and protocol

The study population comprised 440 volunteers of both sexes. Participants were recruited through announcements in the municipality and in all the major commercial establishments in each area studied.

In order to preclude the possibility of participants having age-related hearing loss (prebyacousis), we selected participants aged 21–50 years. Exclusion criteria for all groups were: frequent use of mobile phone; hunters, soldiers and ex-soldiers (to rule out acoustic trauma as a cause of hearing loss); people with a history of ototoxicity, tympanic perforation, HL transmission, congenital hearing loss, hereditary hearing loss and Meniere disease. For the non-smokers group we also excluded those who had smoked previously but had ceased.

The study design required 100 participants in each group. We initially recruited > 550 individuals. A questionnaire was filled in before examination and volunteers were excluded either because they did not meet the inclusion criteria or because there were already enough participants in the particular category. In the noisy area, however, we did not recruit enough persons initially, so a second recruitment was carried out and the number of eligible volunteers amounted to 140 persons in total in that group. These were retained in the study. So, overall, we had 240 current smokers and 200 who had never smoked.

The study sample was divided into 4 groups:

- non-smokers living in quiet areas of Beirut (n = 100);

- smokers living in the same quiet areas (n = 100);
- non smokers living in noisy areas of Beirut (n = 100);
- smokers living in the same noisy areas (n = 140).

Participants were divided into 2 age categories, 21–39 years and 40–50 years. The age categories were initially defined in 10-year intervals, but the first 2 categories (21–29 years and 30–39 years) were combined because the corresponding prevalence of HL as defined in the study was ≤ 3%.

Participants completed a questionnaire covering personal data, home and work address, smoking status, number of cigarettes smoked per day, duration of smoking and all activities or diseases related to hearing. Participants from noisy areas had been living there for ≥ 20 years, and lived or worked on a main street. Those from quiet areas had been living and working in the same place for > 19 years, and had never been in a noisy environment for > 1 hour/day. Non-smokers had never smoked; smokers consumed 20–40 cigarettes or 1 or 2 narghuile (water pipes) per day for ≥ 5 years.

Participants were examined every week for a period of 1 year, between 14.00 and 19.00 every Friday for logistic reasons. Examination included otoscopy, screening pure-tone air-conduction (air conduction hearing threshold and speech reception threshold), and bone-conduction audiometry between 500 Hz and 8000 Hz. Hearing loss was defined as a pure-tone average hearing level in the worse ear of > 25 dB for 500 Hz, 1000 Hz and 2000 Hz, and > 40 dB for 4000 Hz and 8000 Hz [*19,20*]. Hearing tests were performed in a sealed, soundproof room with a calibrated clinical audiometer. The audiologist who conducted the tests was unaware of the smoking and noise status of the person being tested.

Hearing impairment was defined as being in the top third of the hearing loss distribution (at 2 kHz, 4 kHz, and 8 kHz) for their age category, and controls as those in the lowest third of the distribution. Whenever the air conduction threshold

Table 1 **Audition deficiency and exposure to noise, smoking and age: bivariate analysis**

Characteristic	Audition deficiency					
	2000 Hz		4000 Hz		8000 Hz	
	No.	%	No.	%	No.	%
Noise						
Yes (n = 240)	22	9.2	38	15.8	54	22.5
No (n = 200)	13	6.5	23	11.5	32	16.0
PR (95% CI) *P*-value	1.45 (0.71–2.96) 0.30		1.45 (0.83–2.52) 0.19		1.52 (0.94–2.47) 0.09	
Smoking						
Yes (n = 240)	18	7.5	37	15.4	56	23.3
No (n = 200)	17	8.5	24	12.0	30	15.0
PR (95% CI) *P*-value	0.87 (0.44–1.74) 0.70		1.34 (0.77–2.32) 0.43		1.73 (1.06– 2.82) 0.03	
Age (years)						
40–50 (n = 220)	20	9.1	36	16.4	48	21.8
21–39 (n = 220)	15	6.8	25	11.4	38	17.3
PR (95% CI) *P*-value	1.37 (0.68–2.74) 0.38		1.53 (0.88–2.64) 0.13		1.34 (0.83–2.15) 0.23	

PR = prevalence ratio; CI = confidence interval.

in one of these frequencies was > 25 dB hearing level, bone conduction threshold and speech discrimination were also examined. Participants found to have impaired hearing were referred for further evaluation and treatment.

Prevalence ratios (PRs) of hearing loss with 95% confidence interval (CI) were calculated for every factor. A subgroup analysis was then performed using the younger non-smokers not exposed to environmental noise as the reference group; double and triple exposures were evaluated. Finally, a multivariate analysis was performed: logistic regression with hearing loss at every frequency taken as a dependent variable, and age, smoking and noise exposure as independent variables.

Results

Noise measurements

Average noise level, LAeq, 8 h, on crossroads in the noisy residential area was 70–90 dBA, relatively sustained during the day and above the WHO threshold (65 dBA). On crossroads in the quiet residential area, LAeq, 8 h was 45–55 dBA, which was also relatively constant during the day and below the WHO threshold. The statistically significant difference ($P < 0.05$) between noise levels in the 2 areas was important for the assessment of exposure.

Acoustic analysis

When each factor (environmental noise, smoking or age) was analysed alone, no significant association with hearing loss was noted in the study sample at 2000 Hz, 4000 Hz or 8000 Hz, except for smoking, which was associated with hearing loss at 8000 Hz. A PR of 1.73 was found (95% CI: 1.06–2.82; $P = 0.03$) (Table 1).

Subgroup analysis of the 3 factors is shown in Table 2. The lowest estimate of people with HL was found among the reference group: nonsmokers, not exposed to noise, aged 21–39 years (6.0%) and the highest prevalence for smokers, exposed to noise, aged 40–50 years (31.4%). When analysed alone or in combination, smoking, noise and age were not positively associated with hearing loss, except for the combination of smoking, noise and age at 8000 Hz, with a PR of 3.36 (95% CI: 1.25–9.06; $P = 0.01$).

Multivariate analysis is shown in Table 3. The association of smoking with hearing loss at 8000 Hz was maintained (adjusted PR = 1.67; $P = 0.04$), even after adjustment for age and noise exposure.

Discussion

It is well established that long term exposure to noise at work causes HL. Although countermeasures have successfully reduced noise levels in many industries, noise is still a common occupational hazard, and noise-induced HL is one of the major occupational diseases worldwide. Nevertheless, long term exposure to a noisy environment, even if it is not apparently as harmful as occupational noise, should also be taken into consideration [*21,22*]. It is a recognized that if we listen to a sound at 85 db for 8 hours, 88 db for 4 hours or 91 db for 1 hour, we are at risk for hearing loss. Normal conversation is 58 db, busy traffic is 70 db and standing next to running truck engine is 84 db [*18*].

Two aspects of this study may be considered innovative: most of the available studies were performed to test the combination between occupational noise and smoking rather than environmental noise and smoking and despite the small number of the sample, the participants were chosen with precise inclusion/exclusion criteria, and several confounding factors were excluded.

Table 2 **Concomitant exposures to noise, smoking, age and audition deficiency: subgroup analysis**

Subgroup characteristic	Audition deficiency 2000 Hz No.	%	4000 Hz No.	%	8000 Hz No.	%
Noise only						
Yes (n = 50)	4	8.0	5	10.0	7	14.0
No (n = 50)	3	6.0	4	8.0	6	12.0
PR (95% CI) *P*-value	1.33 (0.31–5.65) 1.00		1.25 (0.36–4.39) 1.00		1.17 (0.42–3.23) 0.77	
Smoking only						
Yes (n = 50)	3	6.0	5	10.0	9	18.0
No (n = 50)	3	6.0	4	8.0	6	12.0
PR (95% CI) *P*-value	1.00 (0.21–4.72) 1.00		1.25 (0.36–4.39) 1.00		1.50 (0.58–3.90) 0.40	
Age only (years)						
40–50 (n = 50)	4	8.0	7	14.0	8	16.0
21–39 (n = 50)	3	6.0	4	8.0	6	12.0
PR (95% CI) *P*-value	1.33 (0.31–5.65) 1.00		1.75 (0.55–5.62) 0.34		1.33 (0.50–3.56) 0.56	
Noise + smoking						
Yes (n = 70)	5	7.1	11	15.7	16	22.9
No (n = 50)	3	6.0	4	8.0	6	12.0
PR (95% CI) *P*-value	1.21 (0.27–5.29) 1.00		2.14 (0.64–7.17) 0.21		2.17 (0.78–6.02) 0.13	
Noise + age						
Yes (n = 50)	6	12.0	8	16.0	9	18.0
No (n = 50)	3	6.0	4	8.0	6	12.0
PR (95% CI) *P*-value	2.00 (0.53–7.58) 0.49		2.00 (0.64–6.21) 0.22		1.50 (0.58–3.91) 0.40	
Smoking + age						
Yes (n = 50)	3	6.0	7	14.0	9	18.0
No (n = 50)	3	6.0	4	8.0	6	12.0
PR (95%CI) *P*-value	1.00 (0.21–4.72) 1.00		1.75 (0.55–5.62) 0.34		1.50 (0.58–3.91) 0.40	
Smoking + noise + age						
Yes (n = 70)	7	10.0	14	20.0	22	31.4
No (n = 50)	3	6.0	4	8.0	6	12.0
PR (95% CI) *P*-value	1.74 (0.43–7.09) 0.43		2.88 (0.89–9.33) 0.07		3.36 (1.25–9.06) 0.01	

[a]*Compared to controls.*
PR = prevalence ratio; CI = confidence interval.

In our experimental conditions, there was no correlation between smoking and environmental noise on HL at low frequencies. However, for individuals aged 40–50 years exposed to environmental noise and smoking, a positive correlation was noted at high frequencies. The most significant factor seemed to be smoking, with positive associations on both bivariate and multivariate analyses. The failure to find this association in subgroup analysis was probably on account of the small number of individuals in the subgroups. Nevertheless, the finding of smoking being associated with hearing loss is maintained.

Cigarette smoking, a known cardiovascular disease risk factor, may affect hearing through its effects on antioxidative

Table 3 **Exposure to noise, smoking, age and audition deficiency: multivariate analysis**

Variable	PR_a	95.0% CI	*P*-value
Audition deficiency at 2000 Hertz			
Smoking	0.85	0.42–1.69	0.64
Noise	1.47	0.72–3.01	0.29
Higher age	1.37	0.68–2.75	0.38
Audition deficiency at 4000 Hertz			
Smoking	1.30	0.75–2.27	0.35
Noise	1.42	0.81–2.49	0.22
Higher age	1.53	0.88–2.65	0.13
Audition deficiency at 8000 Hertz			
Smoking	1.67	1.03–2.75	0.04
Noise	1.47	0.90–2.39	0.12
Higher age	1.34	0.83–2.17	0.23

PR_a = adjusted prevalence ratio; CI = confidence interval.

mechanisms or on the vasculature supplying the auditory system [*23,24*]. The development of hearing loss may even be accelerated if the 2 factors had a synergistic effect. A possible biological support for underlying pathogenic mechanisms may be vascular changes and consequent cochlear hypoxia related to smoking and also to long-term intense noise exposure. In fact, carbon monoxide present in the mainstream smoke reduces cochlear blood oxygen levels as a result of capillary vasoconstriction. Noise exposure also induces hypoxia in the cochlea, causing direct lesions or interacting with mechanical, noise-induced impairments [*25*]. Chronic hypoxia may result in cochlear lesions, particularly in the basal, high-frequency region, the most vulnerable part of the cochlea. Age-related degenerative changes may also affect neural fibres and those parts of the cochlea, including vascular structures, which most pronouncedly affect the high frequencies [*26*].

Cigarette burning releases organic solvents such as toluene, styrene, xylene, and also lead and mercury. These substances have been described as independent factors interacting with noise exposure in regard to hearing loss [*27*]. Synergisms have been identified for the combined effects of noise and organic solvents [*28*]. Smoking may also strengthen these ototoxic effects by increasing their access to cochlear areas, where carbon monoxide is present in high concentrations, which leads to an elevated blood flow and vascular permeability as a response [*29*].

Our results are comparable with those of Ferrite and Santana on adult workers [*30*]. They found that age and occupational noise exposure were, separately, positively associated with hearing loss and that the effects of smoking, noise exposure and age on HL are synergistic. However, longitudinal studies with improved assessment of smoking and exposure time and also the use of severity levels of auditory damage should be developed to overcome the methodological limitations of our study.

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