

The effects of cigarette smoking on gingival phenotype and clinical periodontal parameters

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ABSTRACT

Aims: The aim of this study was to evaluate the effects of cigarette smoking on the gingival phenotype and to investigate the relationship between gingival thickness (GT), keratinized tissue width (KTW) and clinical periodontal parameters in smokers.

Methods: A total of 30 systemically healthy volunteers, including 15 smokers (group S) and 15 non-smokers (group NS), were enrolled in the study. GT, KTW and clinical periodontal parameters such as Plaque Index (PI), Gingival Index (GI), probing depth (PD), and bleeding on probing (BOP) were evaluated in the maxillary anterior and first molar regions. GT was measured using the transgingival probing method with a digital caliper (± 0.01 mm accuracy). The KTW was determined by measuring the distance from the gingival margin to the mucogingival junction at the mid-buccal site. Data was analyzed using IBM SPSS Statistics 25.0 software.

Results: In the smoking group, GT and KTW values were significantly higher compared to the non-smoking group ($p < 0.001$ and $p < 0.05$, respectively). The plaque index was also significantly higher among smokers ($p < 0.05$), whereas no significant differences were observed between the groups in terms of GI, PD, and BOP values ($p > 0.05$).

Conclusion: The findings of this study indicate that cigarette smoking leads to an increase in gingival thickness and plaque accumulation. Smoking is an important environmental factor that adversely affects the gingival phenotype, both structurally and functionally. It may result in a gingival phenotype that appears thick and fibrotic but is biologically compromised, with reduced vascularity, healing, and defense capacity. These results emphasize the importance of considering the history of smoking in periodontal treatment planning and in maintaining periodontal health.

Keywords: Gingival phenotype, periodontal health, smoking

INTRODUCTION

The term phenotype is used to describe anatomical characteristics that can vary over time due to external influences such as environmental factors and clinical interventions, whereas biotype refers to tissue characteristics genetically determined by a specific genotype. The gingival phenotype encompasses both gingival thickness (GT) and keratinized tissue width (KTW), integrating not only the individual's gingival biotype but also environmental influences that may cause temporal variations.¹⁻³ In recent years, the clinical importance of gingival phenotype has increased due to its critical role in treatment planning, aesthetic outcomes, and long-term tissue stability.^{4,5} Different gingival phenotypes exhibit variable biological responses to inflammation, trauma, and parafunctional habits and may lead to distinct outcomes in esthetic restorative procedures.² Hwang and Wang⁶ suggested that the durability of thick soft tissues is primarily determined by a high-volume extracellular matrix, dense collagen content, and increased vascularity.

Accordingly, several individual and environmental factors such as age, gender, periodontal disease history, parafunctional

habits, and smoking have been reported to influence the morphological and biological properties of the gingival phenotype. Among these factors, smoking is of particular importance due to its widespread prevalence and multifaceted impact on periodontal tissues.⁷⁻⁹ Cigarette smoking is one of the strongest environmental risk factors in the development and progression of periodontal diseases. Numerous clinical and epidemiological studies have demonstrated that smokers exhibit significantly higher prevalence of gingival inflammation, clinical attachment loss, alveolar bone resorption, and tooth loss compared to non-smokers.¹⁰⁻¹² Exposure to tobacco smoke alters the subgingival microbial composition, resulting in a more pathogenic microbiota. Biofilms from smokers have been shown to be dominated by anaerobic species such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola*, which promote bacterial toxins and proteases that mediate periodontal tissue destruction.^{13,14}

Smoking has also been associated with distinct histopathological changes in the gingival epithelium,

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including increased epithelial thickness and basal cell hyperplasia.^{15,16} Another notable finding is the suppression of the inflammatory response to plaque accumulation and the reduced incidence of bleeding on probing among smokers. Studies reporting decreased gingival bleeding in smokers have attributed this to the modulation of the host immune response by tobacco products.¹⁷

Considering these findings, careful assessment of the gingival phenotype has become an essential component of periodontal treatment planning. Therefore, evaluating the relationship between smoking and gingival phenotype in the general population is clinically important for maintaining periodontal health and improving treatment outcomes.¹⁸ However, clinical studies directly investigating the impact of smoking on the gingival phenotype remain limited. The present study aims to evaluate the gingival phenotype in smokers and to investigate its influence on specific clinical periodontal parameters.

METHODS

Ethics

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the İnönü University Non-interventional Clinical Researches Ethics Committee (Date: 18.11.2025, Decision No: 2025/8571) prior to its initiation. Before the study, all participants were informed in detail about their current condition and the study protocol, and both verbal and written informed consent were obtained.

Study Design

This cross-sectional and comparative study included volunteer participants who applied to the Department of Periodontology, Faculty of Dentistry, İnönü University, and met the predetermined inclusion criteria. Based on the study by Manasa et al.¹⁹ a total of 30 systemically and periodontally healthy individuals aged between 24 and 43 years were enrolled, with 15 participants in each group. Participants with at least 20 natural teeth, including the maxillary and mandibular anterior teeth and first molars, were selected and divided into two groups: smokers (group S, n=15) and non-smokers (group NS, n=15). In both groups, the maxillary anterior and first molar teeth were examined in terms of GT, KTW and clinical periodontal parameters including PI, GI, BOP and PD. Plaque accumulation was assessed using the Silness and Loe PI,²⁰ while gingival inflammation was evaluated using the Loe and Silness GI.²¹ For both indices, four surfaces of each examined tooth (buccal, palatal, mesial, and distal) were evaluated. The PI was scored as follows: 0=no plaque; 1=a thin film of plaque detectable only by running a probe across the tooth surface; 2=moderate, visible plaque accumulation; 3=abundant plaque accumulation. The GI was scored as follows: 0=normal gingiva; 1=mild inflammation without bleeding on probing; 2=moderate inflammation with redness, edema, and bleeding on probing; 3=severe inflammation with marked edema, ulceration, and a tendency toward spontaneous bleeding. For each tooth, the scores obtained from the four surfaces were averaged to calculate the PI and GI scores. BOP was recorded as present or absent following periodontal probing and expressed as a percentage of sites.²² GT was measured at a point 1.5 mm apical to the gingival margin by inserting an endodontic spreader perpendicularly into the gingiva until bone contact was achieved. The penetration depth was

measured with a digital caliper (accuracy: 0.01 mm). The KTW was determined by measuring the distance from the gingival margin to the mucogingival junction at the mid-buccal surface of each tooth, using the same instruments.

All measurements were performed twice for each site by the same calibrated examiner, and the mean of the two values was recorded for analysis.

Inclusion criteria were: Age ≥ 18 years; systemically and periodontally healthy status; for the smoker group, smoking at least 15 cigarettes per day for a minimum of 5 years; for the non-smoker group, no history of smoking; KTW > 2 mm; and not being pregnant or lactating for female participants.

Exclusion criteria included: Use of medications affecting gingival or periodontal tissues; history of systemic diseases such as diabetes mellitus; bruxism; presence of gingival recession in the examined teeth; history of orthodontic treatment; restorative material in the examined area; or previous diagnosis of periodontal disease or periodontitis.

Statistical Analysis

A theoretical power analysis was performed using the G*Power 3.1 software. Assuming a type I error rate (α) of 0.05 and a statistical power ($1-\beta$) of 0.80, a total sample size of 30 participants (15 per group) was determined to be sufficient to detect a significant difference between groups. Statistical analyses were conducted using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). The normality of distribution for continuous variables was assessed using the Shapiro-Wilk test, and the homogeneity of variances was evaluated using Levene's test. When parametric assumptions were met, comparisons between the two independent groups were performed using the independent samples t-test. For variables that did not show a normal distribution, the Mann-Whitney U test was applied. Categorical variables were compared using the Chi-square test. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 30 participants, including 14 females and 16 males, were included in the study. The mean age of the participants was 31.05 ± 2.5 years in the smoking group and 30.91 ± 3.0 years in the non-smoking group, with no statistically significant difference between the groups ($p=0.74$). The gender distribution was equal in both groups (female/male ratio: 7/8) as shown in [Table 1](#).

Table 1. Demographic characteristics of the study groups

	S (n=15)	NS (n=15)	p
Age (Md $\pm$ SD)	31.05 $\pm$ 2.5	30.91 $\pm$ 3.0	0.842 ¹
Gender, n (%)	Female 7 (46.7%)	7 (46.7%)	1.000 ²
	Male 8 (53.3%)	8 (53.3%)	

One-way ANOVA test, Chi-square test (S: > 5 years; ≥ 15 cigarettes/day) ($p < 0.05$). S: Smokers, NS: Non-smokers, Md: Mean, SD: Standard deviation, ANOVA: Analysis of variance

The comparative results of clinical periodontal parameters between smokers and non-smokers are presented in [Table 2](#). The mean PI was 1.98 ± 0.25 in smokers and 1.45 ± 0.30 in non-smokers, indicating a statistically significant difference ($p < 0.05$). The mean GI values were 0.95 ± 0.15 and 0.90 ± 0.18



in groups S and N, respectively, with no significant difference observed ($p>0.05$).

Table 2. Comparison of clinical periodontal parameters between smokers and non-smokers

	S (n=15)	NS (n=15)	p
PI	1.98±0.25	1.45±0.30	<0.001*
GI	0.95±0.15	0.90±0.18	0.41
PD	2.85±0.30	2.80±0.32	0.66
BOP	10.2±2.4	11.4±2.6	0.20

Shapiro-Wilk test, Mann-Whitney U test, Levene's test (* $p<0.05$) (S: >5 years; ≥15 cigarettes/day). S: Smokers, NS: Non-smokers, PI: Plaque Index, GI: Gingival Index, PD: Probing depth, BOP: Bleeding on probing

When PD was evaluated, the mean PD was 2.85±0.30 mm in smokers and 2.80±0.32 mm in non-smokers, showing no statistically significant difference ($p>0.05$). Similarly, bleeding on probing (BOP) values were 10.2±2.4% in smokers and 11.4±2.6% in non-smokers, and the difference was not statistically significant ($p>0.05$).

Values for GT and KTW are presented in **Table 3**. Both GT and KTW were significantly higher in smokers (1.77±0.28 mm and 2.54±0.42 mm, respectively) compared to non-smokers (1.25±0.19 mm and 2.31±0.23 mm) ($p<0.001$; $p<0.05$).

Table 3. Comparison of gingival thickness and keratinized gingival width between groups

	S (n=15)	NS (n=15)	p
GT	1.77±0.28	1.25±0.19	0.0001
KGW	2.54±0.42	2.31±0.23	0.019*

Independent samples t-test, Mann-Whitney U test (* $p<0.05$). S: Smokers, NS: Non-smokers, GT: Gingival thickness, KGW: Keratinized gingival width

Pearson correlation analyses between GT, KTW, and clinical periodontal parameters are summarized in **Table 4**. A positive correlation was observed between GT and PI, while GT showed a negative correlation with BOP. KTW was also positively correlated with PI.

Table 4. Correlation between gingival phenotype parameters and clinical periodontal parameters

	PI	GI	BOP
GT	$r=0.42, p=0.02^*$	$r=0.10, p=0.59$	$r=-0.36, p=0.04^*$
KGW	$r=0.39, p=0.03^*$	$r=0.12, p=0.52$	$r=-0.18, p=0.34$

Pearson correlation analysis (* $p<0.05$). PI: Plaque Index, GI: Gingival Index, BOP: Bleeding on probing, GT: Gingival thickness, KGW: Keratinized gingival width

DISCUSSION

This study was conducted to evaluate the effects of cigarette smoking on the gingival phenotype and clinical periodontal parameters. According to the findings, both GT and KTW values were significantly higher in smokers compared to non-smokers. In addition, the PI was found to be significantly higher among smokers, whereas no statistically significant differences were observed between the groups in terms of GI, PD or BOP.

The gingival phenotype is defined by the buccolingual dimension of the gingiva and the width of the keratinized tissue. This concept encompasses not only genetically determined tissue characteristics but also features that may be

altered by environmental factors and clinical interventions.¹⁹ A systematic review on gingival morphology categorized gingival biotypes into three main groups: thin scalloped, thick flat, and thick scalloped. GT, KTW and bone morphotype are considered the three key parameters for classifying these biotypes, and they play an essential role in the development or progression of mucogingival defects.²³

The gingival phenotype is further influenced by tissue thickness, degree of vascularization, regenerative capacity, and the immune response to inflammatory stimuli, all of which directly affect the outcome of periodontal therapy.² Among the environmental factors influencing the gingival phenotype, cigarette smoking not only exerts deleterious effects on gingival and periodontal tissues at the cellular level but also impairs the healing process, thereby reducing the response to periodontal treatment.²⁴

Nicotine and other toxic components present in cigarettes lead to a reduction in gingival vascularity, decreased cellular oxygenation, and impaired regenerative capacity of connective tissue cells.²⁵ In addition, smoking induces histopathological changes such as epithelial hyperplasia, increased keratinization, and suppression of the inflammatory response, resulting in a thickened gingival phenotype.²⁶ However, clinical studies directly investigating the association between smoking and gingival phenotype remain limited. In this context, the present study aimed to assess the effects of cigarette smoking on the gingival phenotype. Our findings demonstrated that both GT and KTW were significantly greater in smokers than in non-smokers.

In the study conducted by Birlabose et al.,²⁷ a significant increase in GT was observed among smokers. The authors suggested that nicotine may enhance cellular proliferation within gingival tissues, influencing epithelialization and potentially contributing to the thicker gingival phenotype observed in smokers.²⁸ Similarly, a study using the transgingival probing method reported that GT was significantly greater in smokers in both buccal and interdental regions.²⁹ Nevertheless, this thickening has been suggested to represent not a healthy tissue increase but a pathological thickening caused by epithelial hyperkeratosis, inflammatory edema, and fibrotic alterations.²⁵ Therefore, smoking-induced phenotypic changes should be considered as an adverse adaptation rather than a protective mechanism for tissue stability.

In the present study, the PI values were significantly higher in smokers compared to non-smokers. This finding aligns with previous literature indicating that smoking negatively affects oral hygiene and predisposes to increased plaque accumulation. Smoking has been reported to promote plaque retention on tooth surfaces by altering salivary composition and pellicle formation, thereby facilitating oral biofilm development. Furthermore, inadequate oral hygiene habits commonly observed among smokers have also been identified as a contributing factor.^{30,31}

In our study, no statistically significant differences were found between the groups in other periodontal parameters such as GI, PD, and BOP. This may be attributed to the generally healthy periodontal condition of the participants included in the study, as well as to the suppressive effect of smoking on clinical signs of gingival inflammation, which may mask inflammatory findings.³² The inclusion of young and periodontally healthy participants in this study aimed



to minimize potential confounding variables other than smoking. However, several studies have reported higher GI and BOP values in non-smokers compared to smokers.^{29,33,34} These findings have been attributed not only to the vasoconstrictive effects of smoking on the marginal gingival vasculature but also to the poorer oral hygiene frequently observed among smokers.¹⁷ In the present study, although the PI values were significantly higher in smokers, no significant difference was found between the two groups regarding GI. This result can be explained by the vasoconstrictive effects of smoking on periodontal vascular tissues and the inhibition of local inflammatory mediator release.³⁵

Limitations

Among the limitations of this study are the relatively small sample size and the lack of long-term follow-up data due to its cross-sectional design. Therefore, to better understand the effects of smoking on the gingival phenotype, future research should include larger sample sizes and multi-center, prospective studies with long-term follow-up periods.

CONCLUSION

The findings of this study demonstrate that cigarette smoking affects the gingival phenotype by increasing GT. Additionally, higher plaque accumulation was observed among smokers; however, no significant differences were detected in gingival inflammation parameters. This suggests that smoking may mask clinical signs of inflammation in periodontal tissues.

Although smokers may exhibit a clinically thick and fibrotic gingival appearance, compromised vascularization may result in reduced healing capacity and impaired host defense response. Considering smoking habits during clinical evaluation is therefore crucial for accurate assessment of periodontal health and effective planning of surgical and therapeutic procedures.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the İnönü University Non-interventional Clinical Researches Ethics Committee (Date: 18.11.2025, Decision No: 2025/8571).

Informed Consent

Written informed consent was obtained from all individual participants prior to their inclusion in the study. Participants were fully informed about the study's aims, procedures, potential risks and benefits, and their rights-including the right to withdraw at any time without consequence. All participants voluntarily signed a written informed consent form.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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Author Contributions

Concept: ÜGÖ, VET; Design: ÜGÖ, VET; Control: ÜGÖ, VET; Resources: ÜGÖ, VET; Materials: ÜGÖ, VET; Data Collection and/or Processing: ÜGÖ, VET; Analysis and/or Interpretation: ÜGÖ, VET; Literature Review: ÜGÖ, VET; Writing the Article: ÜGÖ, VET; Critical Review: ÜGÖ, VET.

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