Oral Diseases (2007) 13, 71–76. doi:10.1111/j.1601-0825.2006.01249.x

http://www.blackwellmunksgaard.com

ORIGINAL ARTICLE

Association of lip pigmentation with smoking and gingival melanin pigmentation

S Haresaku, T Hanioka, A Tsutsui, T Watanabe

Department of Preventive and Public Health Dentistry, Fukuoka Dental College, Fukuoka, Japan

OBJECTIVE: We investigated the association of lip pigmentation with smoking and melanin pigmentation in the gingiva.
DESIGN: Case–control study.
SETTING: Health check-up in an institute.
SUBJECTS AND METHODS: Photos of 213 males employed in an institution were assessed in terms of pigmentation in lip and gingiva.
MAIN OUTCOME MEASURES: Prevalence and scores of lip and gingival pigmentation and smoking status.
RESULTS: Among subjects displaying lip and gingival pigmentation, 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. 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.
Oral Diseases (2007) **13**, 71–76

Keywords: lip; gingiva; pigmentation; melanin; smoking

Correspondence: Takashi Hanioka, Department of Preventive and Public Health Dentistry, Fukuoka Dental College, 2-15-1 Tamura, Sawara-ku, Fukuoka 814-0193, Japan. Tel/Fax: +81 92 801 0616, E-mail: haniokat@college.fdcnet.ac.jp
Received 3 October 2005; revised 12 December 2005; accepted 18 January 2006

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 (Dummett, 1962; Dummett and Gupta, 1966; Dummett and Barens, 1971; Cicek and Ertas, 2003). The prevalence of melanin pigmentation in the gingiva differs by ethnic group, which is indicative of a hereditary connection (Steigmann, 1965; Fry and Almeyda, 1968; Hedin, 1977; Axell and Hedin, 1982; Araki *et al*, 1983; Hedin and Larsson, 1984; Hanioka *et al*, 1993; Ünsal *et al*, 2001; Sarswathi *et al*, 2003). Gingival pigmentation is evident in subjects receiving anti-malarial drugs (Dencker *et al*, 1976; Main, 1988); however, this phenomenon is rare. Melanin pigmentation is caused by melanin granules in gingival tissue, which are produced in melanosomes of melanocytes (Hedin and Larsson, 1984). Melanin is synthesized from tyrosine and dihydroxyphenylalanine (DOPA) via dopaquinone by the oxidation of tyrosinase (Halaban *et al*, 2001).

Gingival pigmentation has been examined in terms of its association with smoking in various countries, including Israel (Steigmann, 1965), Sweden (Axell and Hedin, 1982), Japan (Araki *et al*, 1983; Hanioka *et al*, 1993), Thailand and Malaysia (Hedin and Axell, 1991), Turkey (Ünsal *et al*, 2001) and India (Sarswathi *et al*, 2003). 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 (Claffey *et al*, 2001) and benzpyrene (Roberto *et al*, 1996) in tobacco smoke relative to melanin. Additionally, a dose–response relationship was detected (Axell and Hedin, 1982; Araki *et al*, 1983). Disappearance of gingival pigmentation was observed following reduction in smoking (Hedin *et al*, 1993). These findings suggest a causal association between smoking and gingival pigmentation; additionally, the specific label of 'smoker's melanosis' was assigned (Hedin, 1977).

Gingival pigmentation is visible because of the presence in the labial area of anterior teeth (Hedin, 1977;

Axell and Hedin, 1982; Hanioka *et al*, 1993: Sarswathi *et al*, 2003). 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 (Axell and Hedin, 1982). The objective of this study was to investigate the association of lip pigmentation with smoking and gingival melanin pigmentation.

Subjects and methods

Digital photos of lip and the labial aspects of frontal teeth, which were produced in a standardized manner (D70, Nikon, Tokyo), were obtained from employees of an institute at Fukuoka, Japan, on the occasion of the annual health check-up. These subjects were medically healthy individuals. 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. Questionnaires, which addressed smoking status, were incomplete in 10 males; consequently, photos of these 10 males and of all participating females were excluded from this investigation. Finally, photos of 213 males (31.8 ± 8.9 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). *K*-statistic for the existence of lip pigmentation was 0.88, which indicated that interexaminer agreement was excellent and that the subjective evaluation of lip pigmentation was reliable.

Gingival pigmentation was scored in each jaw according to the classification of Melanin Index (Hedin, 1977, Figure 1). The index classified pigmentation as follows: 0, no pigmentation; 1, one or two solitary unit(s) 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.

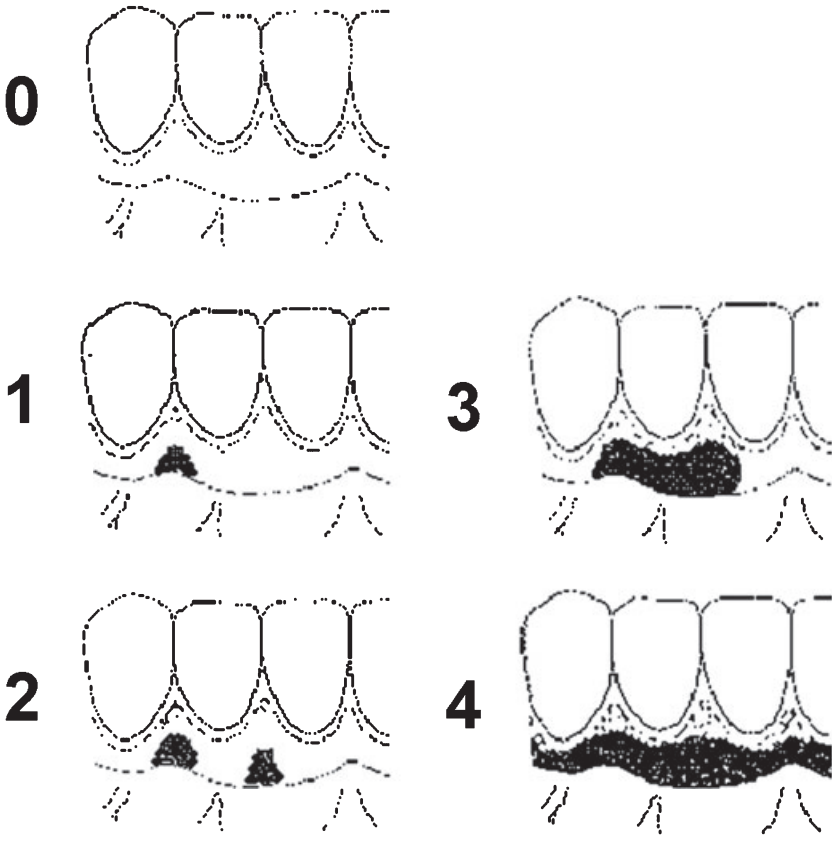

Figure 1 Classification in the Melanin Index for gingival pigmentation; this figure describes in quadrant.

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 (Beck, 1995). Disappearance of pigmentation was observed following reduction of smoking (Hedin *et al*, 1993); additionally, other variables, such as duration of cessation, may influence results of the evaluation. Consequently, FS was excluded from evaluation.

The protocol was approved by the *ad hoc* ethics committee of epidemiological research in Fukuoka Dental College. 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 213 subjects, 73 (33%), 112 (50%) and 28 (13%) were NS, CS and FS respectively (Table 1). Lip and gingival pigmentation was apparent in 143 (67%) and 118 (53%) participants respectively. Prevalence of pigmentation was compared according to the smoking status. FS were excluded in the comparison, as disappearance of pigmentation was observed following reduction of smoking (17). Seventy-three per cent of subjects exhibiting lip pigmentation were CS; in contrast, 33% of subjects lacking lip pigmentation were CS. In the case of gingival pigmentation, 87% and 27% 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, prevalences 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 5.6 (95% confident interval 2.8–11.1) and 17.0 (8.1–36.0), respectively, which differed significantly from those of NS. The difference in prevalence of lip pigmentation between FS and NS was not meaningful, OR = 1.4 (0.6–3.5). OR of FS in terms of gingival pigmentation was 4.5 (1.7–12.0), 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 0.380, 0.377 and 0.387 in lip, and 0.594, 0.640 and 0.632 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.

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

	Lip		*Gingiva*		
Smoking status	*No pigmentation*	*Pigmentation*	*No pigmentation*	*Pigmentation*	*Total*
Never	39 (67)	34 (27)	59 (73)	14 (13)	73 (39)
Current	19 (33)	93 (73)	22 (27)	90 (87)	112 (61)
Subtotal	58 (100)	127 (100)	81 (100)	104 (100)	185 (100)
Former	12	16	14	14	28
Total	70	143	95	118	213

Distributions of lip and gingival pigmentation were significantly associated with smoking status ($P < 0.0001$). Former smokers were excluded.

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	1.4 (0.6–3.5)	1.8 ± 2.2*	50	4.5 (1.7–12.0)
Current (112)	2.1 ± 1.3*	83	5.6 (2.8–11.1)	4.6 ± 3.0*	80	17.0 (8.1–36.0)
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		$r = 0.380$ $P < 0.0001$			$r = 0.594$ $P < 0.0001$	
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		$r = 0.377$ $P < 0.0001$			$r = 0.640$ $P < 0.0001$	
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		$r = 0.387$ $P < 0.0001$			$r = 0.632$ $P < 0.0001$	

n, number of subjects.
*Significantly higher than that of never smokers.

Table 3 Contingency table by score of pigmentation between lip and gingiva for current and never smokers

	Gingiva								
	Current smokers ($P < 0.0001$)					*Never smokers ($P = 0.1728$)*			
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	15	24	22	65	16	7	2	25
3–6	1	1	7	19	28	5	3	1	9
Total	22	17	32	41	112	59	11	3	73

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 89% 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 ($P = 0.1728$). Ninety-seven per cent of subjects lacking lip pigmentation exhibited no pigmentation in gingiva. However, gingival pigmentation was evident in 38% 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 (Aligne *et al*, 2003), paediatric caries (Arbes *et al*, 2001) and melanin pigmentation in the gingiva of children (Hanioka *et al*, 2005) 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 (Roberto *et al*, 1996; Claffey *et al*, 2001). The highly sensitive nature of gingival melanocytes may be beneficial as young smokers could recognize a rather immediate untoward effect of smoking behaviour 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 (Patsakas *et al*, 1981). However, the number of melanocytes did not correlate with visible pigmentation (Schreoder, 1969). Furthermore, inflammatory response to plaque accumulation is suppressed in the gingiva of smokers (Lie *et al*, 1998), 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 (Hammond *et al*, 2003). The image of a mouth was selected by more smokers, especially females and young adults, than were counterpart measures (Environics Research Group Ltd, 2001). Therefore, visible oral symptoms of smokers likely afford the potential with respect to prevention and cessation of smoking.

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 behaviours due to increasing awareness of oral health.

Acknowledgements

This study was supported by the Health and Labour Sciences Research Grant for Research on Cancer Prevention and Health Services (15220801) by the Ministry of Health, Labour and Welfare, Japan (2004).

References

Aligne CA, Moss ME, Auinger P, Weitzman M (2003). Association of pediatric dental caries with passive smoking. *J Am Med Assoc* **289:** 1258–1264.

Araki S, Murata K, Ushio K, Sakai R (1983). Dose–response relationship between tobacco consumption and melanin pigmentation in the attached gingiva. *Arch Environ Health* **138:** 375–378.

Arbes SJ, Ágústsdótir H, Slade GD (2001). Environmental tobacco smoke and periodontal disease in the United States. *Am J Public Health* **91:** 253–257.

Axell T, Hedin CA (1982). Epidemiologic study of excessive oral melanin pigmentation with special reference to the influence of tobacco habits. *Scand J Dent Res* **90:** 434–442.

Beck JD (1995). Issues in assessment of diagnostic tests and risk for periodontal diseases. *Periodontol 2000* **7:** 100–108.

Cicek Y, Ertas U (2003). The normal and pathological pigmentation of oral mucous membrane: a review. *J Contemp Dent Pract* **15:** 76–86.

Claffey DJ, Stout PR, Ruth JA (2001). ^{3}H-nicotine, ^{3}H-flunitrazepam, and ^{3}H-cocaine incorporation into melanin: a model for the examination of drug–melanin interactions. *J Anal Toxicol* **25:** 607–611.

Dencker L, Lindquist NG, Tjalve H (1976). Uptake of ^{14}C-labelled chloroquine and an ^{125}I-labelled chloroquine analogue in some polypeptide hormone producing cell systems. *Med Biol* **54:** 62–68.

Dummett CO (1962). A classification of oral pigmentation. *Mil Med* **127:** 839–840.

Dummett CO, Barens G (1971). Oromucosal pigmentation: an updated literary review. *J Periodontol* **42:** 726–736.

Dummett CO, Gupta OP (1966). Estimating the epidemiology of oral pigmentation. *Q Natl Dent Assoc* **24:** 81–86.

Environics Research Group Ltd (2001). *Evaluation of new warnings on cigarette packages*, Prepared for: Canadian Cancer Society. *Focus Canada* **3:** 1–33.

Fry L, Almeyda JR (1968). The incidence of buccal pigmentation in Caucasoids and Negroids in Britain. *Br J Dermatol* **80:** 244–247.

Halaban R, Cheng E, Svedine S, Aron R, Hebert DN (2001). Proper folding and endoplasmic reticulum to golgi transport of tyrosinase are induced by its substrates, DOPA and tyrosine. *J Biol Chem* **276:** 11933–11938.

Hammond D, Fong GT, McDonald PW, Cameron R, Brown KS (2003). Impact of the graphic Canadian warning labels on adult smoking behaviour. *Tob Control* **12:** 391–395.

Hanioka T, Tanaka M, Tamagawa H, Shizukuishi S (1993). Epidemiologic study of melanin pigmentation in the attached gingiva in relation to cigarette smoking. *J Dent Health* **43:** 40–47. (in Japanese)

Hanioka T, Tanaka K, Ojima M, Yuuki K (2005). Association of melanin pigmentation in the gingiva of children with parental smoking. *Pediatrics* **116:** e186–e190.

Hedin CA (1977). Smoker's melanosis. Occurrence and localization in the attached gingiva. *Arch Dermatol* **113:** 1533–1538.

Hedin CA, Axell T (1991). Oral melanin pigmentation in 467 Thai and Malaysian people with special emphasis on smoker's melanosis. *J Oral Pathol Med* **20:** 8–12.

Hedin CA, Larsson A (1984). The ultrastructure of the gingival epithelium in smoker's melanosis. *J Periodont Res* **19:** 177–190.

Hedin CA, Pindborg JJ, Axell T (1993). Disappearance of smoker's melanosis after reducing smoking. *J Oral Pathol Med* **22:** 228–230.

Lie MA, Timmerman MF, van der Velden U, van der Weijden GA (1998). Evaluation of 2 methods to assess gingival bleeding in smokers and non-smokers in natural and experimental gingivitis. *J Clin Periodontol* **25:** 695–700.

Main JHP (1988). Two cases of oral pigmentation associated with quinidine therapy. *Oral Surg* **66:** 59–61.

Patsakas A, Demetriou N, Angelopoulos A (1981). Melanin pigmentation and inflammation in human gingiva. *J Periodontol* **52:** 701–704.

Roberto A, Larsson BS, Tjarve H (1996). Uptake of 7,12-dimethylbenz(a)anthracene and benzo(a)pyrene in melanin-containing tissues. *Pharmacol Toxicol* **79:** 92–99.

Sarswathi TR, Kumar SN, Kavitha KM (2003). Oral melanin pigmentation in smoked and smokeless tobacco users in India. Clinico-pathological study. *Indian J Dent Res* **14:** 101–106.

Schreoder HE (1969). Melanin containing organelles in cells of the human gingiva. II. Keratinocytes. *J Periodont Res* **4:** 235–247.

Steigmann S (1965). The relation between physiologic pigmentation of the skin and the oral mucosa in Yemenite Jews. *Oral Surg* **19:** 32–38.

Ünsal E, Paksoy C, Soykan E, Elhan AH, Şahin M (2001). Oral melanin pigmentation related to smoking in a Turkish population. *Community Dent Oral Epidemiol* **29:** 272–277.