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Prevalence Study of Melanin Pigmentation Affecting Lip and Gingiva by Cigarette Smoking In Central India Population

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ABSTRACT

Background: Diffuse melanosis of labial mucosa, the anterior facial maxillary and mandibular gingivae, buccal mucosa, lateral tongue, palate, and floor of the mouth is usually seen among cigarette smokers. Most smokers (including heavy smokers) usually fail to show such changes. However, it is probable that in certain individuals, melanin synthesis is stimulated by tobacco smoke products subjects displaying lip and gingival pigmentation.

Material and methods: We have studied 223 individuals of either gender in an institute situated in the central India. We found 73% and 87% respectively, were current smokers, whereas 33% and 27% of individuals lacking pigmentation were current smokers respectively. Odds ratios of current smoking relative to lip and gingival pigmentation were 5.6 (95% confidence interval: 2.8–11.1) and 17.0 (8.1–36.0) respectively.

Results and interpretation: Daily consumption, duration of smoking and lifetime exposure exhibited significant correlation with scores of lip and gingival pigmentation (P< 0.0001). Odds ratios increased in lip and gingival pigmentation upon exposure. In current smokers, scores of lip and gingival pigmentation demonstrated meaningful correlation (P< 0.0001); moreover, 95% of participants with lip pigmentation were positive for gingival pigmentation.

Conclusion: These results indicated the presence of a striking association between smoking and pigmentation in the lip and gingiva, which was stronger with respect to gingival pigmentation. Health professionals could educate smokers, utilizing visible symptoms in the lip and gingiva.

Keywords: Lip; Gingiva; Pigmentation; Melanin; Smoking, Nicotine, Melanosome, Melanocytes.

INTRODUCTION

Brownish or black discoloration, i.e. melanin pigmentation, which occurs as a solitary unit or as a continuous ribbon in gingiva, is distinguishable from other forms of oral pigmentation [8,13,14,15]. The prevalence of melanin pigmentation in the gingiva differs by ethnic group, which is indicative of a hereditary connection[4, 6, 17, 18, 23, 25, 29-31].

Gingival pigmentation is evident in subjects receiving anti-malarial drugs[12, 20]; however, this phenomenon is rare. Melanin pigmentation is caused by melanin granules in gingival tissue, which are produced in melanosomes of melanocytes[29]. Melanin is synthesized from tyrosine and dihydroxyphenylalanine (DOPA) via dopaquinone by the oxidation of tyrosinase[9].

Diffuse melanosis of labial mucosa, the anterior facial maxillary and mandibular gingivae, buccal mucosa, lateral tongue, palate, and floor of the mouth is usually seen among cigarette smokers. Indeed, among dark-skinned individuals who normally exhibit physiologic pigmentation, smoking stimulates a further increase in oral pigmentation.[1] The pigmented areas are brown, flat, and irregular; some are even geographic or map-like in configuration.[2]

The mechanism by which smoking induces the pigmentation remains unknown. Smokeless tobacco (snuff) does not appear to be associated with an increase in oral melanosis. Thus, it is possible that one or more of the chemical compounds incorporated within cigarettes, rather than the actual tobacco, may be causative. Another possibility is that the heat of the smoke may stimulate the pigmentation. Epidemiologic studies suggest that oral melanosis increases prominently during the first year of smoking[1, 2]. If there is a reduction in smoking, the pigmentation may eventually resolve. Histologically, basilar melanosis with melanin incontinence is observed. Unlike other smoking-related oral pathologies, smoker's melanosis is not a pre-neoplastic condition.

Alcohol has also been associated with increased oral pigmentation. In alcoholics, the posterior regions of the mouth, including the soft palate, tend to be more frequently pigmented than other areas. It has been suggested that alcoholic melanosis may be associated with a higher risk of cancers of the upper aero digestive tract.[16, 17, 25]

Diffuse or patchy melanotic pigmentation is also characteristically associated with oral submucous fibrosis[13, 14, 15].

Oral pigmentations are increased significantly in heavy smokers. In one investigation of more than 31.000 Caucasians, 1.5% of tobacco smokers exhibited areas of melanin pigmentation compared with 3% among those not using tobacco. In another study of an ethnically pigmented population, smokers had more oral surfaces exhibiting melanin pigmentation.[25]

Melanin pigmentation in the skin exerts a well-known protective effect against ultraviolet (UV) damage. Investigations of melanocytes located away from sun-exposed areas have shown the ability of melanin to bind to noxious substances. Exposure to polycyclic amines (such as nicotine and the benzopyrenes) has been shown to stimulate melanin production by melanocytes that also are known to bind strongly to nicotine.[6,11,26] It has been suggested that melanin production in the oral mucosa of smokers serves as a protective response against some of the harmful substances in tobacco smoke. This concept is supported by the findings in "reverse" smokers who smoke with the lit end of the cigarette inside the mouth and who demonstrate heavy melanin pigmentation on the palate[19, 23, 26].

Gingival pigmentation has been examined in terms of its association with smoking in various countries, including Israel[30], Sweden[6], Japan[4, 26], Thailand and Malaysia[28], Turkey[31] and India[23]. Excessive melanin pigmentation is correlated with smoking; thus, smoking may stimulate melanin production in gingival tissue. The stimulatory effect could occur as a result of the high-affinity function of nicotine[11] and benzpyrene[22] in tobacco smoke relative to melanin. Additionally, a dose–response relationship was detected[4, 6]. Disappearance of gingival pigmentation was observed following reduction in smoking[28, 29]. These findings suggest a causal association between smoking and gingival pigmentation; additionally, the specific label of smoker's melanosis was assigned[27].

Although any mucosal surface may be affected, smoker's melanosis most commonly affects the anterior facial gingiva (Figure 8- 46). Most people affected by this condition are cigarette users. In contrast, pipe smokers frequently exhibit pigmentations located on the commissural and buccal mucosa. Reverse smokers show alterations of the hard palate. The areas of pigmentation significantly increase during the first year of smoking and appear correlated to the number of cigarettes smoked each day. A higher frequency is seen in females, and it has been suggested that female sex hormones exert a synergistic effect when combined with smoking. Reports from Sweden, Germany, and Japan have shown tobacco smoking to be the most common cause for mucosal pigmentation in light skinned adult populations[1, 2, 23].

Gingival pigmentation is visible in the labial area of anterior teeth[6, 23, 26, 27]. Due to specific localization of gingival pigmentation, smokers may be aware of the health consequences of smoking relative to their own bodies following proper education by health professionals. In a manner similar to gingiva, lip, which is also readily visible, may produce melanin. To the best of our knowledge, no data regarding the association between smoking and lip pigmentation have appeared in the literature since the relationship was first described in a comprehensive study of oral pigmentation[6, 29]. The objective of this study was to investigate the association of lip pigmentation with smoking and gingival melanin pigmentation.

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MATERIAL AND METHODS

Digital photos of lip and the labial aspects of frontal teeth were taken of each individual in routine Dental OPD hours in the period of February 2018 to May 2018 which was produced in a standardized manner by digital camera (SAMSUNG, 13MP camera). The individuals were medically healthy, habitant of smoking more than 2 years and residents of Durg district. Digital images were stored on electronic media, followed by subsequent reproduction on a computer display. These reproductions exhibited size similar to that of the actual mouth. The number of females in the workplace and the smoking rate among females in Japan are small in comparison to those of males. Finally, photos of 223 males (32.7 ± 9.9 years of age, average ± s.d.) were used for analyses.

Lip pigmentation was scored dichotomously (0, 1) for existence of diffuse form of black or brownish discoloration in the vermilion border. Pigmentation was scored in individual sextant of the lip; subsequently, the total score was calculated. This study first addressed lip pigmentation in relation to smoking in a population of certain size; as a result, we examined the reliability of the classification of lip pigmentation. Assessment of pigmentation was calibrated by two examiners employing representative photos. The examiners then evaluated 240 sections of lips in 40 photos (six sextants per individual).

Gingival pigmentation was scored in each jaw according to the classification of Melanin Index[27] (Figure 1). The index classified pigmentation as follows: 0, no pigmentation; 1, one or two solitary units of pigmentation in papillary gingiva without the formation of a continuous ribbon between solitary units; 2, more than three units of pigmentation in papillary gingiva without the formation of a continuous ribbon; 3, one or more short continuous ribbons of pigmentation; and 4, one continuous ribbon including the entire area between canines. Total scores of upper and lower jaws were used for analysis.

Observations of lip and gingival pigmentation were performed separately. Smoking status was withheld from the examiner of pigmentation. Smoking status was defined with a questionnaire: CS denotes an individual who currently smokes more than 100 total pieces; FS describes an individual who previously smoked more than 100 total pieces but does not smoke currently; NS refers to an individual who has never smoked or who had smoked no more than 100 total pieces.

Melanin pigmentation is a visible symptom; thus, smokers could readily recognize the adverse effect of smoking. If CS could be identified on the basis of lip or gingival pigmentation, smokers may actually experience the negative effect of smoking prior to onset of a serious illness attributable to smoking. Therefore, the potential of pigmentation as a screening measure of smoking status was examined. Generally, screening tests are utilized for early detection of non-apparent disease whereas dichotomous classifications, such as negative' and positive' functions, serve to distinguish corresponding disease status. In the present study, two categories,

NS and CS were employed for the evaluation of smoking status with respect to sensitivity and specificity[7]. Disappearance of pigmentation was observed following reduction of smoking[28, 29]; additionally, other variables, such as duration of cessation, may influence results of the evaluation. Consequently, FS was excluded from evaluation. The ethical clearance was approved by institutional ethical committee. Informed consent was obtained from all subjects prior to the study. Associations in distribution between the existence of pigmentation and smoking status and between levels of lip and gingival pigmentation were evaluated with the chi-square test. Relationships between pigmentation scores and levels of exposure to smoking were assessed using the Spearman rank correlation. Difference in mean pigmentation scores between each category of smoking exposure and the reference (NS) was examined with the Dunnett test for multiple comparisons with contrast variable. Statistical significance was set at $P < 0.05$.

RESULTS

Among 223 subjects, 75 (69%), 121 (59%) and 23 (13%) were NS, CS and FS respectively (Table 1). Lip and gingival pigmentation was apparent in 157 (67%) and 109 (53%) participants respectively. Prevalence of pigmentation was compared according to the smoking status. FS were excluded in the comparison, as disappearance of pigmentation was

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observed following reduction of smoking (17). 68% of subjects exhibiting lip pigmentation were CS; in contrast, 35% of subjects lacking lip pigmentation were CS.

In the case of gingival pigmentation, 91% and 31% were CS among individuals with and without pigmentation respectively. To examine the potential of melanin pigmentation as screening test for CS, sensitivity and specificity were calculated. Sensitivity and specificity of the pigmentation test for CS were 0.83 and 0.53 based on the evaluation of lip, and 0.80 and 0.81 based on that of gingiva respectively.

Scores, prevalence's and odds ratios (ORs) adjusted by age of lip and gingival pigmentation were summarized by levels of exposure to smoking including smoking status (Table 2). Mean scores of lip pigmentation in CS were markedly higher than that in NS; however, mean scores of lip pigmentation in FS were similar to that in NS. Mean scores of gingival pigmentation were significantly higher in FS and CS than in NS. ORs of CS in lip and gingival pigmentation were 6.1 (95% confident interval 2.7–11.9) and 16.8 (7.9–35.9), respectively, which differed significantly from those of NS. The difference in prevalence of lip pigmentation between FS and NS was not meaningful OR= 1.6 (0.7–4.1). OR of FS in terms of gingival pigmentation was 5.1 (1.9–13.5), which was significantly different from that of NS.

Lip and gingival pigmentation were compared with respect to levels of exposure in CS involving three types of indices: daily consumption, duration of smoking and lifetime exposure. Correlation coefficients between scores of pigmentation and exposure to smoking were 0.345, 0.399 and 0.356 in lip, and 0.504, 0.670 and 0.658 in gingiva respectively ($P < 0.0001$). NS served as a reference. Mean scores of lip pigmentation for each category of exposure were also higher than those in NS, although differences were not meaningful in the minimum categories of duration of smoking and lifetime exposure. Mean score of gingival pigmentation for each level of daily consumption was approximately nine times greater than that of the corresponding score in NS. This trend was similar, seven to 11 times that of NS, in other categories of exposure. ORs in lip and gingival pigmentation were significantly higher than the reference values in all categories of each index of exposure ORs in lip and gingival pigmentation increased in accordance with the level of exposure to smoking in all indices.

Levels between lip and gingival pigmentation were compared in CS and NS (Table 3). In CS, the correlation in levels between lip and gingival pigmentation was significant ($P < 0.0001$). Gingival pigmentation was absent in 81% of those subjects lacking lip pigmentation.

Ninety-five per cent of subjects displaying lip pigmentation demonstrated gingival pigmentation. In NS, no meaningful association was detected in terms of levels between lip and gingival pigmentation ($p = 0.2112$). 94% of subjects lacking lip pigmentation exhibited no pigmentation in gingiva. However, gingival pigmentation was evident in 41% of those participants characterized by lip pigmentation.

DISCUSSION

Although meaningful correlations between smoking and gingival pigmentation have been demonstrated, the levels of association were not comparable to common measures in different populations. The results of the present study confirmed this relationship and revealed the level of association employing ORs: 5.6 for lip pigmentation and 17.0 for gingival pigmentation. An OR exceeding three is indicative of a relationship that is readily recognized in routine practice; consequently, smoking may be strongly connected to lip and gingival pigmentation. The powerful effects of tobacco smoke may be supported by findings pertaining to the oral effects of passive smoking. To date, periodontal disease[3], pediatric caries[5] and melanin pigmentation in the gingiva of children[26] have been described.

A dose–response relationship was also identified between levels of exposure to smoking and lip and gingival pigmentation. Furthermore, in the minimum categories of exposure to smoking, both scores and prevalence of gingival pigmentation increased relative to the level of NS and approached maximum levels. The dose–response relationship may also indicate high sensitivity of melanocytes in gingival tissue to tobacco smoking. Findings corresponding to the stimulatory mechanism of tobacco smoking in gingiva are limited[11, 22]. The highly sensitive nature of gingival melanocytes may be beneficial as young smokers could recognize a rather immediate

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untoward effect of smoking behavior shortly after initiation to smoking. This study was the first to demonstrate a dose–response relationship between smoking and lip pigmentation.

Strong correlation was detected between smoking and gingival pigmentation; however, lip pigmentation displayed weaker association. Association in terms of prevalence (OR) in lip pigmentation was not meaningful in FS. Furthermore, mean scores of lip pigmentation did not differ significantly between subjects derived from minimum categories of exposure and NS. NS exhibited higher prevalence of lip pigmentation (47%) in comparison to gingival pigmentation (19%); as a result, the weaker association of lip may be explained by differences in the characteristics of pigmentation. Lip may be more susceptible to sources of stimulation other than smoking.

Correlation in terms of levels between lip and gingival pigmentation was apparent in CS. Approximately 95% of smokers with lip pigmentation exhibited gingival pigmentation. Lip is readily observable in comparison to other body parts. Gingiva may also be readily accessible.

Visible symptoms due to smoking in different parts of the body could afford smokers an indicator potentially via which to recognize health consequences of smoking.

Furthermore, oral health professionals could elevate the awareness of smokers in dental practice. High sensitivity of gingival and lip pigmentation during screening of current smoking underscores the suitability of this method. However, clinicians should be reminded that lip and gingival pigmentation is not a flawless indicator of current smoking. Indeed, differentiation between ethnic pigmentation and smoker's melanosis is generally impossible. On the contrary, visible symptoms of lip and gingiva may lead to unnecessary anxiety among NS and FS. The present investigation did not assess gingival inflammation. The density of melanophores in the vestibular epithelium exhibited positive correlation with severity of inflammation (numbers of inflammatory cells) in the attached gingiva but not in the free gingiva[21]. However, the number of melanocytes did not correlate with visible pigmentation[24]. Furthermore, inflammatory response to plaque accumulation is suppressed in the gingiva of smokers[19], who are characterized by more apparent gingival pigmentation than non-smokers. Thus, the relationship between gingival pigmentation and inflammation should be addressed with caution.

A telephone survey in Canada, where graphic warning labels on cigarette packages were first introduced, demonstrated that labels depicting lung cancer and oral diseases were extremely effective with respect to discouraging smoking[10]. The image of a mouth was selected by more smokers, especially females and young adults, than were counterpart measures[16].

Therefore, visible oral symptoms of smokers likely afford the potential with respect to prevention and cessation of smoking.

CONCLUSION

The present study was the first to demonstrate the association of lip pigmentation with smoking and melanin pigmentation in the gingiva; thus, additional investigations involving a pathological approach and employing various variables as possible confounders of smoking are required. The striking relationship between the exposure to smoking and the visible symptom of pigmentation in oral and perioral conditions could potentially influence not only smoking but also oral health behaviors due to increasing awareness of oral health.

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TABLES AND FIGURES

Table 1: Distribution of subjects with or without melanin pigmentation in lip and gingiva by smoking status

	Lip		Gingiva		
Smoking Status	Lip Gingiva	No Pigmentation	Pigmentation	No pigmentation	Total
Never	45 (75)	40 (35)	65 (79)	15 (18)	75 (69)
Current	21 (35)	87 (68)	25 (31)	75 (91)	121 (71)
Subtotal	l51 (100)	131 (100)	89 (100)	109 (100)	193 (100)
Former	15	18	11	11	23
Total	81	157	87	109	223

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Table 2: Comparisons in score, prevalence and odds ratio (OR) and 95% confidence interval (CI) of lip and gingival pigmentation by levels of exposure to smoking.

	Lip pigmentation			Gingival pigmentation		
Levels of exposure (n)	Score	Prevalence (%)	OR (95% CI)	Score	Prevalence (%)	OR (95% CI)
Smoking status						
Never (73)	1.1 ± 1.3	47	1.0(reference)	0.5 ± 1.2	19	1.0 (reference)
Former (28)	1.0 ± 0.9	57	1.6 (0.7–4.1)	1.8 ± 2.2)	50	5.1(1.9-13.5)
Current (112)	2.1 ± 1.3	83	6.1 (2.7–11.9)	4.6 ± 3.0	80	16.8 (7.9–35.9)
Daily consumption (pieces)						
1–19 (37)	1.8 ± 1.4	76	3.9 (1.6–9.7)	4.5 ± 3.3	76	13.5 (5.2–35.3)
20 (58)	2.0 ± 1.3	85	6.0 (2.5–14.0)	4.6 ± 2.9	83	20.4 (8.3–50.6)
>20 (17)	2.8 ± 1.3	94	16.4 (1.3–132)	4.8 ± 3.0	82	20.5 (4.9–85.0)
Correlation	r= 0.345, P < 0.0001			r = 0.504, P < 0.0001		
Duration of smoking (years)						
1–9 (40)	1.6 ± 1.4	70	3.6 (1.4–9.1)	3.4 ± 2.9	70	9.5 (3.4–26.7)
10–19 (36)	2.2 ± 1.3	89	8.9 (2.9–27.9)	5.3 ± 2.9	86	27.2 (8.9–84.6)
>19 (36)	2.4 ± 1.2	92	9.0 (2.2–37.4)	5.2 ± 2.9	86	37.0 (8.5–160)
Correlation	r = 0.399, P < 0.0001			r = 0.670, P < 0.0001		
Lifetime exposure (piece-years)						
1–199 (46)	1.7 ± 1.4	72	3.8 (1.6–9.2)	3.7 ± 3.1	72	10.9 (4.1–28.7)
200–399 (34)	2.1 ± 1.2	88	8.0 (2.5–25.2)	5.6 ± 2.8	88	33.3 (9.8–113
>399 (32)	2.5 ± 1.2	94	13.3 (2.6–66.8)	4.8 ± 2.8	84	33.5 (7.8–143)
Correlation r	r= 0.411, P < 0.0001			r = 0.658, P < 0.0001		

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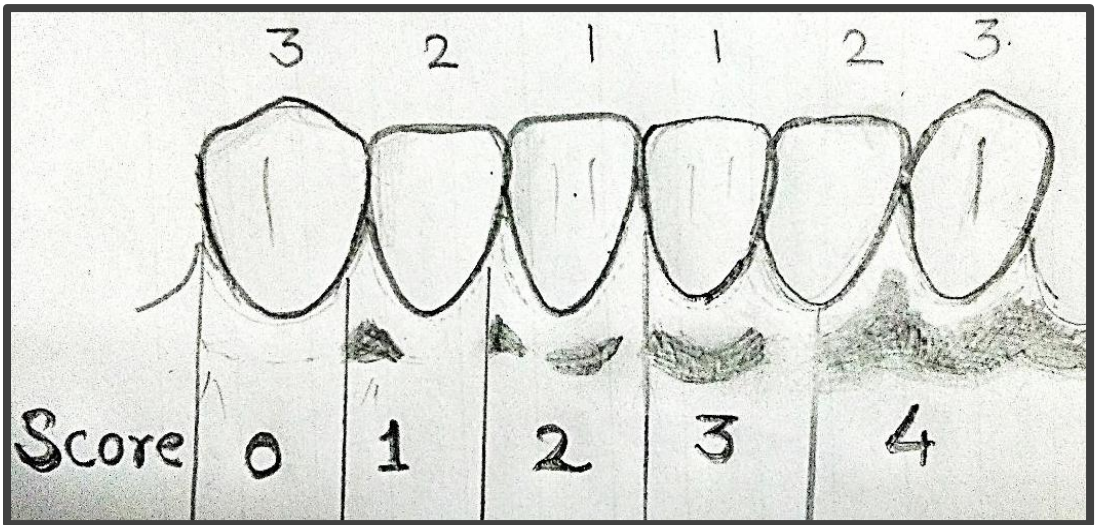
Table 3: Score of pigmentation between lip and gingiva for current and never smokers

Gingiva									
	Current smokers (P < 0.0001)					Never smokers, (P = 0.2112)			
Lip	0	1–3	4–6	7,8	Total	0	1–3	4–6	Total
0	17	1	1	0	19	38	1	0	39
1-2	4	18	25	27	69	16	7	2	27
3–6	1	1	7	19	28	5	3	1	9
Total	22	17	35	41	102	59	11	3	78

Figure 1: Scores of gingival pigmentation.



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