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Effects of Cigarette Smoking on Distortion Product Otoacoustic Emissions

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Abstract

This study measured distortion product otoacoustic emissions (DPOAEs) and DPOAE input/output (I/O) curves to assess the effects of smoking on cochlear function. Twenty-four healthy adults, 12 smokers and 12 nonsmokers in the 20–30 years age range were selected based on self-reported histories of five to eight years of smoking or no smoking, respectively. All subjects received tympanometric screening to rule out middle ear pathology. Conventional (0.25–8 kHz) and ultra high frequency (UHF; 10–20 kHz) audiometry showed normal or age-appropriate thresholds across both groups. DPOAE results showed small, but significant, decline in DPOAE levels without concomitant changes in noise floors in smokers as compared to nonsmokers. I/O detection thresholds were also significantly elevated at high frequencies in smokers as compared to their nonsmoking counterparts. These findings indicate that smokers are at greater risk for cochlear damage than nonsmokers, and that DPOAE amplitudes and I/O detection thresholds may identify early changes in cochlear function in smokers.

Key Words: distortion product otoacoustic emission amplitudes, input/output detection thresholds, cigarette smoking, cochlear function

Abbreviations: DPOAE=distortion product otoacoustic emissions; F=F ratio; I/O=input output; UHF=ultra high frequency

Sumario

Este estudio midió las emisiones otoacústicas por productos de distorsión (DPOAES) y las curvas de ingreso/salida (I/O) de las DPOAE para evaluar los efectos del fumado sobre la función coclear. Veinticuatro adultos, 12 fumadores y 12 no fumadores, en el rango de edad de 20 a 30 años, fueron seleccionados con base en sus historia auto-reportadas de fumado o no fumado en los últimos 5–8 años, respectivamente. Todos los sujetos se sometieron a un tamizaje timpanométrico para descartar patología del oído medio. La audiometría convencional (0.25–8 kHz) y la de ultra-alta frecuencia (UHF, 10–20 kHz) mostraron umbrales normales o apropiados para la edad en ambos grupos. Los resultados de las DPOAE mostraron una caída pequeña pero significativa en los niveles de las DPOAE en los fumadores comparado con los no fumadores, sin cambios concomitantes en el piso de ruido. Los umbrales I/O de detección también estuvieron significativamente elevados en las altas frecuencias en los fumadores, comparado con sus contrapartes no fumadores. Estos hallazgos indican que los fumadores tienen un mayor riesgo de daño coclear que los no fumadores, y que las amplitudes de las DPOAE y los umbrales I/O de detección pueden identificar cambios tempranos en la función coclear de los fumadores.

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Palabras Clave: emisiones otoacústicas por productos de distorsión; umbrales de detección de ingreso/salida; fumado de cigarrillos, función coclear.

Abreviaturas: DPOAE = emisiones otoacústicas por productos de distorsión; F = tasa F; I/O = ingreso/salida; UHF = ultra alta frecuencia

In 2003 the Centers for Disease Control and Prevention estimated that 24.1% of men and 19.2% of women in the United States were cigarette smokers (American Heart Association, 2006), in spite of the well-documented health risks associated with cigarette smoking. Risk for hearing impairment associated with cigarette smoking has also been reported by several studies over the past 30 years. Most of these studies have evaluated correlations between smoking and hearing loss. An epidemiological study conducted by Cruickshanks et al. (1998) showed that the prevalence of hearing loss in current smokers increased from 26% in the youngest age group of 48-59 years to 56% and 71% in the 60-69 and 70-79 year range categories, respectively. In each of these age groups, the prevalence of hearing loss in nonsmokers and ex-smokers was substantially lower ($p < 0.001$). Moreover, as pack years of cigarette smoking (1 pack year=20 cigarettes/day for 1 year) increased from 0 to >40, the prevalence of hearing loss increased from 17% to 29% in the 48-59 years age range, from 36% to 59% in the 60-69 years age group, and from 60% to 74% in the 70-79 year old participants ($p < 0.001$). When the effects of age, gender, education, occupational noise exposure, cardiovascular disease and alcohol consumption were adjusted, current cigarette smokers were gauged to be 1.7 times as likely as nonsmokers to have hearing loss. Thus, cigarette smoking appears to pose a significant risk for hearing impairment.

Similar prevalence rates have been documented by other studies; for example, Sharabi et al. (2002) showed that current and past smokers were approximately twice more likely to have mild conductive and sensorineural hearing losses as compared to nonsmokers. Furthermore, cigarette smoking increased the risk for hearing loss by 43% in younger subjects under the age of 35 years as compared to 17% in subjects over 35 years of age. Likewise, Nakanishi et al. (2000) showed that current smokers (30-59 years old) were at greater risk for high frequency hearing loss (>40 dB HL at 4 kHz) than low frequency loss (>30 dB HL at 1 kHz), as compared to those subjects that never smoked or were ex-smokers, when risk assessment was adjusted for age, body mass index, alcohol consumption, mean blood pressure, serum total cholesterol, high-density lipoprotein cholesterol, triglyceride level, fasting glucose level and hemocrit. When subjects were further categorized into groups based on numbers of cigarettes smoked per day and pack years of smoking, the risk for high frequency hearing loss increased in a dose dependent manner, whereas the risk for low frequency hearing loss remained unchanged.

Although cigarette smoking appears to affect high frequency hearing, there is only one study that reported the effects of smoking on high frequencies over 8 kHz. Cunningham et al. (1983) measured extra- or ultra-high frequency hearing (UHF; 9-20 kHz) along with pure tone thresholds in the conventional frequency range of 0.25-8 kHz in smokers and nonsmokers between 20 and 35 years of age. All subjects in both groups showed hearing thresholds of 15 dB HL or better in the 0.5-8 kHz range, normal otoscopic examinations, no history of ear disease, significant noise exposure, familial hearing loss, or cardiovascular diseases. Smokers had smoked at least one pack of cigarettes per day for at least 6 years, and nonsmokers had never ever been smokers. The results of UHF hearing

measurements showed consistently greater hearing thresholds in smokers as compared to nonsmokers. However, smokers also tended to show greater variability in UHF thresholds as compared to nonsmokers, so that results were statistically significant at only 9 kHz.

Recent research related to occupational noise exposure has also evaluated the joint effects of noise exposure, smoking, and age on hearing loss (Siegelaub et al., 1974; Barone et al., 1987; Virokannas and Anttonen, 1995; Starck et al., 1999; Toppila et al., 2001; Mizoue et al., 2003; Palmer et al., 2004; Ferrite and Santana, 2005; Nomura et al., 2005; Uchida et al., 2005). The results of these studies indicate that smoking, age, and noise exposure together produce a greater risk for hearing impairment than smoking or age or noise exposure alone.

Other studies that examined the effects of smoking among various age groups have shown that smoking produces more adverse effects on hearing in older than in younger adults. For example, Noorhassim and Rampal (1998) showed that pack years of smoking and age served as significant risk factors for hearing impairment. The prevalence rates of hearing impairment for nonsmokers and smokers under 40 years old were 6.9% and 11.9%, respectively, and for nonsmokers and smokers over 40 years of age were 29.7% and 51.3%, respectively. These investigators calculated prevalence rate ratios and showed a multiplicative effect of smoking and aging on hearing impairment.

Several causes for the relationship between smoking and hearing have been proposed. A primary suspected mechanism of hearing impairment associated with smoking is vascular insufficiency of the cochlear end organ (Zelman, 1973). Smoking may reduce blood supply to the cochlea by nicotine-induced vasospasm, atherosclerotic narrowing, and/or thrombotic occlusion of blood vessels (Zelman, 1973; Cunningham et al, 1983; Cruickshanks et al., 1998; Nakanishi et al., 2000). Increased blood viscosity and accompanying hypoxia may also explain the reduced oxygen supply to the cochlea (Maffei and Miani, 1962; Lowe et al., 1980; Browning et al, 1986). Howard et al. (1998) showed that atherosclerotic damage was more prevalent in smokers than nonsmokers and that an increase in the number of pack years of smoking increased the progression of atherosclerotic damage. Thus, atherosclerosis-induced oxygen deprivation may not only affect the cochlear end organ, but also the accompanying spiral ganglion cells.

These mechanisms of hearing damage indicate that the cochlea may be very vulnerable to the effects of smoking and that loss or reduction of blood supply to the cochlea may be the primary mechanism of hearing damage. However, there is no literature on the effects of smoking on cochlear function. In this study we measured DPOAE amplitudes and DPOAE I/O detection thresholds at high frequencies in a group of young smokers and nonsmokers to assess the effects of smoking on cochlear function.

METHODS

Subjects

Twenty-four healthy adults, 12 smokers and 12 nonsmokers (7 females and 5 males/group) in the 20-30 years age range were used in this study. Subjects were included in the study based on self-reported history of smoking status; subjects with a history of 5-8 years of smoking, regardless of the number/packs of cigarettes smoked per day made up the "smokers" group, whereas those with no history of smoking formed the "nonsmokers" group. None of the subjects in either group reported a history of noise exposure or ear diseases. These subjects were recruited from the Western Michigan University (WMU) student community via study announcements posted at approved university locations. Approval from the WMU Human Subjects Institutional Review Board was obtained to conduct this study. Informed consents were obtained from recruited subjects before hearing tests were conducted.

Procedures

Hearing evaluations were performed in the Department of Speech Pathology and Audiology of the College of Health and

Human Services. Tympanometric screening was performed with a Grason Stadler (GSI-38) screening tympanometer to rule out middle ear problems. Those subjects that did not show normal middle ear systems bilaterally were excluded from the study. Selected subjects received a short questionnaire regarding their smoking and hearing health history. Conventional and UHF pure tone sensitivity was determined in a sound-treated room with the Grason Stadler (GSI-61) diagnostic audiometer equipped with TDH-50P and Senheiser HD200 earphones. DPOAEs were measured with an ER-10C probe sealed in the outer ear canal with a disposable foam plug using the Mimosa Acoustics CUBeDIS system (version 5.21).

DPOAE amplitudes were measured from DP-grams plotted by sweeping pairs of pure tones, f1 and f2, at 70 dB SPL (L1=L2) and at 65 and 50 dB SPL (L1-L2=15 dB), at an f2/f1 ratio of 1.2 and a frequency range of 2-8 kHz (3 points/octave, in a high to low frequency descending order). DP-grams were replicated, and the averaged responses were used for data analyses. DPOAE I/O functions were measured as described in Katbamna et al. (1999). Briefly, I/O curves were obtained at f2 frequencies of 2, 4, and 8 kHz using equilevel stimuli between 20 and 80 dB SPL in 10 dB increment steps and an f2/f1 ratio of 1.2. The termination point for averaging at each stimulus level was determined by either the maximum allowable noise floor, which was set to -10 dB SPL, or number of time-locked averages that was set to 100. I/O detection thresholds were obtained for each f2 by determining the intensity level at which the DPOAE amplitude exceeded the noise floor by 6 dB (Kimberley et al., 1997). Since both ears of all subjects were tested, the order of the test ear, i.e., right versus left ear, was determined randomly.

Statistical Analyses

All statistical analyses were performed with the BMDP statistical software (BMDP Statistical Software, Inc, version 7.0, Los Angeles, CA). In the analyses described below, gender effects were not analyzed since the gender distribution was matched subject-for-subject and due to a modest number of subject enrollment in the study. Moreover, earlier studies have shown no significant effects of gender on DPOAE amplitudes (Lonsbury-Martin et al., 1997).

Results of immittance measurements were analyzed with a three-factor analysis of variance (ANOVA), where the between factor was subject groups (smokers vs. nonsmokers) and the two repeated measures were ear side (right vs. left ear) and immittance parameters (ear canal volume, peak admittance, and peak pressure). The hearing threshold data were analyzed with a three-factor ANOVA, i.e., subject group x ear side x hearing level as function of test frequency (thresholds in the 0.25-8 kHz or 10-20 kHz ranges), with repeated measures on the latter two factors. Separate ANOVAs were performed for conventional and UHF hearing test results. For DPOAE amplitude analyses, three separate three-factor ANOVAs (i.e., subject group x ear side x test frequency) were used to measure differences in the signal to noise ratios, the absolute amplitudes of DPOAEs, and the corresponding noise floors. DPOAE I/O detection thresholds were analyzed with a three-factor ANOVA (subject group x test frequency x ear side) with repeated measures on the test frequency and ear side factors. In all analyses, results were considered to be significant if p values were < 0.05, unless otherwise indicated.

RESULTS

Immittance Measurements

Since tympanometric screening was performed to rule out middle ear problems, all subjects were expected to show normal middle ear function bilaterally. Moreover, there were no significant group or ear differences in any of the measured immittance parameters. The repeated measure that analyzed the effect of immittance parameters was expected to be significant ($F=20.04$, $df=2$, $p=0.0001$), since each parameter, i.e., ear canal volume, peak admittance, and peak pressure, represents a different measure within the same tympanogram.

Pure Tone Audiometry

Results of pure tone audiometry showed normal hearing thresholds (<25 dB HL) in the conventional frequency range (0.25-8 kHz; Fig. 1A) and age appropriate thresholds in the UHF region (10-20 kHz; Fig. 1B) across all subjects. In the conventional frequency range, however, the mean hearing thresholds of smokers were consistently poorer than their nonsmoker counterparts; these differences ranged from 2-10 dB as a function of frequency and were maximal at approximately 10 dB at 6 kHz. These differences in hearing profiles of smokers as compared to nonsmokers produced a statistically significant group difference for the three-factor ANOVA ($F=10.91$, $df=1$, $p=0.0032$) for the conventional audiometry results. Furthermore, as seen in Figure 1A, the mean hearing thresholds varied from one test frequency to the next, regardless of subject group, making the effects of test frequency statistically significant ($F=8.95$, $df=7$, $p=0.0001$).

The results of UHF audiometry showed no significant group differences but significant ear differences ($F=29.72$, $df=1$, $p=0.0001$), due to consistently greater thresholds in the left ear as compared to the right ear in both subject groups (Fig. 1B). The three-factor ANOVA also showed significant effects of frequency ($F=319.78$, $df=6$, $p=0.0001$) due to variation in thresholds from one test frequency to the next test frequency and a significant ear side x test frequency interaction ($F=5.38$, $df=6$, $p=0.0001$) due to differences in hearing thresholds across ears as a function of test frequency. Figure 1B displays these effects, i.e., as frequency increases from 10 kHz to 20 kHz, thresholds also increased, whereas ear differences appeared to be variable across frequencies, e.g., interear differences were low in the 10-14 kHz range and at 20 kHz but rather large at 16 and 18 kHz regardless of the subject group.

DPOAE Measurements

DP-grams obtained at high (L1=L2=70 dB SPL) and moderate (L1=65 dB SPL and L2=50 dB SPL) intensity levels for both groups are shown in Figs 2A and 2B, respectively. These figures indicate that mean DPOAE amplitudes measured in smokers are much lower than those measured in nonsmokers across all frequencies. These findings were corroborated by the results of three-factor ANOVAs that showed significant group differences at both high ($F=6.19$, $df=1$, $p=0.0209$) and moderate ($F=9.53$, $df=1$, $p=0.0054$) intensity levels. Although right ear DPOAE

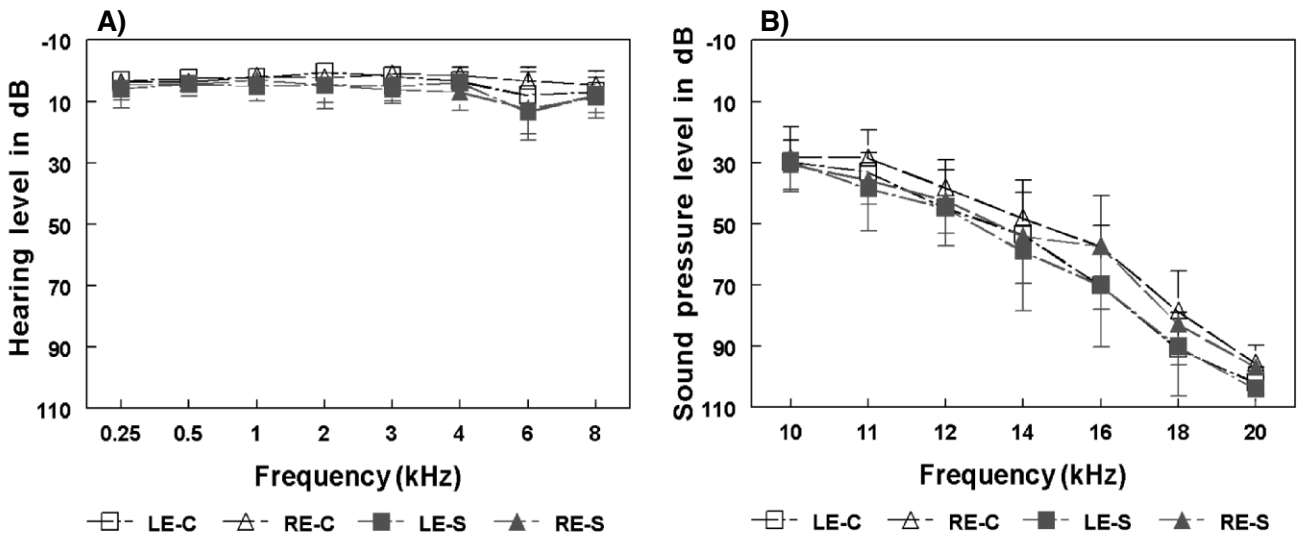

Figure 1. Mean hearing thresholds (standard deviation error bar plotted up for the control group and down for smokers) showing consistently greater thresholds for smokers regardless of test ear (LE-S: left ear-smokers; RE-S: right ear smokers) as compared to the control group of nonsmokers (LE-C: left ear-control; RE-C: right ear-control) in the conventional frequency range (A) and interweaving hearing thresholds for both groups in the UHF region (B). UHF thresholds also show significant ear differences (left ear > right ear) regardless of subject group and interear differences that vary as a function of frequency in both subject groups.

amplitudes were somewhat lower than those measured in left ear at both intensity levels, no significant ear differences were delineated. ANOVAs for both high and moderate level DPOAE measurements also showed significant effects of frequency, since DPOAE amplitudes varied as a function of frequency (high level: F=21.87, df=6, p=0.0001; moderate level: F=24.23, df=6, p=0.0001). In addition, for moderate level measurements, a significant ear side x frequency interaction (F=4.63, df=6, p=0.0003) was measured. This interaction effect is evident in Figure 2B, which shows changing interear differences across the test frequencies, with no differences at 2 and 8 kHz, a maximum difference of 9 dB at 4 kHz, and 3-5 dB differences at the remaining frequencies.

Analyses of noise floor levels at both high and moderate intensity levels showed no significant group differences but significant changes in noise floor across the frequency range (high level: F=67.46, df=6, p=0.0001; moderate level: F=41.74, df=6, p=0.0001). Since noise floors are not identical at each test frequency and each test ear, nor across both subject groups, a significant ear side x frequency interaction (high level: F=2.13, df=6, p=0.0536) and/or a significant ear side x frequency x group interaction (moderate level: F=2.71, df=6, p=0.0164) were expected. These interactive effects are visible in the DP-grams plotted in Figs 2A and 2B respectively.

Based on the above findings of significant group differences in DPOAE amplitudes, but not in noise floor measurements, the signal-to-noise ratios were expected to show significant group differences at both intensity levels (high level: F=6.19, df=1, p=0.0209; moderate level: F=4.60, df=1, p=0.0433). Moreover, significant effects of test frequency (high level: F=21.87, df=6, p=0.0001; moderate level: F=24.86, df=6, p=0.0001) and an ear side x frequency interaction at one or both levels were expected (moderate level: F=2.41, df=6, p=0.0302).

Mean I/O detection thresholds measured at f2 frequencies of 2, 4, and 8 kHz are displayed in Fig 3. This figure shows significantly elevated thresholds in smokers as compared to nonsmokers regardless of test frequency, although interear differences are apparent in the mean data of smokers at 4 kHz and of control subjects at 8 kHz. Thus, although smokers showed a 5 dB interear difference at 4 kHz and nonsmokers showed a 10 dB interear difference at 8 kHz, overall, I/O detection thresholds were greater in smokers by 10 dB at 2 kHz, 8 dB at 4 kHz, and 5 dB at 8 kHz, as compared to the control group. These noticeable differences were substantiated by the three-factor ANOVA that showed significant group differences (F=9.50, df=1,

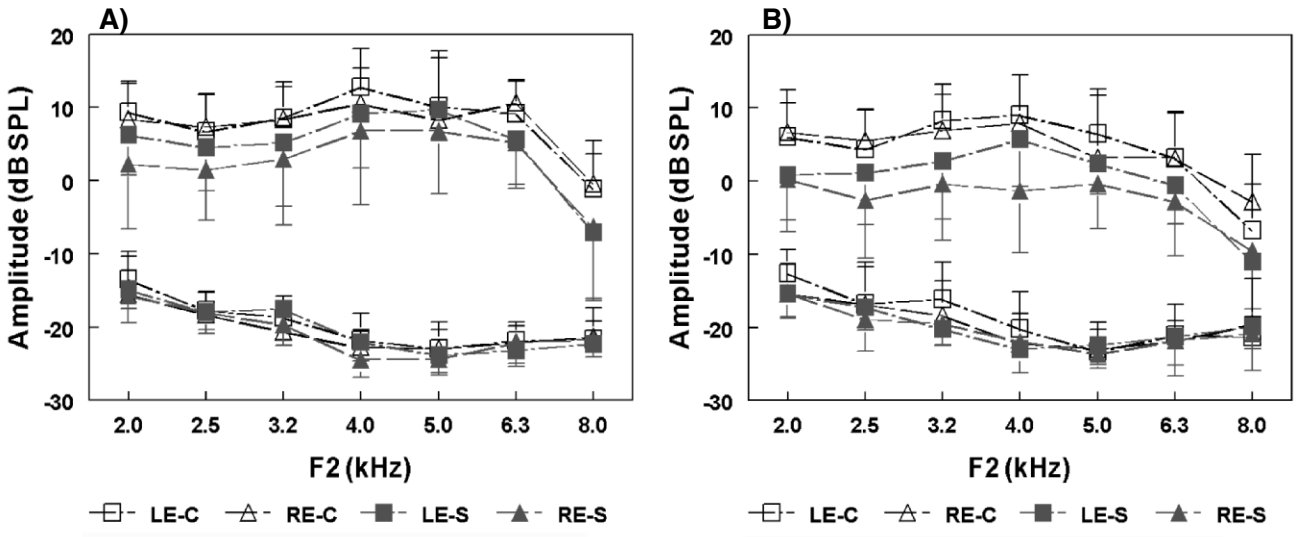

Figure 2. Average DP-grams (standard deviation error bar plotted up for the control group and down for smokers) showing significantly reduced DPOAE amplitudes in smokers (LE-S: left ear-smokers; RE-S: right ear-smokers) as compared to the control group (LE-C: left ear-control; RE-C: right ear-control) at high (A: L1=L2=70 dB SPL) and moderate (B: L1=65 and L2=50 dB SPL) intensity levels, but similar noise floor measurements at both intensity levels.

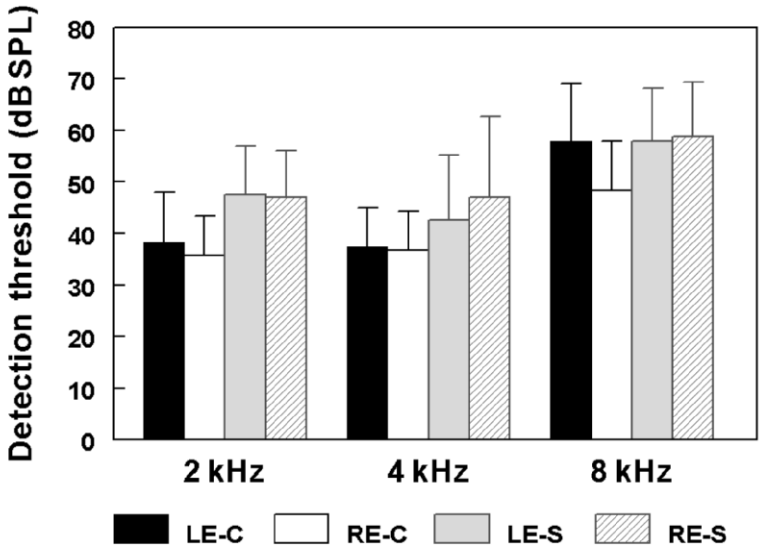

Figure 3. Mean DPOAE I/O detection thresholds (+1SD) showing significantly elevated thresholds at all frequencies in smokers (LE-S: left ear-smokers; RE-S: right ear-smokers) as compared to control nonsmoking subjects (LE-C: left ear-control; RE-C: right ear-control) and distinct interear differences for smokers at 4 kHz and nonsmokers at 8 kHz, explaining the significant ear side x frequency interaction.

p=0.0054) and a significant ear side x group interaction (F=4.43, df=1, p=0.0470). The main effect of test frequency was also significant (F=26.20, df=2, p=0.0001), since the thresholds at 8 kHz were much higher than those obtained at 2 and 4 kHz.

DISCUSSION AND CONCLUSIONS

The major finding of the present study was that cigarette smoking reduced DPOAE amplitudes and increased DPOAE I/O detection thresholds in young persons (20-30 years old), who smoked for 5-8 years, regardless of the admitted numbers/packs of smoked cigarettes. Since these findings were obtained in individuals, who showed normal hearing sensitivity in the conventional (0.25-8 kHz) and UHF (10-20 kHz) range, the results indicate that the effects of cigarette smoking on hearing in young individuals may be first observed in DPOAE measurements and that DPOAEs may provide a sensitive index of cochlear integrity in individuals who smoke. Although DPOAE amplitude reductions measured in smokers were small, in the order of 1-6 dB at high intensity levels (L1=L2=70 dB SPL) and 3-9 dB at moderate intensity levels (L1=65 and L2=50 dB SPL), these results occurred without concomitant changes in noise floors as compared to those measured in nonsmokers and were statistically significant in spite of a modest group size (n=12/group). Thus, these alterations may be attributed to true changes in cochlear function.

Several studies have shown that effects of smoking on hearing may be related to vascular insufficiency of the cochlear end organ. For example, nicotine-induced vasospasm, atherosclerotic narrowing, and/or thrombotic occlusion of blood vessels may reduce blood supply to the cochlea (Zelman, 1973; Cunningham et al., 1983; Cruickshanks et al., 1998; Nakanishi et al., 2000). Howard et al. (1998) showed that smokers were most susceptible to atherosclerotic damage and that as the numbers of pack years of smoking increased, atherosclerotic damage also increased, so that oxygen deprivation may affect both the cochlear hair cells and the spiral ganglion cells. Cigarette smoking has also been shown to increase carbon monoxide in the blood supply (Cunningham et al., 1983) and to increase blood viscosity (Maffei and Miani, 1962; Lowe et al., 1980; Browning et al., 1986). Such changes in the blood supply to the cochlea have been speculated to lead to atherosclerosis and accompanying hypoxic damage to the cochlea (Cunningham et al., 1983). Since

DPOAEs originate from outer hair cells and outer hair cell function is extremely sensitive to changes in blood and oxygen supply, anoxic insults are expected to influence DPOAEs (Whitehead et al., 1992; Frolenkov et al., 1998). Clinical observations of reduced emission amplitudes associated with anoxic insults have been reported previously. For example, lethal anoxia and acute ototoxic drug damage have been shown to produce declines in DPOAE levels (e.g., Whitehead et al., 1992; Frolenkov et al., 1998). Thus, reduced DPOAE amplitudes in smokers may be attributable to chronic hypoxic insult to the cochlea associated with vascular insufficiency in cigarette smokers.

In addition to DPOAE amplitude reductions, smokers also showed significant elevations of DPOAE I/O detection thresholds at high frequencies when compared to their nonsmoking counterparts. Since DPOAEs measured at low stimulus intensity levels reflect the status of the cochlear amplifier (Kemp, 1978), various studies have used DPOAE growth (I/O) functions to assess cochlear metabolic dysfunction. For example, there is now some evidence that age-related hearing loss is a result of deterioration of the stria vascularis and accompanying decline in endocochlear potential (Gratton et al., 1996), and these changes are correlated with reduced output of the cochlear amplifier as measured by DPOAE growth functions (Gates et al., 2002). Thus, age-related hearing loss may show changes in the stria vascularis, without concomitant loss of outer hair cells (Tarnowski et al., 1991). In fact, aged animals raised in quiet have been shown to display the characteristics of metabolic presbyacusis, leading to the hypothesis that hearing loss associated solely with aging may be a direct consequence of an energy starved cochlear amplifier (Schmiedt et al., 2002). Since the effects of cigarette smoking have been correlated to vascular insufficiency, depletion of oxygen to the highly vascularized structure of the cochlea, the stria vascularis, may produce metabolic dysfunction. DPOAE I/O threshold elevations seen in smokers may reflect such changes in the cochlear amplifier.

As stated above, this study also measured pure tone thresholds at conventional and UHFs. Although all subjects in both groups showed normal hearing sensitivity in the conventional range, the mean hearing thresholds for smokers at all frequencies were greater by 2-10 dB as compared to those obtained for nonsmokers, so that these differences were statistically significant. Other researchers, however, have shown significant correlations between hearing loss in the conventional frequency range and smoking. Cruickshanks et al. (1998) showed that the prevalence of hearing loss (pure tone average of 0.5, 1, 2, and 4 kHz greater that 25 dB HL) in smokers increased from 26% in young subjects aged 48-59 years to 56% and 71% in the 60-69 and 70-79 years age range, respectively. Moreover, as pack years of cigarette smoking increased from 0 to > 40, prevalence of hearing loss increased further by 12%, 23%, and 13% in these three age categories respectively. The combined effects of age and smoking have also been documented by other studies, which have shown differences in the prevalence of hearing loss in smokers and nonsmokers with increasing age. Sharabi et al. (2002) showed that current and past smokers were twice as likely to have conductive (bone conduction > 25 dB HL) and sensorineural (air conduction > 25 dB HL) hearing losses compared to nonsmokers, and that younger individuals (< 35 years) were at a greater risk (43%) than individuals over 35 years of age (17%). Likewise, Noorhassim and Rampal (1998) showed that the prevalence of hearing loss in smokers increased by 39% when individuals under 40 years were compared to those over 40 years, whereas similar comparisons in nonsmokers showed an increase of prevalence by only 23%. Since the present study was conducted on much younger subjects (20-30 years age range) and regardless of the numbers of cigarettes or pack years of smoking, the outcomes of the study are not directly comparable to those reported in the prior literature. Furthermore, since the objective of the study was to measure changes in DPOAE profiles, middle ear pathology was ruled out before subject enrollment in the study. Thus, the results of the present study provide

information on the cochlear status of young smokers, rather than the risks for or prevalence of hearing loss.

Although cigarette smoking has been shown to affect high frequencies in the 0.25-8 kHz range (Cruickshanks et al., 1998; Nakanishi et al., 2000; Sharabi et al., 2002) and there is evidence that the cochlear artery that terminates in the high frequency region of the cochlea is very susceptible to the effects of atherosclerotic changes, which are also seen in smokers (Zelman, 1973), only one previous study investigated the effects of smoking on UHFs. Cunningham et al. (1983) showed that UHF hearing was consistently poorer in smokers as compared to nonsmokers, though the results were not statistically significant. The results of the present study also showed no significant differences in UHF hearing of smokers and nonsmokers. However, both subject groups showed significant ear differences where the left ear thresholds were poorer by 2-13 dB as compared to the right ear thresholds, with the greatest difference being 13 dB at 16 kHz. These differences may be explained at least in part by the small sample size and variability of UHF thresholds measured in this study. Results of UHF conducted by Cunningham et al. (1983) also showed substantial variability in the results, although smokers tended to show greater standard deviations than nonsmokers.

In summary, the outcomes of this study indicate that young smokers in the 20-30 years age range who have smoked cigarettes for 5-8 years may be at a greater risk for cochlear damage than nonsmokers and that DPOAE amplitudes and I/O detection thresholds may be useful in detecting early changes in cochlear function in cigarette smokers. These results, however, were based on a modest number of subjects and need further substantiation in a larger pool of young cigarette smokers.

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