

**ORIGINAL ARTICLE**

## **Oral Manifestations among Male Smokers in Banjarmasin, South Kalimantan, Indonesia**

**Maharani Laillyza Apriasari<sup>1</sup>, Ananda Regitha Geneny<sup>2</sup>, Isnur Hatta<sup>3</sup>**

<sup>1</sup>*Departement of Oral Medicine, Faculty of Dentistry, Lambung Mangkurat University, Banjarmasin, Indonesia*

<sup>2</sup>*Faculty of Dentistry, Lambung Mangkurat University, Banjarmasin, Indonesia*

<sup>3</sup>*Departement of Dental Public Health, Faculty of Dentistry, Lambung Mangkurat University, Banjarmasin, Indonesia*

Correspondence e-mail to: [maharaniroxy@gmail.com](mailto:maharaniroxy@gmail.com)

### **ABSTRACT**

**Background:** Smoking is a risk factor for numerous diseases like cardiovascular disease, cancer, and lung disease. It also has negative effects in the oral cavity, including an increased incidence of cancer, oral mucosal lesions, periodontal disease, implant failure, dental caries, and dental staining. **Purpose:** This study aims to analyze the relationship between age, duration, and frequency of smoking with clinical manifestations of the oral cavity in smokers in Banjarmasin. **Methods:** This study utilized an analytical observational approach with a cross-sectional design. Data were gathered from the Dinas Kesehatan Office of Banjarmasin City through anamnesis and clinical oral cavity examination. Fifty-seven participants meeting specific criteria were selected using purposive sampling. Statistical analysis involved the chi-square test to examine the link between age, duration, and frequency of smoking with oral manifestations. The Fisher's exact test was employed if the data did not meet the chi-square test criteria. **Results:** The highest number of smokers were aged  $\leq 40$  years old (52.6%), the duration of smoking was found the most at the duration of  $>10$  years (59.6%), and the frequency of smoking was higher in those who smoked more than 15 cigarettes per day (57.9%). The bivariate analysis showed that there was a significant relationship between the duration of smoking and manifestations of a smoker's palate ( $p = 0.040$ ). A significant relationship between smoking frequency and manifestations of smoker's melanosis was shown ( $p = 0.042$ ). **Conclusion:** The oral manifestations of male smokers included smoker's melanosis, smoker's palate, periodontal disease, and dental staining.

**Keywords:** male-smoker, oral manifestation, nicotine stomatitis, Kalimantan

### **INTRODUCTION**

The estimated number of smokers is expected to reach 1.3 billion globally.<sup>1</sup> Indonesia has the third-highest cigarette consumers after China and India.<sup>2</sup> The results of Riset Kesehatan Dasar (Riskesdas) in 2013 show the national prevalence of smokers in Indonesia was 29%. The average cigarette consumption peak was for the age group of 35-44 at 11.7 cigarettes per day.<sup>2</sup> The prevalence of the number of smokers in South Kalimantan Province was 25.7%, with the prevalence of smoking in the population aged  $\geq 10$  years old in Banjarmasin City at 18.02%.<sup>2</sup> The highest smoking prevalence based on age was in the age group of 30-34 years old (30.01%).<sup>3</sup>

Tar, nicotine, and carbon monoxide are the three most harmful components produced by tobacco combustion<sup>4</sup>. The entry of chemical effects in cigarettes into the body may induce a systemic inflammatory response by stimulating the hematopoietic system, particularly the bone marrow, where the production of erythrocytes and leukocytes increases while the platelets decrease.<sup>5</sup> A long smoking habit impacts many organ systems, including the oral cavity.

The frequency and the length of smoking habits of each person affect the speed at which a person is exposed to abnormalities in the oral cavity caused by the increasing number of free radicals in the body.<sup>6</sup> The age factor also affects the occurrence of abnormalities in the oral cavity, while the elderly will experience various physical setbacks entirely.<sup>7</sup> Pathological conditions of the oral cavity that are frequently found in smokers include periodontal disease, halitosis, caries, dental staining, and the presentations of typical lesions on the soft tissue such as smoker's melanosis, smoker's palate, leukoplakia, and median rhomboid glossitis.<sup>6</sup>

Based on the description above, the researchers conducted a study to find out the manifestations of smoking in smokers and whether there is a relationship related to age, duration of smoking, and frequency of smoking among smokers in Banjarmasin, South Kalimantan, Indonesia.

## MATERIALS AND METHODS

The ethical approval of this study was given by the Health Research Ethics Commission of the Faculty of Dentistry, University of Lambung Mangkurat No. 077/KEPKGFKGULM/EC/I/2020 and a research permit was issued by related agencies. The method used in this study was analytical observational with a cross-sectional design, in which measurements and observations were performed at a particular time. The population in this study was all smokers with a minimum age of 20 years old, with a total of 3,298 people in Banjarmasin. The total research sample was 57 respondents, and the sampling was determined using a purposive sampling technique.

Inclusion criteria were active smokers for at least 1 year, a male who was at least 20 years old, no history of systemic disease, and was willing to become a respondent by signing informed consent. Exclusion criteria include subjects who were not regular smokers, refused to have an oral examination, and had impaired consciousness function. The tools used were examination sheets, stationery, mouth mirror, kidney tray (*Nierbekken*), and flashlights. The materials used were masks, disposable rubber hand gloves, and tissues. Before anamnesis and oral clinical examination, the researchers provide information regarding the advantages of the study and the procedures performed by the respondents. After respondents had approved and signed the informed consent form, the interview (anamnesis) was performed, and the clinical examination of the oral cavity was continued using a dental mirror.

The data were analyzed using the chi-square test to determine the relationship between age, duration, and smoking frequency and clinical manifestations of the oral cavity. However, if the data did not meet the requirements for the 2x2 table, Fisher's exact test would be used.

## RESULTS

A total of 57 respondents were included in this study. The data obtained from this research showed respondents' sociodemographic characteristics, including age, duration of smoking, and frequency of smoking, as shown in Table 1. Table 2 lists the variants of oral manifestations, which mostly caused dental staining. The distribution of oral manifestations is based on age, smoking duration, and frequency. The statistical analysis results are shown in Tables 3, 4 and 5. Table 3 shows that there is a relationship between the smoker's melanosis and the frequency of smoking with  $p < 0.05$ . There is no significant relationship between the smoker's melanosis and age and duration of smoking ( $p > 0.05$ ).

Variables	N	%
Age		
≤40 years	30	52.6%
>40 years	27	47.4%
Duration of smoking		
≤10 years	23	40.4%
>10 years	34	59.6%
Frequency of smoking (daily)		
≤15 cigarettes	24	42.1%
>15 cigarettes	33	57.9%

Table 1. Sociodemographic characteristics of the respondents.

<b>Manifestation</b>	<b>N</b>	<b>%</b>
Smoker's melanosis	36	63.15%
Smoker's palate	28	49.12%
Periodontal disease	39	68.4%
Dental staining	46	80.7%

Table 2. Clinical manifestations in the oral cavity among smokers in Banjarmasin

<b>Smoker's Melanosis</b>			
	<b>Yes</b>	<b>No</b>	<b>Sig.</b>
<b>Age</b>			
≤40 years	60%	40%	0.80
>40 years	66.7%	33.3%	
<b>Duration of smoking</b>			
≤10 years	52.2%	47.8%	0.25
>10 years	70.6%	29.4%	
<b>Frequency of smoking (daily)</b>			
≤15 cigarettes	45.8%	54.2%	0.04
>15 cigarettes	75.8%	24.2%	

Table 3. The Relationship between Age, Duration of Smoking, Frequency of Smoking and Smoker's Melanosis

<b>Smoker's Palate</b>			
	<b>Yes</b>	<b>No</b>	<b>Sig.</b>
<b>Age</b>			
≤40 years	56.7%	43.3%	0.34
>40 years	40.7%	59.3%	
<b>Duration of smoking</b>			
≤10 years	30.4%	69.6%	0.04
>10 years	61.8%	38.25	
<b>Frequency of smoking (daily)</b>			
≤15 cigarettes	41.7%	58.3%	0.48
>15 cigarettes	54.5%	45.5%	

Table 4. The relationship between age, duration of smoking, frequency of smoking and smoker's palate

Table 4 shows that there is a significant relationship between the smoker's palate and the duration of smoking with  $p < 0.05$ . There is no significant relationship between the smoker's palate and age and frequency of smoking ( $p > 0.05$ ). Table 5 shows there is no significant relationship between periodontal disease and age, frequency of smoking, and duration of smoking ( $p > 0.05$ ). Table 6 shows no significant relationship between dental staining and age, smoking frequency, and duration ( $p > 0.05$ ).

Periodontal Diseases			
	Yes	No	Sig.
Age			
≤40 years	63.3%	36.7%	0.55
>40 years	74.1%	25.9%	
Duration of smoking			
≤10 years	56.5%	43.5%	0.19
>10 years	76.5%	23.5%	
Frequency of smoking (daily)			
≤15 cigarettes	70.8%	29.2%	0.96
>15 cigarettes	66.7%	33.3%	

Table 5. The relationship between age, duration of smoking, frequency of smoking and periodontal disease

Dental Staining			
	Yes	No	Sig.
Age			
≤40 years	80%	20%	1.00
>40 years	81.5%	18.5%	
Duration of smoking			
≤10 years	73.9%	26.1%	0.32
>10 years	85.3%	14.7%	
Frequency of smoking (daily)			
≤15 cigarettes	75%	25%	0.49
>15 cigarettes	84.8%	15.2%	

Table 6. The relationship between age, duration of smoking, frequency of smoking and dental staining

## DISCUSSION

This research shows that there was no significant relationship between age and smoker's melanosis and smoker's palate. Oral mucosal lesions in the elderly occur because of other factors of recurrent habits besides smoking, including chewing, ill-fitting dentures, and alcohol consumption.<sup>7</sup> The absence of a significant relationship between age and periodontal disease can be due to organ

function deterioration due to the ageing process.<sup>8</sup> This results in less effective functioning of PMN cells, a decrease in the CD4+/CD8+ ratio, and deterioration in salivary gland function as parenchymal glands are replaced by adipose cells and connective tissues. The atrophy of intermediate duct cell linings further impacts saliva IgA production.<sup>9</sup> In addition to the ageing process, impaired wound healing occurs due to reduced oxygen and nutrient supply to connective tissue and blood vessels and decreased collagen synthesis, rendering individuals more vulnerable to periodontal disease.<sup>10</sup> This study showed no significant relationship between age and dental staining. Smoking becomes one of the extrinsic factors that cause discolouration in tooth enamel. The brownish-black tar staining is due to the tobacco sap from tobacco combustion.<sup>11</sup> Dental staining is also caused by other factors, such as poor oral hygiene, as seen from the great quantity of calculus found during examinations. The surface of this calculus was also exposed to tobacco smoke and discoloured.<sup>12</sup>

In this study, there is no significant relationship between the duration of smoking and the smoker's melanosis. This result is in line with a previous study conducted by Nadeem et al., which reported that smoker's melanosis is mostly found in persons who smoked for more than 14 years.<sup>22</sup> While in this study, the smoking duration grouped roughly  $\leq 10$  years  $\geq$  and more likely covers only a small sample size. Smoker's melanosis occurs due to increased melanin deposition in the oral epithelial layers, primarily as a protective adaptation in response to inflammatory mediators and potential carcinogenic effects caused by nicotine in tobacco products. Nicotine stimulates melanocytes, leading to melanin production.<sup>13</sup> The physiology of melanin pigmentation in the gingiva is affected by the number and size of blood vessels, epithelial thickness, degree of keratinization, and pigments within the gingival epithelium.<sup>14</sup> Yosadi et al. found that the longer the duration of smoking, the higher the melanin content.<sup>23</sup> Therefore, the likelihood of melanosis in the oral cavity increases with the duration of smoking.

There is a significant relationship between the duration of smoking and the smoker's palate. A smoker's palate, also known as nicotine stomatitis, is a lesion caused by physical irritation from cigarette smoke, particularly nicotine or 3-(1-methyl-2-pyrrolidiny) pyridine with the chemical formula of C<sub>10</sub>H<sub>14</sub>N<sub>2</sub>.<sup>15</sup> During the smoking process, the oral mucosa could reach a temperature of 190°C. Subsequently, high temperatures come into contact with palatal mucosa and cause inflammation of the ducts of minor salivary glands.<sup>16</sup> Microscopically, squamous cells in the walls of the ducts of salivary glands undergo hyperplasia, and parakeratosis occurs along the ducts of salivary glands.<sup>17</sup>

In this study, we found no significant relationship between the duration of smoking and periodontal disease. This may be due to other factors that have a stronger influence, including the oral hygiene of the respondents. The primary cause of periodontal disease is tissue irritation due to bacteria in plaque accumulation. The development process of gingival tissue inflammation is affected by the inflammatory process's duration, onset, and intensity, which varies greatly between individuals.<sup>21</sup> Smoking also increases the expression of pro-inflammatory cytokines such as Interleukin-1 (IL-1), contributing to increased tissue damage and alveolar bone resorption. Nicotine content in cigarettes can influence the process of proliferation, binding, and chemotaxis of periodontal ligament cells. This causes some pathogenic microorganisms to bind to epithelium easily.<sup>9</sup> Based on a study by Rohmawati et al., the duration of smoking of  $\geq 10$  years had a three times greater risk of having periodontal disease status,<sup>24</sup> which our study failed to capture due to sample size.

This study also shows no significant relationship between the duration of smoking and dental staining. Dental staining is caused by retention of tar and nicotine components in teeth and furfural reactions of acetaldehyde in cigarette smoke with glycoprotein of the pellicle of amino groups<sup>18</sup>. The absence of a significant relationship between the duration of smoking and the incidence of stain formation in this study was probably caused by the fact that most of the respondents' oral hygiene was poor. It has been reviewed by Bastian and Reade<sup>25</sup> that dental staining was not accumulated and affected by the duration of smoking and the number of cigarettes smoked but may depend on the number of bacteria in the dental plaque that absorbs tobacco combustion products (tar) and attached to the tooth surface.<sup>12</sup> The absence of a significant relationship between smoking frequency and dental staining could be caused by each individual's duration, frequency, and oral hygiene habits. The study conducted by Mubeen et al.<sup>18</sup> found that dental staining was almost twice as much in smokers than

nonsmokers. This is relevant to the study by Khalisa et al., which revealed that respondents who smoked >10 cigarettes daily had more stain formation.<sup>12</sup> Furthermore, another study by Oktanauli et al. also stated that teeth discolouration occurs in all categories of smokers, ranging from light to heavy smokers, regardless of the type of cigarette with filters and non-filters (kretek).<sup>11</sup>

There is a significant relationship between the incidence of smoker's melanosis and the frequency of smoking. The effects of cigarettes on oral mucosa are influenced by numerous factors, including the number of cigarettes smoked, the duration of smoking, the type of cigarettes smoked, and the depth of the cigarette suction.<sup>14</sup> Nicotine in cigarettes stimulates melanocytes in oral mucosa, producing excessive melanin, resulting in brown pigmentation of the buccal mucosa and gingiva.<sup>19</sup> The amount of melanin produced by melanocytes is genetically determined, and several pathways, including the adrenaline/  $\beta$ 2-adrenoceptor/cAMP/MITF pathway, the  $\alpha$ -MSH/MC1R/cAMP/MITF pathway, and the main regulator of the  $\beta$ -endorphin/ $\mu$ -opioid-receptor/PKC $\beta$  isoform signalling pathway, have been identified as main regulators<sup>20</sup>. DOPA chrome, which forms DHI-2-carboxylic acid (DHICA), will produce DHICA-melanin with a light brown colour. In the expression phase, melanosomes are transferred from melanocytes to keratinocytes, skin cells above melanocytes in the epidermis. Subsequently, the melanin colour is finally visible on the surface of the skin<sup>14</sup>. The results of this study are in line with Behura et al.,<sup>6</sup> that smoking >15 cigarettes a day has a risk of oral mucosal lesions 22.9 times higher compared to those who smoke  $\leq$ 15 cigarettes per day.

There is no significant relationship between the frequency of smoking and the incidence of a smoker's palate. The aetiology of a smoker's palate or nicotine stomatitis is the continuous irritating effect of heat and chemicals in cigarette smoke, especially tar, nicotine, and carbon monoxide. The high temperature during cigarette burning leads to inflammation of the minor salivary glands on the palatal mucosa.<sup>17</sup>

## CONCLUSION

Based on the results of this study, it can be concluded that there is no relationship between age and clinical manifestations of smoking in the oral cavity between smokers. Whereas in this study, the duration of smoking significantly contributes to the formation of nicotine stomatitis or smoker's palate, it has less effect on the formation of smoker's melanosis, periodontal diseases, and dental staining. The frequency of smoking, on the other hand, is significantly related to the smoker's melanosis. Further study with more subjects was needed to make a strong conclusion.

## ACKNOWLEDGEMENT

The authors gratefully acknowledge the Dinas Kesehatan Office of Banjarmasin City, where the initial data was collected.

## CONFLICT OF INTEREST

The authors declare no conflicts of interest related to this original report.

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