

Tobacco Use and Its Effects on the Periodontium and Periodontal Therapy

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Abstract

Aim: The purpose of this article is to present a review of the potential biological mechanisms underlying the effects of tobacco smoking on periodontal health and periodontal therapy.

Background: Periodontitis is the result of complex interrelationships between infectious agents and host factors. Environmental, acquired, and genetic risk factors modify the expression of disease and may, therefore, affect the onset or progression of periodontitis.

Review Results: The study of the relationship between periodontal disease and smoking has received increased attention during the last few years. Tobacco smoking has wide spread systemic effects, many of which may provide mechanisms for the increased susceptibility to periodontitis and the poorer response to treatment.

Conclusion: Tobacco smoking is a significant risk factor for periodontal disease.

Clinical Significance: The role smoking plays in periodontal disease should be considered by clinicians and patients during active periodontal therapy and the oral health maintenance phases of care.

Keywords: Tobacco, smoking, periodontitis, literature reviews, risk factors

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Introduction

Forty years have passed since the landmark U.S. Surgeon General's report in 1964 warned that smoking played a causative role in lung cancer and was associated with cardiovascular diseases. Since then, the list of smoking related health effects has grown. A large body of epidemiological research indicates tobacco use is directly related to the incidence and prevalence of a variety of medical problems including cancer, low birth weight, pulmonary, cardiovascular, and gastrointestinal diseases.¹

Dentistry has long been aware of the effects of tobacco on the soft tissues of the oral cavity and the implications of this in clinical practice. In the past 20 years there has been an increasing awareness of the role of tobacco use on the prevalence and severity of periodontal diseases.² As proposed by Gelesky,³ smoking meets the majority of criteria given for causation of a disease. Experimental evidence accumulated over the last two decades has indicated cigarette smoking is a true risk factor for periodontitis. This environmental exposure has been associated with two to three fold increases in the odds of developing clinically detectable periodontitis and subsequent tooth loss.⁴

An overwhelming body of data from multiple cross-sectional and longitudinal studies conducted have demonstrated pocket depth and clinical attachment loss were more prevalent and severe in patients who smoke compared with non-smokers.⁵ It has been estimated there are 1.1 billion smokers worldwide and 182 million



(16.6%) of them live in India.⁶ Tobacco is used in smoking and smokeless forms in India. Among tobacco users, 34% smoke Bidis, 31% are regular cigarette smokers, and 35% use smokeless tobacco.⁷ Bidis are chocolate, mint, or fruit flavored tobacco cigarettes hand-rolled in tendu or temburni leaf and are common throughout India and southeast Asia and are especially appealing to young smokers. The prevalence of smoking among 13-15 year old school going students in India ranges from 19.7-34.5%, even the lowest was considerably higher than the global median of current cigarette smoking (13.9%).¹

Smoking and Acute Necrotizing Ulcerative Gingivitis (ANUG)

An association between necrotizing forms of periodontal disease and tobacco smoking was reported as early as 1947 and has long been considered an etiologic factor in ANUG. Preexisting gingivitis, emotional/psychic stress, and smoking forms a triad of interrelated predisposing factors in the etiology of the disease. Smoking has shown to influence the tissue response to irritation. Karadachi et al.⁸ notes smoking activates the release of epinephrine and promotes contraction of peripheral vessels reducing blood flow to the gingiva. Severe reduction of blood flow induced by sepsis, stress, and smoking may cause loss of vitality to the most vulnerable regions of the gingival epithelium, leading to the onset of ANUG.

Interaction Between Smoking and Systemic Health Status

The combination of smoking with other systemic factors further enhances the risk of periodontal destruction. In an Erie County Pennsylvania study the combination of diabetes and heavy smoking in an individual over the age of 45 years who harbored *P. gingivalis* or *T. forsythensis* resulted in an odds ratio of attachment loss 30 times that of a person lacking these risk factors.⁹ Smoking also increases the risk of attachment and/or bone loss in AIDS and HIV serotype patients.

Age, Sex, and Cigarette Smoking

Carranza⁵ stated women from ages 20 to 39 and men from ages 30 to 59 who smoke cigarettes have twice the chance of having periodontal disease or becoming edentulous as do non-



smokers. Others found no significant difference in loss of alveolar bone height when comparing male and female smokers.⁵ Goultschin et al.¹⁰ found the effects of smoking on periodontal status to be more pronounced in younger women. This was in spite of the fact females and younger subjects were generally periodontally healthier than their male and older counterparts.

Cigarette Smoking and Oral Hygiene

Several studies demonstrated higher levels of oral debris in smokers than in non-smokers. Increased levels of debris observed in smokers have been tentatively attributed to personality traits leading to decreased oral hygiene habits, increased rates of plaque formation, or a combination of the above.¹¹

In this respect it is important to note the plaque accumulation rate and plaque composition in healthy or gingivitis conditions do not seem to differ in smokers as compared to non-smokers.

Effect of Smoking on Plaque

Plaque Development

Early observational studies report smokers showed a higher prevalence of dental plaque than non-smokers, while other studies indicated smoking did not appear to increase the amount of plaque when controlling for other factors.¹²

Oral Flora

A study of the microbiota of the oral mucous membrane and saliva failed to establish a statistically significant trend for smokers to harbor greater proportions of putative periodontal pathogens.¹³ Similarly, an experimental gingivitis study showed no differences between smokers and non-smokers in the alterations to supra-

and subgingival microflora during the change from relative health to experimentally induced gingivitis.¹⁴

Subgingival Microflora in Periodontitis

There are conflicting reports on the effects of smoking on the microflora, which in part is explained by differences in methodology and statistical expression of the data. Some studies report no differences in the prevalence of subgingival bacteria associated with periodontitis.¹ However, data from the large Erie County study showed the proportions of subjects positive for *A. actinomycetemcomitans*, *P. gingivalis*, and *T. forsythesis* were higher among smokers,¹⁵ and there are other reports of a higher prevalence of certain organisms in smokers. Furthermore, increased counts of exogenous flora (*Escheria coli* and *Candida albicans*) have been reported in smokers.¹⁶



Effect of Smoking on Periodontal Tissues

Cigarette Smoking and Gingivitis

Several cross-sectional investigations have indicated smokers may present with lower levels of gingival inflammation to a specific level of plaque than non-smokers.¹¹ Furthermore, development of gingival inflammation in response to experimental plaque accumulation (experimental gingivitis) was less pronounced in smokers than in non-smokers.¹⁷ These cross-sectional and longitudinal data are generally interpreted as an indication cigarette smoking is an environmental exposure capable of modulating gingivitis expression in response to dental plaque.

Cigarette Smoking and Gingival Bleeding

Some early studies suggested smokers expressed less gingival bleeding than non-smokers.^{17,18} They were also able to show a

dose-response effect, which was confirmed in a much larger study of 12,385 general population subjects from the National Health and Nutrition Examination Survey: Part III.¹⁹ This reduced response in smokers has also been elegantly shown in studies using the experimental gingivitis model.¹⁷ These authors cited the potential vasoconstrictive effect of nicotine previously reported by Clarke.¹⁸

Effect of Smoking on Gingival Blood Flow

The data on possible changes of gingival blood flow from the results of human and animal experiments are contradictory.¹⁹ In smokers, gingival blood flow was significantly increased by cigarette smoking.²⁰ However, intravenous administration of nicotine reduces the marginal temperature of gingival sites suggesting a decrease in gingival blood flow which lead to the hypothesis this phenomenon is caused by vasoconstriction induced by nicotine and stress.¹² Later studies²¹ using laser Doppler flow compared the response to smoking a single cigarette in a group of light/occasional smokers and heavier habitual smokers. The changes in gingival blood were not statistically significant, but they did show quite dramatic differences in the response in the skin of the forehead. The light smokers responded with a significant increase in blood flow, but heavy smokers showed no response, indicating a high level of tolerance.

Oxygen Tension in the Gingival Tissues

Oxygen saturation of hemoglobin is affected by cigarette smoking, and attempts have been made to measure this in the gingival tissues. In healthy gingiva smokers appear to have lower oxygen saturation determined by using tissue reflectance spectrophotometry, but in the presence of inflammation converse was shown.¹² The same group of investigators also examined oxygen tension in periodontal pockets and demonstrated oxygen tension was significantly lower in smokers.

Effects on the Gingival Vasculature

The vasculature has also been examined in histological and immunocytochemical studies. In one study researchers found a high proportion of small vessels compared with large vessels in smokers than non-smokers but no difference in the vasculature density.²²

Evidence From Studies on Gingival Crevicular Fluid (GCF)

Smoking may result in lower resting GCF flow rate.²³ The increase in GCF during an experimental gingivitis may be less in smokers.¹⁷ Studies have shown higher levels of TNF- α ²⁴ and decreased levels of IL-1 α and IL-1 β ²⁵ and enzyme elastase in GCF when compared between smokers and non-smokers. For the most part, this research has demonstrated there are lower levels of cytokines, enzymes, and possibly polymorphonuclear leukocytes (PMNs). This correlates with the lower levels of inflammation observed clinically and within the tissues.

Smoking and Fibroblast Function

Gingival Fibroblasts

In vitro studies have shown reduction in the production of Type 1 collagen and fibronectin and an increase in the collagenase activity in the culture media.¹² Cellular changes like disruption of cell orientation, changes in cytoskeleton, presence of large vacuoles, and significant reduction in cell viability have been noticed.¹²

Periodontal Fibroblasts

Periodontal ligament (PDL) fibroblasts growth and attachment to tissue culture plates was inhibited by nicotine at high concentrations (over 1 mg/ml) but no effects were seen at concentrations comparable with plasma levels in smokers.²⁶ Nicotine at high concentrations (100 ng/ml to 25 μ g/ml) was shown by Giannopoulou et al.²⁷ to be cytotoxic by inhibiting the vacuolation and proliferation of fibroblasts. They also confirmed PDL cell proliferation and protein synthesis were inhibited in a dose dependent manner. Cell attachment was significantly less on root surfaces obtained from heavy smokers compared with non-smokers and healthy controls.

Genetic Polymorphism and Smoking

Investigators have looked at genetic variability, its relationship with periodontal disease, and its interaction with smoking. Studies²⁸ looking at tooth loss reported a positive genotype of IL-1 increases the risk for tooth loss by 2.7 times, while smoking increases the risk by 2.9 times. When both were combined, the risk increased to 7.7 times. In another study²⁹ the authors conclude IL-1 genotype positivity is a contributing factor

but a non-essential factor. N-acetyltransferase 2 (NAT-2) polymorphism also affects the population by altering the metabolism of arylamines which may influence the immune response and may act as an immunosuppressant.

Smoking and Host Response

Smoking decreases salivary IgA and serum IgG, and specifically reduces IgG₂ levels against *A. actinomycetemcomitans*.¹ The ability of tobacco products to decrease the proliferating capacity of T and B lymphocytes might contribute to this diminished production of protective antibodies.³⁰ Few investigators have demonstrated suppressed phagocytosis by salivary PMNs and have reported smokers had higher blood counts and reduced chemotaxis of PMNs relative to non-smokers.³¹ In contrast, a few studies have found no significant difference in the chemotaxis ability of PMNs between smokers and non-smokers.³²

Cigarette Smoking and Periodontitis

A multitude of studies investigated the association of smoking status with a variety of periodontal and oral hygiene parameters.^{33,34} Relatively few initial studies have been done which indicate a positive association between smoking and measures of periodontal disease.⁴ The existence of potential confounding factors such as socioeconomic status, education, and, ultimately, the level of oral hygiene was revealed.⁴ Several studies displayed higher levels of oral debris in smokers than non-smokers.⁴ This recognition was the basis for the prevailing opinion in the 1970s and early 1980s that differences in oral hygiene levels could account for the observed differences in the prevalence of periodontal disease in smokers and non-smokers. A variety



of multivariate studies, however, indicated smokers had a significantly higher prevalence of periodontal disease after correcting for potential confounders; oral hygiene in particular.⁴ The current understanding, based on the evidence reviewed in the sections which follow, is although smokers have a tendency to display lower levels of oral hygiene, oral hygiene and/or socioeconomic factors alone are unable to account totally for the observed increase in prevalence and severity of periodontal disease in this group.

Smoking and Periodontitis in Young Adults (≤35 years)

Several studies have shown young adult smokers aged 19-30 years had a higher prevalence and severity of periodontitis compared to non-smokers despite similar or lower plaque levels.^{2,35,36} Haber et al.² reported the prevalence of periodontitis, defined as having a site with attachment loss of ≥ 2 mm and probing depths of ≥ 4 mm, was three to four times higher in young smokers 19-30 years of age compared to non-smokers. The high “periodontal cost” of smoking has been calculated as 27 years of disease progression. In other words, a 32-year-old smoker had similar periodontal attachment loss as a 59-year-old non-smoker.³⁷ Recent statistics indicate more than 23% of high school students are current smokers³⁸ which does not bode well for the future health of this generation.

Smoking and Periodontitis in Adults

In studies where plaque accumulation was similar in smokers and non-smokers, or was adjusted, current smokers had deeper probing depths, greater attachment loss, more bone loss, and fewer teeth.³⁸⁻⁴⁰ Smokers also exhibit more supragingival calculus deposits,⁴¹ while the majority of these studies show a tendency toward decreased clinical signs of inflammation.

The most recent and largest epidemiological study on smoking and periodontal disease is based on data from the National Health and Nutrition Examination Survey (NHANES): Part III⁴² which included a nationally representative sample of 12,329 U.S. adults who were 18 years of age or older. Among this sample, approximately one-half of periodontitis cases were attributable to either current (41.9%) or former (10.9%) smoking.

The investigators also estimated approximately 75% of periodontitis cases were due to smoking among smokers. After adjusting for age, race or ethnicity, income, and educational level, current smokers were four times more likely to have periodontitis as compared to non-smokers.³⁵

Among 20-49 year-olds, the adjusted odds ratio for a mean attachment loss of 1 to 1.99 mm among current smokers was 2.29, whereas the odds ratio for attachment loss ≥ 3 mm was over 18.⁴³ This suggests smoking is particularly important in the etiology of severe periodontal attachment loss. Various other studies^{9,40,44,45} have shown a strong dose-response relationship between the amount smoked and the severity of periodontal destruction which further supports the role of smoking as a risk factor for periodontitis. The most marked difference between smokers and non-smokers in probing depths or attachment loss occurs in the maxillary lingual area and mandibular anterior area, suggesting a local effect of smoking.³⁸



Effects of Smokeless Tobacco on Periodontal Tissues

The relationship between smokeless tobacco use and periodontitis has also been the subject of scientific inquiry; however, there are fewer studies on this subject compared to the number of studies of the association between smoking and periodontitis. The habit of betel chewing with tobacco is a particular form of smokeless tobacco consumption that is predominantly practiced in South Asian countries like India and Sri Lanka. It involves chewing a quid that includes betel leaf, lime, areca nut, and tobacco. The association between tobacco consumed in this form and oral cancer in the Indian subcontinent has been widely

documented over many years. However, relatively few studies have examined the relationship between this practice and periodontitis. In studies done by Amarasena et al.⁴⁶ in a Sri Lankan population has confirmed quantified tobacco use may significantly increase bleeding on probing and periodontal attachment loss.⁴⁷ Other studies have also shown the negative effect of the areca nut on host immunity by affecting PMNs.^{48,49} Areca nut extracts have also been shown to inhibit the growth, attachment, and matrix protein synthesis of cultured human gingival fibroblasts.⁵⁰

Effects Of Cigarette Smoking On Periodontal Therapy

Non-surgical and Surgical Therapy

Numerous studies have shown smoking compromises probing depth and/or attachment gain outcomes following non-surgical or surgical therapy.^{51,52,53} The numerical differences between smokers and non-smokers become more pronounced in probing depths ≥ 5 mm, where smokers demonstrated 0.4 mm⁵¹ to 0.6 mm⁵⁴ less improvement in clinical attachment levels following scaling and root planing. Following flap debridement surgery, smokers experienced up to 1 mm less improvement in clinical attachment levels in probing depths that were initially ≥ 7 mm.⁵⁵

Antimicrobial Therapy in Smokers

Because of the diminished treatment response in smokers, clinicians may recommend adjunctive antimicrobial therapy for smokers. This practice may be justified by evidence suggesting subgingival pathogens are more difficult to eliminate in smokers following scaling and root planing.⁵¹ Soder et al.⁵³ concluded there was little adjunctive effect of systemic metronidazole on non-surgical therapy in smokers. On the other hand, a few studies have reported adjunctive systemic amoxicillin and metronidazole⁵⁵ or locally delivered minocycline microspheres⁵⁵ enhanced the results of mechanical therapy.

A recent study reported a positive response to sub-antimicrobial doxycycline (anticollagenase) therapy in combination with scaling and root planing in a group of severe periodontitis patients that included smokers.⁵⁶ These enhanced results might be due to antimicrobial actions, and

in the case of tetracycline derivatives it could be due to anticollagenase activity. Because gingival fibroblasts show increased collagenase activity when exposed to nicotine *in vitro*, this is an area to be investigated.

Soft and Hard Tissue Grafting

Studies in which recession sites were treated using connective tissue with a partial thickness pedicle graft⁵⁷ and a coronally positioned flap alone, or with a bioabsorbable membrane⁵⁸ found no difference in root coverage between smokers and non-smokers. On the other hand, when guided tissue regeneration procedures were used smokers had significantly less root coverage (57%) compared to non-smokers (78%).⁵⁹

Smoking is detrimental to regenerative therapy in interproximal and function defects, whether treatment includes the use of osseous graft alone, bioabsorbable membrane alone, or bioabsorbable membranes in combination with osseous grafts. The results from various studies have shown less than 50% as much improvement in clinical attachment levels in smokers compared to non-smokers, which amounted to differences ranging from 0.35 mm to 2.9 mm.⁶⁰

Implant Therapy

In the studies reviewed, 0% to 17% of implants placed in smokers were reported as failures as compared to 2% to 7% in non-smokers.¹ The 3-year data demonstrated 8.9% of implants placed in smokers failed as compared to 6% in individuals who had never smoked or had quit smoking.¹ The majority of implant failures in smoking occurred prior to prosthesis delivery.

Effects of Smokeless Tobacco on Periodontal Therapy

The studies which considered the effect of smokeless tobacco on periodontal therapy are

very scanty to null making it very difficult to reach a definitive conclusion on this issue.

Impact of Smoking Cessation on Periodontal Status and Treatment Outcomes

While smoking cessation does not reverse the past effects of smoking, there is abundant evidence to indicate the rate of bone and attachment loss slows after patients quit smoking and the severity of their disease is intermediate compared to current and non-smokers.^{38,42,61-65}

It is encouraging to note former smokers respond to non-surgical and surgical therapy in a manner similar to those who have never smoked.⁵¹ Similarly, implant success rates for past smokers were similar to those who have never smoked.¹

Conclusion

During the past 50 years the available knowledge of the noxious effects of cigarette smoking on the periodontium has significantly progressed. Today there is little doubt cigarette smokers are a high risk group for periodontitis and they respond only partially to periodontal therapy. Tobacco smoking has wide spread systemic effects, many of which may provide mechanisms for the increased susceptibility to periodontitis and the poorer response to treatment. As an environmental factor, smoking interacts with the host and the bacterial challenge associated with periodontal disease. The host genetic and environmental interaction is of the utmost importance. As knowledge of genetic susceptibility to periodontitis increases, there will be further opportunities to explore its relationship with tobacco smoking.

Clinical Significance

The role smoking plays in periodontal disease should be considered by clinicians and patients during active periodontal therapy and the oral health maintenance phases of care.

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