Position Paper

Tobacco Use and the Periodontal Patient*

This 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 1999;70:1419-1427.*

Tobacco use is directly related to a variety of medical problems including cancer, low birth weight, and pulmonary and cardiovascular diseases.[1] In the past 25 years, there also has been an increasing awareness of the role of tobacco use in the prevalence and severity of periodontal diseases[2] and subsequent tooth loss.[3-9] Smoking appears to be one of the most significant risk factors in the development and progression of periodontal disease.[10-16] In the United States, where approximately 25% of the adult population smoke cigarettes,[17] and in other countries where the percentage of smokers may be higher,[18,19] this association between cigarette smoking and periodontal diseases represents a significant public health problem. Although the percentage of adults who smoke has declined in the United States since the 1970s,[20] the rate of decline is less among women and certain minorities,[18-20] and smoking has become more popular among youth. Smoking increased from 27.5% of United States high school students in 1991 to 36.4% in 1997. The use of smokeless tobacco products and cigars, particularly among young males, is another health concern.[21] The use of these tobacco products may also affect periodontal health.[22-25]

The potential impact of tobacco use on periodontal diseases will be discussed in 5 sections: 1) the effect of tobacco smoking on the prevalence and severity of periodontal diseases; 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, cigar and pipe smoking in periodontal diseases; and 5) periodontal effects of tobacco cessation.

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

An association between smoking and acute ulcerative gingivitis (AUG) was demonstrated as early as 1946.[26,27] Epidemiological studies published in the 1980s and 1990s demonstrate an association between smoking and destructive periodontal disease. Results from the first United States National Health and Nutrition Examination Survey (NHANES I) demonstrated that although current smokers had more plaque and periodontal destruction than former or never smokers, the association between periodontal disease and smoking remained after adjusting for oral hygiene and other variables.[28]

In studies in which plaque accumulation was kept to a minimum or adjusted for in both smoking and non-smoking groups, the smoking groups had more sites with deeper pockets[29-31] and greater attachment loss.[10,12,29,32] Differences between current and never smokers in mean attachment level were highly significant and ranged from 0.37 mm among 35-year-olds to 1.33 mm in 75-year-old subjects.[32] A higher prevalence of furcation involvement among current as compared to never smokers has also been noted.[32,33] Compared to never smokers, almost twice as many current smokers had radiographic evidence of furcation involvement.[33] In addition, alveolar bone loss was greater in smokers.[11,34,35] In studies which have expressed alveolar bone height as a percentage of root length, the average bone height in current smokers was 77 to 78%, as compared to 83% in never smokers.[34,35] Similar findings have been reported from other large controlled cross-sectional population studies,[23,36,37] and in several longitudinal studies.[16,24,25,38-40] From these contemporary studies, a general pattern has emerged: smokers have greater attachment and bone loss, increased numbers

of deep pockets and calculus formation, but variable levels of plaque and inflammation, although the bias is towards decreased signs of clinical inflammation.

Depending upon the criteria used to define periodontal disease, smokers are 2.6 to 6 times more likely to exhibit periodontal destruction than non-smokers.[10,12,39,41,42] Regression analyses have shown that the relative risk of attachment loss among 25 to 74 year subjects with a history of moderate smoking (15 to 30 pack years) was 2.77; whereas, for current or former heavy smokers (≥30 pack years), this figure was 4.75.[10] In the same population, the prevalence of severe bone loss, defined as bone level ≥4 mm apical to the cementoenamel junction, was 4.7 times greater among current or former heavy smokers as compared to never smokers.[11]

Several studies have shown a relationship between the amount smoked and the prevalence and severity of periodontitis. A relationship has been demonstrated between the prevalence of moderate to severe periodontal disease and the number of cigarettes smoked per day[2,9-11,37] and to the number of years that the patient has smoked.[9-11,41,43] Attachment loss severity was increased by 0.5% by smoking 1 cigarette per day, while smoking up to 10 and 20 cigarettes a day increased attachment loss by 5% and 10%, respectively.[37] In the Erie County Study population, there was a strong positive relationship between bone loss and mean pack years of smoking; subjects with severe bone loss had a pack year history that was approximately 5 times that of subjects with normal bone levels.[11] Another investigation reported a positive correlation between serum levels of the nicotine metabolite, cotinine, and severity of clinical attachment loss, probing depth and alveolar crestal height in subjects aged 25 to 74 years.[44]

Studies which have focused on subjects less than 40 years of age have shown that smoking has a strong negative impact on periodontal status in young adults.[2,29,45] In one study,[2] current smokers in the 19 to 30 year age range were 3.8 times more likely to have periodontitis as compared to never smokers. Smokers with generalized early-onset periodontitis (G-EOP) had more affected teeth and more attachment loss than G-EOP patients who did not smoke. By contrast, attachment loss was not affected by smoking in younger patients with localized juvenile periodontitis (LJP).[45] It was suggested that the differential effects of smoking on G-EOP and LJP may be explained by the fact that the disease process responsible for attachment loss in LJP may not be of sufficient length to be influenced by smoking. These studies demonstrate that smoking is strongly associated with periodontal destruction in young adults in their 20s and 30s.

Tobacco smoking probably plays a significant role in the development of refractory periodontitis.[46-48] An unusually high percentage of refractory patients are smokers (>90%)[47,48] when compared to the percentage of smokers in the general population (~25%).[47]

EFFECT OF SMOKING ON THE RESPONSE TO PERIODONTAL THERAPY

Smoking has been identified as one of the major predictive variables for response to periodontal therapy.[49,50] In maintenance patients followed at least 5 years, patients who smoked were twice as likely to lose teeth.[49] The majority of studies involving non-surgical therapy have shown less probing depth reduction[51-57] and less attachment gain in smokers as compared to non-smokers.[52,56] Among patients who have been surgically treated for periodontitis and then longitudinally followed, smokers exhibited less reduction in probing depths,[52,53,58] less gain in clinical attachment levels,[52,53] and less gain in bone height [59] than non-smokers.

Root coverage following thick free gingival graft procedures is reportedly diminished by heavy cigarette smoking,[60] and there are conflicting reports on smoking's effect on the success of subepithelial connective tissue grafts.[61,62] Clinical attachment gains are also less in smokers as compared to non-smokers following regenerative procedures.[63-67] While the negative effects of smoking on therapy are evident from these studies, it must be remembered that periodontal therapy produced improvement of periodontal parameters in both smokers and nonsmokers.

Some studies have shown that implant success rates are reduced in smokers.[68-71] In one of these,[69] overall implant success rates up to 6 years among current smokers were approximately 89%, as compared to 95% in non-smokers. Other retrospective data did not identify smoking as a variable associated with implant failure.[72,73] A longitudinal study[74] has shown that marginal bone loss, albeit limited, was greater over a 15-year period around implants supporting mandibular fixed prosthesis in current smokers as compared to former or never smokers.

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, some studies have shown that there is little difference in the level of plaque accumulation in smokers versus non-smokers.[23,30,35] In addition, in cross-sectional studies where plaque levels are controlled to minimum levels in current smokers and never smokers, the level of alveolar bone support is less in current smokers.[35]

Early studies using Gram-staining techniques and/or microscopic examination failed to show a significant difference in the subgingival microbial flora between smokers and non-smokers.[75] Other research reported no significant differences in the percentage recovery of periodontal pathogens from deep pockets between smokers and non-smokers.[42,76] However, cross-sectional data[77] from the large Erie County Study population demonstrated that the proportion of subjects positive for *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis,* and *Bacteroides forsythus* was significantly higher among current smokers as compared to never smokers. Current smokers were 3.1 times more likely to be infected with *A. actinomycetemcomitans* and 2.3 times more likely to be positive for *B. forsythus* than former or never smokers. Furthermore, the relative risk of *B. forsythus* infection increased as the amount of smoking increased; 43% of subjects smoking fewer than 10 cigarettes a day were *B. forsythus* positive as compared to 64% smoking more than 20 cigarettes a day.

A possible explanation for the diminished response to therapy in smokers could be due to differences in pathogen reductions. Some research[78] shows that there is no significant difference in the elimination of periodontal microflora following nonsurgical therapy in current smokers and never smokers, whereas other data[54-57] indicate that certain bacteria were more difficult to eradicate among current smokers. These included *A. actinomycemtemcomitans,*[54,57] *P. gingivalis,*[54,55] and *B. forsythus.*[55]

Effects on the Host Response and Periodontal Tissues

Although bacteria are the primary etiologic factor in periodontal disease, the patient's host response is a determinant of disease susceptibility. In general, smoking could lead to increased periodontal destruction by altering the host response through 2 mechanisms: 1) impairment of the normal host response in neutralizing infection[79] and 2) alterations that result in destruction of the surrounding healthy periodontal tissues.[80]

Smokers appear to have depressed numbers of helper lymphocytes which are important to B cell function and antibody production.[81-83] This was manifested by decreased levels of salivary antibodies (IgA)[84] and serum IgG.[81] Other findings show that smoking is associated with diminished serum IgG2 levels both in white adults[85] and in black subjects with G-EOP.[86] Impaired IgG2 response has been hypothesized to increase the risk of periodontitis, however a cause and effect relationship between smoking and impaired IgG2 response has yet to be established.[85] Serum IgG antibodies to *Prevotella intermedia* and *Fusobacterium nucleatum* also have been reported to be reduced in smokers.[87]

In order for the host to deal efficiently 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.[88-98] For example, it has been shown that tobacco components can impair the chemotaxis and/or phagocytosis of both oral[88,89] and peripheral[90,91] neutrophils. Impairment of phagocytosis has also been reported in neutrophils from smokers with refractory periodontitis.[47] Smoking has been shown to stimulate or impair the oxidative burst of neutrophils.[92-94] In vitro nicotine exposure inhibits neutrophil production of superoxide anion and hydrogen peroxide, which are important to antimicrobial function.[98]

For many years, it has been hypothesized that smoking has a deleterious effect on gingival blood flow. The initial work[99] on the effects of nicotine demonstrated a decrease in gingival blood flow based on heat diffusion studies; however, later research using different methodology for smoke[100] or nicotine exposed tissues[101] yielded contradictory results. The medical literature has demonstrated that exposure to tobacco or nicotine impairs revascularization in soft[102] and hard tissues.[103] Similar phenomena may affect periodontal wound healing.

Nicotine can be stored in and released from periodontal fibroblasts.[104] However, it is unclear whether these fibroblasts exposed to nicotine have an impaired[105] or an enhanced ability[106] to attach to various surfaces. In addition, nicotine may inhibit fibroblast fibronectin and collagen production and increase fibroblast collagenase activity.[107] Nicotine can also suppress the proliferation of cultured osteoblasts while stimulating osteoblast alkaline phosphatase activity.[108]

Tobacco components may also modify the production of cytokines or inflammatory mediators

which play a role in periodontal tissue destruction. Nicotine has been shown to increase release of interleukin-6 by cultured murine osteoblasts.[109] Smokers have been reported to have increased crevicular fluid levels of tumor necrosis factor α.[59] Nicotine has divergent effects on interleukin-1 and prostaglandin E_2 secretion, depending upon the cell type and whether or not bacterial components are present.[98,110-112] Such alterations in host response may affect the reparative and regenerative potential of the periodontium in tobacco users.

ROLE OF SMOKELESS TOBACCO PRODUCTS AND CIGAR AND PIPE SMOKING 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.[113-115] The overall prevalence of current smokeless tobacco use among United States high school students is approximately 9%; however, 20.6% of white high school male students report current smokeless tobacco use.[21] The relationship of smokeless tobacco and oral carcinoma has been well documented.[114] Large-scale studies of athletes [22,113] and adolescents[115-117] who use smokeless tobacco have demonstrated a strong relationship to white oral mucosal lesions. These lesions are commonly found in areas of the mouth where smokeless tobacco products are placed and occur in 50 to 60% of smokeless tobacco users.[22,118,119]

A clear relationship between smokeless tobacco use and generalized periodontal conditions has not been definitively demonstrated.[34] In general, localized attachment loss in the form of gingival recession occurs in 25 to 30% of smokeless tobacco users.[22,118,119] This attachment loss is most prevalent adjacent to the mandibular buccal areas where smokeless tobacco products are commonly placed.[22] In vitro studies have demonstrated that smokeless tobacco extracts affect monocyte and oral keratinocyte production of inflammatory mediators which may play a role in the development of these localized tissue alterations.[120-122]

Between 1993 and 1997, overall cigar consumption in the United States increased almost 50%.[123] This may be due to emergence of cigar smoking among young affluent adults who tend to smoke premium cigars and the increased number of adults over the age of 50, which is a population believed to smoke more cigars than any other segment of our society.[124] The 1997 Youth Risk Behavior Survey[21] reported that the overall prevalence of current cigar use, defined as at least 1 cigar smoked over the past 30 days, was 22%. Regular cigar smoking is associated with cancer of the oral cavity, lung, larynx, and esophagus.[125]

Relative to our knowledge of the effects of cigarettes and smokeless tobacco on the periodontium, there is less information regarding the periodontal effects of cigar and pipe smoking.[23-25,28] In general, a limitation of these studies is the relatively small sample size of cigar and pipe smokers available. In the NHANES I survey conducted between 1971 and 1974, periodontal index scores did not vary among pipe, cigar and cigarette smokers.[28]

The majority of data[23-25] on the periodontal effects of cigar and pipe smoking comes from the Veterans Administration Dental Longitudinal Study which began in 1968. At the baseline dental examination, 862 male subjects were classified as cigarette smokers, pipe/cigar smokers or non-smokers.[23] The latter category included former and never smokers. At the 6-year follow-up,[24] it was concluded that the periodontal status of the cigar/pipe group was intermediate between that of the cigarette and nonsmoking groups.

The most recent data from this cohort included 690 men who returned for examination at least once over a 23-year period.[25] Compared to non-smokers, smokers of cigars had a 1.3 fold risk of tooth loss, and pipe and cigarette smokers each had a 1.6 fold risk. Cigar and cigarette smokers also were at increased risk of experiencing alveolar bone loss compared to non-smokers. Unlike previous reports on this population, this study separated pipe and cigar smokers and included only men who smoked cigars, pipes, or cigarettes exclusively during the follow-up period. This factor, as well as the longer duration of follow-up, may explain this report's finding of a greater negative periodontal impact of cigar and pipe smoking as compared to the earlier studies.

PERIODONTAL EFFECTS OF TOBACCO CESSATION

In general, the periodontal status of former smokers is intermediate between that of never smokers and current smokers.[2,41,126] These findings suggest that while the past effects of smoking on the periodontium cannot be reversed, smoking cessation is beneficial to periodontal health.

A previous history of smoking does not appear to be deleterious to the response of periodontal therapy.

Clinical studies comparing the response of current, former, and never smokers have reported that both never smokers and former smokers respond more favorably to therapy than current smokers and, furthermore, there was no significant difference between former and never smokers with respect to the efficacy of periodontal therapy.[53,55] Similarly, there was no association between number of years since smoking cessation and therapy response. This finding suggests that there is an early benefit of smoking cessation.[55] In a study of the effects of smoking cessation on implant failure, patients who quit smoking for 1 week before and 8 weeks after implant placement had significantly lower failure rates than patients who continued to smoke. Failure rates in these patients were not significantly different from non-smokers.[127] Collectively, these data are encouraging evidence for clinicians and their patients and emphasize the positive impact on smoking cessation on periodontal therapy response. For these reasons, the American Academy of Periodontology strongly recommends inclusion of tobacco cessation in periodontal therapy.[128-130]

The National Cancer Institute has developed tobacco-use intervention strategies for the dental setting.[131,132] Even minimal contact interventions have been shown to improve cessation rates over no-treatment conditions, although there is little doubt that longer person to person interactions have an even greater impact. The level of nicotine addiction, health status, training of the clinician, patient preference, and expense are all variables which affect selection of a particular intervention strategy.[133] When considering the detrimental effects of tobacco use on 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. Tobacco cessation provides benefits by enhancing a patient's overall health as well as reducing their risk of periodontal disease and other oral pathologic alterations.

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, generalized early-onset 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. Several studies have demonstrated that the severity of periodontal disease appears to be related to the duration of tobacco use, smoking status, and amount of daily tobacco intake. The majority of studies on the periodontal microflora of cigarette smokers show no difference between smokers and non-smokers, although recent studies involving large sample sizes suggest that certain periopathogens are more prevalent among smokers. Tobacco products appear to have direct local effects on periodontal tissue and can alter the host response. Smokeless tobacco use induces local clinical attachment loss at the site of intraoral placement, but is not associated with generalized periodontal destruction. Additional data are necessary before definitive conclusions can be drawn regarding the relationship between cigar smoking and periodontal disease. Although smokers benefit from periodontal therapy, clinical improvements are less than those for non-smoking patients. Studies comparing therapy response in former smokers, current smokers, and never smokers demonstrate that former smokers respond in a similar manner to never smokers to periodontal therapy. Based on this evidence, dental health professionals should advise patients of tobacco's negative health effects as well as the benefits of quitting tobacco use, and tobacco cessation counseling should be part of the armamentarium of the dental office.

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