The Journal of International Advanced Otology

J Int Adv Otol 2017; 13(1): 110-2 • DOI: 10.5152/iao.2017.3412

Original Article

Influence of Smoking on Ultra-High-Frequency Auditory Sensitivity

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OBJECTIVE: In this study, an attempt was made to determine the effect of smoking on ultra-high-frequency auditory sensitivity. The study also attempted to determine the relationship between the nature of smoking and ultra-high-frequency otoacoustic emissions (OAEs) and thresholds.

MATERIALS and METHODS: The study sample included 25 smokers and 25 non-smokers. A detailed history regarding their smoking habits was collected. High-frequency audiometric thresholds and amplitudes of high-frequency distortion-product OAEs were analyzed for both ears from all participants.

RESULTS: The results showed that the ultra-high-frequency thresholds were elevated and that there was reduction in the amplitudes of ultra-high-frequency OAEs in smokers. There was an increased risk of auditory damage with chronic smoking.

CONCLUSION: The study results highlight the application of ultra-high-frequency OAEs and ultra-high-frequency audiometry for the early detection of auditory impairment. However, similar studies should be conducted on a larger population for better generalization of the results.

KEYWORDS: Smokers, otoacoustic emissions, auditory threshold

INTRODUCTION

Cigarette smoking has been reported to be one the most common addictions noticed in the general population [1]. In general practice, smoking affects the circulatory and immune systems. There are several studies in the literature suggesting that smoking has adverse effects on the auditory system with elevation of thresholds, abnormal otoacoustic emissions (OAEs), and abnormal auditory evoked potentials [2-4]. Ultra-high-frequency audiometry is extensively used in monitoring the hearing sensitivity as it used to determine early auditory involvement [5]. In addition, the evaluation of OAEs, particularly at high frequencies, is one of the sensitive tests for auditory sensitivity [5, 6].

Previous studies have reported that hearing loss is 1.5-times more likely to be observed in smokers than in non-smokers [1, 4].Tobacco smoke consists of toxic ingredients such as mercury and arsenic, which reportedly damage the hair cells of the cochlea [7]. In addition, tobacco smoke may cause demyelination of nerves of the auditory pathway [7]. Tobacco can also reduce cochlear blood supply and may cause vascular changes affecting the cochlea [8]. Pure tone thresholds are reported to be elevated at normal frequencies [9] and high at frequencies from 9kHz to16 kHz [10, 11]. Distortion-product OAEs are reported to be reduced in amplitude in smokers compared to non-smokers [3, 12]. However, there is a scarcity of literature on differences in high-frequency OAEs between smokers and non-smokers. This is important as high-frequency OAEs are more sensitive that auditory thresholds in the early detection of hearing loss [5, 13]. In addition, there is very limited literature on the correlation between the duration of smoking, number of cigarettes smoked per day, and frequency of smoking on high-frequency OAEs and high-frequency thresholds. Thus, the present study attempted to determine differences across smokers and non-smokers in the amplitudes of ultra-high frequency OAEs and ultra-high frequency auditory thresholds. An attempt was also made to determine if there is any relationship between the nature of smoking and ultra-high frequency OAEs and thresholds.

MATERIALS and METHODS

Participants

Fifty healthy male adults, 25 smokers and 25 non-smokers, between the ages of 18 and 40 years were included. The control group included individuals who never smoked in their life (non-smokers). Individuals with at least a 1 year history of smoking and who continued to smoke were part of the experimental group (smokers). The duration of smoking in the experimental group ranged from 1 year to 15 years. The number of cigarettes smoked per day ranged from 2 to 25, and the frequency of smoking ranged from

Presented in: A part of the manuscript was presented at Indian Speech and Hearing Association Conference held at Kolkata, India from 6th to 8th January 2017.
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Submitted: 19.12.2017 **Revision Received:** 22.02.2017 **Accepted:** 24.03.2017

everyday to once in a week. Ten of 25 individuals in the experimental group reported occasional tinnitus. Participants with no significant otological history, exposure to noise, intake of ototoxic drugs, diabetes, and familial hearing loss were included. The audiometric threshold was less than 25 dB HL from 250 Hz to 6 kHz for all participants. Immittance evaluation showed a normal middle ear function in all participants.

Procedure

The modified Hughson and Westlake procedure was used to determine pure tone thresholds [14]. A two-channel diagnostic audiometer was used to obtain air and bone conduction thresholds and speech identification scores. Phonemically balanced words in Kannada developed for adults by Yathiraj and Vijayalakshmi [15] were used for determining speech identification scores. Recorded word lists were presented through a two-channel diagnostic audiometer at 40 dB SL (re: SRT). An immittance meter, Grason Stadler Inc. Tympstar (Grason-Stadler, Eden Prairie, MN), was used for immittance testing. The better ear of participants was tested to obtain tympanograms, and acoustic reflexes for 500, 1000, 2000, and 4000 Hz pure tones were determined for a probe tone frequency of 226 Hz.

High-frequency thresholds for both ears were determined using a calibrated audiometer with HDA-200 headphones at 8 kHz, 9 kHz, 10 kHz, 12.5 kHz, and 16 kHz. The modified Hughson and Westlake procedure was used again to determine high-frequency thresholds [14]. High-frequency distortion-product OAE measurements were recorded in both ears using the Mimosa Acoustics OAE equipment in a sound-treated room. Appropriate probe fitting was ensured, and the stimulus was calibrated before recording the OAE. High-frequency distortion-product OAEs were recorded at the f2/f1 ratio of 1.22 with the intensity of f1 (L1) at 65 dB SPL and that of f2 (L2) at 55 dB SPL. High-frequency distortion-product OAEs were measured across 8 kHz, 9 kHz, 10.25 kHz, 12.5 kHz, 14 kHz, and 16 kHz.

Ethical Considerations

Approval was taken from the Ethical Approval Committee of the All India Institute of Speech and Hearing, and testing was done using non-invasive procedures. The objectives and procedures of the study were explained to the participants before evaluation, and informed consent was taken from them.

RESULTS

The results were statistically analyzed using IBM Statistical Package for Social Sciences Statistics for Windows, Version 20.0 (IBM Corp.; Armonk, New York). The high-frequency threshold obtained across ultra-high frequencies showed poorer threshold for smokers than non-smokers. The mean and standard deviation (SD) of ultra-high-frequency thresholds in both groups is shown in Figure 1.

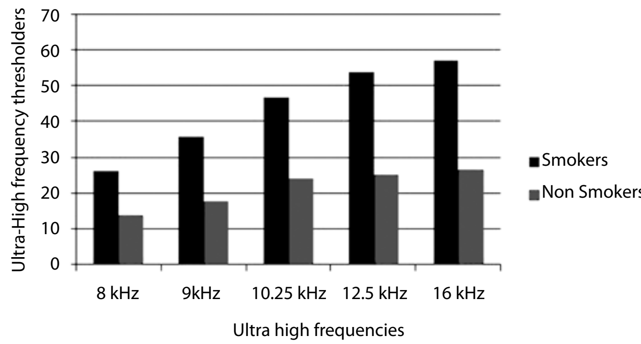

Figure 1. Mean and SD of ultra-high-frequency thresholds in smokers and non-smokers.
SD: standard deviation

The ultra-high frequency distortion-product OAEs also showed a reduction in amplitudes for smokers compared to non-smokers. The mean and SD of distortion-product OAE amplitudes in both groups are shown in Figure 2.

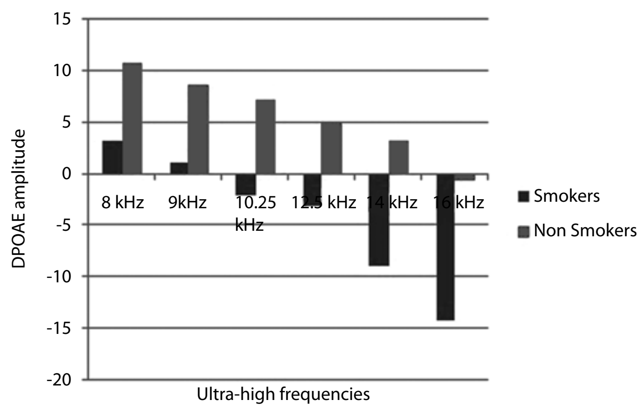

Figure 2. Mean and SD of ultra-high-frequency distortion-product OAE amplitudes in smokers and non-smokers.
SD: standard deviation; OAE: otoacoustic emissions

The results of multiple analysis of variance considering ultra-high frequencies as dependent variables showed that there was a significant elevation in ultra-high frequency thresholds ($p<0.01$) and a significant reduction in ultra-high-frequency OAE amplitudes ($p<0.01$). Further, post hoc analysis of the data using the Sidak test suggested that these differences were significant ($p<0.05$) across all individual frequencies for audiometry and otoacoustic measurements. The amplitudes of ultra-high-frequency OAEs and ultra-high-frequency thresholds were averaged, and the mean differences were analyzed. The result of the independent t-test showed that the average ultra-high-frequency OAE and average ultra-high-frequency threshold in both groups was significantly different ($p<0.01$). In addition, there was a positive correlation between average ultra-high-frequency thresholds and number of years of smoking ($r=0.65$, $p<0.05$), number of cigarettes smoked per day ($r=0.54$, $p<0.05$), and frequency of smoking ($r=0.57$, $p<0.05$). There was a negative correlation between average ultra-high-frequency OAE amplitudes and number of years of smoking ($r=-0.57$, $p<0.05$), number of cigarettes smoked per day ($r=-0.64$, $p<0.05$), and frequency of smoking ($r=-0.61$, $p<0.05$).

DISCUSSION

The present study showed that the ultra-high-frequency thresholds were significantly elevated in smokers. In addition, there was a reduction in ultra-high-frequency OAEs, which was significant. An increase in the number of years of smoking, increase in the frequency of smoking, and increase in the number of individuals who smoke more cigarettes showed a larger elevation of threshold and higher reduction in OAE amplitudes. The free radicals in tobacco such as mercury and arsenic can severely damage hair cells and affect the release of neurotransmitters [7]. The high amounts of carbon monoxide and nicotine restrict blood circulation to the cochlea, which can damage the outer hair cells leading to reduction in OAE amplitudes [9]. Howard et al. [16] reported that smokers have an increased susceptibility to atherosclerotic damage. They also reported increased atherosclerotic damage with an increase in the number of cigarettes smoked and the number of years of smoking. This happens because of oxygen deprivation to outer hair cells of the cochlea and spiral ganglion [16]. Thus, the poorer ultra-high-frequency thresholds and reduced OAE amplitudes can be attributed to a high level of hypoxia at the cochlea along with vascular deficiency.

It has also been reported that ultra-high frequencies are more prone to damage because of the reduced blood supply [3]. Negley et al. [3] reported that carbon monoxide and nicotine in the cigarette reduces oxygen supply to outer hair cells of the cochlea. Previous studies have also shown that high-frequency hearing loss is more common in smokers [7, 17-19]. There is evidence suggesting that the cochlear artery, which supplies the basal region of the cochlea, is susceptible to atherosclerotic changes seen in smokers [19]. This could be one of the important patho-physiological mechanism responsible for ultra-high-frequency damage seen in smokers. Thus, determining ultra-high-frequency OAEs would help in the early detection of auditory dysfunction in smokers. The present study highlights the ill-effects of smoking on the auditory system. However, further studies on a larger group of participants are essential for a better generalization of the results.

CONCLUSION

This study attempted to determine the effect of smoking on ultra-high-frequency auditory sensitivity. It was found that the ultra-high frequency thresholds are elevated and that there was reduction in the amplitudes of ultra-high-frequency OAEs in smokers. The results also showed that chronic smoking increases the risk of auditory damage. Reduced oxygen supply, particularly to the basal end of the cochlea, affects ultra-high-frequency sensitivity. Thus, this study highlights the application of ultra-high-frequency OAEs and ultra-high-frequency audiometry for the early detection of auditory impairment.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of All India Institute of Speech and Hearing, India.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - P.P., G.V., K.K.D., P.K., S.G.; Design - P.P., G.V., K.K.D., P.K., S.G.; Supervision - P.P.; Resources - P.P., G.V., K.K.D., P.K., S.G.; Materials - P.P., G.V., K.K.D., P.K., S.G.; Data Collection and/or Processing - G.V., K.K.D., P.K., S.G.; Analysis and/or Interpretation - P.P., G.V., K.K.D., P.K., S.G.; Literature Search - P.P., G.V., K.K.D., P.K., S.G.; Writing Manuscript - P.P., G.V., K.K.D., P.K., S.G.; Critical Review - P.P.

Acknowledgements: We acknowledge with gratitude Prof. S. R. Savithri, Director, All India Institute of Speech and Hearing, Mysore, for permitting us to conduct the study at the institute. We thank all the participants of the study. We would like to thank the editor and the reviewers for their valuable suggestions to improve the manuscript.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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