A Color Analysis of Smoker’s Melanosis Using a Non-Contact Type Dental Spectrophotometer

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Abstract

Objective: The aim of this study was to measure the gingival color precisely and explore factors affecting gingival pigmentation.

Materials and Methods: Sixty-seven healthy volunteers (22 females and 45 males, average 28 years of age) were recruited for this study. We measured the color of the attached gingiva at the interdental papilla of the upper and lower right central incisors, lateral incisors and canines using a non-contact type dental spectrophotometer. The measured color was quantified according to the CIEL*a*b* 1976 criteria. Oral health indices, such as the plaque control record, gingival inflammation and probing pocket depth, were determined manually. Lifestyle factors that may affect gingival pigmentation, such as smoking and medications, were also recorded according to a questionnaire.

Results: Twenty-one subjects were current smokers, 12 were former smokers and 34 were non-smokers. On the gingival color analysis, the L* and b* values in the current smokers were significantly lower than those in the former and non-smokers in all areas, except the L* value of the lower canine in the former smokers. The other hand, the a* value was lower in the current smokers than in the non-smokers for the upper medial, lower lateral incisor and canine gingiva. Among smokers, the L*, a* and b* values were lower in the patients with a Brinkman index (BI) of ≥100 than in those with a BI<100. In addition, the L* and b* values were lower in the patients with a BI of ≥100 than in those with 0<BI<100.

Conclusions: The brightness and chroma of the gingiva were found to be significantly lower in current smokers than in non- and former smokers and similar between the latter two groups. Our findings suggest that the gingival color is significantly influenced by the smoking status, papillary marginal attached index and BI.

Keywords: Gingival Pigmentation; Smoking; Spectrophotometry

Introduction

The pigmentation of human gingival tissue is derived from melanin granules, which are synthesized in the melanosomes of melanocytes [1,2]. The nicotine present in tobacco activates melanocytes to promote melanin secretion [3]. Therefore, the melanin pigmentation in gingival tissues has a strong correlation with smoking [4,5]. In 1977, Hedin et al. [5] first reported that smokers showed more pigmented gingiva than non-smokers. The authors named this gingival pigmentation “smoker’s melanosis.” Gingival pigmentation related to smoking is often observed in the labial area of the lower medial canine. A previous study also suggested that the severity of gingival pigmentation decreases after smoking cessation, in relation to the number of years after the discontinuation of smoking [3]. These findings indicate that there is a bidirectional association between tobacco smoking and melanin pigmentation in the gingiva and suggest the possibility that a normal gingival pigmentation may be recovered following smoking cessation.

Gingival pigmentation has an aesthetic consideration, and monitoring the gingival color may provide information regarding the biological reaction to smoking and thus be a tool for providing education and support for smoking cessation. Previous studies have reported the application of color quantification of peri-implant soft tissue [6] and the cervical gingiva around ceramic crowns [7] based on the CIELab uniform color space [8] determined using a non-contact type dental spectrophotometer. The aim of this study was to investigate the correlation between changes in the gingival color and various factors, including the smoking status, using a non-contact type dental spectrophotometer.

Materials and methods

Study period and human subjects

Systemically healthy volunteers were recruited from Fukuoka Dental College Hospital between January 2010 and March 2012. After obtaining written informed consent, 67 volunteers were included in this study. The study was performed at the Oro-facial Plastic Medical Center, Fukuoka Dental College, Medical and Dental Hospital, after a protocol review with the permission of the Institutional Review Board of Fukuoka Dental College (Permission # 1653116).

Questionnaire survey

A questionnaire survey was used to collect information for age, gender, smoking status, systemic diseases and medications. The smoking status was defined according to the WHO classification [9]. The Brinkman index (BI) was calculated as the number of cigarettes smoked per day multiplied by the number of years of smoking [10].

Keywords: Gingival Pigmentation; Smoking; Spectrophotometry

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Oral examination

The plaque control record proposed by O'Leally [11] (PCR), papillary marginal attached index [12] (PMA index), periodontal probing depth [13] (PPD) and width of the attached gingiva [14] were assessed by a single examiner. The PPD was determined at the labial and lingual sites of the upper and lower anterior teeth using six measurements. The degree of oral moistness was examined with a moisture checker, the Mucus® device (KISO-Wet, Life Corporation, Japan). All measurement procedures were performed according to the manufacturer’s instructions.

Measurement of the gingival color

The gingival color was measured using a non-contact type dental spectrophotometer, CE100-DC (Crystaley®, Olympus, Japan), which consists of a spectrophotometer to measure the color as well as a cradle and color analysis software program. The contact cap was mounted to the measuring head to shield against any interfering outside light and display only the region of interest. The measured spectrophotometric data were transmitted to a personal computer and used to calculate the L*a*b* according to the CIELab uniform color space classification proposed by the Commission Internationale de l’Eclairage, 1976 [15]. The data obtained via computerized spectrophotometry allowed for quantitative comparisons based on the CIELab parameters. The L*a*b* values were obtained from the three-dimensional color space. L* indicates the brightness, that is, the black-white axis. The higher the L* value, the ‘whiter’ the object. The a* value reflects the red-green axis, with higher values indicating more red within the object. The b* value represents the yellow-blue axis, with higher values signifying more yellow within the object.

Previous studies have demonstrated no differences in laterality with respect to the gingival color [16,17]. Therefore, we measured six spots on the attached gingiva just beneath the papilla, between the central incisors, near the right central incisor and right lateral incisor, near the right incisor and right canine of the maxilla and in the mandibular region (Figure 1). Smoker’s melanosis is most frequently observed in the attached gingiva of the papilla neighboring the lower marginal canine [5]; hence, we set the colorimetric value of this area as a region of interest. The first quartiles of the L*, a* and b* values were calculated with the first quartile group named the 'lower group' and the remaining subjects classified into the 'higher group'. The two groups were compared to assess the correlations for several factors that may affect the gingival color.

Statistical analysis

The data were statistically analyzed according to a one-way ANOVA, the χ² test and Scheffe’s post-hoc test using the IBM SPSS Statistics Ver. 20.0 software package (International Business Machine, USA). The significance level was set at 5%.

Results

The characteristics of the study subjects are shown in Table 1. The distribution of the smoking status determined according to the WHO classification was as follows: 21 current smokers, 12 former smokers and 34 non-smokers. The average age of the participants was 28 years, and no subjects had any systemic diseases or were currently receiving drug therapy. Ninety percent of the smokers were male. The periodontal indicators, including the plaque control record, probing pocket depth and gingival inflammation index, did not differ significantly based on the smoking status.

The gingival color values assessed according to the smoking status are shown in Table 2. There were significant differences in the L* (brightness) and b* (yellow-blue axis) values at all sites among the current smokers, former smokers and non-smokers (p<0.05). For example, the L* and b* values were lower in the current smokers than in the former and non-smokers, with the exception of the L* values for the lower canine in the former smokers. On the other hand, the a* values were lower in the current smokers than in the former and non-smokers for the upper median, lower lateral and canine locations (p<0.05). In contrast, there were no significant differences among the other sites did not show significant differences.

Table 3 shows the gingival color of the lower canine associated with smoker’s melanosis, which was most closely related to the BI. All L*, a* and b* values among the patients with a BI of ≥ 100 were lower than those among the patients with a BI=0 (p<0.05). The L* and b* values were lower among the patients with a BI of ≥ 100 than those with 0<BI<100; however, there were no significant differences in the a* values between these two groups (p>0.05).

The patients in the first quartiles of the L*, a* and b* values for the lower canine were classified into the ‘lower group,’ while the remaining patients were classified into the ‘higher group.’ The correlations between the values and the smoking status in both groups are shown in Table 4. Sixty percent of the patients in the lower group based on the L* value were current smokers, while 59.6% of those in the higher group based on the L* value were non-smokers; this difference was statistically significant (p<0.05). Similarly, 57.1% of the patients in the lower group for the a* value were current smokers, while 58.5% of those in the higher group were non-smokers, also a statistically significant difference (p<0.05). Furthermore, 75% of the patients in the lower group for the b* value were current smokers, while 64.7% of those in the higher group were non-smokers, for a significant difference (p<0.05).

Discussion

The gingival color is influenced by the quantity and width of capillary vessels in the gingiva [18], the thickness of the mucosa [19] and epithelial tissue [20], the degree of keratinization of the epithelial tissue [20] and the amount of intraepithelial melanin [1]. In general, the physiological or pathological amount of melanin in the tissue is the

**Figure 1:** The target site of measuring the color of attached gingiva using non-contact spectrophotometer. 1: Median, 2: Lateral, 3: Canine.
Current smokers were significantly different from non-smokers.

Former smokers were significantly different from non-smokers.

Current smokers were significantly different from former smokers.

The group with a $0 \geq BI$ was significantly different from that with a $BI = 0$.

The group with a $0 < BI < 100$ was significantly different from that with a $0 \geq BI$.

The group with a $BI = 0$ was significantly different from that with a $0 < BI < 100$.

The strongest factor affecting the gingival color [19]. The number of gingival melanocytes is associated with the number of skin melanocytes [21,22], and the prevalence of melanin pigmentation varies among different ethnic groups [21]. In addition to physiological factors, the gingival color is also changed by inflammation [23,24], exposure to ultraviolet rays [1], drugs [25], smoking [4,5] and endocrinological diseases, such as Addison’s disease [26]. The subjects included in this study had the

<table>
<thead>
<tr>
<th>L*</th>
<th>Upper Median</th>
<th>Lower Median</th>
<th>a*</th>
<th>Canine 14.5 ± 2.60</th>
<th>18.2 ± 2.98</th>
<th>18.5 ± 1.96</th>
<th>0.01* 15</th>
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</tr>
<tr>
<td>Canine 13.8 ± 3.06</td>
<td>16.5 ± 2.46</td>
<td>16.7 ± 1.71</td>
<td>&lt;0.05* 15</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canine 14.6 ± 3.07</td>
<td>16.8 ± 1.75</td>
<td>16.8 ± 2.00</td>
<td>&lt;0.05* 15</td>
<td></td>
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</tr>
<tr>
<td>Canine 15.5 ± 2.47</td>
<td>17.8 ± 2.57</td>
<td>18.7 ± 2.01</td>
<td>&lt;0.05* 15</td>
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<tr>
<td>Canine 14.8 ± 2.60</td>
<td>18.2 ± 2.98</td>
<td>18.5 ± 1.96</td>
<td>0.01* 15</td>
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<tr>
<td>Canine 14.5 ± 3.57</td>
<td>17.7 ± 4.07</td>
<td>18.7 ± 1.68</td>
<td>&lt;0.05* 15</td>
<td></td>
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Mean ± S.D. Scheffe’s post-hoc test (p<0.05)

Table 2: L*, a* and b* values of the attached gingiva according to the smoking status.

<table>
<thead>
<tr>
<th>Bl</th>
<th>0</th>
<th>0&lt;Bl&lt;100</th>
<th>≥100</th>
<th>( p )</th>
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</thead>
<tbody>
<tr>
<td>n</td>
<td>34</td>
<td>10</td>
<td>23</td>
<td>---</td>
</tr>
<tr>
<td>L*</td>
<td>48.4 ± 5.13</td>
<td>47.5 ± 6.65</td>
<td>41.6 ± 5.23</td>
<td>&lt;0.05* 13</td>
</tr>
<tr>
<td>a*</td>
<td>22.3 ± 3.45</td>
<td>20.1 ± 2.28</td>
<td>19.4 ± 3.50</td>
<td>&lt;0.01* 13</td>
</tr>
<tr>
<td>b*</td>
<td>18.7 ± 3.16</td>
<td>18.3 ± 3.16</td>
<td>14.5 ± 3.84</td>
<td>&lt;0.01* 13</td>
</tr>
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Scheffe’s post-hoc test (p<0.05)

Table 3: Mean L*, a* and b* values according to the smoking status.
same ethnic background and were not using any drugs or had any metal tattoos. Because the unit of epithelial melanin in the oral mucosa functions to protect the tissue from harmful ultraviolet radiation [1], we conducted our survey from January to March in order to avoid the influence of exposure to ultraviolet rays.

The PCR and PMA index were also examined as potential factors affecting the gingival color due to the effects of inflammation. Consequently, higher PMA values were observed in the subjects with hyperpigmentation, whereas there were no significant differences in the PCR values according to the gingival pigmentation. These results suggest that the PMA index is a marker of inflammation and that a higher PCR value does not necessarily reflect gingival inflammation.

The chemical substances present in tobacco many potential effects on the human body [4,27]. For example, nicotine affects vascular smooth muscle cells by briefly decreasing the blood flow [28,29], as well as inhibiting the growth [30] and inducing the apoptosis [31] of human gingival fibroblasts. Furthermore, it has been previously demonstrated that nicotine activates melanocytes, resulting in increased melanin production [3-5].

Hedin et al. first reported that smoking induces changes in gingival pigmentation, termed ‘smoker’s melanosis’ [5]. In general, the amount of cigarette smoke exposure is associated with the extent of smoker’s melanosis. The most common site of smoker’s melanosis is the gingiva attached to the lower medial canine. In the present study, we also analyzed the relationship between the amount of smoking and changes in the gingival color in various locations. Consequently, the patients with a BI of ≥100 demonstrated significantly lower L* values than those with a BI of <100, indicating that the BI negatively correlates with the brightness (L*) and that a BI of ≥ 100 functions as a threshold for assessing gingival pigmentation. A previous landmark study reported that the risk of lung cancer is increased in individuals with a BI above 400 [32]. Since a BI of 100 is associated with changes in gingival color, assessments of melanosis may provide helpful information for smokers to understand the systemic changes associated with their smoking habits.

A previous study also reported differences in the color of the gingiva between current and non-smokers. In that study, all of the subjects with gingival melanin pigmentation were current smokers [5]. On the other hand, our 21 current smokers were not always included in the lower L*, a* and b* groups. This discrepancy may be due to differences in spectrometric methods and/or the ethnicity of the subjects (Scandinavian vs. Japanese).

Previous studies have also demonstrated that oral pigmentation is related to the smoking status [3-5] and that melanin pigmentation is first observed approximately six months after the initiation of smoking [5]. In addition, Kistiakovsky reported that smoking cessation results in a normal gingival color after five months, while restarting smoking induced gingival melanin pigmentation again within four months [33]. Meanwhile, another study showed that the degree of smoker’s melanosis increases within one year of smoking initiation and subsequently fades to the same level as that seen in the gingiva of non-smokers within three years after smoking cessation [34]. In the present study, the gingival pigmentation did not differ significantly between the former smokers and non-smokers, suggesting that smoking cessation leads to the decoloration of pigmented gingiva. Our findings also indicate that quantitatively measuring the gingival color using a non-contact type dental spectrophotometer can be used to detect marginal changes in this parameter and may be applicable for educating patients regarding smoking cessation, as it easily shows changes relatively soon after smoking/smoking cessation.

**Conclusion**

Factors affecting the gingival color include the PMA, BI and smoking status. In the present study, the L*, a* and b* values were significantly lower in the current smokers than in the former and non-smokers. Moreover, the former smokers tended to have similar values to those of the non-smokers and higher values than those of the current smokers. Measuring the gingival color using a non-contact type dental spectrophotometer is a convenient way to quantify the color of the gingiva, without influences from the surrounding environment. This technique also makes it possible to follow time-dependent changes in gingival pigmentation after both smoking and smoking cessation. Therefore, this method may be a useful tool for guiding education regarding smoking cessation as well as oral hygiene.

**References**

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