Interaction Between Noise and Cigarette Smoking for the Outcome of Hearing Loss Among Women: A Population-Based Study

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Background *We investigated the interaction between exposure to noise and smoking in relation to prevalence of hearing loss among women.*

Methods *A sample of women aged 20–49 years (n = 1,723) from a population-based cross-sectional study carried out in Brazil in 2006 was examined. Hearing loss was assessed using a yes–no validated question. Biological interaction was analyzed using the additive scale and measured with interaction contrast ratio (ICR) and assessment of dose–response relationship.*

Results *The combined effect of exposure to noise and cigarette smoking on hearing loss (adjusted prevalence ratio (PR_{adj}) = 3.94, 95% confidence interval (CI): 2.81, 5.52) was greater than expected based on the additive single effects of smoking (PR_{adj} = 1.39, 95% CI: 1.07, 1.81) and noise (PR_{adj} = 2.66, 95% CI: 1.86, 3.82). ICR estimates were not statistically significant. The prevalence of hearing loss among noise-exposed women increased with duration of smoking (P trend = 0.026), number of cigarettes smoked per day (P trend = 0.034), cumulative tobacco use (P trend = 0.030), and early age at smoking initiation (P trend = 0.047).*

Conclusions *Noise and smoking may have a combined effect on hearing loss but further studies are still needed. A dose–response relation of smoking for the noise effect among women is suggested.* Am. J. Ind. Med. 56:1213–1220, 2013.

KEY WORDS: cross-sectional studies; noise-induced hearing loss; occupational health; smoking; women

INTRODUCTION

Hearing loss is a common condition among the elderly, but its prevalence is increasing in other age groups [Daniel, 2007; Agrawal et al., 2008]. Hearing loss creates a premature preventable deterioration in quality of life and is a great social and economic burden [Nelson et al., 2005]. The major and most common modifiable known cause of hearing loss in adults is exposure to noise, either in workplaces or non-work-related environments [World Health Organization, 1998; Passchier-Vermeer and Passchier, 2000; Nelson et al., 2005; Daniel, 2007]. It has been consistently shown that smoking is also a risk factor for permanent hearing loss [Cruickshanks et al., 1998; Nakanishi et al., 2000; Burr et al., 2005; Nomura et al., 2005a; Fransen et al., 2008; Agrawal et al., 2009]. Occupational

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studies reported poorer hearing thresholds in smokers versus non-smokers among male exposed to occupational noise [Wild et al., 2005; Pouryaghoub et al., 2007], suggesting that tobacco users may be at a greater risk of developing noise-induced hearing loss. The underlying mechanism may be related to an increase in reactive oxygen species levels and a local vascular ischemia [Church and Pryor, 1985; Henderson et al., 2006; Carlsson et al., 2007], which potentiates the effect of noise on cochlear lesions.

Results from studies on the combined effect of noise and smoking on hearing loss are mixed. Evidence of biological interaction has been observed in animal experiments [Ahn et al., 2011]. Two observational studies analyzed this hypothesis using additive models. Mizoue et al. [2003] suggested a greater than additive effect of occupational noise and smoking for hearing loss among male steel-factory workers <50 years of age, but not for the older group. Later, Ferrite and Santana [2005] analyzed a male population of workers from a metal plant and reported similar findings limited to those aged 20–40 years. Both studies did not take into account other important potential confounders in the analysis, and their study populations were restricted to men. Negative results reported from other studies [Palmer et al., 2004; Nomura et al., 2005b; Uchida et al., 2005; Gopinath et al., 2010] may be due to the use of multiplicative models, rather than the application of additive criteria, as has been recommended [Rothman et al., 2008]. None studies have analyzed dose–response of biological interaction. Studies on occupational noise generally exclude women because they usually represent only a small proportion of exposed workers [Nelson et al., 2005]. Few studies to date have examined noise and smoking interaction on hearing loss in populations including women [Palmer et al., 2004; Uchida et al., 2005], and only one reported gender-specific results [Uchida et al., 2005].

This study examined the interaction between noise and cigarette smoking for the outcome of hearing loss in a population-based sample of female adults aged 20–49 years. Worldwide, many individuals are exposed to noise and cigarette smoking, and both exposures are less common in females than males [Nelson et al., 2005; World Health Organization, 2009]. The proportion of females who smoke is approximately 22% in developed countries, higher than estimates from poor regions (9%) [Mackay and Eriksen, 2002]. In the United Kingdom, about 11% of women reported having worked in a noisy job [Palmer et al., 2002], and in the United States the prevalence of noise exposure was 6.7% among women currently employed [Tak et al., 2009]. Compared with men, the duration and intensity of noise exposure are generally lower for women [Nelson et al., 2005]. They also smoke fewer cigarettes per day and begin smoking later in life [Mackay and Amos, 2003], along with particular smoking patterns, such as taking shorter and more puffs per cigarette that affect the composition and concentration of toxins in cigarette smoke [Melikian et al., 2007].

MATERIALS AND METHODS

This cross-sectional study used data from the fourth phase (2006) of a population-based prospective cohort on work conditions and health conducted in the city of Salvador, the capital of Bahia State, Brazil. Salvador is the third largest city in Brazil with 2.7 million inhabitants, and contains higher proportions of African descendants and unregistered workers in the informal economy than other cities in Brazil.

Study Population

The sampling design was one-stage random cluster area sampling, based on sub-areas sampled from the entire city. This sample design was used because no complete database of all household addresses was available, and it allowed easier and safer access for field workers to less affluent areas. Each domicile was visited and basic sociodemographic data for each family member was obtained. All residents in each home aged 10–65 years who declared having paid or unpaid jobs (i.e., at least 8 hr a week spent on household chores) were eligible for further interviews. Trained interviewers using standardized questionnaires during home visits collected sociodemographic, life habits, work conditions, and health status data. A questionnaire on auditory health was included in the 2006 data collection. Based on previous findings showing evidence of the interaction among young and middle age adults [Mizoue et al., 2003; Ferrite and Santana, 2005], and the lack of studies investigating this hypotheses in women, our study population was restricted to females aged 20–49 years. All participants signed an informed consent form. The project was approved by the Internal Review Board of Hospital Prof. Edgard Santos and the Institute of Collective Health, Federal University of Bahia.

Outcome Assessment

Self-reported hearing loss was assessed by the question ''Do you feel you have a hearing loss?'' presenting the options ''no,'' ''yes,'' and ''don't know.'' A positive response was recorded as a case if individuals did not report otologic surgery (past or recommended by a physician), tympanic membrane perforation, congenital hearing loss, or onset of auditory symptoms prior to 16 years of age. The accuracy of self-reported approach as compared with audiometric results was assessed in a substudy conducted with a sample of the parent study population

[Ferrite et al., 2011]. A sensitivity of 78.9% and specificity of 76.1% in detecting mild or greater hearing loss (pure-tone average of audiometric hearing thresholds at 0.5, 1, 2, 3, 4 kHz >25 dB hearing level) in the worse ear was estimated among females.

Exposure Assessment

We classified women as exposed to noise if they reported any lifetime exposure to loud noise, either working in environments where they have to shout to communicate [Neitzel et al., 2009], or engaging in recreational activities involving selected noise sources (firearm without hearing protection, portable music players, amplifiers, firework). Noise exposure was dichotomized as ever exposed to loud noise versus never exposed. Other variables related to noise exposure refer to duration, intensity and lifetime cumulative exposure, each dichotomized with the corresponding median, respectively: duration in years (<4 or ≥4); average number of hours per day (≤8, >8); and cumulative exposure, calculated as the product of the number of hours per day and the duration in years (<24, ≥24).

Smoking was assessed using a question on the number of cigarettes smoked per day over at least 1 month, dichotomized as ever-smoker versus never-smoker. Duration, intensity and lifetime cumulative exposure of smoking were measured as follows: duration in years (<10, ≥10), average number of cigarettes per day (<5, ≥5), and cumulative tobacco use (pack-years) calculated as the product of daily use and duration in years divided by 20 (<3, ≥3), all dichotomized at the median. Other variables related to tobacco use were smoking status (current smoker, former smoker), and age started smoking (≥20, <20 years).

Covariates

Sociodemographic variables included sex, age, skin color (black, non-black), education (elementary or less, high school/college), socioeconomic status (defined in terms of family material possessions and categorized as low or medium/high), and job type (formal, legally registered job contracts; informal, in the informal economy as self-employed, autonomous, or unregistered employees in legal firms; and housework, unpaid, spent at least 8 hr a week on household chores). Known risk factors for hearing loss were exposure to solvents, head injury, high blood pressure, and diabetes.

Statistical Analysis

To evaluate interaction, the prevalence of hearing loss was estimated for: smokers who were unexposed to noise (P_{01}); never-smokers exposed to noise (P_{10}); combined exposure (P_{11}), that is, smokers exposed to noise; and a common referent group composed by non-smokers non-exposed to noise (P_{00}). Unadjusted and adjusted prevalence ratios (PR) and 95% confidence intervals (CI) were estimated using binomial log-linear regression [Spiegelman and Hertzmark, 2005] with the GENMOD procedure in SAS, version 9.1 (SAS Institute, Inc., Cary, NC). Initially, heterogeneity of effect on hearing loss across exposure groups was examined. Then, the expected joint effect for P and PR were calculated as follows: $P_{01} - P_{00} + P_{10} - P_{00} + P_{00}$ and $PR_{01} + PR_{10} - 1$, respectively. The observed joint effect was compared to the expected joint effect under the assumption of pure additivity. Interaction contrast ratio (ICR), also known as the relative excess risk attributable to interaction (RERI), was used to assess the magnitude of the departure from additivity, using the following equation: $PR_{11} - PR_{01} - PR_{10} + 1$ [Hosmer and Lemeshow, 1992; Rothman et al., 2008]. Departure from pure additivity corresponds to ICR > 0 or ICR < 0, as ICR = 0 corresponds to additive effects. Confounders were variables that, separately or in conjunction, changed the main association estimates by 15% or more. Age was included in all models because of its well-established association with hearing loss. We also examined noise effects within subgroups of the smoking level variables and used trend tests across levels of smoking. Trends tests were two-sided and adjusted for the potential confounding factors noted above. All models accounted for the complex sample design.

RESULTS

Of the 1,851 eligible participants, 128 (6.9%) refused to participate, leaving 1,723 women in the study population. The mean age was 33.6 ± 8.6 years. A history of noise exposure was reported by 21.1% of the women (n = 364) and cigarette smoking by 18.6% (n = 320). The most common type of noise exposure was occupational (78.6%) and had lasted for <6 years (68.2%). Most of those who had ever smoked reported smoking <10 cigarettes a day (66.9%), for a period ≤10 years (58.8%), with <10 pack-years of cumulative tobacco use (80.0%). Eighty-two women (4.8%) reported exposure to both factors, corresponding to 22.5% of those exposed to noise and 25.6% of those exposed to smoking.

Women ever exposed to noise were more likely to be black, to have an unregistered job, to be ever smokers, to report exposure to solvents and head injury, and they were less likely to work exclusively at home compared with those who had never been exposed to noise (Table I). Ever smokers were more likely to be older, to have less education, to report exposure to noise, high blood pressure, and diabetes compared with never smokers. The patterns of noise exposure and cigarette smoking did not differ

TABLE I. Sociodemographic Characteristics and Selected Risk Factors for Hearing Loss by Noise Exposure and Cigarette Smoking, Salvador, Brazil, 2006

	Noise exposure				Smoking					
	Never	%[a]	Ever	%[a]	Never	%[a]	Ever	%[a]	Total	%[a]
Variables	**n = 1,359**	**(78.9)**	**n = 364**	**(21.1)**	**n = 1,403**	**(81.4)**	**n = 320**	**(18.6)**	**n = 1,723**	**(100.0)**
Age (in years)										
20–29	541	39.8	136	37.4	611	43.6	66	20.6	677	39.3
30–39	414	30.5	120	33.0	461	32.9	73	22.8	534	31.0
40–49	404	29.7	108	29.7	331	23.6	181	56.6	512	29.7
Mean (SD)	33.6 (8.6)		33.9 (8.4)		32.5 (8.2)		38.7 (8.4)		33.6 (8.6)	
Skin color										
Black/mulatto	770	56.7	241	66.2	809	57.7	202	63.1	1,011	58.7
Non-black	589	43.3	123	33.8	594	42.3	118	36.9	712	41.3
Education										
High school/college	711	52.3	192	52.8	781	55.7	122	38.1	903	52.4
Elementary or less	648	47.7	172	47.3	622	44.3	198	61.9	820	47.6
Socioeconomic status										
Medium/high	599	44.1	171	47.0	640	45.6	130	40.6	770	44.7
Low	760	55.9	193	53.0	763	54.4	190	59.4	953	55.3
Job type										
Formal	399	29.4	122	33.5	439	31.3	82	25.6	521	30.2
Informal	412	30.3	133	36.5	437	31.2	108	33.8	545	31.6
Housework[b]	548	40.3	109	30.0	527	37.6	130	40.6	657	38.1
Noise exposure				n/a	282	20.1	82	25.6	364	21.1
Solvent exposure	15	1.1	21	5.8	27	1.9	9	2.8	36	2.1
Cigarette smoking	238	17.5	82	22.5				n/a	320	18.6
Head injury[c]	24	1.8	14	3.9	31	2.2	7	2.2	38	2.1
High blood pressure[d]	202	14.9	60	16.5	174	12.4	88	27.5	262	15.2
Diabetes[d]	32	2.4	11	3.0	29	2.1	14	4.4	43	2.5

SD, standard deviation; n/a, not applicable.
[a]Percents may not sum to 100 due to rounding.
[b]Non-paid, at least 8 hr a week spent on household chores.
[c]Four women had missing data.
[d]Ever told by a doctor; two women had missing data for high blood pressure.

between groups of single and combined exposure (see Online Supplement, Supplementary Table S1). Participants in each group who have been exposed only to non-occupational noise were similar, 21.3% and 22.0%, respectively for single and combined exposure groups ($P = 0.90$).

The crude prevalence of hearing loss varied from 8.1% in women never exposed to both smoking and noise to 35.3% in women with a history of exposure to these two factors. The adjusted prevalence ratios increase from the single exposure groups, smoking and noise, respectively, to the group of combined exposure, with heterogeneity of effects across groups (Table II). The prevalence of hearing loss was greater in women exposed to smoking only (PR_{adj} 1.39, 95% CI: 1.07, 1.81) and in women exposed to noise only (PR_{adj} 2.66, 95% CI: 1.86, 3.82) in comparison with the doubly unexposed group (reference), whereas in those who smoked and were also ever exposed to noise the prevalence of hearing loss was almost fourfold that of the reference group (PR_{adj} 3.94, 95% CI: 2.81, 5.52). This adjusted joint PR of 3.94 was greater than that expected under the additive model, 3.05. The ICR estimates were not statistically significant.

Positive trends in the prevalence of hearing loss were observed among women ever exposed to noise as a function of duration of smoking (P trend $= 0.026$), average number of cigarettes per day (P trend $= 0.034$), cumulative tobacco use (P trend $= 0.030$), and early age at smoking initiation (P trend $= 0.047$; Table III).

TABLE II. Prevalence, Unadjusted and Adjusted Prevalence Ratios for Joint Effect of Noise Exposure and Cigarette Smoking on Hearing Loss, Salvador, Brazil, 2006

				Unadjusted		Adjusted[a]	
Noise exposure	**Cigarette smoking**	**n = 1,723**	**Prevalence of hearing loss %**	**PR[b]**	**95% CI**	**PR[b]**	**95% CI**
Never	Never	1,121	8.1	1.00	Referent	1.00	Referent
Never	Ever	238	12.6	1.55	1.15, 2.10	1.39	1.07, 1.81
Ever	Never	282	24.1	2.96	2.12, 4.15	2.66	1.86, 3.82
Ever	Ever	82	35.3	4.35	3.13, 6.04	3.94	2.81, 5.52
Expected joint effect			28.6[c]	3.51[d]		3.05[d]	
ICR[e] (95%CI)				0.83	−0.68, 2.34	0.88	−0.47, 2.24

CI, confidence interval; ICR, interaction contrast ratio; PR, prevalence ratio; P, prevalence.
[a]Adjusted for age, job type, solvent exposure and high blood pressure.
[b]All estimates adjusted for sample design.
[c]Expected joint effect for prevalence $= P_{01} - P_{00} + P_{10} - P_{00} + P_{00}$.
[d]Expected joint effect for prevalence ratio $= PR_{01} + PR_{10} - 1$.
[e]Departure from expected joint additive effect, defined as $PR_{11} - PR_{01} - PR_{10} + 1$ (observed–expected joint effect).

TABLE III. Adjusted Prevalence Ratios for Joint Effects of Noise Exposure and Smoking Patterns on Hearing Loss, Salvador, Brazil, 2006 (N = 1,723)

	Noise exposure							
	Never			**Ever**			**Departure from additivity of effects on hearing loss**	
Smoking level	**n = 1,359**	**PR[b,c]**	**95% CI**	**n = 364**	**PR[b,c]**	**95% CI**	**ICR[a]**	**95% CI**
Smoking status								
Never smoker	1,121	1.00	Referent	282	2.66	1.85, 3.82		
Former smoker	135	1.26	0.89, 1.81	39	3.79	2.42, 5.94	0.87	−0.91, 2.65
Current smoker	103	1.56	1.09, 2.24	43	4.07	2.74, 6.04	0.84	−1.05, 2.74
Duration (in years)								
Never smoker	1,121	1.00	Referent	282	2.65	1.85, 3.81		
<10	113	1.10	0.64, 1.89	37	3.49	2.27, 5.35	0.73	−1.07, 2.53
≥10	125	1.67	1.14, 2.47	45	4.45	3.04, 6.52	1.12	−0.78, 3.03
P for trend		0.040			0.026			
Cigarettes per day								
Never smoker	1,121	1.00	Referent	282	2.67	1.86, 3.83		
<5	108	1.28	0.85, 1.94	38	3.20	2.26, 4.55	0.25	−1.58, 2.08
≥5	130	1.48	1.07, 2.05	44	4.54	2.90, 7.11	1.41	−0.40, 3.21
P for trend		0.082			0.034			
Cumulative tobacco use								
Never smoker	1,121	1.00	Referent	282	2.66	1.85, 3.83		
<3 pack-years	135	1.07	0.72, 1.59	40	3.35	2.36, 4.76	0.62	−1.15, 2.39
≥3 pack-years	103	1.83	1.34, 2.50	42	4.54	2.95, 6.97	1.05	−0.84, 2.93
P for trend		0.033			0.030			
Age started								
Never smoker	1,121	1.00	Referent	282	2.66	1.86, 3.81		
≥20 years	158	1.43	0.94, 2.17	45	3.65	2.27, 5.86	0.55	−1.15, 2.26
<20 years	80	1.32	0.78, 2.21	37	4.30	2.86, 6.45	1.33	−0.68, 3.33
P for trend		0.119			0.047			

CI, confidence interval; ICR, interaction contrast ratio; PR, prevalence ratio.
[a]Departure from expected joint additive effect, defined as $PR_{1i} - PR_{0i} - PR_{10} + 1$ for the i^{th} level of smoking exposure (observed–expected joint effect).
[b]Adjusted for age, job type, solvent exposure, and high blood pressure.
[c]All estimates adjusted for sample design.

DISCUSSION

While the observed combined effect of noise and cigarette smoking on hearing loss was above that predicted by a simple additive model, statistical testing did not confirm departure from additivity. A dose–response gradient was observed for smoking and hearing loss among noise-exposed women, positively related to the duration, intensity, cumulative tobacco use, and early age at smoking initiation. Comparison of our results with other studies addressing noise and smoking interaction on hearing loss is difficult because of theoretical and methodological differences. Prior research has applied multiplicative criteria when assessing joint effects, a practice that has been discouraged [Rothman et al., 2008]. Uchida et al. [2005] reported no interaction between noise and smoking for hearing loss in women aged 40–79 years. No other studies have presented results for females separately. Our findings related to the observed combined effects above those predicted by an additive model are in agreement with estimates from studies of individuals under 50 years of age. Specifically, joint effects of noise and smoking on hearing loss are consistent from 40 to 49 [Mizoue et al., 2003] and 20 to 40 years age [Ferrite and Santana, 2005], in male workers. Comparable results could also been calculated using published data of population-based studies that included individuals under 50 years of age [Palmer et al., 2004; Burr et al., 2005; Uchida et al., 2005]. In contrast, negative results were reported from studies using multiplicative models [Nomura et al., 2005b; Gopinath et al., 2010].

The dose–response for smoking found in the present study in women exposed to noise are in agreement with the findings of previous studies conducted with men [Virokannas and Anttonen, 1995; Mizoue et al., 2003; Nomura et al., 2005b; Mohammadi et al., 2010], however, differ from findings of Uchida et al. [2005]. Recent evidence has linked light smoking to negative effects on extra-high-frequency auditory thresholds in adults aged 21–23 years [Ohgami et al., 2011].

The biological evidence for joint effects of smoking and hearing loss involves similarities between the causal pathways, ischemia, and oxidative stress [Church and Pryor, 1985; Henderson et al., 2006; Carlsson et al., 2007], and the sites where cochlear lesions predominate [Carlsson et al., 2007]. Smoking leads to oxidative stress, induces free radicals, and decreases blood antioxidant levels [Yanbaeva et al., 2007]. Both smoking and noise increase the reactive oxygen species levels [Huang et al., 2005; Carlsson et al., 2007]. Coherently, experimental studies have shown that exposure to carbon monoxide may interrupt antioxidant mechanisms or increase the generation of reactive oxygen species, potentiating the effect of noise on cochlear lesions [Morata, 2002; Fechter and Pouyatos, 2005]. Although present in low concentrations, other ototoxic substances in the mainstream smoke (e.g., styrene, toluene) may participate in the etiology of hearing lesions when combined with noise, considering the findings among workers environmentally exposed to ototoxic chemicals and noise [Fechter and Pouyatos, 2005; Sliwinska-Kowalska et al., 2007]. Recently, Ahn et al. [2011] reported that mice exposed to noise only recover hearing prenoise levels after 2 weeks, while those exposed to smoking plus noise showed a significantly higher loss of hearing, and thresholds did not return to the prenoise levels until 4 weeks later. Control mice unexposed to both smoking and noise, and mice exposed to smoking only, showed no change in hearing threshold. Hence, smoking may act increasing susceptibility to noise-induced hearing loss.

As mentioned above, statistically significant departures from pure additivity were not observed in our ICR results. Assessment of interaction in epidemiologic studies is often limited because study power is often insufficient to support analysis across the multiple subgroups needed for assessment of interaction [Greenland, 1983; Marshall, 2007; Rothman et al., 2008]. Therefore, further analyses exploring this hypothesis are needed. This study has other limitations. Notably, the cross-sectional design precludes temporality when examining the effect of noise and smoking on hearing loss. Similar to other population-based studies [Palmer et al., 2002; Agrawal et al., 2008], the assessment of occupational noise exposure was self-reported using a single question. This question has previously found to have acceptable validity for exposures over 85 dB(A) [Neitzel et al., 2009]. Pure-tone audiometry is the gold-standard for definition of hearing loss, but self-reported measures have been used in wide-scale investigations [Nondahl et al., 1998; Sindhusake et al., 2001]. Validity of self-reported hearing loss was examined in a subset of this study population and results demonstrated good validity [Ferrite et al., 2011], although it could result in underestimation of smoking-related high frequency hearing loss. Restriction to women and to those from 20 to 49 years of age limits the generalization of the findings.

Although the study findings were not conclusive for the hypothesis of this biological interaction among women, further studies are needed as this might be relevant for general public health interventions, workplace risk control, and prevention. Their level of exposure to both risk factors is rising. Cigarette smoking rates among women are increasing in developing countries [Mackay and Eriksen, 2002; World Health Organization, 2009]. Female participation in sectors of the workforce that involve exposure to noise is also increasing [International Labour Office, 2009]. Despite the advances in the control of occupational risk factors, noise remains the main cause of permanent hearing loss in non-elderly age groups, and new

technologies such as portable music players have increased exposure to noise at young age. Noise and smoking may jointly affect hearing in a dose–response manner, and these findings are relevant to public health in view of the increasing global prevalence of these exposures.

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