



CODEN [USA]: IAJPBB

ISSN: 2349-7750

**INDO AMERICAN JOURNAL OF  
PHARMACEUTICAL SCIENCES**<http://doi.org/10.5281/zenodo.3250316>Available online at: <http://www.iajps.com>

Research Article

**STUDY TO DETERMINE THE ASSOCIATION BETWEEN  
SMOKING AND PERIODONTITIS****<sup>1</sup>Dr Ayesha Aftab and <sup>2</sup>Dr Fahima Maqbool <sup>3</sup>Dr Sayyeda Tatheer Fatima**<sup>1</sup>Dental Surgeon Sharif Medical and Dental College Lahore<sup>2</sup>BDS Bolan University Of Medical And Health Sciences Quetta<sup>3</sup>DOW University of Health Sciences Karachi

Article Received: April 2019

Accepted: May 2019

Published: June 2019

**Abstract:**

*Smoking is an important factor in periodontitis. Many studies have found that active smoking is closely associated with the prevalence and severity of periodontal disease. The purpose of this study was to examine the association of smoking with the prevalence of periodontal disease. **Material & Methods:** A Cross-sectional descriptive study was conducted over a period of 6 months from Oct 2018 to March 2019, in the Dental Department of Services Hospital Lahore. The study was approved by the Ethical Review Board/ Committee of Services Hospital Lahore. Informed consent was taken from the patients. A total of 500 smokers were selected through questioning during screening from the population of patients presenting to outdoor/ diagnosis department. The smokers underwent a further detailed periodontal examination and were screened for the presence of periodontal disease. They were also cross questioned to rule out other causative factor related to periodontitis such as poor oral hygiene and diabetes. **Conclusion and Clinical Significance:** Smoking is a significant risk factor for impaired periodontal health. This needs to be considered before treatment planning for good prognosis and affective results of the provided treatment. Hence this habit must be diagnosed during history taking, evaluation and patient should be instructed to minimize and ultimately stop cigarette smoking for best treatment outcome.*

**Key words:** Smoking; periodontitis; periodontal disease; risk factor.

**Corresponding author:****Dr Ayesha Aftab,**

Dental Surgeon Sharif Medical and Dental College Lahore

QR code



Please cite this article in press Ayesha Aftab *et al.*, *Study To Determine The Association Between Smoking And Periodontitis.*, *Indo Am. J. P. Sci.*, 2019; 06(06).

### INTRODUCTION:

Periodontitis is associated with a number of factors (Table 1). Smoking is a risk factor of several diseases such as lung cancers, myocardial infarctions, cardiovascular disease, chronic ischemic heart diseases, and strokes. It also affects the prevalence, extent, and severity of periodontal diseases. Many studies have shown the association between and periodontitis. Due to the high number of smokers in many countries, the association between cigarette smoking and periodontal diseases is a significant public health problem. The aim of this research was to examine evidence for the association between smoking and periodontal disease, to discuss its pathology and its adverse effects on the periodontium.

A total of 500 smokers were selected from the patients presenting to outdoor/ diagnosis dept of Dental Services Hospital, Lahore. The smokers underwent a detailed periodontal exam to evaluate the presence of periodontitis. It was noted that the accumulation of carbon debris on tooth surfaces of smokers leads to increased plaque buildup. Mature supra or sub gingival plaque leads to periodontitis. Moreover, a decrease in the vascular supply of the underlying epithelium and a resulting decrease in the concentration of WBC's and reduced immune response favored the progression of periodontal disease.

Of the smokers who were screened for periodontitis, a large percentage (68 %) had some form of periodontitis whether it was bone loss, pocket formation or mobile teeth.

### MATERIAL & METHODS:

A Cross- sectional descriptive study was conducted over a period of 6 months from Oct 2018 to March 2019, in the Dental Department of Services Hospital Lahore. The study was approved by the Ethical Review Board/ Committee of Services Hospital Lahore. Informed consent was taken from the patients. A total of 500 smokers were selected through questioning during screening from the population of patients presenting to outdoor/ diagnosis department of Dentistry, Services Hospital Lahore. The smokers underwent a further detailed periodontal examination and were screened for the presence of periodontal disease. They were also cross questioned to rule out other causative factor related to periodontitis such as poor oral hygiene and diabetes.

### DISCUSSION AND LIMITATIONS:

Many authors suggested that tobacco smoking is associated with increased accumulation of supragingival and subgingival dental calculi.

Although smokers had more calculus deposits than nonsmokers, the effect of smoking was independent of the number of present calculus debris. There have been consistent reports of more calculi in smokers than in nonsmokers [5].

In three-day-old plaque, the proportion of gram-positive bacteria was statistically higher in smokers in comparison with nonsmokers [3]. There are some controversial findings. Bergstrom et al. found no difference in mean plaque index scores amongst 285 musicians (31% smokers and 69% nonsmokers). In addition, they found no quantitative difference in the growth rates of plaque between smokers and nonsmokers [4].

In another study, PDL cells were plated for one day and then were treated with various concentrations of smoke extract (CSE). It was concluded that CSE causes cell death at concentrations equal to or greater than 5%. PDL cells-induced collagen gel contraction was reduced at CSE concentrations of 1.5%. CSE selectively increased the expression of collagen V $\alpha$ 3, decreased collagen XI $\alpha$ 1, and increased the expression of matrix metalloproteinase 1 (MMP1), MMP3, and to a lesser extent MMP2 and MMP8. CSE also increased the expression of integrins  $\alpha$ 1,  $\alpha$ 2, and  $\alpha$ 10 (collagen receptors) as well as integrin  $\alpha$ 9 (a tenascin receptor) (28).

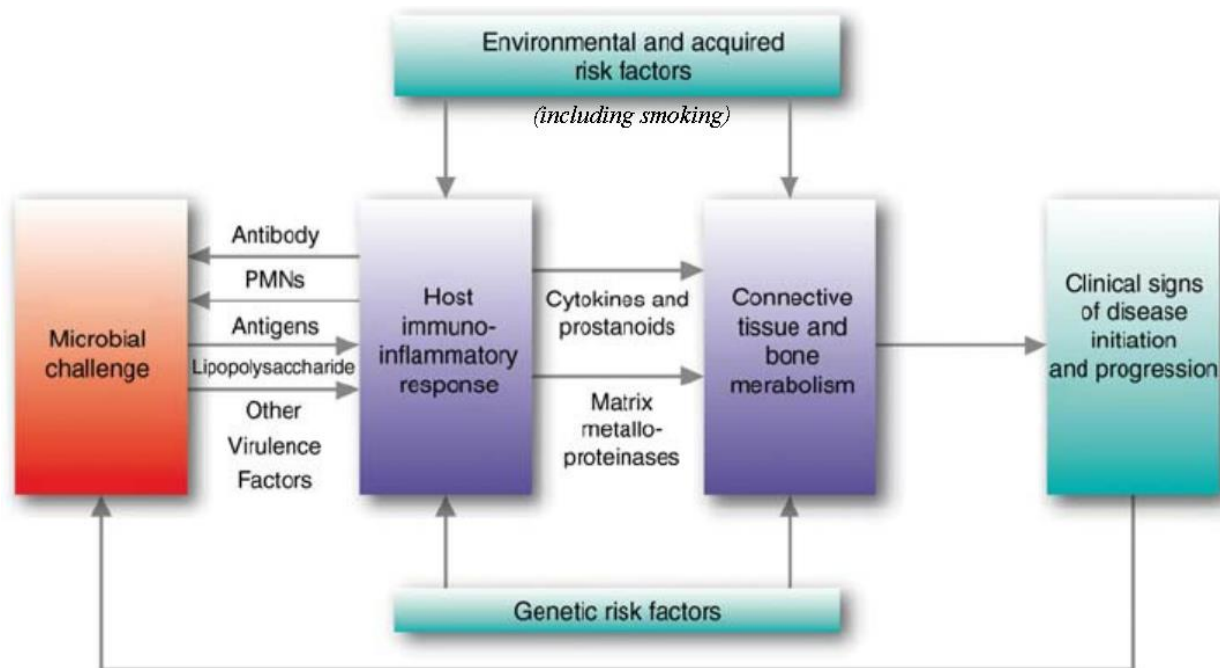
Bergstrom et al. declared that probing depth (PD), alveolar bone loss, and tooth mobility were significantly increased in smokers (4). It has been noted that cigarette smokers had significantly fewer teeth than nonsmokers. The effect of smoking in tooth loss is due to periodontal disease rather than tooth decay. Gomes et al. showed that visible plaque index, gingival bleeding index, and bleeding on probing were similar in smokers and nonsmokers; however, regardless of tooth surface, periodontal PD in buccal/lingual sites and clinical attachment loss were more in smokers than in nonsmokers (20). Severe periodontal disease with increased bone loss, greater periodontal attachment loss, gingival recession, and periodontal pocket formation are more frequent in smokers (48). Many authors declared that effect of cigarette consumption and periodontal attachment loss were dose dependent. Destructive effects of smoking on periodontal tissues may be mainly from systemic side effects and almost independent of the site within the mouth although some additional local effects may be present in areas such as anterior palatal sites (49). Comparing clinical parameters of periodontal disease between smokers and nonsmokers showed that clinical symptoms were greater in smokers. The symptoms were presented by

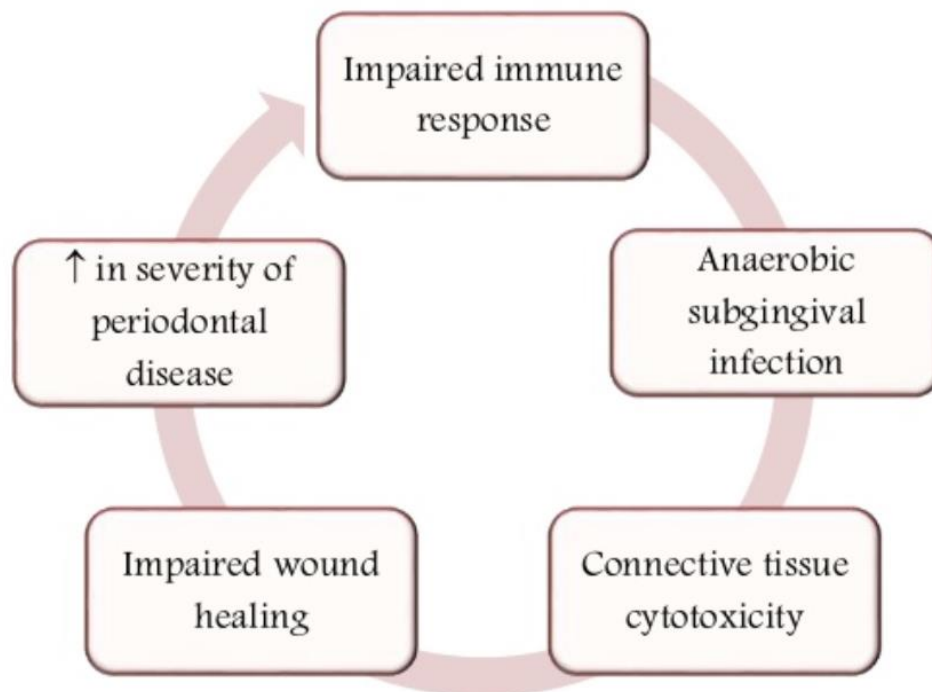
a greater number of perio pockets and gingival recession, excessive loss of epithelial attachment, and accelerated alveolar bone loss; however, gingival bleeding was reduced in these patients <sup>(48, 51)</sup>. An additional analysis of gingival tissue adjacent to

periodontitis sites showed that MMP-2 levels were higher in the exposed than in unexposed animals. This finding suggests that MMP-2 might be one of the molecules responsible for the increased tissue degradation in the periodontal tissues of smokers.

*Table 1 –Risk factors associated with periodontal disease*

1. Genetic predisposition
2. Smoking and tobacco use
3. Mal-aligned or crowded teeth
4. Parafunctional habits e.g. Grinding or clenching of teeth
5. Hormonal imbalance e.g. during pregnancy
6. Medicines such as Phenytoin, Cyclosporine and Nefidipine
7. Systemic Diseases e.g. Diabetes
8. Poor nutrition e.g Scurvy



*Mechanism of periodontal disease progression by smoking***CONCLUSIONS:**

Tobacco smoking has widespread systemic effects, many of which may provide mechanisms for increased susceptibility to periodontitis and the poor response to treatment. The frequency of smoking directly affects the prevalence of periodontal disease. The smoking status of the family members may also be relevant to both behavioral influences and the potential consequence of passive smoking. Smoking changes the normal bacterial population in the mouth and human immune response that leads to destruction of the supporting tissues of the tooth. More research needs to be done to study the exact means by which smoking affects the health of periodontium. It has been noted that a down regulation of anti-inflammatory factors along with an upregulation of proinflammatory cytokines is involved. Different byproducts of cigarette smoke might have different effects on the progression of periodontitis. Moreover, treatments in patients with periodontal disease must be focused on understanding the association between genetic and environmental factors. We can identify our patients' risks and achieve better results only through customizing dental treatment to suit individual needs better. To pursue strategies of prevention, early detection of disease, and prompt

intervention, the dental profession should continue to target and educate patients regarding the effects of smoking on periodontal health. In this way, dentistry will also make a significant contribution to the general health and well-being of our youth and future generation.

**Conflict of Interest.**

The authors do not report any conflict of interest in this study. No financial help of any sort was received in funding this study

**REFERENCES:**

1. Torzabab P, Khalili Z, Ziaei N Smoking and Periodontal Diseases Avicenna J Dent Res. 2013 December; 5(2): e20218.
2. Tonetti MS. Cigarette smoking and periodontal diseases: etiology and management of disease. *Ann Periodontol.* 1998;3(1):88–101.
3. Wilson TG, Jr. Effects of smoking on the periodontium. *Quintessence Int.* 1998;29(4):265–6.
4. Bergstrom J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. *J Periodontol.* 1991;62(4):242–6.

5. Martinez-Canut P, Lorca A, Magan R. Smoking and periodontal disease severity. *J Clin Periodontol*. 1995;**22**(10):743–9.
6. Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *J Periodontol*. 2000;**71**(12):1874–81.
7. Bergstrom J. Tobacco smoking and subgingival dental calculus. *J Clin Periodontol*. 2005;**32**(1):81–8.
8. Leroy K, Brion JP. Developmental expression and localization of glycogen synthase kinase-3beta in rat brain. *J Chem Neuroanat*. 1999;**16**(4):279–93.
9. Preber H, Bergstrom J. Occurrence of gingival bleeding in smoker and non-smoker patients. *Acta Odontol Scand*. 1985;**43**(5):315–20.
10. Anil S. Study of the patterns of perio destruction in smokers with chronic periodontitis. *Indian J Dent Res*. 2008;**19**(2):124–8.
11. Powell JT. Vascular damage from smoking: disease mechanisms at the arterial wall. *Vasc Med*. 1998;**3**(1):21–8.
12. Bergstrom J, Persson L, Preber H. Influence of cigarette smoking on vascular reaction during experimental gingivitis. *Scand J Dent Res*. 1988;**96**(1):34–9.
13. Palmer RM, Scott DA, Meekin TN, Poston RN, Odell EW, Wilson RF. Potential mechanisms of susceptibility to periodontitis in tobacco smokers. *J Periodontol Res*. 1999;**34**(7):363–9.
14. Mirbod SM, Ahing SI, Pruthi VK. Immuno histochemical study of vestibular gingival blood vessel density and internal circumference in smokers and non-smokers. *J Periodontol*. 2001;**72**(10):1318–23.
15. Holmes LG. Effects of smoking and/or vitamin C on crevicular fluid flow in clinically healthy gingiva. *Quintessence Int*. 1990;**21**(3):191–5.
16. Kinane DF, Radvar M. The effect of smoking on mechanical and antimicrobial periodontal therapy. *J Periodontol*. 1997;**68**(5):467–72.
17. Rawlinson A, Grummitt JM, Walsh TF, Ian Douglas CW. Interleukin 1 and receptor antagonist levels in gingival crevicular fluid in heavy smokers versus non-smokers. *J Clin Periodontol*. 2003;**30**(1):42–8.
18. Alavi AL, Palmer RM, Odell EW, Coward PY, Wilson RF. Elastase in gingival crevicular fluid from smokers and non-smokers with chronic inflammatory periodontal disease. *Oral Dis*. 1995;**1**(3):110–4.
19. Chen X, Wolff L, Aeppli D, Guo Z, Luan W, Baelum V, et al. Cigarette smoking, salivary/gingival crevicular fluid cotinine and periodontal status. A 10-year longitudinal study. *J Clin Periodontol*. 2001;**28**(4):331–9.
20. Gomes SC, Piccinin FB, Oppermann RV, Susin C, Nonnenmacher CI, Mutters R, et al. Periodontal status in smokers and never-smokers: clinical findings and real-time polymerase chain reaction quantification of putative periodontal pathogens. *J Periodontol*. 2006;**77**(9):1483–90.
21. Sayers NM, James JA, Drucker DB, Blinkhorn AS. Possible potentiation of toxins from *Prevotella intermedia*, *Prevotella nigrescens*, and *Porphyromonas gingivalis* by cotinine. *J Periodontol*. 1999;**70**(11):1269–75.
22. Umeda M, Chen C, Bakker I, Contreras A, Morrison JL, Slots J. Risk indicators for harboring periodontal pathogens. *J Periodontol*. 1998;**69**(10):1111–8.
23. Apatzidou DA, Riggio MP, Kinane DF. Impact of smoking on the clinical, microbiological and immunological parameters of adult patients with periodontitis. *J Clin Periodontol*. 2005;**32**(9):973–83.
24. Haffajee AD, Cugini MA, Dibart S, Smith C, Kent RL, Jr, Socransky SS. The effect of SRP on the clinical and microbiological parameters of periodontal diseases. *J Clin Periodontol*. 1997;**24**(5):324–34.
25. Giannopoulou C, Roehrich N, Mombelli A. Effect of nicotine-treated epithelial cells on the proliferation and collagen production of gingival fibroblasts. *J Clin Periodontol*. 2001;**28**(8):769–75.
26. James JA, Sayers NM, Drucker DB, Hull PS. Effects of tobacco products on the attachment and growth of periodontal ligament fibroblasts. *J Periodontol*. 1999;**70**(5):518–25.
27. Wendell KJ, Stein SH. Regulation of cytokine production in human gingival fibroblasts following treatment with nicotine and lipopolysaccharide. *J Periodontol*. 2001;**72**(8):1038–44.
28. Giannopoulou C, Geinoz A, Cimasoni G. Effects of nicotine on periodontal ligament fibroblasts in vitro. *J Clin Periodontol*. 1999;**26**(1):49–55.
29. Bulmanski Z, Brady M, Stoute D, Lallier TE. Cigarette smoke extract induces select matrix metalloproteinases and integrin expression in periodontal ligament fibroblasts. *J Periodontol*. 2012;**83**(6):787–96.
30. Ryder MI, Hyun W, Loomer P, Haqq C. Alteration of gene expression profiles of peripheral mononuclear blood cells by tobacco smoke: implications for periodontal diseases. *Oral Microbiol Immunol*. 2004;**19**(1):39–49.

31. Giannopoulou C, Cappuyns I, Mombelli A. Effect of smoking on gingival crevicular fluid cytokine profile during experimental gingivitis. *J Clin Periodontol.* 2003;**30**(11):996–1002.
32. Morozumi T, Kubota T, Sugita N, Itagaki M, Yoshie H. Alterations of gene expression in human neutrophils induced by smoking cessation. *J Clin Periodontol.* 2004;**31**(12):1110–6.
33. Hodge PJ, Teague PW, Wright AF, Kinane DF. Clinical and genetic analysis of a large North European Caucasian family affected by early-onset periodontitis. *J Dent Res.* 2000;**79**(3):857–63.
34. Meisel P, Siegemund A, Dombrowa S, Sawaf H, Fanghaenel J, Kocher T. Smoking and polymorphisms of the interleukin-1 gene cluster (IL-1alpha, IL-1beta, and IL-1RN) in patients with periodontal disease. *J Periodontol.* 2002;**73**(1):27–32.
35. Koundouros E, Odell E, Coward P, Wilson R, Palmer RM. Soluble adhesion molecules in serum of smokers and non-smokers, with and without periodontitis. *J Periodontal Res.* 1996;**31**(8):596–9.