

## Evaluation and comparison of melanin gingival hyperpigmentation in smokers and non-smokers: A comparative cross-sectional study

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### Abstract

**Background:** Gingival hyperpigmentation refers to the color of gingiva which is primarily influenced by melanin deposition in the basal and suprabasal layers of the epithelium and various other exogenous and endogenous factors. Smoking is a well-established extrinsic factor contributing to increased melanin production and thus gingival hyperpigmentation, a condition commonly termed as “smokers melanosis” Despite its clinical relevance, limited comparative data exists evaluating gingival pigmentation among smokers and non-smokers. This study aims to assess the prevalence and severity of gingival melanin pigmentation among smokers and non-smokers.

**Materials and methods:** In this comparative cross-sectional study 100 adults aged 18 years and above were divided equally into two groups: Smokers (Group I) and Non-smokers (Group II). Gingival pigmentation was assessed in the anterior labial region of the gingiva using the Dummet-Gupta Oral Pigmentation Index (DOPI). Data was analyzed using statistical tests.

**Results:** Smokers (Group I) demonstrated a higher prevalence and severity of gingival pigmentation compared to Non-smokers (Group II). The mean DOPI scores were  $[1.252 \pm 0.743]$  in smokers and  $[0.376 \pm 0.425]$  in non-smokers, with a statistically significant difference ( $p < 0.0001$ ).

**Conclusion:** Smoking is significantly associated with increased gingival melanin pigmentation. The findings highlight the need for awareness regarding the oral effects of tobacco use. Statistical data reveal a clear association between smoking habits and oral pigmentation changes. Individuals with longer duration and higher frequency of smoking tend to exhibit greater intensity of pigmentation that suggests a dose dependent relationship.

Further longitudinal studies are recommended to evaluate the reversibility of pigmentation following smoking cessation and to better understand the biological mechanisms involved in gingival pigmentation.

**Keywords:** Gingival hyperpigmentation, melanin, smoker’s melanosis, DOPI Index, tobacco, oral health, comparative cross-sectional study

### Introduction

Gingival pigmentation refers to the discoloration of the gingival tissues caused by the deposition of various endogenous and exogenous pigments within the oral epithelium and connective tissue. The normal color of gingiva varies among individuals and is influenced by several factors such as vascular supply, epithelial thickness, degree of keratinization and the presence of pigments. Among all pigments, melanin is considered the most common endogenous pigment responsible for physiological oral pigmentation. Melanin is synthesized by melanocytes located in the basal and suprabasal layers of the epithelium and is transferred to surrounding keratinocytes through melanosomes. The degree of melanin pigmentation may differ according to race, genetic predisposition and environmental influences. Although physiological pigmentation is benign in nature, excessive melanin deposition can lead to gingival hyperpigmentation, which may become an esthetic concern for many individuals [1].

Melanin pigmentation of the gingiva may occur due to physiological causes or may be associated with systemic conditions, medications, syndromes and environmental factors. Among the various extrinsic factors, tobacco

smoking has been identified as one of the most important contributors to increased gingival pigmentation. Tobacco smoke contains nicotine and polycyclic amines such as benzopyrene, which stimulate melanocytic activity and melanin synthesis as a protective mechanism against noxious agents. This increased melanocytic stimulation results in diffuse brown to black pigmentation of the gingiva, commonly referred to as “smokers melanosis”. The pigmentation is usually observed more prominently on the anterior labial gingiva and is reported to be directly related to the duration and frequency of smoking habits [2].

Smoking is recognized as a major contributory factor in the development of a variety of oral health conditions, including periodontal diseases, oral mucosal abnormalities, oral malignancies, and alterations in oral pigmentation. Continuous exposure of the oral tissues to the toxic constituents of cigarette smoke can induce both morphological and physiological changes within the oral environment. Among these manifestations, gingival hyperpigmentation is often regarded as one of the earliest clinically observable effects of tobacco use. Consequently, its presence may provide valuable evidence of chronic tobacco exposure during routine dental examination. Several

investigations have reported a considerably greater occurrence of oral pigmentation among smokers than non-smokers, supporting a strong relationship between smoking and increased melanin accumulation in gingival tissues. In addition, previous research has documented a reduction in pigmentation following smoking cessation, suggesting that smoker's melanosis may be partially reversible when tobacco exposure is discontinued [3].

Reliable evaluation of gingival pigmentation depends on the use of standardized clinical indices that allow consistent assessment and comparison across individuals and studies. The Dummett–Gupta Oral Pigmentation Index (DOPI) is among the most frequently employed tools for this purpose. This index categorizes gingival pigmentation according to its intensity, ranging from the absence of pigmentation to pronounced clinical pigmentation. Owing to its ease of application, reproducibility, and clinical relevance, DOPI has been extensively utilized in epidemiological research and comparative clinical studies. The use of such objective assessment methods enables clinicians to quantify pigmentation severity and explore potential etiological factors, including the influence of smoking on oral soft tissues [4].

The pathophysiology of smoker's melanosis is deeply rooted in the oxidative stress induced by the inhalation of toxic chemicals. The oral epithelium reacts to this chronic irritant through the upregulation of melanin production, which serves as a physiological defense mechanism to neutralize free radicals. This mechanism explains why clinical pigmentation is often more pronounced in the anterior labial gingiva, which is the site of maximum direct contact with cigarette smoke during inhalation [5].

Gingival hyperpigmentation serves as an objective, visible marker of systemic tobacco exposure that can be utilized to facilitate patient engagement in smoking cessation. By documenting the extent of pigmentation, clinicians can provide a tangible, visual representation of oral health impact. Longitudinal observations have shown that the cessation of tobacco use leads to a gradual reduction in pigmentation, reinforcing the reversible nature of this condition and serving as a motivational metric for the patient's recovery progress [6].

Clinical evaluation of gingival pigmentation plays an important role in both diagnosis and treatment planning in dental practice. Several indices have been developed to assess the degree of oral pigmentation, among which the Dummett–Gupta Oral Pigmentation Index is widely accepted because of its simplicity and reproducibility. Accurate assessment of pigmentation severity assists clinicians in comparing pigmentation patterns among different population groups and evaluating treatment outcomes following smoking cessation or depigmentation therapy [7].

In recent years, increasing emphasis has been placed on oral esthetics and patient awareness regarding gingival appearance. Gingival hyperpigmentation, although medically benign, may negatively affect smile esthetics and psychological confidence in certain individuals. As a result, awareness regarding the etiological role of tobacco smoking in gingival discoloration has become important not only from a preventive perspective but also in improving patient education and motivation toward tobacco cessation [8].

Despite the availability of studies on smoker's melanosis, there exists limited data that compares the severity and

distribution of gingival hyperpigmentation among smokers and non-smokers. There are also limited studies that demonstrate the influence of frequency, duration and passive smoking on gingival hyperpigmentation. Therefore, further studies are required to understand better the relationship between tobacco smoking and gingival melanin pigmentation. The present study was undertaken to evaluate and compare melanin gingival hyperpigmentation among smokers and non-smokers using the Dummett–Gupta Oral Pigmentation Index.

## Methodology

### Study Design and Setting

The present study was a comparative cross-sectional study conducted in the Department of Public Health Dentistry at M.A. Rangoonwala College of Dental Sciences and Research Centre, Pune. The study was carried out after obtaining approval from the Institutional Ethics Committee (IEC) and all infection control protocols were strictly followed throughout the study period. The null hypothesis ( $H_0$ ) for the present study stated that there would be no significant difference in gingival melanin pigmentation between smokers and non-smokers.

### Study Participants

A total of 100 participants aged 18 years and above were recruited for the study and divided into two groups:

**Group I:** Smokers ( $n = 50$ )

**Group II:** Non-smokers ( $n = 50$ )

Participants were selected from patients visiting the outpatient department of Public Health Dentistry. Individuals with a history of systemic diseases affecting oral pigmentation, previous gingival depigmentation therapy, medication-induced pigmentation, or any other oral mucosal pathology affecting gingival color were excluded from the study. Participants who were unwilling to participate were also excluded.

All participants were informed about the nature and purpose of the study and written informed consent was obtained prior to clinical examination.

### Sample size

The present comparative cross-sectional study was conducted among 100 participants, comprising 50 smokers and 50 non-smokers. The sample size was calculated based on a comparison of mean Dummett–Gupta Oral Pigmentation Index (DOPI) scores between smokers and non-smokers using the formula:

$$n = \frac{2\sigma^2(Z_{\alpha/2} + Z_{\beta})^2}{d^2}$$

Based on previous studies evaluating gingival pigmentation among smokers and non-smokers, the calculated minimum sample size was determined to be adequate for detecting statistically significant differences between the two groups. To improve the reliability of the study, a total sample size of 100 participants was included.

### Sampling Method

Participants who fulfilled the inclusion criteria were selected using convenience sampling.

**Study procedure**

Participants were interviewed using a structured questionnaire to record demographic details including age, gender, smoking status, frequency of smoking, duration of smoking habit, quantity of tobacco consumption per day, passive smoking exposure, and self-awareness regarding gingival discoloration.

Clinical examination was carried out under adequate illumination using a mouth mirror and periodontal probe. Gingival melanin pigmentation was assessed on the attached gingiva, particularly in the anterior region, using the Dummett–Gupta Oral Pigmentation Index (DOPI).

The DOPI scoring criteria used were as follows:

- **Score 0:** No clinical pigmentation (pink gingiva)
- **Score 1:** Mild clinical pigmentation (light brown color)
- **Score 2:** Moderate clinical pigmentation (medium brown or mixed pink and brown color)
- **Score 3:** Heavy clinical pigmentation (deep brown or bluish-black color)

Participants in the smoker group were further categorized according to smoking frequency, duration of tobacco use, quantity of tobacco consumed per day, and passive smoking exposure to evaluate their association with gingival pigmentation severity.

All examinations were performed by a single trained examiner to minimize inter-examiner variability and ensure consistency in recording clinical findings.

**Statistical analysis**

The collected data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) version XX (IBM Corp, Armonk, NY, USA).

Descriptive statistics were expressed as mean, standard deviation (SD), frequencies, and percentages.

Normality of data distribution was assessed using the Shapiro–Wilk test. Intergroup comparison of mean DOPI scores between smokers and non-smokers was performed using the independent t-test. Comparison of categorical variables was carried out using the Chi-square test.

For subgroup analysis among smokers, the Kruskal–Wallis test was used to compare DOPI scores according to smoking frequency, duration, and quantity of tobacco use. Correlation between smoking-related variables and gingival pigmentation was evaluated using Spearman’s correlation coefficient.

A p-value of less than 0.05 was considered statistically significant.

**Result**

**Table 1:** Demographic Characteristics and Clinical Findings of Study Participants

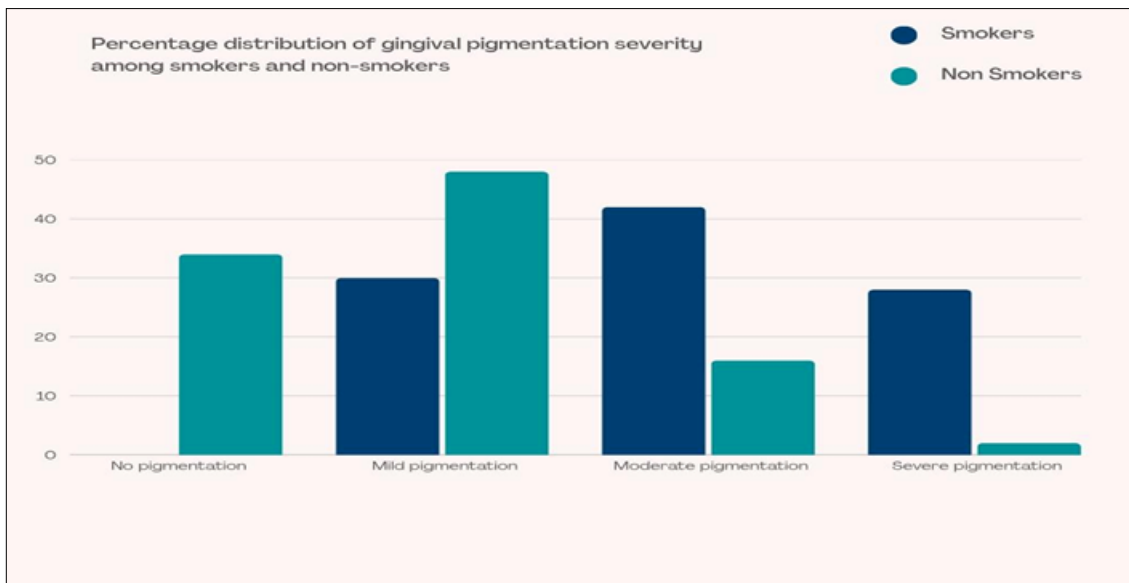
Parameters	Group I	Group II
Age (years)		
Mean ± SD	40.88 ± 13.22	39.82 ± 11.93
Minimum	19	19
Maximum	72	65
Gender Distribution		
Females - n (%)	5 (10%)	8 (16%)
Males- n (%)	45 (90%)	42 (84%)
Means DOPI ± SD	1.252 ± 0.743	0.376 ± 0.425

Age was compared using the Mann-Whitney U test, and sex was compared using the Chi-square test (p < 0.0001 which is considered statistically significant)

**Table 2:** Distribution of DOPI Scores - Smokers vs Non-Smokers (N=100)

DOPI Category	Score	Smokers Percentage	Non-Smokers Percentage	Statistical Result
No pigmentation	0	0.0%	34.0%	p < 0.0001
Mild pigmentation	1	30.0%	48.0%	
Moderate pigmentation	2	42.0%	16.0%	
Heavy pigmentation	3	28.0%	2.0%	
Total	0-3	100%	100%	

Distribution was compared using the Mann - Whitney U test (p < 0.0001 which is considered statistically significant)



**Fig 1:** Distribution of DOPI score among smokers and non-smokers

**Table 3:** Correlation of DOPI Scores with Smoking Frequency Among Smokers (n=50)

Smoking Frequency	Percentage	Mean DOPI ± SD	Statistical Result
Daily (Score 1)	50.0%	1.640 ± 0.707	p < 0.0005
Occasionally (Score 2)	36.0%	1.000 ± 0.581	
Rarely (Score 3)	10.0%	0.560 ± 0.365	
Never (Score 4)			

Distribution compared by Kruskal-Wallis test (p < 0.0005 which is considered statistically significant)

**Table 4:** Correlation of DOPI Scores with Duration of Tobacco Use Among Smokers

Smoking Duration	Percentage	Mean DOPI ± SD	Statistical Result
< 6 months	16.0%	0.625 ± 0.552	p < 0.0003
6 months - 2 years	44.0%	1.118 ± 0.547	
2-5 years	24.0%	1.783 ± 0.720	
5-10 years	12.0%	1.833 ± 0.816	
> 10 years	4.0%	0.300 ± 0.000	

Distribution compared by Kruskal-Wallis test (p < 0.0003 which is considered statistically significant)

**Table 5:** Mean DOPI With Respect to Daily Tobacco Consumption Among Smokers

Tobacco Units/Day	Percentage	Mean DOPI ± SD	Statistical Result
1-5 units/day	54.0%	1.163 ± 0.552	p < 0.0001
6-10 units/day	18.0%	2.156 ± 0.678	
11-15 units/day	6.0%	1.867 ± 0.231	
16-20 units/day	2.0%	0.500 ± 0.000	
>20 units/day	20.0%	0.570 ± 0.397	

Distribution compared by Kruskal-Wallis test (p < 0.0001 which is considered statistically significant)

**Table 6:** Mean DOPI Scores among Smokers and Non-Smokers Through Passive Smoking

Group/Passive Smoking	Percentage	Mean DOPI ± SD	Statistical Result
Smokers			
Exposed (Yes)	42.0%	1.543 ± 0.720	p < 0.0152
Not Exposed (No)	58.0%	1.041 ± 0.697	
Non-Smokers			
Exposed (Yes)	40.0%	0.565 ± 0.318	p < 0.0002
Not Exposed (No)	60.0%	0.250 ± 0.445	

Distribution compared by Mann-Whitney U test (two-tailed) (p < 0.0152 and p < 0.0002 for Smokers and Non-Smokers respectively which is considered statistically significant)

**Table 7:** Self-Reported Discoloration Awareness vs. Measured DOPI - Smokers and Non-Smokers

Self Reported Awareness	Percentage	Mean DOPI ± SD	Statistical Result
Smokers			
Did Not Notice (No)	36.0%	1.683 ± 0.520	p < 0.0014
Noticed Discoloration (Yes)	36.0%	1.083 ± 0.926	
Unsure	28.0%	0.914 ± 0.431	
Non-Smokers			
Did Not Notice (No)	28.0%	0.621 ± 0.500	p < 0.0075
Noticed Discoloration (Yes)	64.0%	0.266 ± 0.374	
Unsure	8.0%	0.400 ± 0.115	

Distribution compared by Kruskal-Wallis test (p < 0.0014 and p < 0.0075 among Smokers and Non-Smokers which is considered statistically significant)

A total of 100 participants were included in the present comparative cross-sectional study, comprising 50 smokers (Group I) and 50 non-smokers (Group II).

Table 1 shows that the mean age of participants in the smoker group was 40.88 ± 13.22 years, while the mean age in the non-smoker group was 39.82 ± 11.93 years. Male participants constituted the majority in both groups. The mean Dummet-Gupta Oral Pigmentation Index (DOPI) score was significantly higher among smokers (1.252 ± 0.743) compared to non-smokers (0.376 ± 0.425), indicating increased gingival melanin pigmentation among individuals with smoking habits.

As shown in table 2, the distribution of DOPI scores indicated that none of the smokers presented with an

absence of pigmentation, whereas 34% of non-smokers showed no pigmentation. Moderate and heavy pigmentation were predominantly observed among smokers, with 42% exhibiting moderate pigmentation and 28% exhibiting heavy pigmentation. In contrast, the majority of non-smokers demonstrated either no pigmentation or only mild pigmentation. The difference in pigmentation distribution between the two groups was found to be statistically significant (p < 0.0001).

Table 3 shows that analysis of smoking frequency revealed that participants with daily smoking habits demonstrated the highest mean DOPI scores (1.640 ± 0.707), whereas occasional and rare smokers showed comparatively lower scores. A statistically significant association was observed

between smoking frequency and severity of gingival pigmentation ( $p = 0.0005$ ), suggesting that increased exposure to tobacco smoke contributes to greater melanin deposition within gingival tissues.

Similarly, Table 4 shows that the duration of tobacco use was significantly associated with gingival pigmentation severity. Participants with smoking duration between 2–5 years and 5–10 years demonstrated higher DOPI scores compared to those with shorter duration of smoking. Statistical analysis showed a significant relationship between duration of smoking and gingival pigmentation ( $p = 0.0003$ ), indicating a cumulative effect of long-term tobacco exposure on melanocytic activity.

Table 5 demonstrates that evaluation of daily tobacco consumption showed that individuals consuming greater numbers of tobacco units per day exhibited comparatively higher pigmentation scores. Participants consuming 6–10 tobacco units daily showed the highest mean DOPI score ( $2.156 \pm 0.678$ ). The association between quantity of tobacco consumed and gingival pigmentation was statistically significant ( $p < 0.0001$ ), supporting a dose-dependent relationship between tobacco use and pigmentation intensity.

Table 6 shows how passive smoking exposure also appeared to influence gingival pigmentation. Smokers exposed to passive smoking demonstrated higher mean DOPI scores ( $1.543 \pm 0.720$ ) compared to smokers without passive exposure ( $1.041 \pm 0.697$ ). Among non-smokers, individuals exposed to passive smoking showed greater pigmentation scores than those not exposed. These findings were statistically significant among both smokers ( $p = 0.0152$ ) and non-smokers ( $p = 0.0002$ ).

Assessment of self-awareness regarding gingival discoloration, as depicted in Table 7, showed that several participants with clinically evident pigmentation were either unaware of the discoloration or uncertain about its presence. Among smokers, participants who did not notice discoloration exhibited comparatively higher mean DOPI scores than those who noticed pigmentation. Similar findings were observed among non-smokers. Statistical analysis demonstrated significant associations between self-reported awareness and measured DOPI scores in both groups (smokers:  $p = 0.0014$ ; non-smokers:  $p = 0.0075$ ).

Overall, the results of the present study demonstrated that gingival melanin hyperpigmentation was significantly more prevalent and severe among smokers compared to non-smokers. Increased smoking frequency, longer duration of tobacco use, greater quantity of tobacco consumption, and passive smoking exposure were all associated with higher gingival pigmentation scores. These findings suggest a strong association between tobacco exposure and gingival melanin pigmentation.

The null hypothesis ( $H_0$ ) stated that there would be no significant difference in gingival melanin pigmentation between smokers and non-smokers. However, the findings of the present study demonstrated statistically significant differences in mean DOPI scores and pigmentation distribution between the two groups ( $p < 0.0001$ ). Smokers exhibited markedly higher gingival pigmentation compared to non-smokers. Therefore, the null hypothesis was rejected, and the alternative hypothesis ( $H_1$ ) was accepted, indicating that smoking has a significant association with increased gingival melanin hyperpigmentation.

## Discussion

The present study aimed to evaluate and compare gingival melanin hyperpigmentation in smokers and non-smokers using the Dummett–Gupta Oral Pigmentation Index (DOPI). The results demonstrated that smokers exhibited substantially higher levels of gingival pigmentation than non-smokers, suggesting a significant relationship between tobacco consumption and increased melanin accumulation in gingival tissues.

Melanin pigmentation of the gingiva is mainly determined by the activity of melanocytes located within the basal layer of the oral epithelium. While physiological pigmentation is a normal finding in many individuals, environmental influences such as tobacco smoking can modify both its extent and intensity. Cigarette smoke contains compounds including nicotine and benzopyrene that may stimulate melanocytic activity, leading to enhanced melanin synthesis as a defense mechanism against chemical irritation. Consequently, this increased production of melanin manifests clinically as smoker's melanosis. These findings are consistent with the observations of Hedin, who was among the first to report the link between smoking and pigmentation changes within the oral cavity [2].

A significantly greater mean DOPI score was recorded among smokers compared with non-smokers. The majority of smokers presented with moderate to severe pigmentation, whereas most non-smokers displayed either minimal pigmentation or none at all. This indicates that smoking is associated not only with the occurrence of gingival pigmentation but also with an increase in its severity. Similar outcomes were documented by Almas and Sadig, who observed more pronounced and widespread pigmentation in smokers than in non-smokers [9].

The findings further revealed that the severity of pigmentation increased with the duration of smoking. Individuals with a longer smoking history demonstrated higher DOPI scores than those who had smoked for a shorter period. This trend suggests a cumulative effect of prolonged exposure to tobacco smoke on gingival tissues. Hedin and Axéll likewise reported greater pigmentation among long-term smokers, emphasizing the influence of sustained tobacco exposure on melanocyte stimulation and activity [10].

Participants who were exposed to environmental tobacco smoke, regardless of whether they were smokers or non-smokers, demonstrated relatively higher gingival pigmentation scores compared to those without such exposure. While the detrimental systemic consequences of passive smoking have been extensively documented, its influence on oral pigmentation has received comparatively little attention. The present findings support previous observations by Hanioka *et al*, who identified a positive association between passive tobacco smoke exposure and increased oral melanin pigmentation [11].

The cosmetic impact of gingival hyperpigmentation is another factor that warrants consideration, especially among individuals who place importance on dental appearance and smile aesthetics. Although smoker's melanosis is typically a harmless and symptom-free condition, noticeable gingival discoloration can negatively influence self-esteem and overall satisfaction with one's appearance. With the growing emphasis on esthetic dentistry, interest in depigmentation techniques—including scalpel-based procedures, laser treatment, and cryosurgery—has increased considerably.

Consequently, smoking cessation should be encouraged not only to improve oral and general health but also to help preserve desirable gingival aesthetics [12].

Variations related to age and gender may additionally affect the extent and presentation of gingival pigmentation in smokers. Younger individuals who begin smoking early and continue the habit for extended periods may develop pigmentation sooner because of heightened melanocyte responsiveness. In contrast, older smokers may exhibit more generalized or diffuse pigmentation patterns as a result of long-term tobacco exposure. Although demographic variables were not specifically assessed in the current study, they may partially explain differences in pigmentation severity and should be explored in future research examining tobacco-associated oral pigmentation [13].

An additional consideration is the potential association between smoking-related gingival pigmentation and periodontal health. Tobacco use has been shown to reduce tissue vascularity, modify immune function, and disrupt normal gingival metabolism, all of which contribute to periodontal breakdown. As a result, gingival pigmentation in smokers may occur alongside other smoking-related periodontal alterations. This highlights the importance of conducting a thorough periodontal assessment in individuals with a history of tobacco use [14].

The behavioral and psychological consequences of smoking-related oral alterations should also be considered. When patients become aware of gingival discoloration during routine dental visits, they may be more receptive to counseling aimed at reducing or discontinuing tobacco use. Previous studies have suggested that visually demonstrating the oral manifestations of smoking can enhance patient motivation and improve adherence to tobacco cessation recommendations. For this reason, gingival pigmentation can be utilized as a valuable clinical tool in patient education and smoking cessation initiatives [15].

The current study additionally evaluated participants' perception of gingival discoloration. It was observed that many smokers with clinically evident pigmentation either failed to recognize the discoloration or regarded it as insignificant. Because smoker's melanosis typically develops without any symptoms, affected individuals may remain unaware of the condition despite its noticeable clinical appearance. Nevertheless, gingival pigmentation represents an early and readily identifiable marker of tobacco exposure that can be detected during routine oral examinations.

A notable strength of this investigation was the application of the Dummett–Gupta Oral Pigmentation Index (DOPI), which offered a practical and standardized approach for grading the severity of gingival pigmentation. Furthermore, the study considered several tobacco-related variables, including smoking duration, frequency of smoking, quantity of tobacco consumed, and exposure to passive smoking. Evaluating these factors provided a more comprehensive understanding of the determinants associated with gingival pigmentation.

Despite these strengths, several limitations must be considered when interpreting the results. The cross-sectional nature of the study restricts the ability to establish a definitive cause-and-effect relationship between smoking and gingival pigmentation. In addition, the sample was drawn from a single institution, which may limit the applicability of the findings to broader populations. Other

potentially influential factors, such as skin pigmentation, oral hygiene status, and the use of alternative tobacco products, were not investigated in detail. Future longitudinal research involving larger and more diverse populations may help clarify the progression of smoker's melanosis and evaluate the extent to which pigmentation regresses following smoking cessation.

To summarise, the findings of the present study support a significant association between tobacco smoking and increased gingival melanin hyperpigmentation. The severity of pigmentation appeared to rise with greater tobacco exposure, particularly with increased smoking frequency, longer duration of use, and higher quantities of consumption. These observations suggest that gingival pigmentation may serve as a practical clinical marker of tobacco exposure and reinforce the importance of incorporating tobacco cessation awareness and counseling into routine dental practice.

### Conclusion

Within the limitations of the present study, it can be concluded that gingival melanin hyperpigmentation is significantly more prevalent and severe among smokers compared to non-smokers. Tobacco smoking appears to stimulate melanocyte activity, resulting in increased melanin production and deposition within the gingival epithelium.

The study findings support the concept of "smoker's melanosis," demonstrating a clear association between smoking habits and oral pigmentation changes. Individuals with a longer duration and higher frequency of smoking tending to exhibit greater pigmentation intensity, which suggests a dose-dependent relationship.

Although gingival pigmentation is generally considered a benign condition, its presence may serve as an important clinical indicator of tobacco exposure and smoking-related oral alterations. Early identification of these changes during routine dental examinations may help dental professionals counsel patients regarding the harmful effects of tobacco use and encourage smoking cessation.

The findings of this study also emphasize the importance of creating awareness about the oral manifestations of smoking, not only among patients but also within public health dentistry programs. Further longitudinal studies with larger sample sizes are recommended to evaluate the reversibility of pigmentation following smoking cessation and to better understand the biological mechanisms involved.

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