Received: 14 July 2021 | Revised: 24 August 2021 | Accepted: 12 September 2021

DOI: 10.1002/lio2.671

ORIGINAL RESEARCH

Laryngoscope Investigative Otolaryngology

Olfactory dysfunction in passive vs active smoking

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Abstract

Background: The aim of this study is to assess the olfactory functions of passive smokers compared to active smokers and nonsmokers.

Methods: This prospective case-control study included 30 nonsmokers, 30 passive smokers, and 30 active smoker participants. All groups were matched for gender and age. The Sino-Nasal Outcome Test 22 (SNOT-22) and Sniffin' Sticks test battery were administered to all subjects. Threshold (T), discrimination (D), and identification (I) scores were noted. Olfactory function was subjectively assessed as 0: severe dysfunction and 5: no problem.

Results: Overall, TDI scores of active smokers (24.78 ± 3.02) and passive smokers (24.90 ± 2.45) were significantly lower than nonsmokers (34.23 ± 3.46). There was no statistically significant difference between passive smokers and smokers ($F_{(2,87)} = 13.47$, $P < .001$)). All subscores are negatively affected by active or passive smoking. The greatest impact of smoking was on threshold scores ($\eta^2_T = 0.719$), followed by identification ($\eta^2_I = 0.353$) and discrimination ($\eta^2_D = 0.282$) scores. SNOT-22 and TDI scores were weakly ($r = -.352$) correlated as subjective assessment, and TDI scores were moderately correlated (r: .539) (P values < .001). Age and pack-years cigarette dosage had a negative effect on the TDI score (TDI = 26.386 − (0.084 × age) − (0.072 × Pack.Year)) according to stepwise linear regression model ($F = 10.187$; $P = .001$).

Conclusions: Passive smoking has nearly the same adverse effect on olfactory function as active smoking. The threshold scores are the most negatively affected. The olfactory effect of cigarette smoke may not be directly related to nasal inflammation. Olfactory neuronal pathways should be investigated to elucidate the exact pathophysiology.

Level of Evidence: 3b.

KEYWORDS
olfaction disorders, secondhand smoking, smell, smoking

1 | INTRODUCTION

The olfactory function is a warning system against poisonous gas, fire, or spoiled foods, in addition to its actual effect on the quality of life (QoL), resulting from the pleasure of eating and drinking.[1,2] Aging, male sex, upper respiratory infections, trauma, toxicity exposure, sinonasal, and neurological disorders all contribute significantly to the etiology.[3]

Cigarette smoke causes inflammation in the respiratory tract and structural changes in the epithelium, such as decreased mucociliary activity, goblet cell hyperplasia, and squamous metaplasia.[4,5] Passive smoking is defined as the inhalation of smoke from tobacco products used by others.[6] Passive smoking, like active smoking, has been linked to cancer and cardiovascular disease. Cigarette smoke exposure has been associated with otitis media, rhinosinusitis, bronchitis, and allergic infiltration in children due to inflammation and reduced ciliary function in the respiratory mucosa.[5,7] Previous reports indicated that smoking is one of the major causes of olfactory dysfunction.[8-10] However, the consequences of passive smoking on olfactory function were much less well investigated. No studies have analyzed the factors associated with objective olfactory test results in a regression model in passive smokers. To the best of our knowledge, no study combines passive and active smoking in olfactory function in the literature. The primary aim of this study was to investigate and compare the impact of active and passive smoking on olfaction using objective psychophysical measures. The secondary endpoint of this study was to examine the contributing factors affecting olfactory dysfunction in individuals who are passively or actively exposed to cigarette smoke.

2 | MATERIALS AND METHODS

2.1 | Participants

This prospective case-control study included 90 nonsmokers, passive smokers, and smoker volunteers tested between December 2020 and February 2021. To be enrolled in the study, participants had to meet the following inclusion criteria: adults between 18 and 65 years of age, cooperate to the test, and are willing to participate in the study. Since the study was carried out on hospital staff, there is no heterogenicity in educational status and socioeconomic level distribution among the groups. The exclusion criteria included participants who had upper respiratory disease within the previous 1 month, sinonasal disorders, nasal surgery history, head trauma, pregnancy, and any known systemic or psychiatric disease. The participants had no abnormal findings in their anterior rhinoscopy and nasal endoscopy.

The duration of active or passive smoking and the number of cigarettes exposed per day were recorded. The number of packs smoked per day was multiplied by the number of years that smoking or exposure occurred to calculate the cigarette dose in pack-years. All active smokers have been smoking, and all passive smokers have been exposed to smoke for at least 5 years. Individuals who have been exposed to cigarette smoke for at least 5 years and whose partners or relatives smoke at home (not on the balcony) were included in the passive smoking group. Because the passive smoking group members are voluntary health care professionals, they are exposed to cigarette smoke for an average of 6 hours each day during the evening time hours at home. Participants were divided into three equal groups: active smokers, passive smokers, and nonsmokers (with no exposure to environmental smoke, no use of tobacco products). Written informed consent was obtained prior to the interview. The protocol of this study was approved by the local institutional ethical committee (OMU KAEK 2020/675) and conducted in accordance with the Declaration of Helsinki. All participants in the study provided their written informed consent. The participants have consented to the submission of their data to the journal.

2.2 | Subjective assessment of sinonasal and olfactory function

The Turkish version of the Sino-Nasal Outcome Test 22 (SNOT-22)[11] was administered to all patients to assess the sinonasal symptoms and their effect on the QoL. The questionnaire has 22 items that assess sinonasal and otologic symptoms and sleep and emotional functions. Each question was graded on a scale of 0 (no problem) to 5 (severe problem). The lower score defines a better sinonasal status.

Subjective smell scoring was obtained with a five-point Likert scale coded as follows: 0: severe problem and 5: no problem with smell sensation.

2.3 | Psychophysical assessment of olfactory function

The olfactory function was tested using an extended Sniffin' Sticks odor test battery (Sniffin' Sticks, Burghart Messtechnik GmbH, Wedel, Germany) in a well-aerated room. This well-known and validated psychophysical olfactory test is divided into three subtests: odor threshold (T), odor discrimination (D), and odor identification (I).

For the threshold test, 16 triplets of pens, including one diluted *n*-butanol pen and two dilution solvent pens, were presented in ascending order of odor concentration. The single-staircase technique was used to determine the thresholds. The correct identification of the diluted *n*-butanol pen twice in a triplet resulted in the reversal of the staircase leading to the higher dilution step. The first misidentification of the correct pen resulted in a reversal of the staircase to the subsequent lower dilution. The test was repeated seven times more before seven reversals were detected. The threshold score was calculated as the average of the staircase's last four reversals. The discrimination test was conducted using the following sets of odorants, two of which contained the same odor and one contained a different odor. The participant was tested while his/her eyes closed and asked to recognize the different odors. The discrimination score was the sum of correctly identified pens. In the last subtest, odor identification was evaluated by 16 commonly recognized odor-filled pens, and the participant was asked to identify the odor from a list of four descriptors. The total number of correct answers represented the identification test score. Cumulative olfactory function is determined by adding the three

TABLE 1 Demographic data and smoking information of participants

		Nonsmoker (n: 30)	Passive smoker (n: 30)	Active smoker (n: 30)	Test statistics	*P*
Age (median; min-max)		42.0 (20-62)	40 (18-58)	39 (25-56)	0.136	.934[a]
Sex	Female n (%)	18 (60.0%)	22 (73.3%)	15 (50%)	3.52	.172[b]
	Male n (%)	12 (40.0%)	8 (26.7%)	15 (50%)		
Pack-year exposure (mean ± SD)		—	16.81 ± 12.07	14.60 ± 12.22	0.779	.483[c]

Note: n and % indicate number and percentage within column, respectively.
[a]Kruskal-Wallis test.
[b]Pearson Chi-square test with Yates adjustment.
[c]Independent samples *t* test.

TABLE 2 Descriptive statistics of olfactory test and SNOT-22 scores according to groups

	Nonsmoker (n: 30)	Passive smoker (n: 30)	Active smoker (n: 30)	Total (n: 90)
TDI	34.23 ± 3.46[a]	24.90 ± 2.45[b]	24.78 ± 3.02[b]	28.00 ± 5.35
Threshold	7.76 ± 1.84[a]	3.13 ± 1.14[b]	3.15 ± 1.03[b]	4.68 ± 2.58
Discrimination	13 ± 1.26[a]	10.73 ± 1.62[b]	10.66 ± 2.24[b]	11.46 ± 2.05
Identification	13.47 ± 1.68[a]	11.03 ± 1.69[b]	10.96 ± 1.42[b]	11.82 ± 1.96
Subjective olfaction score	4.86 ± 0.34[a]	4.03 ± 0.96[b]	3.39 ± 0.98[b]	4.27 ± 0.91

Note: There is no significant difference between groups with the same superscript letter for each row after pairwise comparisons (Dunnett T3 test).
Abbreviations: n, number; TDI, overall score of threshold-discrimination-identification; SNOT-22, Sino-Nasal Outcome Test-22.

TABLE 3 Multiple analysis of variance results

	Type III sum of squares	Mean square	*F*	*P*	η^2	R^2
TDI	1761.14	880.57	97.17	<.001	0.691	.684
Threshold	426.28	213.14	111.3	<.001	0.719	.713
Discrimination	105.87	52.93	17.15	<.001	0.283	.266
Identification	121.75	60.87	23.70	<.001	0.353	.338
Subjective olfaction	15.76	7.88	11.76	<.001	0.213	.195

Note: Pillai's trace = 0.776. R^2: adjusted *R* square; η^2: partial eta square; *F*: test statistics.
Abbreviations: SNOT-22, Sino-Nasal Outcome Test-22; TDI, overall score of threshold-discrimination-identification.

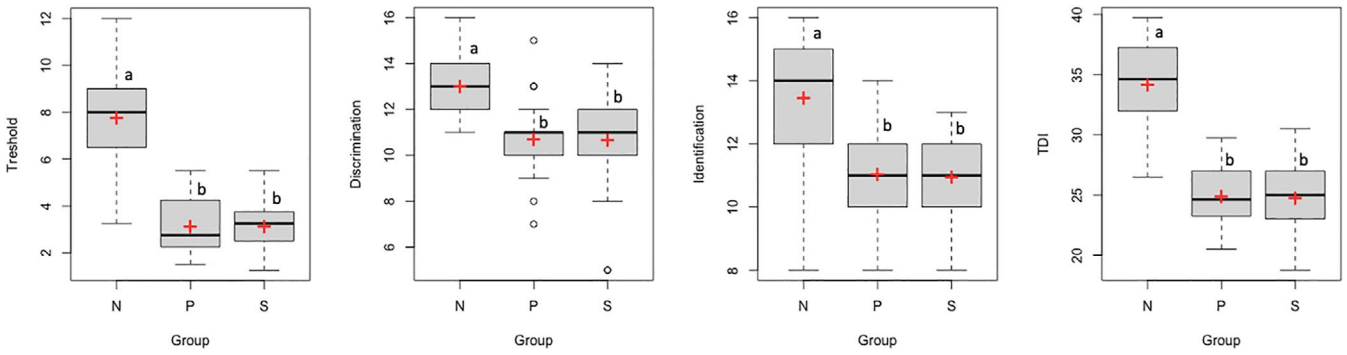

FIGURE 1 Sniffin' Sticks threshold, identification, discrimination, and overall threshold-discrimination-identification (TDI) scores in nonsmokers (N), passive smokers (P), and active smokers (S). Boxes indicate the first and third quartiles, and median observations are denoted by a line in each box. Mean values are demonstrated by a "+" in the boxes (multivariate analysis of variance [MANOVA]). Different letters (a,b) indicate that mean scores significantly differ between groups in pairwise comparisons (post hoc Dunnett T3 test)

TABLE 4 The regression model's significance

Source	df	Seq SS	Contribution	Adj SS	Adj MS	*F*-value	*P*-value
Enter method							
Age	1	94.320	0.037	25.735	25.735	3.300	.073
SNOT-22	1	306.920	0.120	0.058	0.058	0.010	.932
Subjective score	1	408.030	0.160	29.061	29.061	3.720	.057
Gender	1	0.020	0.000	3.961	3.961	0.510	.478
Smoking status	2	1085.830	0.426	485.498	242.749	31.110	**<.001**
Pack.Year	3	30.050	0.012	30.051	10.017	1.280	.286
Stepwise method							
Age	1	94.320	0.037	49.110	49.115	6.330	**.014**
Subjective score	1	670.450	0.263	55.790	55.787	7.190	**.009**
Smoking status	2	1125.540	0.442	1125.540	562.772	72.570	**<.001**

Abbreviations: Adj SS, adjusted sum of squares; df, degree of freedom; *F*, test statistics; Seq SS, sequential sums of squares; SNOT-22, Sino-Nasal Outcome Test-22. significant *p* values in *p* value column is bold.

subtest scores and is referred to as the TDI score. TDI scores ≤15 refer to anosmia, between 16 and 30 refer to hyposmia, and TDI ≥31 defines normosmia.[12]

2.4 | Statistical analysis

The data were analyzed and visualized by R Studio (version 1.2.5019). Parametric tests were used when the variables were normally distributed according to the Kolmogorov-Smirnov tests. Any skewness or kurtosis statistic below an absolute value of 2.0 was considered as the distribution was normal.[13] The homogeneity of variances variance-covariance matrices were tested using Levene's test and Box's *M* test, respectively; as a result, Pillai's trace criterion was considered. Continuous variables were reported as mean (±SD). A one-way multivariate analysis of variance (MANOVA) with a post hoc Dunnett T3 test was conducted to compare groups. Pearson correlation test was performed to investigate the relationship between subjective olfactory assessment and TDI scores. A multivariate linear regression model was constructed to identify estimators of overall TDI score in passive smokers and smoker patients. The analysis was conducted using nested linear regression independent from the connection between package year and smoking status (dependent variable = TDI score).

All tests were two-tailed and statistical significance was set at the $P < .05$ level.

3 | RESULTS

Participants included 30 active smokers (15 men, 15 women; median age = 39 years), 30 passive smokers (8 men, 22 women; median age = 40 years), 30 nonsmoker control subjects (12 men, 18 women; median age = 42 years) matched for gender and age. There was no significant difference in age ($P = .934$) or in gender distribution between groups ($P = .172$). The mean exposure duration for active smokers was 14.60 (±12.22) pack-years and 16.81 (±12.07) pack-years for passive smokers ($P = .483$) (Table 1). None of the participants were found to be anosmic (TDI ≤15).

There was a statistically significant difference in olfactory test and SNOT-22 scores based on smoking status in MANOVA model ($F_{(2,87)} = 13.47$, $P < .001$; Pillai's trace = 0.776). Nonsmokers had a mean TDI value of 34.23 (±3.46), while passive smokers had a mean value of 24.90 (±2.45), and active smokers had a mean value of 24.78 (±3.02). Table 2 demonstrates the descriptive statistics of all domains of the Sniffin' Sticks test and subjective scoring scores as MANOVA results are summarized in Table 3. TDI scores of active and passive smokers were significantly lower than nonsmokers (Dunnett T3 test, $P < .001$). However, there was no statistically significant difference between passive smokers and smokers (Dunnett T3 test, $P = .988$) (Figure 1).

All indices of olfactory function were negatively affected by smoking or passive exposure. While the greatest effect determined on the threshold scores ($\eta^2_T = 0.719$), followed by identification ($\eta^2_I = 0.353$) and discrimination ($\eta^2_D = 0.283$) scores. Smoking also had an impact on subjective olfactory scores ($\eta^2_s = 0.213$). There was a moderate positive correlation between subjective scoring and TDI scores ($r = .539$, $P < .001$).

There was no statistically significant difference between nonsmokers (3.87 ± 3.14) and passive smokers (5.93 ± 3.75) in SNOT-22 scores. Active smokers (3.39 ± 0.98) had higher SNOT-22 scores than the other two groups ($P < .001$; one-way analysis of variance, Dunnett T3 test).

When the factors affecting the TDI score are examined in the final model, age ($\beta = -.072$, $P = .014$), active ($\beta = -8.435$, $P < .001$), or passive ($\beta = -8.485$, $P < .001$) smoker and subjective evaluation ($\beta = -.996$, $P = .009$), was found to have a significant effect on the total TDI score. Gender and SNOT-22 score and package.year did not have a significant effect on the total TDI score. The coefficients and regression outcomes that describe quantitatively the exact relationship of TDI score with age, gender, smoker type, package-year dose, and SNOT-22 scores are presented in Tables 4 and 5.

TABLE 5 Factors affecting the threshold-discrimination-identification score

Factor	β	SE	95% CI	*T*-value	*P*-value	R^2
Enter methods						
Constant	32.320	2.660	(27.04; 37.61)	12.170	**.000**	72.76
Age	−0.056	0.031	(−0.1178; 0.0054)	−1.820	.073	
SNOT-22	0.007	0.080	(−0.1525; 0.1663)	0.090	.932	
Subjective score	0.796	0.412	(−0.025; 1.616)	1.930	.057	
Gender (ref: male)[a]						
Female	0.454	0.638	(−0.815; 1.724)	0.710	.478	
Smoking status (ref: nonsmoker)						
Passive	−7.900	1.170	(−10.23; −5.57)	−6.750	**<.001**	
Active	−7.730	1.160	(−10.04; −5.42)	−6.660	**<.001**	
Pack.Year						
Nonsmoker	−0.017	0.068	(−0.1523; 0.1194)	−0.240	.810	
Passive smoker	−0.055	0.043	(−0.1414; 0.0310)	−1.270	.206	
Active smoker	−0.070	0.046	(−0.1619; 0.0225)	−1.510	.136	
Stepwise methods						
Term						
Constant	32.15	2.340	(27.49; 36.81)	13.730	**<.001**	72.93
Age	−0.072	0.029	(−0.1292; −0.0152)	−2.520	**.014**	
Subjective score	0.996	0.371	(0.258; 1.734)	2.680	**.009**	
Smoker (ref: nonsmoker)						
Passive	−8.485	0.783	(−10.041; −6.930)	−10.840	**<.001**	
Active	−8.435	0.796	(−10.018; −6.852)	−10.590	**<.001**	

Note: Dependent variable: overall TDI score. Bold prints in *P* value column indicate significant effect.
Significant *P* values in *P* value column is bold.
Abbreviations: β, standardized regression coefficient; CI, confidence interval; SE, standard error; SNOT-22, Sino-Nasal Outcome Test-22; *T*, test statistics; TDI, overall score of threshold-discrimination-identification.
[a]Included in the regression model as: gender (male = 0, female = 1); smoker type (passive = 1, smoker = 2, nonsmoker = 0); and subjective score (0 = very problematic smell sensation, 5 = no problem).

4 | DISCUSSION

The most important finding of our study is that passive smoking, the same as active smoking, has a negative impact on olfactory functions. Another significant finding is that the most affected score in the test battery by smoking is the threshold ($\eta^2_T = 0.719$), which is followed by identification ($\eta^2_I = 0.353$) and discrimination ($\eta^2_D = 0.283$) subscores. The findings of our study can be interpreted in several different ways. First, cigarette smoke can cause inflammation in the nasal and olfactory mucosa and disrupt the smell. Alternatively, other underlying pathophysiological mechanisms can lead to smoking-related olfactory dysfunction.

Tobacco use is associated with inflammation of the respiratory tract and structural changes in the epithelium, including reduced mucociliary activity, goblet cell hyperplasia, squamous metaplasia, and mucosal edema.[4,5,14-16] Histological examinations of smokers' olfactory epithelium revealed squamous metaplasia and altered morphology of olfactory receptor neurons.[17] In the study of Vent et al., smoke-exposed animals, had a significantly higher rate of olfactory receptor neuron apoptosis when compared to controls.[8] As a result of smoking, Sahin et al. observed increased apoptosis in rat olfactory neurons.[18] In smoke-exposed rabbits, Iskander et al. found a loss of sustentacular cell microvilli, a reduction in the distribution of specialized cilia on olfactory receptor cells, and respiratory metaplasia.[19]

Passive smoking can be just as hazardous in many ways as active smoking. The effect of passive smoking on the nasal mucosa and nasal mucociliary clearance has been demonstrated.[6,20,21] However, the impact of passive cigarette smoking on olfactory function has yet to be determined in adults. Nageris et al. examined the impact of passive smoking on olfactory identification in children and identified a negative effect.[22] Although Schubert et al. discovered a nonsignificant increased risk of dysfunction in nonsmokers exposed to high levels of environmental tobacco smoke compared to those who were not exposed, Ranf et al. found no such association.[23,24]

Active or passive smoking harmed both the overall score and each subscore in the present study. Our group of nonsmokers scored comparable to those in Tekeli et al.'s normative value research.[25] There was no substantial difference in Sniffin' Sticks scores between those who smoked and those who were exposed to smoke in our study. The most significant effect of smoke exposure was detected on the

threshold score. There could be several possible reasons for this situation. The effect of smoking on the central neural system and its consequences on cognitive functions becomes subject to debate. There is also evidence that smoking influences olfaction by affecting central neural pathways that are directly involved in olfactory function. For example, smokers were found to have significantly smaller olfactory bulb volumes than nonsmokers.[26] Nicotine inhibits neurogenesis while promoting glia genesis.[27] A decrease in olfactory bulbus may be explained by insufficient afferent input caused by changes in the olfactory epithelium caused by smoke. Fritz et al. suggested that smokers' brains had significantly less gray matter volume in the olfactory gyrus than nonsmokers' brains.[28]

Another theory on smoke-related smell loss is that smoking causes neuroepithelial damage in the olfactory field. The cell bodies of olfactory sensory neurons (OSN) are found in the epithelium, and their dendrites protrude into the nasal passage, and this unprotected location causes neuronal apoptosis.[8] Despite OSN apoptosis, the maturation of progenitor cells in the olfactory epithelium provides neuronal regeneration in mammals.[29] In animal research, Vent et al. discovered that smoking enhances caspase-3 activation, a proteolytic effector enzyme involved in apoptosis.[8] Dinc et al. discovered that while threshold scores were unchanged after stopping smoking, discrimination and identification scores improved. They hypothesized that insufficient regeneration of olfactory epithelium was responsible for the lack of improvement in the odor threshold score.[9]

Tobacco smoke has been proven to be harmful to cognitive function in studies.[30,31] It has been established before that there is a connection between olfactory and cognitive function.[31,32] Our research groups consisted of participants with an average age of 40 and no known illness. Their young age and good psychophysical health may explain why identification and discrimination scores are less affected.

Smoking-caused microvascular damage may also play a role in pathophysiology. Özmen et al. asserted that the vasculogenic effect of smoking is responsible for olfactory and erectile dysfunction. They identified a connection between erectile and olfactory dysfunction in smoking men as opposed to nonsmoking adult men.[33] Siegel et al. also obtained results that will support the microvascular theory by investigating the effect of smoking on cardiovascular disease concomitantly with smell loss. According to their findings, the risk of a heart attack increases in smokers who have olfactory dysfunction.[34]

According to our results, age and being a smoker (active or passive) were found to decrease the TDI score. Gender, and SNOT-22 scores had no effect on the TDI score. Katotomichelakis et al. discovered that cigarette smoking was associated with olfactory dysfunction.[10] As in our study, they found a direct negative correlation between olfactory function and the amount smoked. They did not, however, include passive smokers in their regression model. Frye et al. observed a significant dose-response relationship between pack-years of smoking and olfactory function in smokers.[35] Other research shows that even after quitting smoking, the olfactory function was not equal to that of nonsmokers, implying that restoring olfactory function may not be enough if smoking was prolonged.[9] A meta-analysis was conducted by Ajmani et al. revealed that age is a risk factor for impaired olfaction in smokers.[4] Indeed, both age and pack-years are indicators of time spent actively or passively exposed to cigarette smoke. According to our regression model, age, subjective scoring of olfaction and being a the effect of smoking status overall TDI score. Additional research can be conducted to determine which factors contribute to olfactory function in passive and active smokers by including other factors in the regression model.

Active smokers are, of course, more directly and intensely exposed to cigarette smoke particles. Hutson et al. discovered that active cigarette smoking was associated with lower SNOT-22 scores.[36] In our study, active smokers, had higher SNOT-22 values than passive smokers and nonsmokers. These findings support previous research examining the relationship between smoking and rhinosinusitis, and they are interpreted as indicating that the inflammatory effect of passive smoking is limited.[37]As a result, we might speculate that the cause of smoke-related odor loss was not solely related to inflammatory pathology it should also be related to olfactory neuronal regeneration pathology. There was also no difference in subjective scoring between passive and active smokers. The correlation between TDI scores and subjective scoring was found to be moderately significant (*r*: .539). This demonstrates that patient-reported measures and psychophysical tests are not alternatives for each other, but rather complementary assessment methods.

According to our study, passive smoking is just as effective as active smoking in terms of olfactory loss. It is well understood that smoking is harmful not only to the person who smokes but also to those around them. In research conducted by Fjaeldstad et al., the etiology of olfactory impairment and smoking were connected. In their series, which included many patients, they discovered that current smoking, but not former smoking, was involved with posttraumatic olfactory loss.[15] A similar study design can be used to investigate the effect of passive smoking on already existing olfactory dysfunction. Elimination of active or passive exposure to cigarette smoke should be prioritized in patients who already have a loss of smell for any reason.

Demographic, cognitive, and systemic factors all contribute to the rate of olfactory dysfunction declines. It is well established that olfactory function weakens with aging.[38] In our study, a significant correlation was found between decreasing TDI scores and increasing age. Although some olfactory loss may be a natural component of aging, our findings emphasize the detrimental effect of smoking on the olfactory system's functionality.

One of the limitations of our study is that olfactory function was not evaluated subjectively by a validated and standardized survey. Since there is no validated questionnaire translated into our language, we decided to use SNOT-22 and subjective scoring of olfaction to compensate for this deficiency partially. Inability to use the olfactory-related QoL or olfactory disorders questionnaire might also be regarded as a restriction. We could not utilize culturally adapted translations of these measures into our language since they are not yet validated. The effect of passive smoking on olfactory bulbous volume and demonstration of histopathological changes with electron microscopy can be considered in prospective studies. Future works using functional MRI to demonstrate the effects of passive smoking on cognitive

functions and the olfactory cortex may support our findings. Comparing other commonly used tobacco products (electronic cigarettes, pipes, etc.) and cigarettes may also be the subject of another study.

5 | CONCLUSION

Passive smoking has nearly the same effect on the olfactory function as active smoking. Smoking has a greater impact on olfactory thresholds in particular. Both aging and passive or active smoking have been demonstrated to decrease olfactory function in this study. Disrupted sinonasal functioning in active smokers may create this situation, but olfactory dysfunction induced by passive smoking is a problem that has to be addressed further.

ACKNOWLEDGMENTS

The authors express their sincere gratitude to all participants who agreed to take part in the study and thus contributed to science. The authors would like thank to Dr Naci Murat (e-istatistik Company) for his significant contributions to statistical analyses of this study.

CONFLICT OF INTEREST

The authors declare there are no conflicts of interest—financial or otherwise—related to the material presented herein.

AUTHOR CONTRIBUTIONS

Senem Çengel Kurnaz, **Emel Tahir**, and **Esra Kavaz**: Conceptualization. **Senem Çengel Kurnaz**, **Emel Tahir**, and **Esra Kavaz**: Data curation. **Emel Tahir**: Formal analysis; **Senem Çengel Kurnaz**, **Emel Tahir**, and **Esra Kavaz**: Investigation. **Senem Çengel Kurnaz** and **Esra Kavaz**: Methodology. **Senem Çengel Kurnaz**, **Emel Tahir**, and **Esra Kavaz**: Project administration. **Senem Çengel Kurnaz**, **Emel Tahir**, and **Esra Kavaz**: Resources. **Emel Tahir**: Software. **Emel Tahir**: Supervision. **Emel Tahir**: Visualization. **Senem Çengel Kurnaz**, **Emel Tahir**, and **Esra Kavaz**: Roles/writing - original draft. **Senem Çengel Kurnaz**, **Emel Tahir**, and **Esra Kavaz**: Writing - review and editing.

DATA AVAILABILITY STATEMENT

Availability of data and material: The data of the study can be shared on demand and it was uploaded to Dryad digital data repository as; Tahir, Emel (2021), Passive Smoking and Olfaction, Dryad, Dataset, https://doi.org/10.5061/dryad.vt4b8gtrw.

ETHICS STATEMENT

Approval for the study was granted by the institutional ethical committee (OMU KAEK 2020/675). All procedures performed in this study involving human participants were in accordance with the 1964 Declaration of Helsinki and its later amendments.

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How to cite this article: Çengel Kurnaz S, Tahir E, Kavaz E. Olfactory dysfunction in passive vs active smoking. *Laryngoscope Investigative Otolaryngology*. 2021;6(5):932-939. doi:10.1002/lio2.671