

Position Paper

Tobacco Use and the Periodontal Patient*

THIS POSITION PAPER WAS PREPARED BY THE Research, Science and Therapy Committee of The American Academy of Periodontology and is intended for the information of the dental profession. The purpose of the paper is to provide the reader with a general overview of the relationship of tobacco use and periodontal diseases. This paper will review the epidemiological and clinical findings that have led to our understanding of the role of tobacco use in relation to periodontal diseases and their treatment. In addition, this paper will review the possible underlying mechanisms for these effects from tobacco use. The practitioner can use this information in treatment decisions and in giving advice to the patients who use tobacco products. *J Periodontol* 1996;67:51-56.

INTRODUCTION

A large body of epidemiological research indicates that tobacco use is directly related to the incidence and prevalence of a variety of medical problems including cancer; low birth weight; and pulmonary, cardiovascular, and gastrointestinal disease.¹ In the past 20 years, there also has been an increasing awareness of the role of tobacco use on the prevalence and severity of periodontal diseases² and subsequent tooth loss.³⁻⁸ Recent studies have demonstrated that smoking may be one of the most significant risk factors in the development and progression of periodontal disease.⁹⁻¹³ In the United States, where approximately 26% of the adult population smoke cigarettes,¹⁴ and in other countries where the percentage of smokers may be higher,^{1,15,16} this association between cigarette smoking and periodontal diseases represents a significant public health problem. Although the percentage of adults who smoke appear to be gradually declining in the United States and in other countries,¹⁶ the rate of decline is less among women and certain minorities.^{1,15} Furthermore the use of smokeless tobacco products, particularly among young people, appears to be increasing.¹⁷ The use of smokeless tobacco products may also affect periodontal health.¹⁸

In the discussion regarding the potential impact of tobacco use on periodontal diseases, this informational paper will focus on five areas: 1) the effect of tobacco smoking on the prevalence and severity of periodontal dis-

eases; 2) the effects of smoking on response to periodontal therapy; 3) mechanisms of periodontal disease progression in smokers; 4) the role of smokeless tobacco products in periodontal diseases; and 5) smoking cessation and disease prevention.

THE EFFECT OF TOBACCO SMOKING ON THE PREVALENCE AND SEVERITY OF PERIODONTAL DISEASES

An association between smoking and the prevalence of acute ulcerative gingivitis (ANUG) was demonstrated as early as 1946.^{19,20} More recently, a similar relationship has been shown between tobacco smoking and the "ANUG-like" lesions in HIV-infected individuals.²¹ However, in earlier cross-sectional studies assessing chronic gingivitis and periodontitis, the relationship between smoking and the prevalence and severity of periodontal diseases was often contradictory and inconclusive.²²⁻³³ Some investigators reported less gingival inflammation or no difference in smokers when compared to non-smokers,^{22,23,34} while other studies demonstrated greater gingival inflammation in smokers.²⁴⁻²⁶ Some observers reported either an increase^{22,25-28} or no difference^{29,30,34} in the amounts of plaque in smokers versus non-smokers. Gingival crevicular fluid flow was reported to increase immediately following smoking³⁵ but diminished in chronic smokers versus non-smokers.³⁶ A direct relation between smoking and probing depths and bone loss^{22,31-33,37,38} was reported by some researchers, whereas others reported no clear relationship.^{25,28} It has been suggested that differences in attachment loss between smokers and non-smokers was probably due to poorer oral hygiene with greater accumulations of plaque in smokers.^{26,28,31,38}

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In the past 10 years, cross-sectional studies on larger groups of patients have demonstrated a clearer relationship between smoking and periodontal diseases. In some of these studies, the plaque accumulation levels were adjusted between smokers and non-smokers.³⁹⁻⁴² In studies in which plaque accumulation was kept to a minimum in both smoker and non-smoker groups, the smoker group had more sites with deeper pockets.^{10,39,40,42} In addition, alveolar bone loss was greater in smokers, especially in older patients.⁴¹ Similar findings have been reported from other large controlled cross-sectional population studies⁴³⁻⁴⁵ and in several longitudinal studies.^{9,10,46-47} From these more recent studies, a general pattern has emerged: smokers have greater bone loss, increased numbers of deep pockets, and increased calculus formation, but have the same or less gingival inflammation, and the same levels of plaque accumulation.

This pattern of smoking effects is also evident from recent studies in which general disease categories were used and the risk of developing disease was calculated using odds ratios.^{9,10,38,48,49} In one study, current and former smokers were significantly more prevalent in an American periodontal practice population of patients with moderate and advanced periodontitis when compared to referring general dental practices and the general population.⁴⁸ A relationship has also been demonstrated between the prevalence of moderate to severe periodontal disease and the number of cigarettes smoked per day^{2,9,10,38,49,50} and to the number of years that the patient has smoked.^{9,10,48,49}

THE EFFECT OF SMOKING ON RESPONSE TO PERIODONTAL THERAPY

Since there seems to be a strong relationship between smoking and periodontal disease, one would expect that smokers would require more periodontal treatment. This has been demonstrated by one group of investigators who reported that smokers had higher Community Periodontal Index of Treatment Needs (CPITN) scores⁵¹ than nonsmokers. Cigarette smoking appears to alter the response to various forms of periodontal therapy. For example, it has been reported that in patients who had been surgically treated for periodontitis and then longitudinally followed, smokers exhibited significantly deeper post-therapy probing depths^{52,53} and less gain in clinical attachment levels⁵³ than non-smokers. With scaling and curettage, the clinical reduction in probing depth has been reported to be similar between smokers and non-smokers in posterior areas but significantly less in anterior areas in smokers.⁵⁴ Smokers have also been reported to have a poorer success rate with soft tissue graft procedures⁵⁵ and certain bone graft and implant procedures.⁵⁶

Tobacco smoking may also play a significant role in the development of refractory periodontitis.^{57,58} An unusually high percentage of refractory patients are smokers

(>90%) when compared to the percentage of smokers in the general population (~30%).⁵⁸

MECHANISMS OF PERIODONTAL DISEASE PROGRESSION IN SMOKERS

Microbiology

One hypothesis regarding the role of tobacco smoking in the development of periodontal diseases is that when compared to non-smokers, smokers have more plaque or harbor different or more virulent types of plaque bacteria. However, studies have shown that there is generally little difference in the level of plaque accumulation in smokers versus non-smokers.^{40,41,43} In addition, in cross-sectional studies where plaque levels are controlled to minimum levels in smokers and non-smokers, the level of alveolar bone support is less in smokers.⁴¹

On the other hand, several studies have shown that smokers do seem to have greater accumulations of calculus.^{19,20,42,45} Calculus might either act as a local tissue irritant or it might create a local environment that promotes the colonization and growth of certain kinds of pathogenic bacteria. It has been shown that the periodontal pockets of smokers are more anaerobic.⁵⁹ This anaerobic environment could conceivably promote the growth of periodontopathic anaerobic Gram-negative species in subgingival plaque. However, early studies using Gramstaining techniques and/or microscopic examination failed to show a significant difference in the subgingival microbial flora between smokers and non-smokers.⁵⁹ In more recent studies, no significant differences in the percentage recovery of *A. actinomycetemcomitans*, *P. gingivalis*, *P. intermedia*, or *B. forsythus* from deep pockets were found between smokers and non-smokers.^{60,61} Therefore, to date there have been no conclusive studies to show that smoking adversely affects periodontal tissues by altering the microbial composition of plaque.

Effects on the Host Response and Periodontal Tissues

Since smoking does not appear to have a major effect on the periodontal bacterial flora, some investigators have examined the role of smoking in altering the periodontal host response. In general, smoking could lead to increased periodontal destruction by altering the host response through two mechanisms: 1) tobacco smoking could impair the normal function of the host response in neutralizing infection⁶² and 2) tobacco smoking could alter the host response resulting in destruction of the surrounding healthy periodontal tissues.⁶³ Several studies have shown that cigarette smoking may exert both of these types of effects on the host response. For example smokers have decreased levels of salivary antibodies (IgA)⁶⁴ and serum IgG antibodies to *P. intermedia* and *F. nucleatum*.⁶⁵ In addition, smokers appear to have depressed numbers of

helper lymphocytes which are important components of the immune system.^{66,67}

In order for the host to efficiently deal with bacterial infections, fully functional neutrophils are required. It has been well documented that tobacco smoke and its individual components can have deleterious effects on various neutrophil functions.⁶⁸⁻⁷⁶ For example, it has been shown that tobacco smoke can impair the chemotaxis and phagocytosis of both oral^{68,69} and peripheral^{70,71} neutrophils. Impairment of phagocytosis has also been reported in neutrophils from smokers with refractory periodontitis.⁵⁸ Smoking has been shown to stimulate⁷² or impair⁷³ the oxidative burst of neutrophils. Similar effects have been observed with higher concentrations of individual tobacco components such as nicotine, acrolein, and cyanide.⁶⁹

Perhaps the most extensively studied tobacco substance is nicotine. Nicotine at low concentrations can stimulate neutrophil chemotaxis,⁷⁵ but at higher concentrations will impair phagocytosis.⁷⁶ Nicotine is only one of over 2,000 potentially toxic substances in tobacco smoke that may have harmful effects on the periodontal tissues.⁷⁷ Many of these other substances may also have harmful effects on the periodontal tissues by either altering the host response or by directly damaging the normal cells of the periodontium. It has been shown that low dosages of nicotine can be stored in and released from periodontal fibroblasts.⁷⁸ However, it is unclear whether these fibroblasts exposed to nicotine have an impaired⁷⁹ or an enhanced ability⁸⁰ to attach to various surfaces. Nicotine can also suppress the proliferation of cultured osteoblasts while stimulating osteoblast alkaline phosphatase activity.⁸¹ These *in vitro* alterations of cells of the periodontium by nicotine may also occur *in vivo* and thus affect the reparative and regenerative potential of the periodontium in tobacco users.

Another area of investigation into the effects of tobacco smoke and tobacco components on periodontal tissues relates to their potential ability to reduce gingival blood flow. Earlier studies on the effects of nicotine using a heat diffusion technique demonstrated a decrease in gingival blood flow.⁸² However, a more current study on the effects of cigarette smoke using laser Doppler probes has yielded contradictory results.⁸³ Inconclusive results have also been obtained in studies of possible thermal damage to periodontal tissues from cigarette smoke.⁸⁴ A relationship between smoking to bone mineral content,^{85,86} particularly osteoporosis, has been demonstrated by several studies. However a direct relationship between smoking, osteoporosis, and periodontal disease remains unclear.

Thus tobacco products may induce or exacerbate various forms of periodontal disease by direct local damage to periodontal tissues and/or by altering the host response. The changes in the host response may impair neutralization of infection and/or give rise to enhanced destruction of healthy periodontal tissues. In addition,

tobacco products may also alter the normal repair mechanisms in the periodontium.

THE ROLE OF SMOKELESS TOBACCO PRODUCTS IN PERIODONTAL DISEASES

Smokeless tobacco products such as snuff and chewing tobacco are popular among young adults and in older women living in rural Southern areas in the United States.⁸⁷⁻⁸⁹ The relationship of smokeless tobacco and oral carcinoma has been well documented.⁸⁸ In addition, a relationship of smokeless tobacco use to cardiovascular mortality has been reported.⁹⁰ However, the relationship of smokeless tobacco to various forms of periodontal disease is less clear. Large-scale studies on athletes who use smokeless tobacco demonstrated a strong relationship to oral leukoplakia.^{87,89} These lesions are commonly found in areas of the mouth where smokeless tobacco products are placed.

Although individual cases of ANUG, gingivitis, gingival recession, and periodontitis have been reported in smokeless tobacco users⁹¹⁻⁹³ a clear relationship between smokeless tobacco use and generalized periodontal conditions has not been demonstrated. In one study of large numbers of young smokeless tobacco users, there was a significant increase in the prevalence of localized gingival recession and attachment loss.¹⁸ This attachment loss was especially noted in the mandibular buccal areas where smokeless tobacco products were placed. Some investigators have proposed that local exposure to high concentrations of tobacco products in smokeless tobacco may play a role in both the local periodontal host response and the clinical attachment loss.^{18,87,94} In addition, the high concentrations of nicotine in smokeless tobacco cause alterations in gingival blood flow.⁹⁵ Recent *in vitro* studies have demonstrated that smokeless tobacco extracts affect the secretion of monocyte inflammatory mediators.⁹⁴

PREVENTION

This paper has reviewed studies that have shown that the severity of periodontal disease is related to the quantity and duration of smoking. At present there are no published studies measuring the effectiveness of smoking cessation programs in reducing the incidence and severity of periodontal diseases. However, both the prevalence and severity of periodontal disease is greater in current smokers when compared to patients who have quit smoking or never smoked.^{2,9,10,48,96} The observations of these studies imply that smoking cessation may slow or halt the progression of periodontal disease. Periodontists in the United States and other countries are more likely to give this type of advice regarding smoking cessation to patients when compared to general dentists.⁹⁷ Smokers who are medically healthy may be more likely to visit their dentist as opposed to their physician. When considering the detrimental effects of tobacco use in a variety of medical

conditions,¹ smoking cessation advice given by the dentist may be beneficial to the smoking patient from a number of different health aspects. Another concern is that in the United States, 80 to 90% of regular smokers start smoking by the age of 18.¹⁵ Dentists can give advice to this young population considering the use of tobacco products and the relationship of tobacco and periodontal diseases. Thus, tobacco cessation advice may have broad long-term benefits for patients who use tobacco as well as the nonsmoking population.

SUMMARY

Clinical and epidemiological studies support the concept that tobacco use is an important variable affecting the prevalence and progression of periodontal diseases such as adult periodontitis, refractory periodontitis, and ANUG. In studies in which plaque levels were adjusted between smokers and non-smokers, greater probing depths, clinical attachment loss, and bone loss have been reported in smokers. In addition, smokers do not appear to respond as well as non-smokers to various forms of periodontal therapy. Smokeless tobacco products appear to induce local clinical attachment loss at the site of intra-oral placement.

Published studies on the periodontal microflora of smokers have not demonstrated significant differences when compared to non-smokers. On the other hand, tobacco products appear to have direct local effects on periodontal tissue and can alter the host response. Several studies have demonstrated that the severity of periodontal disease appears to be related to the duration of the tobacco use, smoking status, and amount of daily tobacco intake. Although there are no published studies that specifically measure the effects of smoking cessation programs and periodontal disease, the results from recent epidemiological studies imply that smoking cessation may improve the periodontal status and the outcome of periodontal treatment.

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