EFFECT OF SMOKING ON HIGH FREQUENCY DISTORTION PRODUCT OTOACOUSTIC EMISSIONS

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This Dissertation is submitted as a part of fulfilment

for the Degree of Master of Science in Audiology

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July 2020

CERTIFICATE

This is to certify that this dissertation entitled '**Effect of smoking on high frequency distortion product otoacoustic emissions'** is the bonafide work submitted in part fulfillment for the Degree of Master of Science (Audiology) of the student with Registration No: **18AUD033**. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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CERTIFICATE

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DECLARATION

This is to certify that this Master's dissertation entitled **'Effect of smoking on high frequency distortion product otoacoustic emissions'** is the result of my own study under the guidance of **Ms Mamatha N.M**, Assistant Professor in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier in other University for the award of any Diploma or Degree.

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Dedicated to my parents, Mamatha ma'am, brother & friends for their constant guidance for pursuing my degree &and last but not the least MYSELF

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Abstract

Purpose: Cigarette smoking has become a common trend all over the world and the literature have concluded that smoking is also found to harm the auditory system, which is noted as an elevation in the hearing threshold and abnormal otoacoustic emissions. Hence the present study was aimed to determine the effect of smoking on high frequency distortion product emission (8points/octave).

Methods: Two groups of participants were taken for the study. Group I consisted of 18 smokers with a history of smoking for duration of 1year to 15 years and smoking minimum of 10 cigarettes per day and Group II consisted of 18 non-smokers who doesn't have any history of smoking. All the participants in both the groups underwent a detailed case history, pure tone Audiometry, immittance and high frequency distortion product otoacoustic emissions (DPOAE) testing.

Results: All the smokers included in the study had reduction in both DP amplitude and SNR values and the reduction was more evidently seen in the ultra-higher frequencies region above 11.2 kHz. The effect of smoking across frequency and between frequency was also seen in both DP amplitude and SNR, these effects were also seen more in the ultra-higher frequency region i.e., above 11.2 kHz.

Conclusion: The effect of smoking on DPOAE's is seen more at higher frequencies, which was evidenced through reduction of DP amplitude and SNR in the smoker group. This suggests that there is an adverse effect of smoking on higher frequency auditory sensitivity which cannot be seen in the audiogram of routine audiological test battery. Hence, from the current study it can be concluded that, it's important to include the extended high frequency DPOAE's in routine test battery which can provide better clarity about the occurrence of high frequency damage to cochlea more descriptively.

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Chapter 1

Introduction

Cigarette smoking has become a common trend all over the world, and it has been reported that tobacco is consumed by approximately 1.3 billion of the world's population (Shafey, Dolwick & Guindon, 2003). The risk of developing smoking-related diseases, such as cancer, heart disease, stroke, and respiratory illnesses, has been extensively researched (Gopal, Herrington, & Pearce, 2009)**,** but relatively little data exists on the specific auditory mechanisms that are affected by smoking. Many of the hazardous health effects of smoking is found to be depended on the exposure history, which includes the age at which the smoking began and the number of cigarettes which were smoked per day, the degree of inhalation, and the presence of cigarette characteristics such as the tar and the nicotine content (Fletcher & Peto, 1977).

The literature have concluded that smoking is also found to harm the auditory system, which is noted as an elevation in the hearing threshold, abnormal otoacoustic emissions as well as auditory evoked potentials (Jedrzejczak, Koziel, Kochanek, & Skarzynski2015). Hence, cigarette smoking is also considered to be highly associated with the reason for the development of hearing loss (Fransen et al. 2008).

Tobacco smoking is found to affect the inner ear through several mechanisms that can be categorized as direct or indirect. Toxic cigarette substances are noted to induce direct oxidative damage mediated by reactive oxygen species and free radicals that are capable of damaging many cellular components such as DNA, protein, and lipids, leading to neurosensory hearing

loss that affects mainly the higher frequencies. Indirect damage is mediated by the vasospastic effect caused by nicotine, by the acceleration of atherosclerosis in the vascular loops of the auditory system and by the increased levels of carboxyhemoglobin and increased blood viscosity. All these factors are observed to reduce oxygen perfusion in the organ of corti. Moreover, smoking can act as a risk factor for noise-induced hearing loss since smokers simultaneously exposed to noise in the workplace were affected by a higher degree of hearing loss compared to non-smokers (Zeilinger et al. 2013).

A hospital-based study done by Kumar et al. (2013) on 148 subjects (age: 20 to 60 years), among which 108 were smokers and 40 were age-matched non-smokers. The smoking history of all the subjects was taken in detail and their audiometric thresholds were measured. They found that the mild form (26-40 dB loss) was the most common (56.5%), while the severe type was the least common (2.8%) in the smokers and with 65.7% of the smokers and 15% of the non-smokers having a hearing impairment. Also, as the age increased, the percentage of the affected individuals increased, with more significant percentages of the smokers being affected in comparison to non-smokers. The most common type of hearing loss in the smokers was the sensorineural type (77.5%), followed by the mixed hearing loss (18.3%), while the mixed type was found in the non-smokers. They concluded that the severity of the hearing loss in smokers increased with an increase in the use of number of bidis/cigarettes.

It has also been reported that the hearing sensitivity of smokers is more likely to be reduced 1.5 times compared to non- smokers. Tobacco is found to cause vascular changes that can affect the cochlea and can also result in

reduced blood supply to the cochlea (Lowe, Drummond, Forbes, & Barbenel, 1980). Also, the toxic ingredients such as mercury and arsenic which can be seen in tobacco smoking can cause the degeneration of cochlear hair cells and also demyelination of nerves of auditory pathway(Cruickshanks et al. 1998) and also reduction in amplitude of DPOAE and wave V of auditory brainstem response (Gopal et al., (2009).

Negley, Katbamna, Crumpton, & Lawson (2007) examined the distortion product otoacoustic emissions (DPOAE) old smokers aged 20-30 years who smoked for5-8 years with no history of noise exposure or any middle ear disorder and had normal hearing sensitivity in the convectional audiometric frequency range (250 Hz – 8 kHz). The results showed a reduced emission and amplitude in the frequency that ranged from 2-8 kHz in smokers and mean input-output detection threshold that was measured at the f2 frequency of 2, 4, and 8 kHz. The elevation of the input-output detection threshold in smokers was found to be significant compared to non-smokers. It has been evidenced that the cochlear stria-vascularis may undergo age-related changes in the absence of any outer hair cell damage and any change could be detected by the input-output curve. Hence, it was concluded that the elevation of DPOAE input-output detection threshold in smokers was found to be are reflection of metabolic changes.

Otoacoustic emissions (OAEs) provide an index of cochlear function and are linked to outer hair cells' health (Kemp, 2002).Damage to the cochlea can be found through DPOAEs. The sound-induced vibrations by the OHCs in the cochlea, which are by-products of compressive non-linear amplification, which is found to enhance both the frequency resolution of hearing and

sensitivity and are referred to as DPOAEs (Robles & Ruggero, 2001;Moore, 2007).

Dreisbach et al. (2006) checked for the Repeatability of high-frequency DPOAEs in normal-hearing adults. DPOAEs were measured in 25 subjects (14 female &11 male) with normal behavioural hearing thresholds, normal middle ear function, and presence of acoustic reflexes at 1 kHz evoked by contralateral stimulation. Behavioural hearing thresholds were measured through 16 kHz, using Bekesy tracking. Each subject attended four trials, in which a complete set of data was collected, more significant variability was found at the higher frequencies (>8 kHz) for DPOAE level measurements. The average DPOAE level differences-between-trials for the higher and lower frequencies for the four different stimulus level conditions were 5.15 (SD = 4.40 dB) and 2.80 (SD = 2.70 dB) dB, respectively. The conclusion was that DPOAE level data obtained at frequencies higher than 8 kHz were more variable than at low frequencies, the higher frequencies were found to be repeatable. These results encourage the exploration of high-frequency DPOAE measures to be used as an objective test for monitoring ototoxicity in humans.

1.1 Need for the study

Smoking is found to have a significant influence on hearing function, especially on the cochlear apparatus and TEOAE is found to be a sensitive method used for very early detection of hearing loss, even in the absence of subjective complains when no threshold change was indicated on the audiogram. It has been reported that TEOAE amplitude is reduced in 76.6% of smokers and 3.33% of non-smokers and altered audiograms in 6.7% of smokers

and 3.33% of non-smokers in individuals aged 30-59 years (Gegenava, Japaridze, Sharashenidze, Jalabadze, and Kevanishvili. 2016).

It has been reported that there is a deterioration of behavioral hearing thresholds between 2000 and 10000 Hz frequencies with being more significant at 8000 Hz in smoker group compared to non-smoker group. Also the DPOAE amplitude of smokers was decreased infrequencies such as 1000, 2000, 4000 and 6000 Hz (Rogha et al.2015).

Munjal et al. (2017) revealed a statistically significant difference between chronic male smokers (age24-40 years) and the non-smoker group (age20-31 years) for behavioral thresholds (right ear: 250 Hz, 500 Hz, 2000 Hz and 4000 Hz; left ear: 250 Hz-8000 Hz). Also smokers were found to have different forms of degree of hearing loss that was shown in extended high-frequency audiometry. DPOAE and TEOAE results also revealed a statistically significant difference between the two groups at 2 kHz and 4 kHz and no significant difference was observed at 0.5 kHz, 1.4 kHz, 2 kHz, 3 kHz, 4 kHz, and 8 kHz. The study also revealed that chronic smokers were found to have reduced amplitude for OAEs, especially in high frequencies compared to non-smokers. Hence, the results here suggest the need for OAEs & HFA as essential tools for early identification of hearing loss related to smoking.

Prabhu, Varma, Dutta, Kumarand Goyal (2017)showed that the ultra-high-frequency behavioural thresholds (8 kHz, 9 kHz, 10 kHz, 12.5 kHz, &16 kHz)were elevated and ultra-high-frequency DPOAEs (8 kHz, 9 kHz, 10.25 kHz, 12.5 kHz, 14 kHz, &16 kHz.) amplitudes were reduced in smokers whose smoking duration ranged from 1-15 years.

The high-frequency OAEs are more sensitive than behavioural auditory thresholds in the early detection of hearing loss and cochlear damage. And in the previous studies, very less studies have been carried out in assessing the effect of smoking ultra-high frequency OAE's. Also, there is no quantification about up to which frequency the reduction in OAE amplitude is seen in smokers. Hence, the current study was planned to study the effect of smoking on high frequency DPOAE's (8 points/octave).

1.2 Aim of the study

The study aimed to determine the effect of smoking on high frequency DPOAE 8points/octave.

1.3 Objectives of the study

1. Comparison of DPOAE amplitude and SNR between smokers and non-smokers
2. Comparison of DPOAE amplitude across and between frequency in smokers (8 kHz-16 kHz)
3. Comparison of DPOAE SNR across and between frequency in smokers (8 kHz-16 kHz)

Chapter 2

Review of literature

2.1 Smoking and its effect on health

Cigarette smoking is reported to be a severe public health problem, and many studies have shown its various harmful effects on human physiological functions. Smokers are found to be at a higher risk of having bacterial respiratory infections and both acute and chronic viral diseases; oral, laryngeal, esophageal, pancreatic, renal, and bladder cancer; circulatory diseases such as arteriosclerosis, aortic aneurism, stroke, and multiple organ disorders. Tobacco toxicity effect was directly proportional to the number of cigarettes smoked and inversely proportional to the age at which the habit was initiated. The smoke from a cigarette is found to contain more than 4000 chemicals and has over 4500 complex chemicals in them, including carbon monoxide (CO), nicotine, and carbon dioxide (Gopal, Herrington, & Pearce et al. 2009).After cigarette smoke inhalation, carbon monoxide (CO) displaces the oxygen in red blood cells forming carboxyhemoglobin (COHb).In the form of COHb, the cigarette-derived CO has a conservative half-life of approximately five to six hours though it may remain in the bloodstream for up to 24 hours (Stewart 1970).

The risk of developing smoking-related diseases, such as cancer, heart disease, stroke, and respiratory illnesses, has been extensively researched. However, relatively little data exists on the smoking and its specific effect on auditory mechanisms.

2.2 Smoking and its effect on the auditory system

Hearing sensitivity of smokers is 1.5 times more likely to be reduced compared to non-smokers. Cigarette smoking is also considered to be highly associated with the development of hearing loss (Fransen et al., 2008).Prenatal and neonatal exposure to nicotine from smoking has shown to alter or diminish the functioning of the cortical nicotinic acetylcholine receptors and thus leading to long-term adverse effects on auditory-cognitive functions in adult rats(Slotkin et al.2007, Liang et al., 2006).Exposure to cigarette smoke was known to be associated with a 4.9 times increase in the prevalence of hearing deficits (Lyons, 1992).According to canter for disease control (CDC), 18-24year-old used tobacco products more than any other age group (Agaku et al., 2014). A decrease in the cardiac output is expected to affect the inner ear micro-circulation, which results in adverse effects on both the auditory and vestibular functions.

It has been reported that increased smoking is found to reduce the amount of available oxygen reaching the bloodstream in the human body, and thus, the amount of oxygenated blood that reaches the vital organs decreases (Moliterno et al., 1994). There is an excellent association between the vestibule and the cochlear structure as they share a continuous membranous labyrinth and also has a similar receptors cell (Zhou, Wu, & amp; Wang, 2016).

A longitudinal study has reported the risk of acquiring hearing loss, mainly at high frequencies. It is directly related to the number of cigarettes a day a smoker has and the time for which the subject has been a smoker (Nomura et al. 2005). Former smokers tend to develop high-frequency hearing loss, thus suggesting that the harmful effects of smoking upon hearing are cumulative and permanent (Nakanishi 2000).

A study carried out with animals in a laboratory observed the infliction of cochlear damage after exposure to cigarette smoking (Stewart 1976). Hawkins (1971) using animals found nicotine receptors in hair cells, thus indicating that smoking may have a direct ototoxic impact upon hair cell function and reduce the potential of the hearing neurotransmission organ. As less oxygen is available to the organ of corti, there is less energy available for the cochlea, and possibly more hair cell gets injured (Stewart, 1976; Chung & Browning et al. 1986).

Oto-acoustic Emission (OAE) is found to provide direct insight into outer hair cell cochlear amplification; indicates middle ear integrity and regular cochlear biologic mechanism activity. The efferent innervations made up of a large number of fibers that form the central and lateral efferent system. The medial efferent system is connected to the innervations of outer hair cells, while the lateral system is related to inner hair cells. The release of acetylcholine in the synaptic cleft through the medial olivocochlear efferent tract modulates the motion of outer hair cells (Bonfils, Bertrand & Uziel 1988).

Oto-acoustic emissions provide an index of cochlear function and are linked to outer hair cells' health (Kemp, 2002). Damage to the cochlea can be found through DPOAEs. The sound-induced vibrations by the OHCs in the cochlea, which are the by-products of compressive non-linear amplification, are found to enhance both the frequency resolution of hearing and sensitivity and are referred to as DPOAEs (Robles & Ruggero, 2001; Moore, 2007).

Laura Elizabeth et al. (2006) checked for the replicability of high-frequency DPOAEs in normal-hearing adults and concluded that higher frequencies were found to be repeatable for both paradigms tested. These results encourage the exploration of high-frequency DPOAE measures to be used as an objective test for monitoring

ototoxicity in humans. Testing subjects receiving ototoxic therapies is a necessary step in determining if monitoring high-frequency DPOAEs will successfully predict ototoxic effects.

Smokers are more susceptible to get hearing loss compared to non-smokers (Cruick shanks et al. 1998).According to a study on the Bangladeshi population, it has been shown that smokers had significantly higher hearing thresholds at 4, 8, and 12 kHz frequencies compared to non-smokers (Sumit et al. 2015). Negley et al. 2007 assessed the DPOAE amplitude and input-output function in smokers. The results showed a reduced emission and amplitude in 2-8 KHz range in smokers and meant input-output detection, which was measured at f2 frequency of 2, 4, and 8 kHz. The elevation in amplitude of smokers was found to be significant compared to non-smokers.

There are some studies in the literature which have concluded that smoking is found to harm the auditory system which leads to an elevation in the hearing threshold, abnormal OAEs as well as auditory evoked potentials (Jedrzejczak et al. 2015). Hence, cigarette smoking is also considered to be highly associated with the reason for the development of hearing loss (Fransen et al., 2008).

The previous studies have reported that the hearing sensitivity of smokers is 1.5 times more likely to be reduced compared to non- smokers. Tobacco is found to cause vascular changes that can affect the cochlea and also results in reduced blood supply to the cochlea (Lowe, Drummond, Forbes, & Barbenel, 1980).Also, the toxic ingredients such as mercury and arsenic which can be seen in tobacco smoking can cause the degeneration of cochlear hair cells and also demyelination of nerves of auditory pathway (Cruickshanks et al. 1998) and also reduction in amplitude of DPOAE and wave V of auditory brainstem response (Gopal et al.2009).

Kumar, Goyal, Varma, and Dutta (2017) assessed the influence of smoking on contra lateral suppression of DPOAEs on 25 smokers and non-smokers. The results of the study showed that the amount of suppression was reduced in smokers in all the frequencies (1000 Hz, 2000 Hz, 3000 Hz, 4000 Hz, 5000 Hz, &6000 Hz) suggesting an efferent auditory system dysfunction, and increase in the duration of smoking(1 to 15years), and the frequency of smoking which was every day to once in a week and the number of cigarettes smoked correlated negatively with the amount of suppression, concluding the chronic effect of smoking on the efferent auditory system.

Gegenava, Japaridze, Sharashenidze, Jalabadze, and Kevanishvili (2016) compared the TEOAE magnitude in 30 smokers and 30 non-smokers within the age range of 30-59 years. The OAEs were performed on each subject using Madsen Capella's-OAE/middle ear analyzer-GN Otometrics. After OAE testing, each subject was performed routine pure-tone audiometry and tympanometry. Results revealed that the amplitude was reduced in 76.6% of smokers, and 3.33% of non-smokers and audiogram measurements showed altered audiograms in 6.7% of smokers and 3.33% of non-smokers. They concluded that smoking has a significant influence on hearing function, especially on the cochlear apparatus, and TOEAE is also a sensitive method that can be used for very early detection of hearing loss, even when there are neither any subjective complaints nor some changes on the audiogram.

Rogha et al. (2015) evaluated the hearing threshold of the smoker group versus non-smoker using pure tone audiometry, TEOAE, and DPOAEs. In smoker group, smoking intensity (number of cigarettes smoked per day) was categorized in three-level: mild (<10 cigarettes smoked per day), moderate (10-20 cigarettes smoked per day) and severe (>20 cigarettes smoked per day) and use of other

tobacco products and drugs that were being used were recorded. Results showed deterioration of hearing threshold at frequencies between 2000 Hz and 10000 Hz was significant, particularly at 8000 Hz in the smoker group compared with the non-smoker group. Also, the DPOAE amplitude of smokers was decreased in respective frequencies 1000, 2000, 4000, and 6000 Hz. However, TEOAE amplitude decreased at all frequencies in a smoker than a non-smoker and results were not statistically significant. Overall results showed significant differences between smoking intensity (mild &severe level) and the hearing threshold at 2000, 4000 and 8000 Hz, but statistically no significant difference was found between smoking intensity and DPOAE/TEOAE test results.

Prabhu, Varma, Dutta, Kumar, and Goyal, (2017) analyzed high-frequency audiometric threshold and high-frequency DPOAE for both smokers having smoking duration that ranged from 1-15 years and non-smokers aged from 18 to 40 years.. The results showed that the ultra-high-frequency thresholds were elevated and there was a reduction in the amplitudes of ultra- high-frequency OAEs in smokers. Harkrider et al., (2001)investigated the role of cholinergic mechanisms in the auditory system by assessing the acute effects of nicotine, an acetylcholinomimetic drug, on aggregate responses within the auditory pathway.

In a single-blind procedure, auditory responses were obtained from 20 normal-hearing, non-smokers (10 male) under two conditions (nicotine & placebo). After the drug session, plasma tests revealed a subject's nicotine concentration and theeffects of nicotine on early, exogenous responses of the auditory system (oto-acoustic emissions &auditory brainstem potentials) were assessed. Results indicated that transdermal administration of nicotine to non-smokers does not significantly affect cochlear activity but does acutely affect the neural transmission of acoustic

information. Overall, OAEs were unaffected by transdermal nicotine, while wave I of the auditory brainstem response was significantly increased in latency and decreased in amplitude.

Mehrparvar et al. (2015) carried out a cohort study on 224 workers who were exposed to noise and were divided into two groups: Smokers and non-smokers and measured DPOAE response amplitudes. Assessed the simultaneous effect of noise and smoking on standard pure tone audiometry (PTA) and DPOAEs. One hundred and five subjects were smokers (case group), and 119 individuals were non-smokers (control group). All the subjects were exposed to 91.08 ± 2.29 dBA [time-weighted average (TWA) for an eight work shift]. Mean DPOAE response amplitude at frequencies higher than 1,000 Hz was significantly higher in smokers compared to non-smokers. From this study, it can be observed that smoking can aggravate the effect of noise on hearing in DPOAEs.

The effects of cigarette smoking on auditory thresholds, OAE and their inhibition effect by the efferent olivocochlear medial system were assessed by Paschole and de Azevedo (2009)where they performed high-frequency audiometry, TEOAEs, and suppression effect on 144 adults from both genders, between 20 and 31 years of age, consisting of both smoking and non-smoking individuals. The results of the study indicated that smokers presented worse auditory thresholds in the frequencies of 12,500 Hz in the right ear, and 14,000 Hz in both ears. It was also noted that individuals with smokers presented a lower OAE response level in the frequencies of 1,000 Hz in both ears and 4,000 Hz in the left ear. Among the smokers, cochlear dysfunction and tinnitus were found to be more. Moreover, it was concluded that smoking has harmful effects on hearing, as the smoker group had worse auditory thresholds in high frequencies (above 8000Hz), lower response levels

to TEOAEs, and higher suppression levels when compared to non-smokers. Additionally, it was found that the smoker groups were affected more by tinnitus and cochlear disorders.

From the review, it is clear that smoking is found to have an adverse effect on the auditory system which leads to an elevation of hearing threshold, abnormal OAE's as well as auditory evoked potentials. Reduced DP amplitude in smokers is reported up to 8 kHz but there are very few studies have been carried out DPOAE's including frequencies above 8 kHz. Hence, the present study was aimed to measure DPOAE at higher frequency range (8 kHz-16 kHz) with 8 points per octave in individuals with smokers having history of various smoking duration, which might further through light on smoking and its effects on the cochlear system.

Chapter 3

Methods

The present study was conducted to assess the effect of smoking on distortion product otoacoustic emission with 8 points per octave and the effect of frequency on amplitude and SNR of DPOAE's. To meet the aim of the study, the following method is followed.

3.1 Selection of participants

Thirty-six participants in the age range of 20 to 40 years were recruited for the study. Two groups of participants were included, where the group I consisted of 18 individuals with a history of smoking for 1year to 15 years and group II (control) consisted of 18 individuals who never had a history of smoking.

The following Inclusion criteria were included for the selection of participants for Group I. Smoking at least a pack consisting of 10 cigarettes per day.

- No history of any middle ear pathology (ear discharge, ear pain, etc.)
- No medical problems such as hypertension and diabetes etc.
- No history of noise exposure.
- Absence of history of any relevant otological problems.
- No history or presence of any neurological problems.
- Should not be trained musicians.

The participants included in the control group (group II) were age-matched to the experimental groups and fulfilling the following criteria was considered.

- No history of any middle ear pathology (ear discharge, ear pain, etc)
- Subjects should have a normal pure tone, speech recognition threshold and speech identification scores should be within 90-100%
- Normal otoscopic findings with 'A' type tympanogram and standard acoustic reflex threshold, both ipsilateral and contralateral.
- No medical problems such as hypertension and diabetes etc.
- Absence of history of any relevant otological problems.
- No history or presence of any neurological problems.

3.2 Instrumentation

- Calibrated Inventis Piano Plus was used to perform threshold estimation (pure tone audiometry &speech audiometry) with calibrated TDH 39 headphones for AC threshold and calibrated B-71 bone vibrator for BC threshold.
- Calibrated GSI Tympstar Immittance meter was used to measure tympanometry with a probe tone frequency of (226 Hz) and the acoustic reflex threshold for probe tone frequency of 226Hz. Both ipsilateral and contralateral acoustic reflexes will be measured using 500, 1000, 2000, and 4000 Hz pure tones.
- OAE measurements were done using a calibrated Mimosa Acoustics OAE system after ensuring proper probe fit.

3.3 Test environment

All the participants were subjected to tests in an acoustically treated room, which meets the ambient noise level criteria specified by ANSI S3.1-1999 (R2008).

3.4 Procedure

3.4.1 Case History

A detailed case history was taken from all the participants to rule out any pathological conditions of the auditory system and to make a count of smoking histories such as the number of cigarettes per day and the duration of smoking.

3.4.2Pure tone and speech Audiometry

Using the modified Hughson and Westlake procedure (Carhart&Jerger, 1959), air-conduction threshold with the TDH 39 headphones and bone conduction thresholds with B-71bone vibrator were obtained for octave frequencies from 250 to 8000 Hz and 250 to 4000 Hz respectively to investigate the hearing sensitivity of each participant. Phonemically Balanced Kannada Word Test by Yathiraj&Vijayalakshmi (2005)was used to obtain speech identification scores (SIS).

3.4.3Immittance Evaluation

Both tympanometry and acoustic reflex thresholds were measured using Garson Stadler Inc. Tympstar (GSI-TS, Eden Prairie, MN) to record tympanogram and acoustic reflex threshold for probe tone frequency of 226Hz.Both ipsilateral and contralateral acoustic reflexes were measured using

500, 1000, 2000 and 4000 Hz pure tones, to rule out middle ear pathology and confirm the presence/absence of sensorineural component.

3.4.4Recording of DPOAE

DPOAE's with two frequencies F1(lower frequency) and F2(higher frequency) was selected for the recording of DPOAE. The two frequencies F1 &F2 stimulate the cochlea and produce a small distortion. The distortion product occurs at a frequency lower than the F2 or F1. The typical distortion product in the human ear that is largest occurs at 2F1-F2. There is also a partial reflection component to the DPOAEs.

After ensuring proper and adequate probe fit OAE measurements was done through a calibrated Mimosa Acoustics OAE system in a sound-treated room, 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, 9.5 kHz, 10.3 kHz, 11.2 kHz, 12.3 kHz, 13.4 kHz, 14.6 kHz, and 15.9 kHz, and the stimulus tones were swept at rate of 8 points per octave.

3.4.5DPOAE Analysis

An absent DPOAE is classified, if the DPOAE is not present at two or more f2 frequencies with sufficient signal-to-noise ratio (SNR) with at least 6 dB SNR and present DPOAE response must show a DPOAE with greater than 6 dB SNR at approximately 70 percent of the collected data points. (Hall, Chase, Baer and Schwaber1994)

Chapter 4

Results

The aim of the current study was to compare the DPOAE amplitude and SNR values in two groups (smokers &non-smokers). The smoker and non-smoker group consisted of 18 participants each respectively. The DPOAE amplitude (DP amplitude) and DPOAE SNR (DP SNR) values that were obtained for eight frequencies (8 kHz-16 kHz) were compared between the two groups. All the data obtained was analyzed using statistical package of social science (SPSS) software version 2.0. The Shapiro Wilk's test of normality was administered to check whether the raw data is normally distributed or not, and it was found that, the data was not normally distributed ($p < 0.05$).Hence, the non-parametric tests were chosen for further analysis and the results of the study are explained. The following statistical analysis was carried out to compare the obtained data.

- Descriptive statistics was performed to examine the mean and standard deviation (SD) of DP amplitude and DP SNR of both smokers and non-smokers.
- Wilcoxon signed ranks test was administered to check for the ear difference in DP amplitude and SNR values.
- Mann-Whitney test for group comparison was administered on combined ear results to check for the difference of DP amplitude and DP SNR values between two groups since there was no difference between the ears.
- Friedman's test was done to see the effect of frequencies on DP amplitude and SNR values and Wilcoxon Signed Rank test for pair wise comparison across frequencies.

The results of the data are presented in the following headings:

1. Comparison of DP amplitude and DP SNR values between smokers and non–smokers.
2. Comparison of DP amplitude between frequencies in smokers (8 kHz – 16 kHz).
3. Comparison of DP SNR between frequency in smokers (8 kHz – 16 kHz)

4.1 Comparison of DPOAE amplitude and SNR between smokers and non-smokers

Descriptive statistics was done to find the mean and standard deviation of DP amplitude and SNR values at eight test frequencies (8 kHz, 9.5 kHz, 10.3 kHz, 11.2 kHz, 12.3 kHz, 13.4 kHz, 14.6 kHz & 15.9 kHz) in both smokers and non-smokers in each ear individually. The mean and standard deviation of DP amplitude of both right ear and left ear are represented in Figure 4.1 and 4.2 and DP SNR values of both right ear and left ear given in Figure in 4.3 and 4.4.

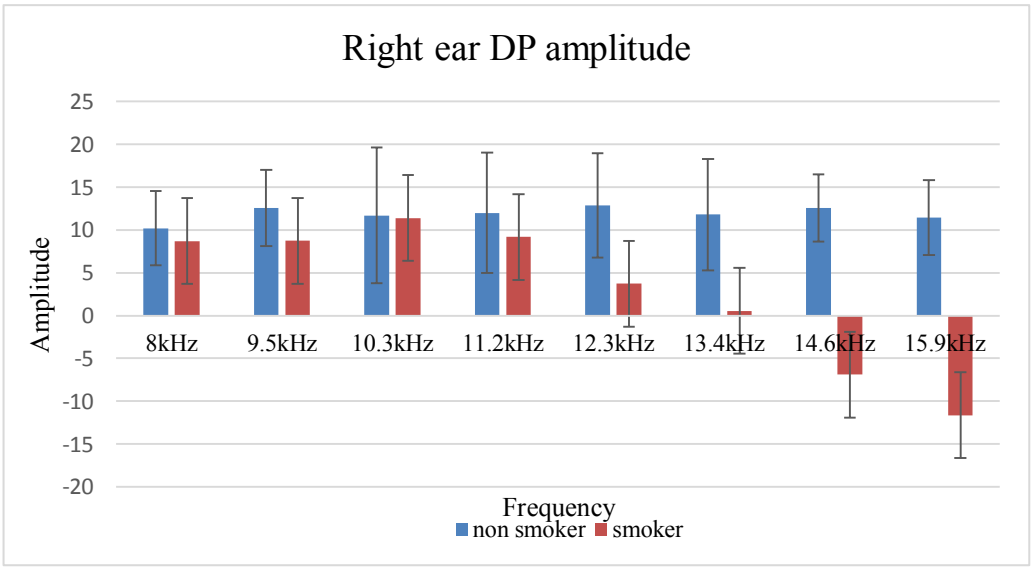

Figure 4.1 Representing mean and standard deviation of right ear DP amplitude of both smokers and non-smokers

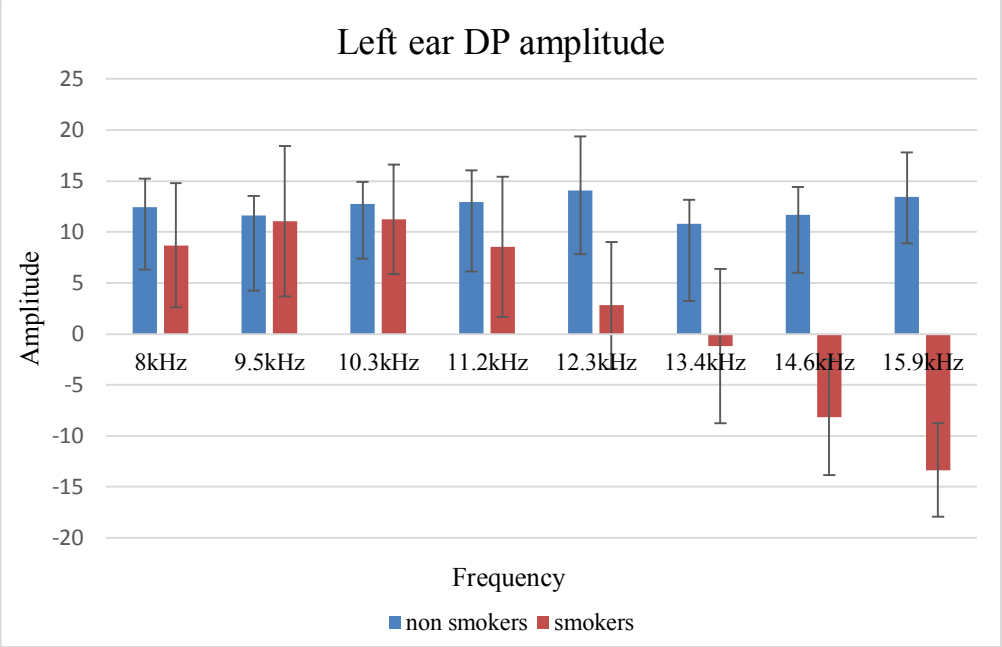

Figure 4.2 Representing mean and standard deviation of left ear DP amplitude of both smokers and non-smokers

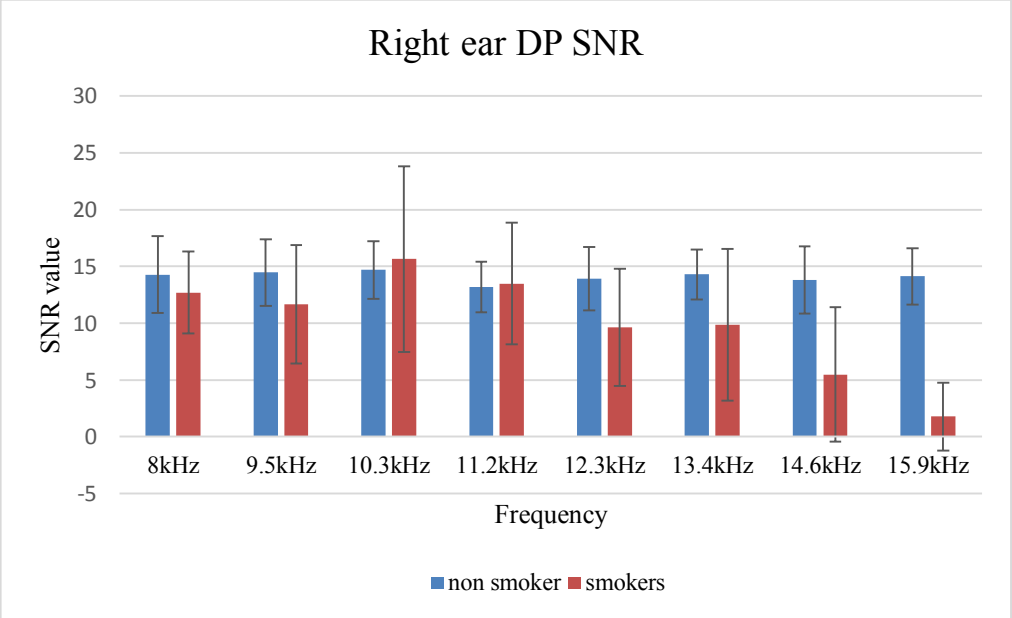

Figure 4.3 Representing mean and standard deviation of right ear DP SNR values of both smokers and non-smokers

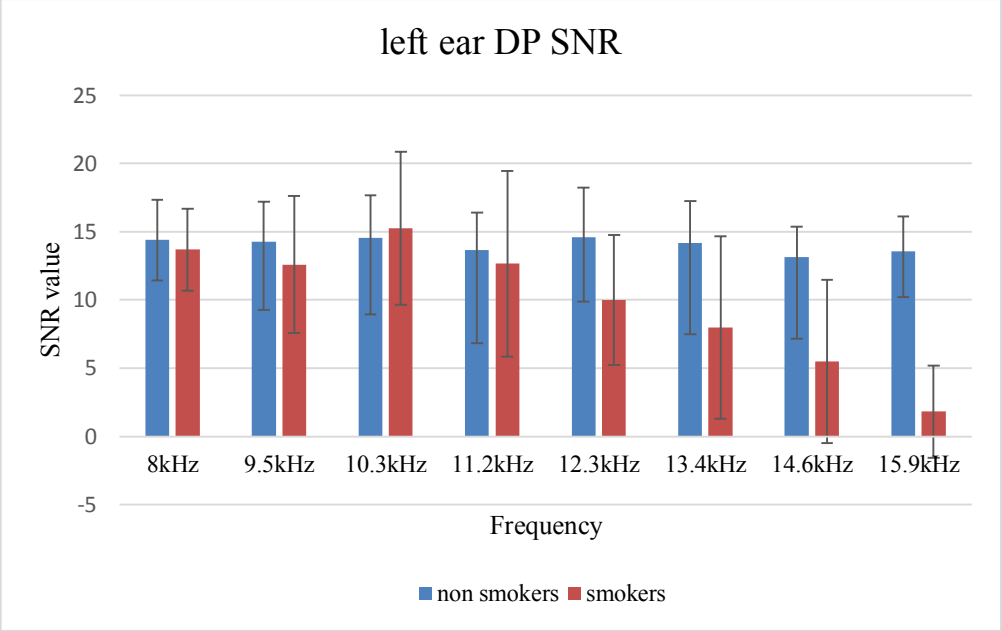

Figure 4.4 Representing mean and standard deviation of left ear SNR values of both smokers and non-smokers

From the Figure 4.1 and 4.2 it can be seen that the mean DP amplitude of smokers for both right and left ears were reduced from 8 kHz to 16 kHz when compared with mean amplitudes of non- smokers. It can also be observed that, the DP amplitude started to reduce from 11.2 kHz and was found to be absent from 13.4 kHz.

Figure 4.3 and 4.4 depicts that mean DP SNR value of both right and left ear of smokers group was gradually been reducing when it was compared with mean DP SNR values of non-smokers group. It can also be noted that, the DP SNR were found to be reduced as the frequency increased and this was more evident after 14.6 kHz.

Before doing the group comparison Wilcoxon signed ranks test was administered to check for the difference between the ears in DPOAE amplitude and SNR values for both smokers and non-smokers group and the results are shown in table 4.1.1 and table 4.1.2

Table 4.1

Test statistics for comparison of DP amplitude between the ears in smokers and non-smokers

Table 4.1.1

DP amplitude of smokers and non-smokers

Frequencies (left ear v/s right ear)	Smokers		Non -smokers	
	Z	p value	Z	p value
L8kHz- R8kHz	-.41	.67	-2.69	.00
L9.5kHz - R9.5kHz	-.84	.39	-1.96	.05
L10.3kHz - R10.3kHz	-.23	.81	-1.23	.21
L11.2kHz - R11.2kHz	-.08	.93	-1.20	.22
L12.3kHz - R12.3kHz	-.80	.42	-.58	.55
L13.4Kamp - R13.4kHz	-.76	.44	-1.08	.27
L14.6kHz - R14.6kHz	-1.11	.26	-1.39	.162
L15.9kHz - R15.9kHz	-1.61	10	-1.82	.07

Table 4.1.2

DP SNR of Smokers and non-smokers

Frequencies (left ear v/s right ear)	Smokers		Non-smokers	
	Z	p value	Z	p value
L8KkHz - R8kHz	-.89	.37	-.90	.36
L9.5kHz - R9.5kHz	-.80	.42	.00	1.00
L10.3kHz - R10.3kHz	-.19	.84	-.19	.84
L11.2kHz - R11.2kHz	-.47	.63	-.45	.64
L12.3kHz - R12.3kHz	-.54	.58	-.59	.55
L13.4kHz - R13.4kHz	-1.63	.102	-.47	.63
L14.6kHz - R14.6kHz	-.024	.98	-.71	.47
L15.9kHz - R15.9kHz	-.052	.95	-.61	.53

From the table 4.1.1 and 4.1.2itcan be seen that there was no significant difference ($p < 0.05$) found between smokers and non-smokers group in both DPOAE amplitude and SNR between right and left ear across most of the frequencies. Hence for further analysis, both right and left ear DPOAE amplitude and SNR across frequencies were combined and the combined mean and standard deviation of both amplitude and SNR values across different frequencies of both smokers and non-smokers are given in table 4.2 and 4.3 respectively. The same results are represented in Figure 4.5 and 4.6.

Table 4.2

Descriptive statistics showing both mean and standard deviation of smokers and non-smokers for DPOAE amplitude from 8 kHz to 16 kHz

Ears combined DPOAE amplitudes	Non-smokers		Smokers	
	Mean	SD	Mean	SD
*F8Kamp	11.28	11.28	8.70	5.22
*F9.5Kamp	12.10	12.10	9.89	6.10
*F10.3Kamp	12.21	12.21	11.32	6.67
*F11.2Kamp	12.48	12.48	8.85	6.84
*F12.3Kamp	13.44	13.44	3.27	6.08
*F13.4Kamp	11.30	11.30	-.327	7.01
*F14.6Kamp	12.12	12.12	-7.51	4.85
*F15.9Kamp	12.45	12.45	-12.51	4.49

*F= combined ear result

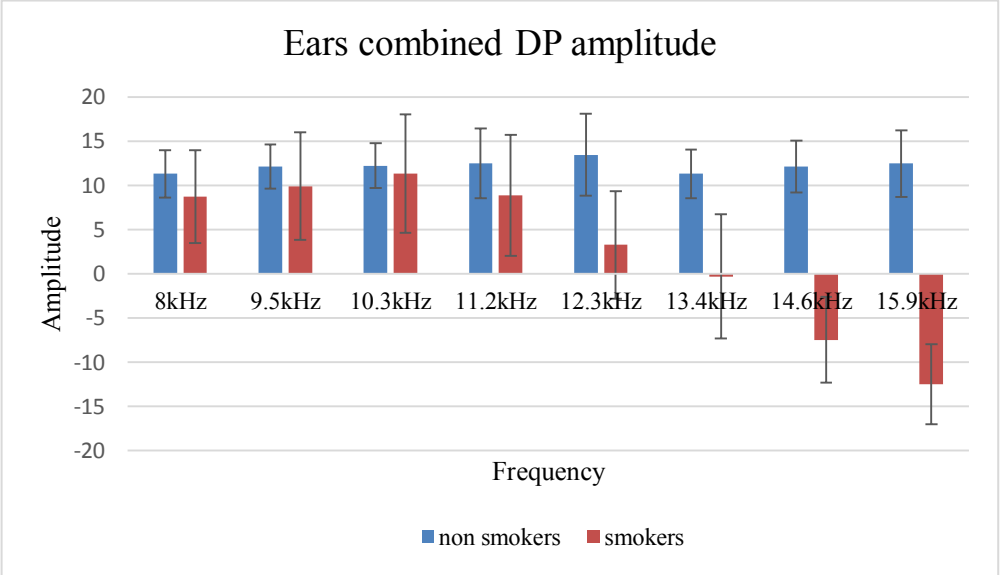

Figure 4.5 Representing mean and standard deviation of DP amplitude of both smokers and non-smokers

Table 4.3

Descriptive statistics comprising both mean and standard deviation of DP SNR from 8 kHz to 16 kHz in smokers and non-smokers

DPOAE SNR values	Non – smokers		Smokers	
	Mean	SD	Mean	SD
*F8Ksnr	14.34	14.34	13.19	3.30
*F9.5Ksnr	14.36	14.36	12.12	5.05
*F10.3Ksnr	14.61	14.61	15.46	6.91
*F11.2Ksnr	13.42	13.42	13.07	6.05
*F12.3Ksnr	14.27	14.27	9.81	4.88
*F13.4Ksnr	14.23	14.23	8.91	6.65
*F14.6Ksnr	13.49	13.49	5.49	5.86
*F15.9Ksnr	13.85	13.85	1.79	3.14

*F= combined ear result

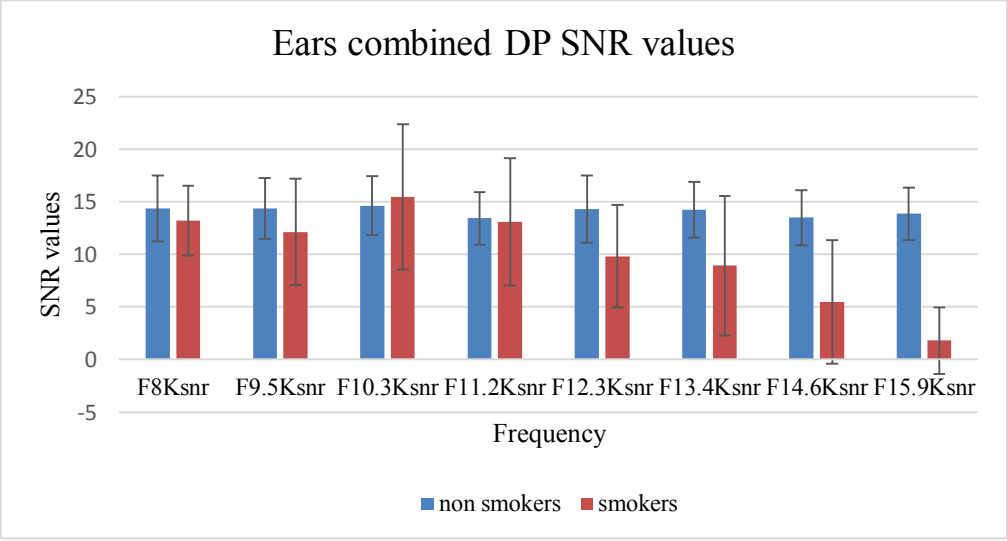

Figure 4.6 Representing mean and standard deviation of DP SNR values of both smokers and non-smokers

From Table 4.2 and Figure 4.5, it is evident that, that the mean DP amplitude of smokers group were reduced when compared to non-smokers and it's also seen that as the frequency increases from 8 kHz to 16 kHz the DP amplitude in the smokers group got reduced gradually when compared with non-smokers.

Also, from Table 4.3 and Figure 4.6, it is evident that, the mean DP SNR values of smokers group were reduced when compared to non-smokers. It can also be seen that as the frequency increases from 8 kHz to 16 kHz the DP SNR reduced in the smokers group compared to non- smokers.

Overall, it was found that the mean amplitude of smokers are reduced when compared to non-smokers after 11.2 kHz and also the SNR value is reduced in smokers after 12.3 kHz when compared to non-smoker which is also depicted in the above tables (4.2 &4.3) and Figure (4.5 &4.6)

The mean scores indicated that there exists a difference in mean scores (DP amplitude & DP SNR) between smokers and non- smoker. In order to see if there exists a significant difference between the two groups, Mann-Whitney test was done for both amplitude and SNR which is shown in table 4.4.

Table 4.4

Z and p value obtained for comparison of DP amplitude and DP SNR between smokers and non-smoker

DP amplitude			DP SNR		
Frequencies	Z	p value	Frequencies	Z	p value
*F8Kamp	-3.41	.00*	*F8Ksnr	-1.31	.19
*F9.5Kamp	-3.32	.00*	*F9.5Ksnr	-2.34	.01*
*F10.3Kamp	-2.00	.04*	*F10.3Ksnr	-.84	.39
*F11.2Kamp	-2.87	.00*	*F11.2Ksnr	-1.52	.12
*F12.3Kamp	-5.90	.00*	*F12.3Ksnr	-3.72	.00*
*F13.4Kamp	-5.94	.00*	*F13.4Ksnr	-4.83	.00*
*F14.6Kamp	-7.27	.00*	*F14.6Ksnr	-5.12	.00*
*F15.9Kamp	-7.24	.00*	*F15.9Ksnr	-7.24	.00*

*F= combined ear result and*significant level ($p < 0.05$)

The Mann-Whitney test results showed that there was significant difference ($p < 0.05$) of DPOAE amplitude at all the test frequencies (8 kHz – 16 kHz) between smokers and non-smokers except at 8 kHz, 10.3 kHz and 11.2 kHz.

For comparing DP amplitude across various frequencies in both smokers and non-smokers, Friedman's test was done. The results showed that there was significant difference found in DPOAE amplitude across all the test frequencies for smokers [χ^2 (7, n = 35) = 178.16, $p < 0.05$] group and there was no significant difference found in amplitude for non-smoker [χ^2 (7, n = 36) = 14.104, $p > 0.05$] respectively.

4.2 Comparison of DPOAE amplitude between frequencies (8 kHz-16 kHz) in smokers

Since there was a significant difference noted in DPOAE amplitude across all the frequencies in smoker group, Wilcoxon signed rank test was administered to see if there exists a significant difference and to see which frequency groups had significant difference in DPOAE amplitude and the results are depicted in table4.5.

Table 4.5

Z and p values for within group comparison of DPOAE amplitude of smokers between different frequency combinations

Frequencies	Z	p value	Frequencies	Z	p value
F9.5Kamp - F8Kamp	-.56	.57	F12.3Kamp - F10.3Kamp	-4.81	.00*
F10.3Kamp - F8Kamp	-.99	.31	F13.4Kamp - F10.3Kamp	-5.10	.00*
F11.2Kamp – F8Kamp	-.17	.86	F14.6Kamp - F10.3Kamp	-5.23	.00*
F12.3Kamp - F8Kamp	-3.28	.00*	F15.9Kamp - F10.3Kamp	-5.16	.00*
F13.4Kamp - F8Kamp	-4.16	.00*	F12.3Kamp - F11.2Kamp	-4.63	.00*
F14.6Kamp - F8Kamp	-5.16	.00*	F13.4Kamp - F11.2Kamp	-5.18	.00*
F15.9Kamp - F8Kamp	-5.16	.00*	F14.6Kamp - F11.2Kamp	-5.23	.00*
F10.3Kamp - F9.5Kamp	-.66	.50	F15.9Kamp - F11.2Kamp	-5.15	.00*
F11.2Kamp - F9.5Kamp	-.89	.37	F13.4Kamp - F12.3Kamp	-3.07	.00*
F12.3Kamp - F9.5Kamp	-3.82	.00*	F14.6Kamp - F12.3Kamp	-5.23	.00*
F13.4Kamp - F9.5Kamp	-4.43	.00*	F15.9Kamp - F12.3Kamp	-5.16	.00*
F14.6Kamp - F9.5Kamp	-5.23	.00*	F14.6Kamp - F13.4Kamp	-5.12	.00*
F15.9Kamp - F9.5Kamp	-5.16	.00*	F15.9Kamp - F13.4Kamp	-5.13	.00*
F11.2Kamp - F10.3Kamp	-2.91	.00*	F15.9Kamp - F14.6Kamp	-4.91	.00*

*significant level ($p < 0.05$)

From table 4.5 it is clear that except for the frequency combinations (9.5 kHz- 8 kHz, 10.3 kHz-8 kHz, 11.2 kHz – 8 kHz and 10.3 kHz – 9.5 kHz, 11.2 kHz – 9.5 kHz) rest all other test frequencies had significant difference in DPOAE amplitude when they were paired and compared with rest of the frequencies. Thus it can be seen that the significant difference is only at high frequencies i.e., above 11.2 kHz.

For comparing the DP SNR across the frequency in both smokers and non-smokers, Friedman's test was done. The results showed that there was a significant difference found in DP SNR across all the test frequencies for smokers [$\chi^2(7, n = 35) = 114.84, p < 0.05$]group and there was no significant difference found in DP SNR for non-smoker [$\chi^2(7, n = 36) = 6.70, p > 0.05$].

4.3 Comparison of DP SNR between frequencies (8 kHz – 16 kHz) in smokers

Since there was a significant difference noted in DP SNR across all the frequencies in smoker group, Wilcoxon signed rank test was administered if there exists a significant difference within group comparison to see which frequency groups had significant difference in DP SNR and the results are depicted in table 4.6

Table 4.6

Z and p value for within group comparison of DP SNR value of smokers between different frequency combinations

Frequencies	Z	p value	Frequencies	Z	p value
F9.5Ksnr - F8Ksnr	-.82	.40	F12.3Ksnr - F10.3Ksnr	-4.66	.00*
F10.3Ksnr - F8Ksnr	-1.44	.14	F13.4Ksnr - F10.3Ksnr	-4.93	.00*
F11.2Ksnr - F8Ksnr	-.48	.62	F14.6Ksnr - F10.3Ksnr	-5.01	.00*
F12.3Ksnr - F8Ksnr	-3.75	.00*	F15.9Ksnr - F10.3Ksnr	-5.16	.00*
F13.4Ksnr - F8Ksnr	-3.45	.00*	F12.3Ksnr - F11.2Ksnr	-3.09	.00*
F14.6Ksnr - F8Ksnr	-4.88	.00*	F13.4Ksnr - F11.2Ksnr	-3.85	.00*
F15.9Ksnr - F8Ksnr	-5.16	.00*	F14.6Ksnr - F11.2Ksnr	-4.87	.00*
F10.3Ksnr - F9.5Ksnr	-2.37	.01*	F15.9Ksnr - F11.2Ksnr	-5.11	.00*
F11.2Ksnr - F9.5Ksnr	-.33	.73	F13.4Ksnr - F12.3Ksnr	-1.67	.09
F12.3Ksnr - F9.5Ksnr	-2.62	.00*	F14.6Ksnr - F12.3Ksnr	-3.74	.00*
F13.4Ksnr - F9.5Ksnr	-2.49	.01*	F15.9Ksnr - F12.3Ksnr	-4.86	.00*
F14.6Ksnr - F9.5Ksnr	-3.78	.00*	F14.6Ksnr - F13.4Ksnr	-3.82	.00*
F15.9Ksnr - F9.5Ksnr	-5.09	.00*	F15.9Ksnr - F13.4Ksnr	-4.52	.00*
F11.2Ksnr - F10.3Ksnr	-2.20	.02*	F15.9Ksnr - F14.6Ksnr	-3.23	.00*

*significant difference ($p < 0.05$)

From the table 4.6 It's shown that except these frequency (9.5 kHz – 8 kHz) (10.3 kHz – 8 kHz) (11.2 kHz – 8 kHz) and (11.2 kHz – 9.5 kHz) rest all the test

frequencies had significant difference in SNR value when they are paired with rest of the test frequencies

Overall the statistical test results for group comparison between smokers and non-smokers revealed that

- There was a significant difference found in DP amplitude between smoker and non-smoker group in all the test frequencies.
- There was a significant difference in DP SNR between smoker and non-smoker group except at 8 kHz, 10.3 kHz and11.2 kHz.
- There was no significant ear difference seen in both DP amplitude and DP SNR.
- There was no significant effect of test frequencies on DP amplitude and DP SNR in non-smoker group whereas; smoker group showed a significant effect of test frequencies on DP amplitude and DP SNR.
- Between test frequency comparison showed that there was significant difference in DP amplitude in all the stimulus pairs compared (starting from above 11.2 kHz), except for other stimulus pairs such as 9.5 kHz – 8 kHz; 10.3 kHz – 8 kHz; 11.2 kHz – 8 kHz and 10.3 kHz – 9.5 kHz; 11.2 kHz – 9.5 kHz.
- Similarly, there was a significant difference in DP SNR values in all the stimulus pairs compared (starting from above11.2 kHz), except for other stimulus pairs such as 9.5 kHz – 8 kHz; 10.3 kHz – 8 kHz; 11.2 kHz – 8 kHz and 11.2 kHz – 9.5 kHz.

Chapter 5

Discussion

The aim of the present study was to study the effect of smoking on DPOAE's with eight points per octave. The objectives of the study were to measure the DP amplitude in smokers and to compare them with non-smokers, to measure the DP SNR values in smokers and to compare them with non-smokers and to check for the effect of frequency as well as between test frequencies on DP amplitude and DP SNR in both smokers and non-smokers. The results obtained in the study are discussed below.

5.1 Comparison of DPOAE amplitude and SNR between smokers and non-smokers

The results of the present study show that overall the smokers group had reduced DP amplitude when compared to non-smokers in all the frequencies tested (8 kHz-16 kHz). However, this reduction in DP amplitude is found to be more in the higher frequency region i.e., (12.3 kHz, 13.4 kHz, 14.6 kHz &15.9 kHz). Similar findings are obtained for DP SNR values where; smokers had overall reduced DP SNR especially at higher frequency i.e.,(11.2 kHz, 12.3 kHz, 13.4 kHz, 14.6 kHz and 15.9 kHz). In other words, the reduction of DP amplitude and DP SNR values of smokers is found to be directly proportional to the stimulus frequency, showing that effect of smoking is more on higher frequency region. In comparison of sensitivity of DP amplitude and DP SNR towards smoking, the DP amplitude showed effect of smoking in all the stimulus frequencies considered however, more effect was seen in higher frequencies (i.e., 12.3 kHz, 13.4 kHz, 14.6 kHz & 15.9 kHz). Whereas DP SNR showed the effect of smoking only

above 11.2 kHz but didn't have any effect or reduction in SNR value in frequencies below 11.2 kHz.

The results of the present study are in agreement with the study done by Prabhu, Varma, Dutta, Kumar and Goyal (2017) where they have got similar findings i.e., reduction of DP amplitude in smokers when compared to non- smokers and this effect was more evident in higher frequencies (i.e., 12.5 kHz,14 kHz, 16 kHz) which is similar to the present study. The reduction of DP amplitude in the smokers group was attributed to the high amounts of carbon monoxide and nicotine which is found to restrict blood circulation to the cochlea in turn damaging the outer hair cells. However, the damage is happening to the OHCs that are noted to be initiating from the basal region of the cochlea. Hence the effect of smoking can be seen initially in the higher frequencies. The study done by Negley et al (2007) concludes that ultra-high frequencies are more prone to damage because of the reduced blood supply and reported that carbon monoxide and nicotine in the cigarette reduces oxygen supply to outer hair cells of the cochlea. Evidence also suggests that the cochlear artery, which supplies the basal region of the cochlea, is susceptible to atherosclerotic changes seen in smokers (Zelman,1973).

The similar results were also supported by Rogha et al. (2015) where the study considered 32 individuals and some of the factors such as smoking intensity (number of cigarettes smoked per day) which categorized in three-level: mild (<10 cigarettes smoked per day), moderate (10-20 cigarette smoked per day) and severe (>20 cigarettes smoked per day) and use of other tobacco products and drugs are being used were recorded. Their study results revealed that 1000, 2000, 4000, and 6000 Hz had reduced DP amplitude. Munjal et al. (2017) considered 100 chronic smokers and the duration of smoking ranged for more than 5 years and the results

revealed that there was reduction of amplitude that was seen only in 2 kHz and 4 kHz. Negley, Katbamna, Crumpton& Lawson (2007) considered 12 smokers and 12 non-smokers who had history of 5-8 years duration of smoking, regardless of the number/packs of cigarettes smoked per day. Their study results depicted that there was reduction in DPOAE amplitude at all frequency tested (i.e., from 2 kHz-8 kHz).

Ali, Ismail and Ismail (2019) measured DPOAE in 15 individuals where the duration of smoking was not specified and the results showed that the reduction of DP amplitude was seen in 1 kHz, 2 kHz, 3 kHz, 4 kHz, 6 kHz. Similarly, Mehrparvar et al. (2015) also supported present study that showed reduction of DP amplitude in individuals who had history of exposure to noise and smoking, where both smokers and non-smokers suffered from hearing loss. But DPOAE response amplitude was significantly lower among the smokers and abnormal response amplitude was observed most frequently at 6,000 Hz followed by 4,000 Hz in both ears. Overall from above supporting studies it's seen that the reduction of DP amplitude was reported only till 8 kHz.

The results of the present study on DP SNR values are in agreement with study done by Jedrzejczak, Koziel, Kochanek, & Skarzynski, (2015) who considered 24 smokers. And determined the average number of cigarettes smoked per day (8.7 cigarettes; range, 1 to 20 cigarettes), the reduction of DP SNR values were reported till 8 kHz.

5.2 Comparison of DP amplitude and DP SNR across frequency (8 kHz-16 kHz) and between test frequencies

The results indicated no significant effect of test frequencies on DP amplitude and SNR in non-smoker group. This could be due to the nonlinear aspect of the outer hair cells functioning which involves active functioning of stereocilia which is found to be intact in non-smoker group (Brownell, 1983), where as smoker group showed a significant effect of test frequencies on DP amplitude and SNR. Between test frequency comparison showed that there was significant difference noted in amplitude in all the stimulus pairs compared (from above11.2 kHz) except 9.5 kHz – 8 kHz; 10.3 kHz – 8 kHz; 11.2 kHz – 8 kHz and 10.3 kHz – 9.5 kHz; 11.2 kHz – 9.5 kHz. Similarly, there was a significant difference seen in DP SNR in all the stimulus pairs compared (from above11.2 kHz) except 9.5 kHz – 8 kHz; 10.3 kHz – 8 kHz; 11.2 kHz – 8 kHz and 11.2 kHz – 9.5 kHz.

The significant difference that was found between test frequencies could be attributed to the fact that there was more reduction of DP amplitude and SNR's in the higher frequency region (i.e., 11.2 kHz), whereas the lesser reduction in DP amplitude and DP SNRs resulted in no significant difference between frequencies 9.5 kHz – 8 kHz; 10.3 kHz – 8 kHz; 11.2 kHz – 8 kHz and 10.3 kHz – 9.5 kHz; 11.2 kHz – 9.5 kHz and9.5 kHz – 8 kHz; 10.3 kHz – 8 kHz; 11.2 kHz – 8 kHz and 11.2 kHz – 9.5 kHz. The study done by Negley et al. (2007) concludes that ultra-high frequencies are more prone to damage because of the reduced blood supply and reported that carbon monoxide and nicotine present in the cigarette is also found to reduce oxygen supply to outer hair cells of the cochlea. Other evidence also suggests that the cochlear artery, which supplies the basal region of the

cochlea, is more susceptible to atherosclerotic changes seen in smokers (Zelman, 1973).

There are not many studies that have empirically examined the effect of between test frequencies on DP amplitude and SNR in smokers.

Chapter 6

Summary and Conclusion

Cigarette smoking has become a common trend all over the world, and it has been reported that tobacco is consumed by approximately 1.3 billion of the world's population (Shafey, Dolwick & Guindon, 2003).Tobacco smoking is found to affect the inner ear through several mechanisms either directly or indirectly. Toxic cigarette substances are noted to induce direct oxidative damage mediated by reactive oxygen species and free radicals that are capable of damaging many cellular components such as DNA, protein, and lipids, leading to neurosensory hearing loss that affects mainly the higher frequencies. Indirect damage is mediated by the vasospastic effect and is found to be caused by nicotine, by the acceleration of atherosclerosis in the vascular loops of the auditory system and by the increased levels of carboxyhemoglobin and increased blood viscosity. All of these factors are observed to reduce oxygen perfusion in the organ of corti. Moreover, smoking can act as a risk factor for noise-induced hearing loss since smokers simultaneously exposed to noise in the workplace were affected by a higher degree of hearing loss compared to non-smokers (Zeilinger et al. 2013). Since the literature towards the DPOAE in high frequencies in smoking population is very limited, the current study was planned with an aim of determining the effect of smoking on DPOAE using high frequency test stimulus (8points/octave). The objectives of the study were

1. Comparison of DPOAE amplitude and SNR between smokers and non-smokers.

2. Comparison of DPOAE amplitude across frequency in smokers (8 kHz - 16 kHz) and between test frequencies.
3. Comparison of SNR across frequency in smokers (8 kHz-16 kHz) and between test frequencies.

In order to achieve these objectives of the study, HF DPOAE was recorded in 18non-smokers and 18 smokers whose duration of smoking varied from 1year to 15 years. The DP amplitude and DP SNR values were recorded for all the participants. Descriptive statistics was done to find out the mean and SD of DP amplitude and DP SNR values. Results of descriptive statistics shows that mean DP amplitude and DP SNR values were lesser in smoker group when compared to non-smoker group in all the frequencies tested (8 kHz to 16 kHz). It was also observed that the reduction in DP amplitude and SNR value was noted to be more evident in higher frequencies (starting from above 11.2 kHz) compared to lower frequencies.

Since the data didn't follow the normal distribution, non-parametric tests such as Man Whitney U test, Friedman, Wilcoxon signed rank tests were administered to check for the significant difference in DP amplitude and DP SNR values between smoker and non-smoker group. Results obtained from non-parametric tests reveal that

- There was a significant difference found in DP amplitude between smoker and non-smoker group in all the high frequencies tested.
- There was a significant difference in DP SNR between smoker and non-smoker group except at 8 kHz, 10.3 kHz and11.2 kHz.
- There was no significant ear difference seen in both DP amplitude and DP SNR values.

- There was no significant effect of test frequencies on DP amplitude and DP SNR values in non-smoker group whereas; smoker group showed a significant effect of test frequencies on DP amplitude and DP SNR.
- Between test frequency comparison showed that there was significant difference in DP amplitude in all the frequency pairs compared (above11.2 kHz), except 9.5 kHz – 8 kHz; 10.3 kHz – 8 kHz; 11.2 kHz – 8 kHz and 10.3 kHz – 9.5 kHz; 11.2 kHz – 9.5 kHz.
- Similarly, there was a significant difference in DP SNR in all the frequency pairs compared (above11.2 kHz), except 9.5 kHz - 8 kHz; 10.3 kHz - 8 kHz; 11.2 kHz -8 kHz and 11.2 kHz - 9.5 kHz

Thus it can be concluded that the effect of smoking is seen more at higher frequency DPOAE's as there was reduced DP amplitude and DP SNR in smoker group. This suggests that there is adverse effect of cigarette smoking on higher frequency auditory sensitivity which cannot be seen in the audiogram used in routine audiological test battery. Hence, from the current study it can be concluded that, it's important to include the extended high frequency DPOAE's in routine test battery which can provide better clarity about the occurrence of damage to high frequencies to cochlea more descriptively.

Implications of the study

1. High frequency DPOAE can be used as an additional tool in routine physiological test battery assessment of hearing as it gives clearer picture about adverse effect of smoking on the cochlea.
2. The findings of the study can be used in creating awareness and counseling the general population regarding hazardous effect of smoking on hearing sensitivity.

Future directions

1. To investigate the correlation between different duration of smoking and its effect on HF DPOAE.
2. Longitudinal studies can be done by taking duration of smoking and more number of individuals to investigate the relationship between smokers and nonsmokers.
3. Study can be conducted on females to see the effect of smoking on high frequency DPOAE and its relation with gender.

Limitations of the study

1. Sample size included in the study is less.
2. The study considered only male population and hence gender effect of smoking on high frequency DPOAE could not be studied.
3. Effect of different duration of smoking on high frequency DP OAE was not compared.

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