

Impact of Smoking Intensity and Duration on Audiometric Thresholds and Sensorineural Hearing Loss: A Cross-Sectional Study among Adult Iraqi Males

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ABSTRACT

Background: This cross-sectional study aimed to examine the effect of smoking intensity (pack-years) and duration on hearing thresholds and sensorineural hearing loss (SNHL) occurrence among adult Iraqi males.

Methods: 100 males (50 smokers, 50 non-smokers), aged 18-45 years, were recruited at Mustansiriyah University, Baghdad, Iraq. Data collected from September 2024 to January 2025 included demographics, smoking history (pack-years), and nicotine dependence (Fagerström Test). Participants underwent otoscopic and physical examination, tympanometry, and pure-tone audiometry, measuring hearing thresholds at low frequencies (500-2000 Hz) and high frequencies (3000-8000 Hz). Logistic regression was used to assess smoking's association with SNHL.

Results: SNHL was detected in 16% of smokers and none of the non-smokers ($p = 0.003$). Multivariate analysis showed that smoking duration (AOR = 1.21; 95% CI: 1.04-1.45; $p = 0.011$) and pack-years (AOR = 1.38; 95% CI: 1.07-1.88; $p = 0.021$) independently predicted SNHL, after adjusting for age and occupation. Smokers had poorer hearing thresholds, especially at high frequencies (3000-8000 Hz). A smoking duration ≥ 24.5 years predicted SNHL with 87.5% sensitivity and 90.5% specificity, though this cutoff is exploratory due to the small number of SNHL cases ($n=8$). Fagerström Test results showed no significant association between nicotine dependence and SNHL.

Conclusion: Smoking duration and intensity are associated with elevated hearing thresholds and high-frequency SNHL in adult males. The findings highlight smoking's harmful auditory effects, but the small sample size and cross-sectional design limit causality. Generalizability may be limited as only male participants were included. Further longitudinal studies including both genders are needed to confirm these findings.

Keywords: Smoking Intensity, High Frequency SNHL, Smoking Duration, Nicotine Dependence

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INTRODUCTION

Hearing loss is a significant public health concern, currently affecting approximately 466 million individuals all over the world, and its prevalence is anticipated to rise in the coming decades. It is a national health issue that profoundly impacts an individual's physical and mental well-being.^{1,2} Among the various risk factors contributing to hearing impairment, smoking has emerged as a significant concern. The pathophysiology of hearing impairment caused by smoking is complex, involving both direct and indirect effects on the auditory system. Tobacco smoke contains harmful components, particularly nicotine and carbon monoxide, which are hypothesized to have ototoxic effects via vasoconstriction and oxidative stress. Smoking can reduce blood flow to the cochlea by constricting its tiny blood vessels, limiting the supply of oxygen and nutrients to the delicate hair cells, and inducing hypoxia that further impairs cochlear metabolism.³ Free radicals present in tobacco smoke induce oxidative stress, damaging cochlear hair cells and auditory nerve fibers. This imbalance between reactive oxygen species and the body's antioxidant defences can lead to cell dysfunction and death, which is particularly harmful because hair cells cannot regenerate. Smoking also triggers inflammatory responses in the cochlea, contributing to chronic cochlear damage and potentially increasing susceptibility to additional auditory complications.⁴ Nicotine disrupts neurotransmitter systems, including dopamine, acetylcholine, and glutamate, impairing auditory signal processing, auditory brainstem responses, and central auditory functions.⁵ Collectively, these mechanisms can cause irreversible cochlear injury and increase the risk of permanent SNHL.

Risk factors for hearing impairment from smoking include lifestyle habits, genetics, environmental exposures, and health conditions. Smoking duration and intensity are key contributors, as long-term and heavy smokers face greater risk from prolonged tobacco toxin exposure. Second-hand smoke also poses serious risks, especially for non-smokers in smoking environments.⁶ According to the Centers for Disease Control and Prevention (CDC), a current smoker is someone who has smoked within the past 30 days, while a former smoker has smoked at least 100 cigarettes in their lifetime but is no longer smoker.⁷ The classification of hearing loss is based on the degree of impairment, ranging from normal hearing (≤ 25 dB HL) to profound hearing loss (≥ 91 dB HL).⁸ Several epidemiological studies have explored the association between smoking and hearing impairment. For instance, a study demonstrated a dose-response relationship, where smokers consuming ≥ 10 cigarettes per day showed significantly greater deterioration in hearing thresholds.⁹ Similarly, another study found that higher cigarette consumption correlates with a greater risk of hearing impairment among Japanese adults.¹⁰ The concept of pack-years, which quantifies lifetime tobacco exposure, is essential for under-

standing the cumulative effects of smoking on health.¹¹ Pack-years are calculated as (number of cigarettes smoked per day/20 \times years of smoking). Based on this calculation, smokers can be broadly classified as light, moderate, or heavy smokers, reflecting increasing cumulative tobacco exposure and potential auditory risk.¹²

The Fagerström Test for Nicotine Dependence (FTND) is a widely used, standardized, six-item questionnaire designed to assess the physical and psychological dependence on nicotine, based on factors such as smoking habits and difficulty in quitting. It has been utilized to evaluate nicotine dependence among young adults in Beirut, Lebanon, and its association with hearing loss was investigated, highlighting potential auditory risks linked to nicotine addiction.¹³

This research aims to fill the gap in the literature regarding the specific impact of smoking intensity and duration on audiometric thresholds and SNHL in men. This study seeks to provide valuable insights that can inform public health interventions and smoking cessation programs aimed at reducing the burden of hearing loss, with a specific focus on adult male participants due to the lack of gender-specific data in previous research. Understanding these relationships is essential for developing targeted strategies to mitigate the auditory health risks associated with smoking.

METHODOLOGY

Study design and setting: This cross-sectional study was conducted at the Otolaryngology and Audio vestibular Consultation Unit, College of Medicine, Mustansiriyah University, Baghdad, Iraq. Data collection took place from September 10, 2024, to January 31, 2025, with the overall study duration extending until September 10, 2025.

Participants: The study included 100 adult male participants, comprising 50 current smokers and 50 non-smokers. Participants were recruited using a convenience sampling technique. Recruitment was carried out by announcing the study within the consultation unit and selecting accompanying visitors who met the inclusion and exclusion criteria. Before enrolment, the study objectives were explained, emphasizing the importance of hearing assessment and the potential impact of smoking on auditory health. Inclusion criteria were adults aged 18-45 years, current smokers (active) of manufactured cigarettes, and asymptomatic individuals without complaints of hearing loss. Exclusion criteria included individuals with conductive or mixed hearing loss, history of middle ear disease or ear surgery, ototoxic medication use, noise exposure (occupational and non-occupational), passive or ex-smokers, users of electronic cigarettes or hookah devices, those with systemic diseases associated with SNHL such as hypertension, diabetes mellitus, hyperlipidaemia, autoimmune disorders (e.g., lupus, rheumatoid arthritis), thyroid disease, chronic kidney disease, cardiovascular disorders, neuro-

logical disorders affecting the auditory nerve (e.g., multiple sclerosis, migraine), as well as individuals with congenital or familial hearing loss.

Data collection tools: Data collection involved a structured questionnaire to gather demographic information and smoking history. Smoking intensity was evaluated using the Pack-years index, and participants were categorized as light (1-20), moderate (21-40), or heavy (>40) pack-years.¹²

Nicotine dependence was assessed using the Fagerström Test for Nicotine Dependence (FTND). Scores were classified as: very low (0-2), low (3-4), medium (5), high (6-7), and very high (8-10).¹³

Physical examination: A detailed otoscopic examination was performed to assess the external auditory canal and tympanic membrane.¹⁴ Tuning fork tests (512 Hz) were conducted, including the Rinne and Weber tests, to evaluate air and bone conduction, ensuring participants met the inclusion criteria for normal hearing.¹⁵

Audiometric examination: Audiometric evaluation was conducted using standard pure tone audiometry (PTA) with an Amplivox Model 240 diagnostic audiometer, using calibrated DD45 earphones for air conduction and a B-71 bone vibrator for bone conduction in a sound-attenuated booth. Testing covered frequencies from 250-8000 Hz, which includes the full range of speech-relevant sounds and high-frequency tones that are often affected early in SNHL. Results were graphically represented on an audiogram.¹⁶ The modified Hughson-Westlake method was employed to determine the softest sound participants could reliably detect.⁸ Tympanometry was used to exclude any middle ear pathology, utilizing the OTOWAVE Tympanometer (Amplivox Ltd 102, UK).¹⁷ Hearing loss was classified based on frequency range: Low-frequency hearing loss was defined as an average threshold more than 25 dB at 500, 1000, and 2000 Hz, and High-frequency hearing loss as an average threshold more than 25 dB at 3000, 4000, 6000, and 8000 Hz.¹⁸

Quality Assurance: All audiological instruments were calibrated according to ANSI S3.6 2018 standards, background noise levels adhered to ISO standards, and all audiometric tests were conducted by the same examiner to ensure consistency and reliability.

Statistical analysis: All analyses were conducted using IBM SPSS Statistics version 26. Descriptive measures summarized the demographic variables and audiometric findings. Continuous data were expressed as means with standard deviations, while categorical data were reported as frequencies and proportions. Comparisons between smokers and non-smokers were carried out using independent samples t-tests for continuous outcomes and chi-square tests for categorical ones. To evaluate the independent association between smoking and SNHL, a binary logistic regression analysis was employed, where SNHL status (present/absent) was the outcome variable.

Predictors included smoking duration, pack-years, age, and occupational category. Adjusted odds ratios (AOR) with corresponding 95% confidence intervals (CI) were presented. A p-value <0.05 was considered statistically significant.¹⁹

Sample size justification: A priori power analysis was performed using a two-sided chi-square test with an α of 0.05 and power of 80%. To detect a minimum difference of 16% in the prevalence of SNHL between smokers and non-smokers, a total of 94 participants (47 per group) were required. Accordingly, the final sample size of 100 participants (50 smokers and 50 non-smokers) was considered sufficient to achieve adequate statistical power.

Ethical considerations: Approval for this study was granted by the Ethics Committee of the College of Medicine, Mustansiriyah University, Baghdad on October 8, 2024 (Approval No. 7968). All participants provided informed consent, with assurances of confidentiality and voluntary involvement throughout the research process.

RESULTS

This study was conducted to assess the correlation between smoking intensity and duration in smokers, and audiometric thresholds as well as the occurrence of SNHL among 100 male participants (50 current smokers and 50 non-smokers).

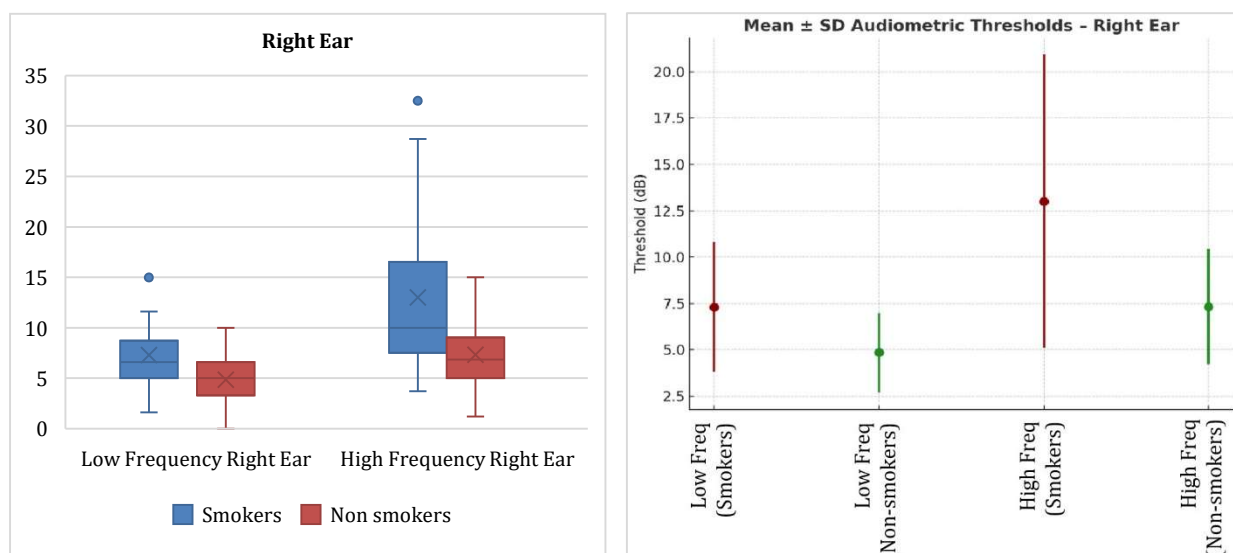
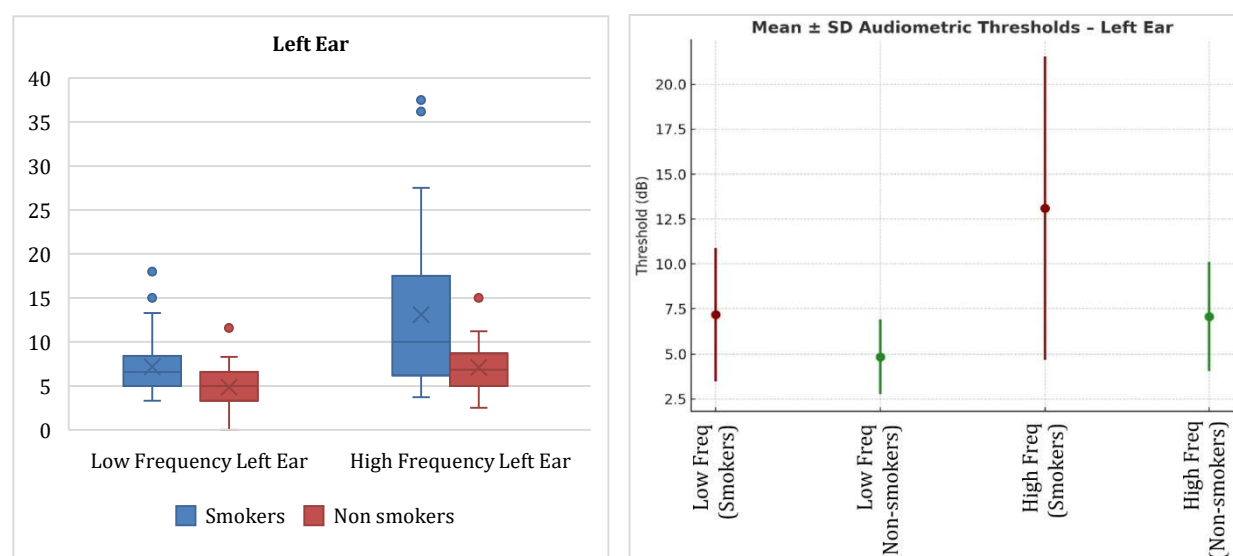
Demographic characteristics: (Table 1) outlined the demographic characteristics of the participants. The mean age was 32.98 ± 8.26 years, with no significant difference between smokers and non-smokers ($p = 0.981$). All participants were male and urban residents. Most participants were employees (52.0%), with no significant association between occupation and smoking status ($p = 0.722$). Educational level also showed no significant difference between smokers and non-smokers ($p = 0.437$).

Audiometric thresholds: Figures 1 and 2 illustrate the comparison of audiometric thresholds at low and high frequencies between smokers and non-smokers. Overall, smokers demonstrated significantly higher thresholds compared to non-smokers, indicating reduced auditory sensitivity. In the right ear, the mean thresholds were 5.60 ± 2.01 dB for smokers versus 3.2 ± 1.8 dB for non-smokers at low frequencies, and 15.64 ± 7.32 dB versus 9.11 ± 4.5 dB at high frequencies. Similarly, in the left ear, smokers showed thresholds of 5.50 ± 2.13 dB compared to 3.5 ± 2.0 dB for non-smokers at low frequencies, and 16.35 ± 8.12 dB versus 9.48 ± 4.7 dB at high frequencies. Error bars representing standard deviations were included in the figures to highlight the variability within each group.

A hearing threshold >25 dB was used to define SNHL. SNHL was detected in 8 participants (8.0%), all of whom were smokers, and was classified as mild, high-frequency hearing loss.

Table 1: Demographic Characteristics of the Study Participants: A comparison Between Smokers and Non-Smokers (n=100)

Variable	Total (n = 100) (%)	Non-Smokers (%)	Smokers (%)	P value
Age (years)				
20-29	36 (36.0)	18 (50.0)	18 (50.0)	-
30-39	38 (38.0)	19 (50.0)	19 (50.0)	
≥ 40	26 (26.0)	13 (50.0)	13 (50.0)	
Mean ± SD	32.98 ± 8.26	33.00 ± 8.26	32.96 ± 8.32	0.981
Gender				
Male	100 (100)	50 (50.0)	50 (50.0)	-
Female	0 (0.0)	0 (0.0)	0 (0.0)	
Occupation				
Employee	52 (52.0)	28 (53.8)	24 (46.2)	0.722
Student	28 (28.0)	13 (46.4)	15 (53.6)	
Worker	20 (20.0)	9 (45.0)	11 (55.0)	
Residency				
Urban	100 (100)	50 (50.0)	50 (50.0)	-
Rural	0 (0.0)	0 (0.0)	0 (0.0)	
Educational level				
Illiterate	15 (15.0)	8 (53.3)	7 (46.7)	0.437
Primary school	35 (35.0)	15 (42.9)	20 (57.1)	
Secondary school	30 (30.0)	14 (46.7)	16 (53.3)	
College or higher	20 (20.0)	13 (65.0)	7 (35.0)	

**Figure 1: Audiometric Thresholds in addition to error bars for the Right Ear Between Smokers and Non-Smokers****Figure 2: Audiometric Thresholds and error bars for the Left Ear Between Smokers and Non-Smokers**

No significant difference was observed between right and left ear high-frequency thresholds among participants with SNHL (smokers) ($p = 0.529$). A significant association was found between smoking status and SNHL ($p = 0.003$) (Table 2).

Table 2 Association Between Tobacco Status and SNHL (n=100)

SNHL	Non-Smokers (n=50) (%)	Smokers (n=50) (%)	P value
No	50 (100.0)	42 (84.0)	0.003
Yes	0 (0.0)	8 (16.0)	

(Table 3) illustrates the association between smoking variables and SNHL. A significant association was found between smoking pack-years and the presence of SNHL ($p = 0.016$), where 35.7% of moderate smokers and 28.6% of heavy smokers had SNHL compared to only 3.4% of light smokers. In contrast, no significant association was observed with the Fagerström score ($p = 0.699$). The mean duration of smoking was significantly longer among participants with SNHL (25.5 ± 5.0 years) than those without (12.7 ± 7.5 years, $p = 0.0001$). Similarly, participants with SNHL were significantly older (43.0 ± 3.5 years) compared to those without (31.1 ± 7.6 years, $p = 0.0001$).

Table 3: Association between Smoking Variables and SNHL Among Smokers (n=50)

Variables	SNHL		P value
	Absent (%)	Present (%)	
Pack\year Classification (mean \pm SD)			
Light smokers: 7.4 ± 2.1 pack-years	28 (96.6)	1 (3.4)	0.016
Moderate smokers: 18.6 ± 3.5 pack-years	9 (64.3)	5 (35.7)	
Heavy smokers: 32.8 ± 4.2 pack-years	5 (71.4)	2 (28.6)	
Fagerström score Classification			
very low	6 (100)	0 (0)	0.699
low	6 (75)	2 (25)	
medium	6 (75)	2 (25)	
high	5 (83.3)	1 (16.7)	
very high	19 (86.4)	3 (13.6)	
Duration of smoking (Mean \pm SD)	12.74 \pm 7.519	25.50 \pm 5.043	0.0001
Age of the participants	31.05 \pm 7.574	43.00 \pm 3.505	0.0001

Table 4: Effect of Smoking Pack-Years on Low- and High-Frequency Hearing Thresholds in Both Ears among Smokers (n = 50)

Hearing threshold (in decibels dB)	Pack\year			P value
	Light (Mean \pm SD)	Moderate (Mean \pm SD)	Heavy (Mean \pm SD)	
Right Ear Low- Frequency Threshold	5.600 \pm 2.0114	9.986 \pm 4.1961	9.014 \pm 3.4585	0.0001
Right Ear High- Frequency Threshold	9.207 \pm 5.3021	17.836 \pm 8.6550	19.086 \pm 7.8259	0.0001
Left Ear Low-Frequency Threshold	5.5045 \pm 2.12786	9.9786 \pm 4.66331	8.5429 \pm 3.51798	0.0001
Left Ear High- frequency Threshold	9.0448 \pm 5.14612	18.1857 \pm 9.40923	19.8071 \pm 9.20536	0.0001

Table 5: Multivariate Logistic Regression Assessing Independent Predictors of SNHL

Variable	AOR	95% CI	P-value
Smoking Duration	1.21	1.04 - 1.45	0.011
Pack-Years	1.38	1.07 - 1.88	0.021
Age	1.02	0.95 - 1.10	0.420
Occupation (Worker)	1.09	0.66 - 2.23	0.520

Correlation Analysis:

Correlation Between Smoking Intensity (Pack-Years) and Hearing Thresholds

Audiometric thresholds (mean \pm SD) increased significantly with higher pack-year exposure ($p = 0.0001$). The most pronounced differences were observed at high frequencies: in the right ear, thresholds were elevated from 9.207 ± 5.30 dB in light smokers to 19.086 ± 7.83 dB in heavy smokers, and in the left ear from 9.045 ± 5.15 dB to 19.807 ± 9.21 dB. At low frequencies, thresholds also increased with greater pack-year exposure, although the differences were less marked compared to high frequencies (Table 4).

To assess the independent association between smoking exposure and SNHL, a binary logistic regression analysis was performed. The results demonstrated that both smoking duration and pack-years were significant predictors of SNHL after adjusting for age and occupation. Specifically, each additional year of smoking duration increased the odds of SNHL by 21% (AOR = 1.21; 95% CI: 1.04-1.45; $p = 0.011$), and each additional pack-year increase the odds by 38% (AOR = 1.38; 95% CI: 1.07-1.88; $p = 0.021$). Age and occupational status did not show statistically significant associations with SNHL in this model. (Table 5).

Correlation Between Smoking Duration and Hearing Thresholds Figures 3 illustrate a statistically significant positive relationship between the duration of smoking and hearing thresholds across all frequency ranges in both ears. In the right ear, longer smoking duration was significantly linked to higher thresholds at low ($R^2 = 0.55$, slope = 0.31, $p < 0.0001$) and high frequencies ($R^2 = 0.53$, slope = 0.33, $p < 0.0001$). Similarly, in the left ear, smoking duration showed a positive correlation with low-frequency ($R^2 = 0.47$, slope

= 0.30, $p < 0.0001$) and high-frequency thresholds ($R^2 = 0.55$, slope = 0.36, $p < 0.0001$). These results indicate that extended periods of smoking are significantly associated with deteriorating auditory function.

A smoking duration ≥ 24.5 years predicted SNHL with 87.5% sensitivity and 90.5% specificity, though this cutoff is exploratory due to the small number of SNHL cases ($n=8$). Figure 4.

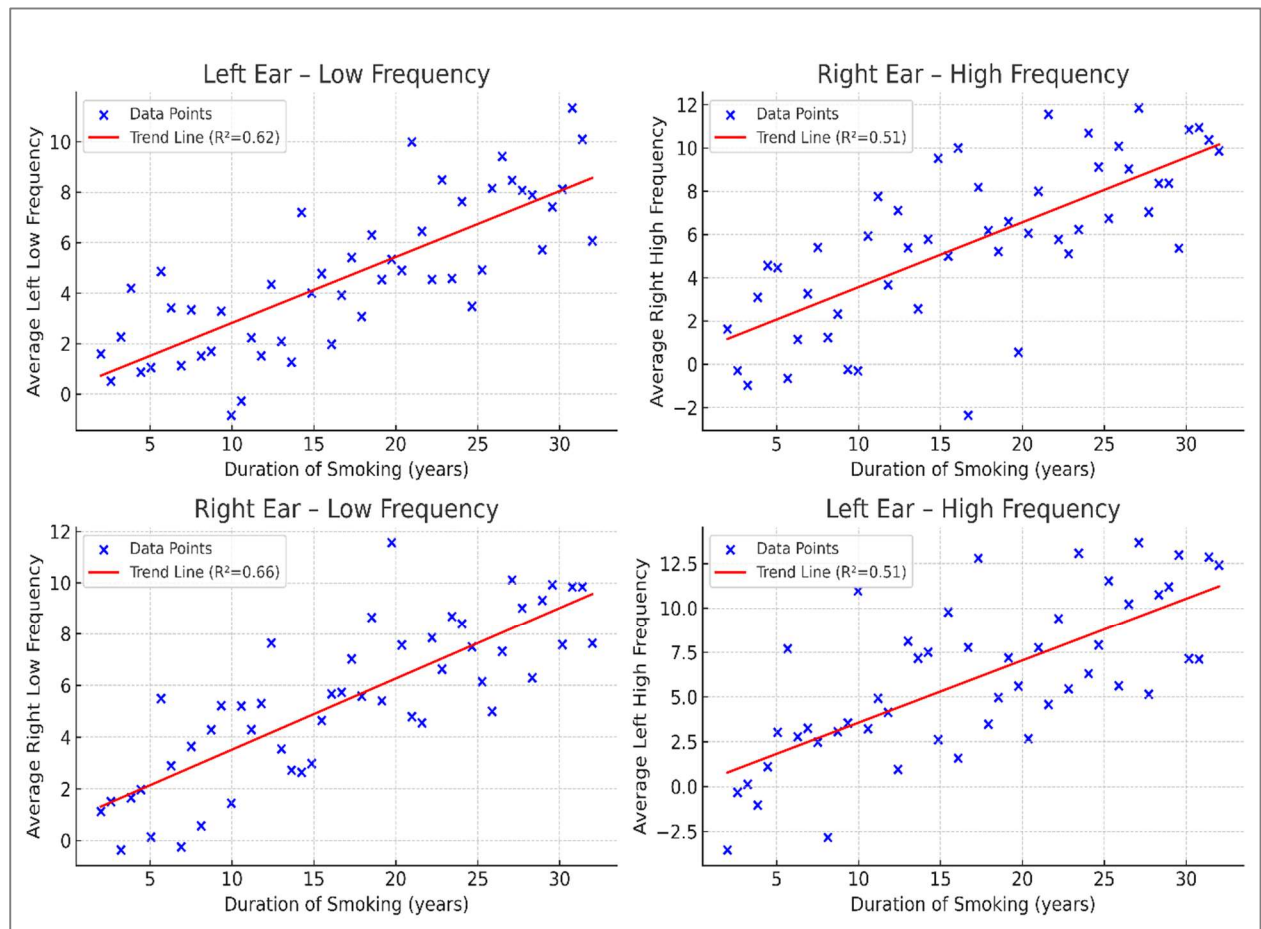


Figure 3: Correlation Between Smoking Duration and Low- and High-Frequency Hearing Thresholds in Both Ears among smokers (no=50)

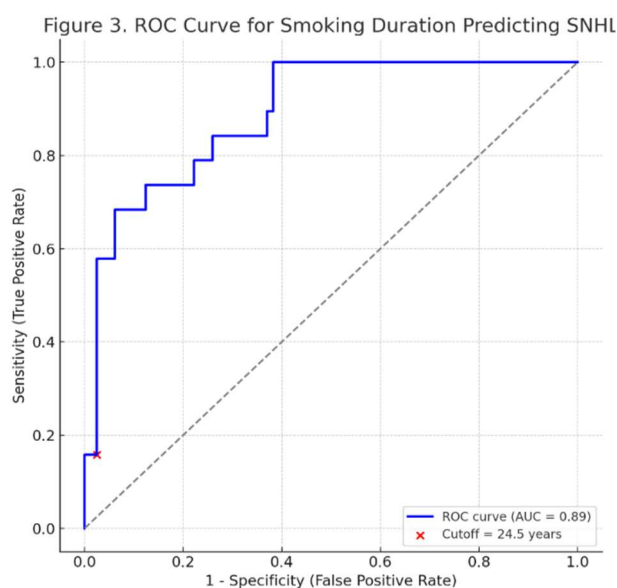


Figure 4: ROC curve analysis to determine the cut-off duration value suspected to developed SNHL among smokers

DISCUSSION

The present study aimed to investigate the correlation between smoking intensity and duration and audiometric thresholds in men, particularly focusing on the occurrence of SNHL among smokers compared to non-smokers. The findings indicate a significant association between both smoking intensities, measured in pack-years, and smoking duration (in years), and elevated audiometric thresholds across all frequency ranges, particularly at high frequencies ($p = 0.0001$). This aligns with previous research that has established a dose- and time-dependent relationship between smoking and hearing impairment, reinforcing the notion that increased smoking intensity and longer duration exacerbate auditory dysfunction.

Demographic Characteristics of the Study Participants: No significant differences were found between smokers and non-smokers regarding age ($p = 0.981$), consistent with previous studies reporting similar age distributions and minimizing age as a confounder in smoking-related SNHL.^{20,21} All participants were male

urban residents, reducing gender and rural environment bias. However, urban noise exposure may act as a subtle contributor to hearing decline, even among non-smokers. Although individuals with known occupational or recreational noise exposure were excluded, chronic exposure to city sounds, such as traffic, construction, and public transport, may still influence hearing thresholds over time, potentially confounding the isolation of smoking's effect. This is supported by a study of 150 males, which emphasized that environmental noise in urban settings contributes to hearing impairment in both smokers and non-smokers.²² Occupational status did not differ significantly between smokers and non-smokers ($p = 0.722$), consistent with previous research.²³ However, smoking in combination with occupational noise may synergistically increase the risk of hearing loss, as reported in a previous study.¹⁸ Educational level showed no significant difference between smokers and non-smokers ($p = 0.437$), although a previous study reported a significant association between education and SNHL ($p = 0.023$), likely reflecting differences in the studied populations.²⁴ The potential confounder was controlled in the current study through exclusion criteria, enhancing internal validity. The equal group sizes and demographic homogeneity further strengthen the internal consistency of the findings.

Impact of Smoking on SNHL and Audiometric Thresholds: This study demonstrates that smoking significantly influences audiometric thresholds and the development of SNHL. A significant association was observed between smoking and SNHL ($P = 0.003$), highlighting the impact of smoking on auditory function. This finding aligns with previous study, demonstrating that smokers tend to have higher hearing thresholds and are more susceptible to auditory damage.¹⁸ Higher smoking intensity, measured by pack-years, was significantly associated with SNHL ($p = 0.016$), consistent with previous study.²⁵ Similarly, longer smoking duration was associated with more severe hearing loss, in line with previous research.²⁶

Age was also a significant factor; participants with SNHL were notably older (43.00 ± 3.505 years) compared to those without SNHL (31.05 ± 7.574 years, $p = 0.0001$), supporting by findings of prior study.²⁷

All SNHL cases in this study were of mild degree (26-40 dB), aligning with prior study.²⁸ In our study, SNHL among smokers was similar in both ears, with no significant difference between the right and left sides high-frequency thresholds ($p = 0.529$). This bilateral symmetry suggests that smoking-related ototoxicity affects both cochleae systemically. Substances such as nicotine and carbon monoxide may induce cochlear damage through vasoconstriction, hypoxia, and oxidative stress. The high-frequency regions at the basal turn of the cochlea are particularly sensitive. This explains why hearing loss often manifests similarly in both ears, with possible involvement of the auditory nerve in advanced stages or after prolonged exposure. These findings emphasize the importance of assessing

both ears for a comprehensive evaluation of SNHL in smokers and support the hypothesis of smoking-induced cochlear dysfunction.³

Furthermore, the finding that all SNHL cases occurred among smokers reinforces the hypothesis that smoking is a strong independent predictor of auditory dysfunction.²⁹ No SNHL was observed among non-smokers in our study. It is important to note that participants with potential confounding factors, including significant exposure to second-hand smoke, were excluded from the analysis. Therefore, the absence of SNHL in this group likely reflects the limited exposure to second-hand smoke. These findings emphasize the importance of minimizing exposure to second-hand smoke to protect auditory function and highlight a potential area for future research in populations with higher exposure.

Correlation Between Smoking Intensity (Pack-Years) and Hearing Thresholds: This study found a significant correlation between smoking intensity (pack-years) and elevated hearing thresholds across all frequencies ($P = 0.0001$), with the most pronounced effects observed at high frequencies among heavy smokers. Specifically, thresholds increased progressively with higher pack-year exposure, confirming a dose-dependent relationship. These findings are consistent with previous research reinforcing the evidence that greater smoking intensity contributes to progressive auditory impairment.²⁵

Nicotine Dependence and Hearing Loss: No significant association was found in the present study between nicotine dependence, measured via the Fagerström Test and SNHL ($p = 0.699$). However, a trend of increased SNHL prevalence was observed among individuals with low to moderate dependence. This paradox may indicate that biological susceptibility, smoking patterns, or limitations of the Fagerström scale could obscure the actual risk. Additional factors, such as the relatively small sample size and reliance on self-reported nicotine dependence, may also contribute to the lack of statistical significance. These results partially align with previous study, which reported a 1.73-fold higher risk of hearing loss among smokers.¹³ Overall, our findings highlight the need for further studies with larger samples and objective measures of nicotine dependence to clarify its potential relationship with SNHL.

Correlation Between Smoking Duration and Hearing Thresholds: A significant correlation was found in the current study between smoking duration and elevated thresholds at both high and low frequencies. These findings were in line with prior research, showing that prolonged smoking duration was associated with increased risk and severity of SNHL, particularly at high frequencies.^{28,30}

The ROC curve analysis, following established methodology for defining optimal cut-points³¹, identified a smoking duration of ≥ 24.5 years as a threshold for predicting SNHL with high sensitivity (87.5%) and specificity (90.5%). This suggests that long-term

smoking may represent a critical risk factor for auditory damage, a finding supported by a meta-analysis, which reported a linear relationship between smoking duration and hearing loss, further emphasizing the cumulative impact of prolonged smoking on auditory function.³²

Independent Association of Smoking with SNHL

Multivariate Findings: The addition of logistic regression analysis in this study allowed for a more robust evaluation of the association between smoking and SNHL, adjusting for key confounders such as age and occupation. Although alcohol and caffeine intake were not assessed in the present study, they are recognized as potential confounders of hearing thresholds based on previous research.^{33,34} While these factors were not measured in our work, future studies should consider including them to more accurately isolate the effects of smoking on hearing loss. Results indicated that both smoking duration and pack-years were significant independent predictors of SNHL. However, due to the limited number of SNHL cases (n = 8), these estimates have low precision, and confirmation in larger cohorts is necessary.

LIMITATIONS

This study examined the association between smoking and SNHL in adult males. However, limitations should be noted. First, the population included only male urban residents from a single center, which limits generalizability to women, rural populations, and other geographic regions. Second, due to the cross-sectional design, causal relationships cannot be established. Third, reliance on the Fagerström Test without biochemical verification may reduce the accuracy of assessing nicotine dependence. Additionally, the relatively small sample size limits the robustness of subgroup and stratified analyses. Finally, this was a single-center study, and larger, multicenter, and gender-inclusive investigations are needed to validate and extend these findings.

CONCLUSION

This study demonstrates a significant association between cigarette smoking and SNHL, particularly at high frequencies. Both smoking duration and intensity (measured in pack-years) were independently associated with elevated audiometric thresholds. Notably, all cases of SNHL were observed among smokers, while no hearing loss was detected in non-smokers. These findings underscore the importance of incorporating hearing assessments into routine health evaluations for smokers and raise awareness about the impact of smoking on auditory health. However, due to the small sample size, single-center design, and reliance on self-reported smoking data, the results should be interpreted with caution. Furthermore, future longitudinal studies are needed to establish causality, as the cross-sectional design represents a key limitation

of the present study. Biochemical validation of smoking exposure (e.g., cotinine measurements) is recommended to enhance the accuracy of self-reported data.

Individual Authors' Contributions: NKR conceived and designed the study, performed data analysis, and wrote the initial manuscript draft. MRD contributed to data collection and interpretation.

Availability of Data: The dataset used in this study is available from the corresponding author upon reasonable request.

Declaration of Non-use of Generative AI Tools: This article was prepared without the use of generative AI tools for content creation, analysis, or data generation. All findings and interpretations are based solely on the authors' independent work and experience.

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