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Özgün araştırma

Sigara Kullanımının İşitmeye Etkisi: Buz Dağının Görünmeyen Kısmı

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Öz

Amaç Sigara içmenin işitme üzerindeki potansiyel etkisine ilişkin önceki çalışmalarda çelişkili sonuçlar elde edilmiştir. Çelişkili sonuçların bir nedeni olarak genişletilmiş yüksek frekanslı işitme eşiklerini değerlendiren çalışma sayısının sınırlı olması gösterilebilir. Bu nedenle, bu çalışma, genişletilmiş yüksek frekanslı işitme eşikleri kullanarak sigara içmenin işitme üzerindeki potansiyel etkisini değerlendirmeyi amaçlamaktadır.
Gereç ve Yöntem Bu çalışmaya 20-30 yaşları arasında normal işiten (0.125-8 kHz işitme eşiği ≤ 15 dB HL) toplam 80 katılımcı dahil edildi. Tüm katılımcıların her iki kulağındaki genişletilmiş yüksek frekanslı işitme eşikleri 9-20 kHz aralığında değerlendirildi. Sigara içenlerin sigara tüketimi Brinkman indeksine göre belirlendi.
Bulgular Saf ses işitme eşikleri açısından çalışma ve kontrol grupları arasında istatistiksel olarak anlamlı bir fark elde edilmiştir. Bu fark sağ kulakta 9 kHz, sol kulakta 10 kHz'den sonra istatistiksel olarak anlamlı düzeyde artmaya başlamıştır. Ayrıca, Brinkman İndeksi ile genişletilmiş yüksek frekans işitme eşikleri arasında istatistiksel olarak anlamlı düzeyde yüksek derecede pozitif bir korelasyon elde edilmiştir.
Sonuç Sonuçlar sigara içmenin işitme üzerinde, özellikle yüksek frekanslı işitme eşikleri üzerinde potansiyel bir etkisi olduğunu düşündürmektedir. Ayrıca bu çalışma sigara kullanımı durumunda saf ses işitme eşiklerinin hangi frekanstan itibaren etkilendiğini gösteren ilk çalışmadır. Klinisyenler, işitmeyi etkileyebilecek olası durumları gözden kaçırmamak için rutin odyolojik değerlendirmede kullanılan frekans aralığı ile sınırlı kalmayıp genişletilmiş yüksek frekans işitme eşiklerini ihmal etmemelidir.

Anahtar kelimeler: Sigara kullanımı; İşitme kaybı; İşitme sağlığı; Genişletilmiş yüksek frekans işitme eşikleri; Brinkman indeksi

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Original Research

The Effect of Smoking on Hearing: The Invisible Part of the Iceberg

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Abstract

Objectives Previous studies have had conflicting results regarding the potential effect of smoking on hearing. One reason for the conflicting results may be the limited number of studies evaluating extended high-frequency hearing thresholds among these studies. Therefore, this study aims to evaluate the potential effect of smoking on hearing using extended high-frequency hearing thresholds.
Materials and Methods A total of 80 participants aged 20-30 with normal hearing (0.125-8 kHz hearing thresholds ≤ 15 dB HL) were included in this study. Extended high-frequency hearing thresholds in each ear of all participants were evaluated in the range of 9-20 kHz. The cigarette consumption of smokers was determined according to the Brinkman Index.
Results There was a significant difference between the study and control groups in pure-tone hearing thresholds. This difference started to increase after 9 kHz in the right ear and 10 kHz in the left ear. Also, there was a highly positive correlation between Brinkman Index and hearing thresholds at all extended high-frequencies.
Conclusion The present study suggests that smoking has a potential effect on hearing, especially on high-frequency hearing thresholds. Also, this is the first study to show from which frequency pure tone hearing thresholds are affected in smoking. Clinicians should not be limited to the frequency range used in the routine audiological evaluation and should not ignore extended high-frequency hearing thresholds not to overlook possible conditions that may affect hearing.

Keywords Smoking; Hearing loss; Hearing health; Extended high-frequency hearing; Brinkman Index

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Introduction

Sense of hearing is the most important factor that provides opportunities for individuals' social communication. Conditions such as ageing, exposure to noise, and use of ototoxic drugs can cause hearing loss. In particular, noise exposure is one of the most important environmental factors that can cause hearing loss (Hong et al., 2013). However, smoking can lead to life-threatening chronic diseases and the risk of hearing loss (Ohgami et al., 2011).

There are discussions in the literature on the mechanism of the effect of smoking on the inner ear. Previous studies showed that nicotine directly affects the inner ear (Harkrider et al., 2001). In addition, different studies stated that the level of carboxyhemoglobin is high in smokers. Therefore, the oxygen level that the organ of Corti can use may decrease (Sung et al., 2013). In another study, they showed that the inner ear blood perfusion of participants exposed to high noise and smoked at the same time could decrease more due to tobacco. As a consequence, hearing loss may occur because the amount of oxygen required for cellular metabolism is not sufficient (Raub & Benignus, 2002; Tavanai & Mohammadkhani, 2017). In another study, they suggested that diastolic blood pressure increased in smokers. As a result, arterial hypertension could also affect the organ of Corti (Figueiredo et al., 2015; Mosnier et al., 2001).

There are differences in the results of studies on the effect of smoking on hearing. Ohgami et al. compared 14 smokers and 37 non-smokers with 1, 4, 8 and 12 kHz pure-tone hearing thresholds (PTTs) (Ohgami et al., 2011). As a result of the study, there was no difference between the 1, 4 and 8 kHz PTTs between the two groups. In comparison, the 12 kHz PTTs were higher in the smoking group. Cunningham et al. compared 18 smokers and 25 non-smokers using extended high-frequency hearing thresholds (EHFTs) between 8 and 18 kHz. As a result of the study, the PTTs of the smoking group were higher than the control group. However, this difference in hearing thresholds was not statistically significant (Cunningham et al., 1983). Oliveira et al. compared 30 smokers and 30 non-smokers using PTTs between 0.25 and 18 kHz. As a result of the study, the PTTs of the smoking group at all frequencies were statistically significantly higher (de Oliveira, 2009). On the other hand, Pearson et al. evaluated the effects of risk factors for hearing loss, such as age, blood pressure, smoking, and alcohol use, on hearing thresholds in participants using PTTs between 0.5 and 8 kHz. They could not find any factor that would cause a statistically significant increase in hearing thresholds except for blood pressure (Pearson & Morrell, 1996). Also, Karlsmose et al., in their study evaluating the hearing thresholds of the participants between 0.25 and 8 kHz, could not find a relationship between smoking and hearing loss (Karlsmose et al., 2000).

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In previous studies, it is seen that while some of the researchers consider the high-frequency hearing thresholds in smokers, some ignore this and investigate the effect of smoking on hearing. Therefore, conflicting results have emerged regarding the potential effect of smoking on hearing. However, it is thought that factors such as smoking, which affect hearing, cause metabolic problems in the body and affect extra high-frequency hearing thresholds more than the hearing thresholds used in routine audiometry frequencies (Lasso de la Vega et al., 2016; Makishima, 1978; Manju, 2014; Thottan, 2019) Therefore, this study aims to evaluate the potential effect of smoking on hearing with EHFTs in participants who do not have a history of hearing loss, such as noise exposure and ototoxic drug use.

Material and Methods

Ethical approval of this study was obtained from the Non-Interventional Clinical Research Ethics Committee (GO20/835) and was completed in accordance with the standards set by the Declaration of Helsinki. The present research's type is a case-control study.

Participants

The study and control groups were formed from individuals who applied to the announcement created for the study on the university campus, which met the criteria for participation in the study. A total of 65 participants aged 20-30 with normal hearing (0.125-8 kHz hearing thresholds ≤ 15 dB HL) were included in this study. The study group consisted of 37 (21 Males, 16 Females) participants with a mean age of 24.9 ± 3.35 who smoked for one year or longer. The control group consisted of 43 non-smokers (22 Females, 21 Males) with a mean age of 24.7 ± 4.9 years. It was considered that there were no conditions (noise exposure and history of ototoxic drug use) that could cause differences in hearing thresholds between the groups for all participants. The function and appearance of all participants' external and middle ears were examined by otoscopy and tympanometry and defined as normal. The cigarette consumption of smokers was determined according to the Brinkman Index (BI). This index was obtained by multiplying the number of cigarettes consumed per day by the participants and the number of years they smoked. All assessments were applied alternately, from a study group to a control group. Individuals who agreed to participate in the study were informed about the purpose of the study, and their written consent was obtained.

Pure Tone Audiometry

Pure tone audiometry was performed in a soundproof room with Interacoustics AC-40 audiometer and calibrated TDH-39P (for 0.125-8 kHz) earphones, Sennheiser HDA200 (for 9-20 kHz), and Radioear B-71 bone vibrator. The participants were given a button to put on

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appropriate headphones. Participants were asked to respond to the sounds they heard through the headphones by pressing the button. The descending method was used when determining hearing thresholds. First, it was confirmed that the participants' air conduction hearing thresholds of 0.125-8 kHz were lower than 15 dB HL. Afterwards, it was confirmed that there was no difference of more than 10 dB hl between the bone conduction hearing thresholds and the air conduction hearing thresholds. After individuals met the study criteria, EHFTs were evaluated at 9, 10, 11.2, 12.5, 14, 16, 18, and 20 kHz, respectively.

Statistical Analysis

The sample size of the study was determined by the G*Power program. It should include a minimum of 15 participants from each group with a 5% type I error level and 95% power to detect a clinically significant difference, according to the power analysis. Data were evaluated with SPSS version 24.0 (IBM Inc., Armonk, NY, USA). In comparing two numerical independent data groups, to decide which non-parametric or parametric tests will be used, whether the data is normally distributed or not was evaluated by visual (Histogram, Detrended Plot) and analytical methods (Skewness/Kurtosis, Shapiro-Wilk, Coefficient of Variation). Descriptive analyses of the study data, observed to be normally distributed, were given using the mean and standard deviations. Independent samples t-test was used to compare the hearing thresholds of the study and control groups. The assumption of homogeneity of variance was checked via Levene's test ($p<0.001$). The Repeated Measure ANOVA test was used to compare the differences between the hearing thresholds of the study and control groups according to the frequencies in each ear. The Greenhouse-Geisser correction was used when the sphericity assumption was not met. The relationship between two normally distributed data sets was evaluated with Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

Results

Descriptive Statistics of the Participants

A total of 80 individuals, 38 female and 42 male, were included in our study. The study group consisted of 37 (21 Male, 16 Female) smokers, and the control group consisted of 43 (22 Female, 21 Male) non-smokers. While the mean age of the study group was 23.08 ± 3.16, the mean age of the control group was 24.7 ± 4.9. There was no statistically significant difference between the groups regarding age and gender ($p>0.05$). The BI values of the participants were between 40 and 220, and the average BI value was 116,43. Demographic information and BI values of the study group are shown in Table 1.

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Hearing Thresholds Differences Between Groups

The PTTs (0.125-8 kHz) were less than 15 dB HL. Considering the PTTs in the right ears of the participants, there were significant differences between the study and control groups in terms of 11.2, 12.5, 14, 16, and 20 kHz PTTs ($p<0.05$). No significant difference was found in pure-tone hearing thresholds of 9, 10, and 18 kHz ($p>0.05$). The distribution of the average hearing thresholds for each frequency in the 9-20 kHz range in the participants' right ear according to the groups and the statistical differences between the groups are shown in Table 2.

Considering the PTTs in the left ears, all frequencies' PTTs differ statistically between the study and control groups ($p<0.05$). The distribution of the average hearing thresholds for each frequency in the 9-20 kHz range in the left ear of the participants according to the groups and the statistical differences between the groups are shown in Table 3.

In addition, the average hearing threshold differences between the study and control groups at each frequency for the right ear are shown in Table 4. There was a significant difference between the averages of PTTs differences from 9-10 kHz, 10-11.2 kHz, and 11.2-12.5 kHz ($p<0.01$). There was no significant difference between the averages of PTTs between 12.5 and 20 kHz ($p>0.05$). The frequency ranges where these values differ statistically are shown in Figure 1.

The average hearing threshold differences between the study and control groups at each frequency for the left ear are shown in Table 5. There was a significant difference between the averages of 10-11.2 kHz PTTs ($p<0.01$). There was no significant difference between the averages of PTTs differences between 9-10 kHz, and 11.2- 20 kHz ($p>0.05$). The frequency ranges where these values differ statistically are shown in Figure 2.

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Table 1: Demographic information and BI values of the study group

Participants No	**Sex**	**Age**	**BI**
1	M	21	96
2	F	21	85
3	F	22	90
4	M	26	150
5	M	26	180
6	M	27	140
7	F	20	80
8	F	19	60
9	M	18	72
10	F	28	200
11	M	30	220
12	F	27	180
13	M	25	160
14	F	21	100
15	F	20	60
16	M	19	80
17	M	18	40
18	F	21	48
19	M	26	150
20	M	23	120
21	F	20	51
22	M	21	66
23	F	22	90
24	M	20	100
25	M	23	144
26	M	24	160
27	F	27	190
28	M	21	50
29	F	26	200
30	M	25	120
31	M	22	91
32	F	23	85
33	F	26	180
34	M	25	80
35	M	27	120
36	F	19	144
37	M	25	126

BI: Brinkman Index

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Table 2: Distribution of average hearing thresholds for the right ear in the range of 9-20 kHz by groups

Frequency	Group	N	Mean (dB)	Std.	P-value
9 kHz	Control	43	9,06	9,25	0,82
	Study	37	6,35	9,20	
10 kHz	Control	43	5,18	8,13	0,61
	Study	37	7,06	12,25	
11.2 kHz	Control	43	2,78	10,17	**0,04***
	Study	37	13,24	19,68	
12.5 kHz	Control	43	-1,94	10,16	**<0.01***
	Study	37	17,06	23,78	
14 kHz	Control	43	-1,83	12,15	**0,03***
	Study	37	15,59	18,93	
16 kHz	Control	43	-2,39	15,12	**0,03***
	Study	37	16,47	25,23	
18 kHz	Control	43	-2,22	11,90	0,10
	Study	37	16,24	25,18	
20 kHz	Control	43	-1,94	12,3	**0.01***
	Study	37	17,94	21,8	

Std.: standard deviation

***** Independent samples t-test

Table 3: Distribution of average hearing thresholds for the left ear in the range of 9-20 kHz by groups

Frequency	Group	N	Mean (dB)	Std.	P-value
9 kHz	Control	43	3,06	9,41	**0.01***
	Study	37	11,76	10,74	
10 kHz	Control	43	1,39	9,36	**0,03***
	Study	37	10,88	14,92	
11.2 kHz	Control	43	-1,94	8,59	**<0.01***
	Study	37	17,35	22,78	
12.5 kHz	Control	43	-2,78	10,03	**<0.01***
	Study	37	17,94	22,50	
14 kHz	Control	43	-1,94	12,26	**<0.01***
	Study	37	20,00	24,17	
16 kHz	Control	43	-2,50	15,26	**0,04***
	Study	37	18,24	27,09	
18 kHz	Control	43	-1,18	9,92	**<0.01***
	Study	37	20,59	27,43	
20 kHz	Control	43	0,83	12,27	**<0.01***
	Study	37	24,12	26,23	

Std.: standard deviation

***** Independent samples t-test

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Table 4: The difference values between the study and control group average hearing thresholds according to the frequencies for the right ear

Frequency	N	Mean (S-C)	Std.
9 kHz	37	-3,82	12,18
10 kHz	37	1,47	14,87
11.2 kHz	37	9,70	21,89
12.5 kHz	37	18,52	23,30
14 kHz	37	18,52	29,93
16 kHz	37	19,12	20,70
18 kHz	37	18,23	22,56
20 kHz	37	19,41	18,61

Std.: standard deviation

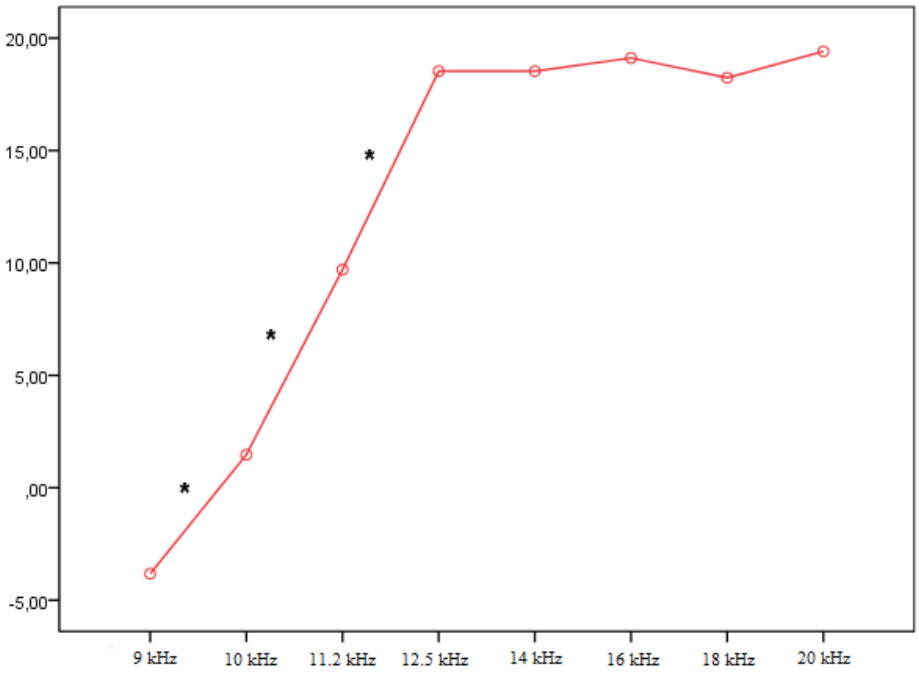

Figure 1: The difference values between the control and study group average hearing thresholds according to the frequencies for the right ear

Table 5: The difference values between the control and study group average hearing thresholds according to the frequencies for the left ear

Frequency	N	Mean (S-C)	Std.
9 kHz	37	9,41	13,90
10 kHz	37	10,00	27,32
11.2 kHz	37	19,11	23,86
12.5 kHz	37	20,58	23,10
14 kHz	37	20,88	24,05
16 kHz	37	22,05	20,16
18 kHz	37	22,94	19,45
20 kHz	37	22,64	21,87

Std.: standard deviation

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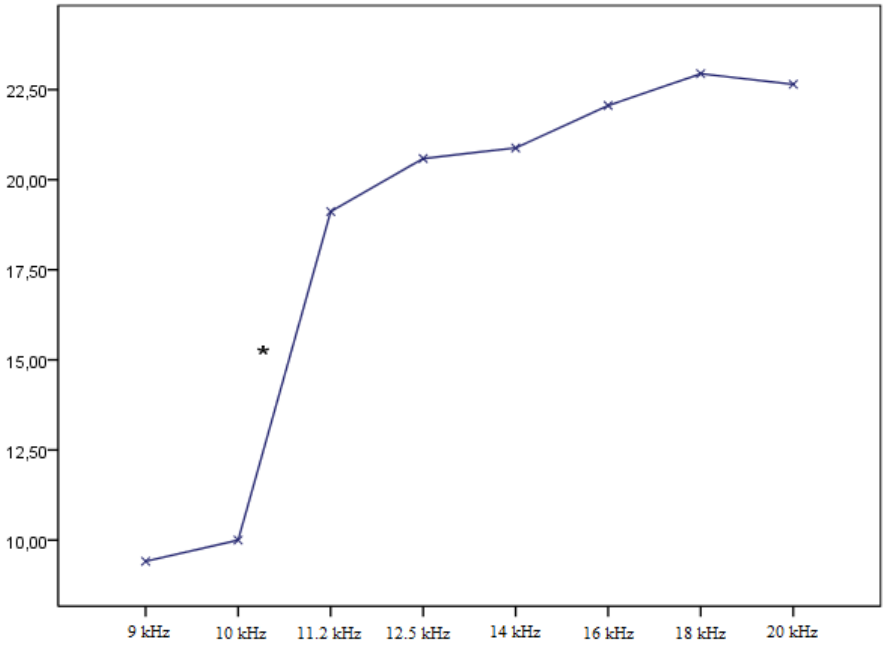

Figure 2: The difference values between the control and study group average hearing thresholds according to the frequencies for the left ear

The relationship between the hearing thresholds and the Brinkman Index of the participants in the study group was investigated using Pearson's correlation. As a result of the analysis, the correlation coefficients and statistical significance values are shown in Table 6. According to this, there was a weak correlation between Brinkman Index and 9 kHz hearing thresholds (r= 0.37, p<0.05) and a moderate positive correlation with 10 kHz (r=0.44, p<0.01). There was a highly positive correlation between the Brinkman Index and hearing thresholds of 11.2 (r=0.69), 12.5 (r=0.74), 14 (r=0.69), 16 (r=0.73), 18 (r=0.70) and 20 kHz (r=0.67) (p<0.01).

Table 6: Statistical analysis of hearing thresholds and Brinkman Index results from the study group

		9 kHz	**10 kHz**	**11.2 kHz**	**12.5 kHz**	**14 kHz**	**16 kHz**	**18 kHz**	**20 kHz**
BI	**Correlation Coefficient**	0,37*	0,44**	0,69**	0,74**	0,69**	0,73**	0,70**	0,67**
	P-value	**0,03**	**0,00**	**0,00**	**0,00**	**0,00**	**0,00**	**0,00**	**0,00**
	N	37	37	37	37	37	37	37	37

BI: Brinkman Index

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Discussion and Conclusion

In this study, there was no difference in the mean age of the participants between the smoking group and the control group. Considering the previous studies, it is seen that the age variable alone causes the degeneration of the cochlea, and as a result, it may cause an increase, especially in the EHFTs (Ciorba et al., 2011; Huang & Tang, 2010). In the current study, it was considered that the mean age did not differ between the groups to evaluate the effect of smoking on hearing independently of age.

In the present study, EHFTs in the right and left ears of the smoking and control group were compared. Considering the PTTs in the right ears of the participants, there was a significant difference between the two groups at 11.2, 12.5, 14, 16 and 20 kHz. On the other hand, in the left ear, there was a significant difference between the two groups at all frequencies.

There are several hypotheses in the literature regarding the pathophysiology of the effect of smoking on the inner ear. Hultcrantz et al. reported that nicotine in cigarettes directly affects the inner ear and causes hearing loss (Hultcrantz et al., 1982). Stewart et al. showed that carboxyhemoglobin levels increased in smokers. They stated that the increase in carboxyhemoglobin level causes a decrease in the oxygen level that the organ of Corti can use, and thus hearing loss may occur (Stewart, 1976). In another study, they stated that noise exposure might cause an increase in the cellular metabolism of the cochlea and that smoking may decrease the perfusion of the cochlea. Consequently, they stated that hearing loss might occur due to the inability to meet the required amount of oxygen for the cellular metabolism needs of individuals exposed to high-intensity noise and who smoke (Hawkins Jr, 1971). Another hypothesis suggested that diastolic blood pressure increased in smokers and that arterial hypertension could also affect the organ of Corti (Pyykkö et al., 1989). In addition to the cigarette mentioned above effects on the cochlea, studies show that cochlear perfusion is more intense in the basal region of the cochlea, which is responsible for high-frequency hearing than in the apex region (Chen et al., 2013). Therefore, potential metabolic problems can be expected to affect the basal region of the cochlea more than the apex. As an indicator of this situation, in the present study, unlike previous studies, we tried to show in which frequency range cigarette smoking affects hearing thresholds more. According to the results, the mean values of the differences between the hearing thresholds of the control and research groups in both ears increase as the frequency increases. This increase is statistically significant in the frequency range of 9-10 kHz, 10-11.2 kHz, 11.2-14 kHz in the right ear, and 10-11.2 kHz in the left ear. According to these results, considering that the participants' 0.125-8 kHz hearing thresholds are within normal limits, it can be thought that smoking creates a difference in high-

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frequency hearing thresholds in individuals. This difference increases significantly, especially after 9 and 10 kHz. Also, in the current study, a correlation was observed between the hearing thresholds of each frequency evaluated regardless of the ear and the Brinkman Indices. The fact that this correlation is weak at 9 kHz hearing thresholds, moderate at 10 kHz hearing thresholds, and high at and higher at 11.2 kHz and above may indicate that the negative effect of smoking increases towards the basal region in the cochlea.

In addition, when PTTs were compared between the groups, it was observed that smoking created more hearing threshold differences between the groups in the left ear. This finding may indicate that smoking has more negative effects on the left ears of the participants. It can be seen that there are findings in previous studies that could explain this finding. Moffat et al. treated individuals with bilateral symmetrical hearing loss with gentamicin. As a result of the study, it was shown that the improvement in hearing levels in the right ear was more significant than in the left ear (Moffat & Ramsden, 1977). In another study, it was reported that noise-induced hearing loss was more common in the left ear than in the right ear (Berg et al., 2014). Schmidt et al., on the other hand, showed that the left ears of the participants who received cisplatin treatment were significantly more affected than their right ears (Schmidt et al., 2008). It has been stated that conditions such as noise-induced hearing loss, ototoxic and age-related hearing loss cause damage to the cochlea through reactive oxygen-based molecules (Henderson et al., 2006; Peters et al., 2000). It has also been shown that reactive oxygen-based molecules can cause damage to the cochlea through nicotine in smokers (Ahn et al., 2011; Gatto et al., 2014; Stewart, 1976). Considering all these findings, it can be concluded that either reactive oxygen-based molecules affect the left ear more than the right ear or the hearing loss caused by these molecules may suggest that the right ear has a more advanced healing mechanism. However, it is not easy to reach a definite conclusion with the data we have.

The limitations of the present study can be summarised. In the current study, the participants' smoking habits, previous disease history, etc., were obtained through the subjective histories of the patients. The accuracy of the information has been directly accepted without relying on any objective data. In addition, although we consider that there is no difference between the average age of the groups so that age-related hearing loss does not affect the study results, there are many pathologies that affect the high-frequency hearing thresholds of individuals. For this reason, we think that the results should be interpreted carefully and that the effect of this situation can be reduced by increasing the number of participants included in the study. İn this study, the effect of smoking on hearing was demonstrated by the difference in

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hearing thresholds. Future studies may evaluate the higher levels of the auditory system, considering the potential effects of smoking on EHFTs.

To the best of our knowledge, the present study is the first study in which high-frequency hearing thresholds are evaluated, including 20 kHz, which draws attention to the frequency after which smoking begins to affect hearing. At the same time, the relationship between smoking level (with Brinkman Index) and EHFTs were evaluated for the first time with this study. There is a difference between the smoker's group and the non-smoker's group in terms of EHFTs in the current study. This difference started to increase after 9 kHz in the right ear and 10 kHz in the left ear. Also, a relationship was found between the participants' Brinkman Indexes and EHFTs. These findings may indicate the potential effect of smoking on hearing in individuals, especially on EHFTs. Since high-frequency hearing thresholds evaluated outside of routine audiometric examination may reflect factors that may affect the hearing system, such as smoking, it is recommended to evaluate EHFTs to avoid missing some pathologies even in patients who are clinically evaluated as normal hearing in the 0.125-8 kHz range.

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Conflict of interest

The authors declare that they have no conflict of interest.

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