

## Prevalence of Oral Mucosal Lesions in Male Smokers and Nonsmokers

Fatemeh Ahmadi-Motamayel, Parisa Falsafi<sup>1</sup>, Zahra Hayati<sup>1</sup>, Farzad Rezaei<sup>2</sup> and Jalal Poorolajal<sup>3,\*</sup>

Dental Research Center, Department of Oral Medicine, School of Dentistry, Hamadan University of Medical Sciences, <sup>1</sup>Department of Oral Medicine, School of Dentistry, Hamadan University of Medical Sciences, <sup>2</sup>Department of Oral and Maxillofacial Surgery, School of Dentistry, Hamadan University of Medical Sciences, <sup>3</sup>Research Center for Health Sciences and Department of Epidemiology & Biostatistics, School of Public Health, Hamadan University of Medical Sciences, Hamadan, Iran

Tobacco smoking is one of the most important risk factors for the development of oral mucosal lesions such as leukoplakia and hairy tongue. Controversy exists in the literature, however, about the prevalence of oral lesions in smokers. The aim of this study was to evaluate oral lesions in male smokers compared with nonsmokers in Hamadan. A total of 516 male participants were assessed, 258 of whom were smokers and 258 of whom were healthy nonsmokers. The prevalence of lesions was evaluated by clinical observation and biopsy. We found that the most prevalent lesions among smokers were gingival problems and coated tongue; smokers had significantly more lesions than did nonsmokers. Malignant and premalignant lesions were found in a higher age range. Among all participants in our study, we found a large number of oral mucosal lesions in smokers that had a strong correlation with smoking. Dental services need to implement care and health education for smokers to promote health.

**Key Words:** Mouth; Smoking; Prevalence

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

### Article History:

received 1 May, 2013  
revised 10 June, 2013  
accepted 21 June, 2013

### Corresponding Author:

Jalal Poorolajal  
Research Center for Health Sciences  
and Department of Epidemiology &  
Biostatistics, School of Public Health,  
Hamadan University of Medical  
Sciences, Hamadan, Iran  
TEL: +98-0811-8381084  
FAX: +98-0811-8381085  
E-mail: Poorolajal@umsha.ac.ir  
Poorolajal@yahoo.com

## INTRODUCTION

Tobacco smoking and cigarettes cause DNA damage and increase the risk for oral cancer. Smoking is an important risk factor for oral diseases such as oral cancer, periodontal disease, cleft lip, cleft palate, alveolar bone loss, and black hairy tongue.<sup>1-3</sup> The evidence showing that the use of tobacco is the major risk factor for oral cancer and potentially malignant lesions of the mouth is obvious.<sup>4</sup>

Cigarettes contain over 4,000 constituents of chemicals and free radicals such as nicotine, ammonia, acrolein, phenols, acetaldehyde, benzopyrene, nitric oxides, carbon monoxide, polonium, radium, and thorium that can cause cellular damage.<sup>5,6</sup> Tobacco smoking is one of the most important risk factors for oral mucosal lesions (OMLs).<sup>7</sup> Cigarette smoking is also associated with oral leukoplakia, smoker's melanosis, frictional hyperkeratosis, nicotinic stomatitis or smoker's palate, black hairy tongue, and squamous cell carcinoma (SCC)<sup>8-10</sup> Smoking is a significant risk factor for periodontal disease. Tobacco contains a huge number of carcinogens, but the most significant of them are

the polycyclic aromatic hydrocarbons, aromatic amines, and nitrosamines.<sup>11</sup>

In 2011 Gönül et al.<sup>12</sup> studied OMLs in smokers and alcoholic patients and concluded that coated tongue was common among smokers and alcoholic patients. The results of research by Sujatha et al.<sup>13</sup> in 2012 showed that oral leukoplakia was the most common OML among smokers. Saintrain et al.<sup>14</sup> in 2012 concluded that OMLs are more common in smokers. Pipe and cigarette smoking along with consumption of alcohol, poor oral hygiene, and the use of poorly fitted prostheses are risk factors associated with the development of oral carcinoma, and the most affected sites are the tongue and floor of the mouth.<sup>14</sup> A previous smoking habit is recognized as an addiction that exposes people to many toxic substances.<sup>14</sup>

Tobacco smoking is responsible for many oral cavity conditions including nonmalignant, precancerous, and malignant lesions. Most of these oral lesions and conditions can be detected by a simple visual inspection of the oral cavity.<sup>15</sup> Controversy remains among these studies in the literature, however. Thus, the purpose of the present study was

to determine the prevalence of oral soft tissue lesions in smokers compared with nonsmokers.

**MATERIALS AND METHODS**

A total of 516 men (258 nonsmokers and 258 smokers, with an age range of 20-50 years) participated in this study. The case group used to smoke at least 5 cigarettes a day, since at least 5 years ago. The control group was selected from healthy nonsmokers who attended the Oral Medicine Department of Hamadan Dental School for routine dental examinations. Participants who had systemic disease (cardiovascular, endocrine, gastrointestinal, oral, or respiratory disease), who consumed alcohol, or who were under drug treatment were excluded.

The study protocol was approved by the ethics committee of Hamadan University of Medical Science. All participants were informed and approved that the study goals had been explained to them.

The case and control groups were examined by post-graduate students of the Department of Oral Medicine, who used a light, mouth mirror, gauze, and Universal Periodontal Probe (Elephant and Castle, London, United Kingdom). The diagnosis was made on the basis of medical history, clinical features, and investigations according to World Health Organization guidelines. Questionable lesions were investigated further, such as by biopsy. Because of controversy owing to the inconclusive results of similar studies, the purpose of the present study was to determine the prevalence of oral soft tissue lesions in smokers com-

pared with healthy nonsmokers. Lesions were recorded only if they were observed at the time of examination. The prevalence of oral mucosal developmental anomalies was not evaluated in this study.

Data were collected during 5 months of follow-up and were assessed by use of chi-square tests and logistic regression models at the 0.05 significance level by use of SPSS-19 statistical software to assess the effects of smoking habits on oral lesions. We considered adjusted odds ratios (ORs) to assess covariate effects on oral lesions.

**RESULTS**

The mean age of the participants was 29.26±7.359 years. In all 258 smoker participants, the most common lesions were coated tongue [258 (100%)], gingivitis [148 (57.3%)], oral melanosis [120 (46.5%)], and periodontitis [110 (42.6%)]. Furthermore, 3 (1.1%) of the participants had SCC and 7 (2.7%) had oral leukoplakia. Nearly all 258 participants had some levels of halitosis and dental stains. In comparison with smokers, the most common OMLs in the control group (nonsmokers) were coated tongue [91 (35.2%)], gingivitis [83 (32.1%)], oral melanosis [25 (9.6%)], and periodontitis [15 (5.8%)]; leukoplakia [2 (0.7%)] and SCC [0 (0%)] were low in nonsmokers (Table 1). Oral halitosis was uncommon in the control group. The total number and intensity of oral lesions was far greater in smokers than in the control group. The prevalence of OMLs was statistically different between the two groups.

The effect of smoking on oral lesions was evaluated by

**TABLE 1.** Prevalence of oral lesions among smokers (cases) and nonsmokers (controls)

Variables	Cases (N=258)		Controls (N=258)		Odds ratio		p value
	Yes	No	Yes	No	Statistics	95% CI	
Gingivitis	148	110	83	175	2.83	1.94, 4.13	0.001
Periodontitis	110	148	15	243	12.04	6.65, 23.00	0.001
Melanosis	120	138	25	233	8.10	4.93, 13.64	0.001
Friction keratosis	19	239	2	256	10.17	2.41, 90.71	0.001
Leukoplakia	7	251	2	256	3.57	0.67, 35.45	0.092
Nicotinic stomatitis	6	252	0	258	ND	-	-
Squamous cell carcinoma	3	255	0	258	ND	-	-
Coated tongue	258	0	91	167	ND	-	-

CI: confidence interval, ND: no data (could not be measured).

**TABLE 2.** Odds ratio (OR) estimates of the effect of smoking habits and 1-year increase in age on oral lesions by logistic regression analysis

Lesion	Effect of age adjusted for smoking			Effect of smoking adjusted for age		
	OR	95% CI	p value	OR	95% CI	p value
Gingivitis	1.01	0.99, 1.04	0.179	2.85	1.96, 4.13	0.001
Periodontitis	1.00	0.97, 1.04	0.787	12.06	6.70, 21.71	0.001
Melanosis	1.00	0.97, 1.03	0.952	8.13	4.98, 13.28	0.001
Frictional keratosis	1.03	0.97, 1.10	0.321	11.07	2.50, 49.03	0.002
Leukoplakia	1.02	0.92, 1.12	0.739	3.13	0.60, 16.26	0.174

CI: confidence interval.

using OR estimates (Table 1). According to these results, the OR estimates in smokers over nonsmokers were 2.83 for gingivitis, 12.04 for periodontitis, 8.10 for melanosis, 10.17 for friction keratosis, and 3.57 for leukoplakia. OR estimates were not calculated for nicotinic stomatitis or SCC because none of the subjects in the control group had these lesions. ORs were also not calculated for coated tongue because all smokers had the lesion.

The effects of smoking on oral lesions adjusted for age and the effects of a 1-year increase in age adjusted for smoking habit are shown in Table 2. Aging had no significant effect on oral lesions, whereas the effect of smoking was statistically significant for lesions other than leukoplakia.

## DISCUSSION

Tobacco smoking has many deleterious effects on the oral mucosa<sup>13</sup> and is one of the most important risk factors for oral cancer. Early detection and screening of smokers is very important. In this study, 3 of the 258 smokers (1.1%) had SCC. Thus, implementation of regular dental services and care and health education is essential in high-risk patients.<sup>14</sup> In one study, the most common OMLs were fissured tongue, coated tongue, aphthae, linea alba, glossitis, leukoplakia, candidiasis, and macroglossia.<sup>12</sup> In this study, we did not evaluate oral mucosal developmental anomalies such as fissured tongue, linea alba, lip pit, and Fordyce granules; we assessed only lesions. However, coated tongue was also common in our study.

We selected the same age range for the two groups and did not evaluate the effect of age on oral lesions. The results of another study showed that smoking and age are significant risk factors for OMLs.<sup>12</sup> Also in another study, smokers were about 7 years younger than nonsmokers.<sup>16</sup>

To improve primary and secondary prevention according to our study, the results showed that smokers had greater and more severe lesions than did nonsmokers; some of these lesions were premalignant and altered oral, dental, and gingival health. Therefore, young adults should be informed more extensively about the negative and deleterious effects of tobacco smoking on oral health.<sup>17</sup> Smoking or addiction is very low among Iranian women because of cultural statues in Iran. Thus, we chose our participants from among men only. Fordyce granules were the most frequent in smokers and OMLs were more prevalent among men than women.<sup>18</sup> Another study showed that the most prevalent normal variations and oral lesions were as follows: Fordyce granules, fissured tongue, leukoedema, varices, frictional hyperkeratosis, smoker's palates, aphthous stomatitis, oral submucosal fibrosis, oral malignant leukoplakia, median rhomboid glossitis, oral candidiasis, lichen planus, traumatic ulcer, stomatitis, geographic tongue, betel chewers mucosa, irritation fibroma, angular cheilitis, and herpeslabialis.<sup>18</sup> In this study, we only examined lesions that were present in the oral mucosa at the time of examination. We did not assess the history of herpes or aphthous ulceration.

The prevalence of cancer in our study was 1.1%. Another study concluded that the prevalence of cancer is 0.03%, and their results showed a correlation between habits and lesions that was similar to our results.<sup>19</sup>

In this study we selected only cigarette smokers and did not evaluate other habits such as alcohol and tobacco chewing. In another study, OMLs differed according to some habits, but were most prevalent in smokers.<sup>20</sup> Therefore, close follow-up and systematic evaluation is more necessary in smokers. It is also necessary to educate oral hygienists, dentists, and medical specialists about how to treat smokers.<sup>20</sup>

In a study of leukoplakia, the prevalence of leukoplakia was 0.59%, and smoking and chewing tobacco were significant predictors of leukoplakia. In our study, leukoplakia was observed in 2.7% of patients.<sup>21</sup> In another study also smoking had a huge correlation with the frequency of leukoplakia in the oral cavity.<sup>22</sup> Smoking tobacco is the most responsible factor for the development of intraoral white lesions. These lesions are more frequent in tobacco chewers and pipe smokers than in those who smoke cigarettes, and the amount of daily consumption and period of addiction are also important.<sup>14</sup> However, leukoplakia (14%) was the most common mucosal condition in another study.<sup>13</sup> This difference may be explained by patient selection in this study, in which cigarette smokers were selected from those who had used cigarettes for at least 5 years or more. However, the number of cigarettes used by the smokers in these two studies may have differed.

A study showed that heavy smokers exhibit worse periodontal tissue breakdown and less bleeding tendency, which was similar to our study. Smoking is a major environmental factor that is associated with accelerated periodontal destruction despite regular oral hygiene habits.<sup>23</sup>

After analyzing the data, we concluded that OMLs are significantly more common in smokers. According to the literature, these outcomes were the same as in other studies.<sup>24-27</sup> Dentists must be aware of the effect of smoking on the development of oral pathologic lesions and encourage smokers to quit.<sup>14</sup>

Tobacco smoking increases the number of aneuploid nuclei in the oral epithelium<sup>28</sup> and causes oral malignant and premalignant lesions. Therefore, inspection of the oral cavity to find lesions caused by tobacco is a good way to initiate tobacco cessation.<sup>15</sup>

In this study, the most common OML was coated tongue and gingival problems, and OMLs were statistically higher in smokers than in nonsmokers. Smoking may be a predisposing factor in the development of gingival problems. In our study, we found oral malignant and premalignant lesions in smokers. Thus, the oral mucosa should be examined carefully, especially in smokers, even if the patients did not attend with the complaint of oral lesions. Careful periodical assessment of OMLs in smokers seems to be essential because of the side effects and lesions caused by smoking and to improve primary and secondary prevention.

## REFERENCES

1. Khalili J. Oral cancer: risk factors, prevention and diagnostic. *Exp Oncol* 2008;30:259-64.
2. Bánóczy J, Gintner Z, Dombi C. Tobacco use and oral leukoplakia. *J Dent Educ* 2001;65:322-7.
3. Khor GH, Siar CH, Jusoff K. Chromosome 17 aberration of oral squamous cell carcinoma in Malaysia. *Global J Health Sci* 2009;1:150-6.
4. Warnakulasuriya S, Sutherland G, Scully C. Tobacco, oral cancer, and treatment of dependence. *Oral Oncol* 2005;41:244-60.
5. Pasupathi P, Saravanan G, Farook J. Oxidative stress biomarkers and antioxidant status in cigarette smokers compared to nonsmokers. *J Pharm Sci & Res* 2009;1:55-62.
6. Pryor WA, Hales BJ, Premovic PI, Church DF. The radicals in cigarette tar: their nature and suggested physiological implications. *Science* 1983;220:425-7.
7. Hayes RB, Bravo-Otero E, Kleinman DV, Brown LM, Fraumeni JF Jr, Harty LC, et al. Tobacco and alcohol use and oral cancer in Puerto Rico. *Cancer Causes Control* 1999;10:27-33.
8. Greenburg MS, Glick M, Ship JA. *Burket's Oral Medicine*. 11th Ed. 2008;77-106.
9. Bánóczy J, Gintner Z, Dombi C. Tobacco use and oral leukoplakia. *J Dent Educ* 2001;65:322-7.
10. Meraw SJ, Mustapha IZ, Rogers RS 3rd. Cigarette smoking and oral lesions other than cancer. *Clin Dermatol* 1998;16:625-31.
11. Ding YS, Zhang L, Jain RB, Jain N, Wang RY, Ashley DL, et al. Levels of tobacco-specific nitrosamines and polycyclic aromatic hydrocarbons in mainstream smoke from different tobacco varieties. *Cancer Epidemiol Biomarkers Prev* 2008;17:3366-71.
12. Gönül M, Gül U, Kaya I, Koçak O, Cakmak SK, Kılıç A, et al. Smoking, alcohol consumption and denture use in patients with oral mucosal lesions. *J Dermatol Case Rep* 2011;5:64-8.
13. Sujatha D, Hebbar PB, Pai A. Prevalence and correlation of oral lesions among tobacco smokers, tobacco chewers, areca nut and alcohol users. *Asian Pac J Cancer Prev* 2012;13:1633-7.
14. Saintrain MV, Holanda TG, Bezerra TM, de Almeida PC. Prevalence of soft tissue oral lesion in elderly and its relations with deleterious habits. *Gerodontology* 2012;29:130-4.
15. Benomar S, Boutayeb S, Nitassi S, Hassam B, Ismaili N. Tobacco-associated lesions of the oral cavity and motivation for smoking cessation: a study of 121 cases. *Presse Med* 2009;38:1746-9.
16. Pentenero M, Giaretti W, Navone R, Rostan I, Gassino L, Broccoletti R, et al. Evidence for a possible anatomical subsite-mediated effect of tobacco in oral potentially malignant disorders and carcinoma. *J Oral Pathol Med* 2011;40:214-7.
17. Morger R, Ramseier CA, Rees TD, Bürgin WB, Bornstein MM. Oral mucosal findings related to tobacco use and alcohol consumption: a study on Swiss army recruits involving self-reported and clinical data. *Oral Health Prev Dent* 2010;8:143-51.
18. Mathew AL, Pai KM, Sholapurkar AA, Vengal M. The prevalence of oral mucosal lesions in patients visiting a dental school in Southern India. *Indian J Dent Res* 2008;19:99-103.
19. Lay KM, Sein K, Myint A, Ko SK, Pindborg JJ. Epidemiologic study of 600 villagers of oral precancerous lesions in Bilugyun: preliminary report. *Community Dent Oral Epidemiol* 1982;10:152-5.
20. Mehrotra R, Thomas S, Nair P, Pandya S, Singh M, Nigam NS, et al. Prevalence of oral soft tissue lesions in Vidisha. *BMC Res Notes* 2010;3:23.
21. Saraswathi TR, Ranganathan K, Shanmugam S, Sowmya R, Narasimhan PD, Gunaseelan R. Prevalence of oral lesions in relation to habits: Cross-sectional study in South India. *Indian J Dent Res* 2006;17:121-5.
22. Lima JS, Pinto Ddos S Jr, Sousa SO, Corrêa L. Oral leukoplakia manifests differently in smokers and non-smokers. *Braz Oral Res* 2012;26:543-9.
23. Vouros ID, Kalpidis CD, Chadjipantelis T, Konstantinidis AB. Cigarette smoking associated with advanced periodontal destruction in a Greek sample population of patients with periodontal disease. *J Int Acad Periodontol* 2009;11:250-7.
24. Kleinman DV, Swango PA, Pindborg JJ. Epidemiology of oral mucosal lesions in United States schoolchildren: 1986-87. *Community Dent Oral Epidemiol* 1994;22:243-53.
25. Dundar N, Ilhan Kal B. Oral mucosal conditions and risk factors among elderly in a Turkish school of dentistry. *Gerontology* 2007;53:165-72.
26. Lin HC, Corbet EF, Lo EC. Oral mucosal lesions in adult Chinese. *J Dent Res* 2001;80:1486-90.
27. Castellanos JL, Díaz-Guzmán L. Lesions of the oral mucosa: an epidemiological study of 23785 Mexican patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2008;105:79-85.
28. Souto GR, Caliar MV, Lins CE, de Aguiar MC, de Abreu MH, Mesquita RA. Tobacco use increase the number of aneuploid nuclei in the clinically healthy oral epithelium. *J Oral Pathol Med* 2010;39:605-10.