

SMOKING AND PERIODONTAL DISEASE A REVIEW

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Summary. *The role of smoking as a contributory factor in the progression of the periodontal disease process has long been suspected. Nowadays, a large number of studies have been published in the dental literature regarding this possible role. Much of the literature has also indicated that smokers affected with periodontitis respond less favorably to periodontal treatment be it non-surgical, surgical or regenerative. This paper will review the current literature regarding the effects of smoking on various aspects of the periodontal disease process and present an explanation for the possible association between smoking and the progression of periodontitis.*

Key words: *Smoking, periodontal disease*

Introduction

Periodontal diseases are a group of conditions affecting the supporting structures for the dentition. The periodontal tissue consist of a specialized form of oral mucosa known as gingiva, which has a keratinized epithelium and covers the alveolar bone. There is an epithelial attachment between the enamel of the tooth and the marginal gingivae which is formed from the fusion of reduced enamel epithelium and the oral epithelium and is known as junctional epithelium when tooth eruption is completed (1)

Inflammation of the marginal gingival tissues is a common condition and its extent and severity can be variable. This condition known as gingivitis can be modified by systemic and local influences and is plaque induced. It can be reversed if improved oral hygiene measures are introduced (2).

Chronic periodontitis is the result of a response of the host to bacterial aggregations on the tooth surfaces. The outcome of this is an irreversible destruction of the connective tissue attachment, which results in periodontal pocket formation and eventual loss of alveolar bone (3).

Tobacco smoking is an addictive habit first introduced into Europe. Smoking is now recognized as the most important cause of preventable death and disease. Currently, the most of adult population smoke cigarettes. The number of cigarette smokers is slowly declining, but those who do smoke are smoking more. However, nowadays, the smokers are changing to lower-tar brands. Consumption is rising in developing countries, particularly where tobacco production bring great economic benefits, and it will probably continue to rise for the foreseeable future.

Toxity of tobacco smoke

Hundreds of different compounds have been identified in tobacco smoke and some occur in concentrations judged to be harmful to health (4). Some of these substances are indisputably carcinogenic, and smoking has been implicated in the aetiology of oral neoplasia (5). Tobacco smoke also contains such noxious substances as benzanthracene and hydrogen cyanide, which undoubtedly have anti-bacterial properties.

Among the substances found in tobacco smoke is the alkaloid nicotine, which appears to be responsible for the dependence that characterizes the smoking habit (6). During smoking, nicotine is rapidly absorbed into the bloodstream, where 30% remains in its free form. It is highly lipid-soluble and readily penetrates cell membranes. Nicotine has actions on almost all the organs of the body (7).

Nicotine is considered the most pharmacologically active compound in tobacco smoke. Most is absorbed through the lung alveoli, but nicotine can also be absorbed, though more slowly, through the oral mucosa in sufficient quantities to have a pharmacological effect (8). Nicotine has pronounced effects on the cardiovascular system. During smoking it increases the heart rate, cardiac output, and blood pressure by autonomic stimulation, which also effects peripheral vasoconstriction (9). There is also evidence that nicotine acts directly on blood vessels and capillaries to produce vasoconstriction (7).

For the last few decades, dentists and dental researchers have become more aware of the critical role of smoking on the incidence and severity of periodontal disease (10) and smoking is now considered a risk factor in periodontal disease. There is a long history of association between

tobacco smoking and periodontal disease. In 1983, Ismail et al. (11) analyzed smoking and periodontal disease and found that after adjusting for potential confounding variables such as age, oral hygiene, gender and socioeconomic status, smoking remained a major risk indicator for periodontal diseases. Locker and Leake (12) found that among Canadians, smoking was one of the most consistent predictors of periodontal disease experience.

Plaque formation

The early studies that examined the relation between smoking and oral cleanliness consistently found that smokers had poorer oral hygiene than non-smokers (13, 14). Macgregor (15) measured the area of stained plaque, and the proportion of gingival margin in contact with plaque in 64 smokers and 64 non-smokers, matched for age and sex. In both sexes, smokers had significantly more plaque than non-smokers, and there was a trend towards increased plaque deposits with increasing cigarette consumption.

However, others have reported contrary findings. Feldman (16), in the study of periodontal measures, found significantly less plaque in smokers than in non-smokers. Bergstrom and Eliasson (17) similarly found no difference in mean PI index scores amongst 285 musicians (31% smokers and 69% non-smokers). Bergstrom and Preber (18) studied the rate of plaque growth in 20 dental students, 10 of whom were smokers, and 10 non-smokers. Again, there was no quantitative difference between the growth rates of plaque in smokers and non-smokers.

Toothbrushing behaviour in smokers and non-smokers

Why smokers have more plaque than non-smokers is not altogether clear, but recent studies suggest that oral hygiene behaviour in smokers may be less favourable than in non-smokers. Toothbrushing behaviour has a marked effect on oral cleanliness; people who brush their teeth frequently have less plaque than those who brush less frequently or only occasionally (19). The toothbrushing efficiency of smokers was much less and the calcium concentration in the dental plaque of smokers was found to be significantly higher than in non-smokers (20, 21). There is opinion that male smokers spent significantly less time brushing their teeth, and had significantly more plaque remaining on their teeth after toothbrushing than age-matched, male, non-smokers (22).

Danielsen (23), following the experimental gingivitis model in healthy adult volunteers with good oral hygiene, found no significant differences in the rate of plaque accumulation between 12 smokers and 16 non-smokers.

These findings are compatible with the extrovert behaviour patterns observed in smokers; they tend to show

less well-developed health-related behaviour than non-smokers, but more developed grooming behaviour, at least in adolescence. Thus behavioural differences between smokers and non-smokers may largely account for the poorer oral cleanliness found in tobacco smokers.

Smoking and gingival inflammation

Gingival inflammation which develops through several phases, so that it further affects the other parts of parodont, and finally turns into periodontitis. The changes at the level of blood vessels appear as the first manifestations of gingival inflammation (*initial lesion*). There is dilatation of capillaries and increase of blood flow. In the *early lesion*, the initial lesions are becoming more marked, inflammatory infiltrate also increases, which clinically brings about the enlargement of gingiva. This is followed by changes in the cell population, with increasing in the number of lymphocytes and macrophages. *Developed lesion* occurs as the consequence of dental plaque persistence, when some of the bacteria may penetrate into the host tissue. Perivascular accumulation of chronic inflammatory cells is evident in this stage. The increasing number of cells in the chronic inflammatory content is followed by the loss of collagen in the affected connective tissue. However, in this stage, there is still no loss of the bone or connective tissue attachment (24).

Smoking does not normally lead to striking gingival changes. A reduction in clinical signs of gingivitis has been reported in smokers and this effect has been shown to be independent of plaque levels (25). Heavy smokers may have greyish discoloration and hyperkeratosis of the gingiva: an increased number of keratinized cells has been found in the gingiva of smokers. Changes in the epithelium were described as keratotic, hyperkeratotic and hyperplastic (26).

Smoking has long been considered an etiologic factor in acute necrotizing ulcerative gingivitis (ANUG). Rowland (27) in a series of studies determined that tobacco smoking was a factor in ANUG and that with the increase in the use of tobacco there was an increase in frequency of ANUG. The tar in the smoke exerted a direct irritating effect on the gingiva giving rise to gingivitis, and that nicotine could cause contraction of the capillaries (28).

Smoking and gingival bleeding

Bleeding from the gum margin is an important early symptom of gingivitis, and gingival bleeding on probing is now widely used in clinical examination as a means of identifying active lesions in periodontal disease.

Although smoking is known to produce peripheral vasoconstriction, in some subjects this is preceded by vasodilatation. In any particular instance, the effect produced is probably related to the degree of inhalation of the tobacco smoke and the rate of nicotine absorption (29).

Nicotine from cigarette stimulates the sympathetic ganglia to produce neurotransmitters including catecholamines (30). These affect the alpha-receptors on blood vessels which in turn causes vasoconstriction. The vasoconstriction of peripheral blood vessels caused by smoking can also effect the periodontal tissue (31) as smokers have less overt signs of gingivitis than non-smokers and clinical signs of gingival inflammation such as redness, bleeding and exudation are not as evident in smokers. The vasoconstrictive actions of nicotine may be responsible for the decreased gingival blood flow. Bergstrom et al. (32) have found less gingival bleeding in smokers than in non-smokers, due to vasoconstriction of gingival vessels, but may also be attributable to the heavier keratinization of the gingivae in smokers

More recently Palmer and colleagues (33) measured gingival blood flow, using a laser Doppler technique, and their data did not support the view that smoking compromised blood flow in the periodontal tissues. Tobacco use has also been associated with reduced permeability of peripheral blood vessels (34).

While there might be some controversy regarding the effect of tobacco consumption on the gingival vasculature, the clinical relevance is clear. Prolonged and heavy smoking can reduce gingival bleeding and therefore mask the clinical marker of bleeding on probing often used by dentists to monitor periodontal health.

Smoking and oral microorganisms

Smoking has important effects on oral bacteria. Although smokers have more plaque than non-smokers, there is no evidence to suggest that smoking increases the rate at which plaque develops. Cigarette smoking could cause a lowering of the oxidation-reduction potential (Eh), and this could cause an increase in anaerobic plaque bacteria (35). There was a statistically significant increase in the proportion of Gram-positive to Gram-negative bacteria in 3-day-old plaque from smokers, when compared with the non-smokers (36).

Tobacco smoke contains phenols and cyanides, which can account for antibacterial and toxic properties. Smokers harboured significantly higher levels and were at significantly greater risk of infection with *Tanarella forsythensis* than non-smokers (37). Adjusting for disease severity, *Porphyromonas gingivalis* was also more likely to subgingivally infect smokers than non-smokers (38). However, there was not a significantly higher relative risk for infection with this bacterium.

Calculus formation

Smokers had more calculus than non-smokers, but the effect of smoking was independent of the amount of calculus present. There have been consistent reports of more calculus in smokers than in non-smokers from the earliest epidemiological studies (39). Some authors reported that significantly more pipe smokers than ciga-

rette smokers had supragingival calculus. This might be because the pH of pipe smoke is higher than that of cigarette smoke, and because pipe smokers circulate the smoke around the mouth, whereas cigarette smoke tends to be inhaled (40). Moreover, the smoking cycle is much longer in pipe smokers than in cigarette smokers, causing pipe smokers to salivate more (41).

Reports that calculus formation is more abundant in smokers may be due to the increased salivary flow rates. There is an increased calcium concentration in fresh saliva in smokers following smoking (42). Nicotine affects the exocrine glands by an initial increase in salivary and

bronchial secretions that are followed by inhibition of the secretions. The calcium phosphates found in supragingival calculus are in the main derived from the saliva. The organic components may also arise from this source, the proteins and polypeptides constituting the major fraction (43). The increased amount of calculus found in smokers might therefore be due to an effect of tobacco smoke upon properties of saliva.

Smoking and periodontal disease

Periodontitis is defined as "inflammatory disease of supportive tissue of teeth caused by specific microorganisms which lead to progressive destruction of periodontal membrane and alveolar bone, with formation of periodontal pockets and gingival recession (44).

Opinions have been divided about the effect of smoking on chronic inflammatory periodontal disease. Earlier reviews of the epidemiology of periodontal disease concluded that smoking was a possible causative factor.

Few studies have conclusively demonstrated any relevant microbiological changes in the periodontal tissues attributable to smoking. Some authors (45, 46) using self-reported smoking data, investigated the relationship between periodontal pathogens and cigarette consumption. They reported an increased risk for smokers to have subgingival infection with *Porphyromonas gingivalis* although this was not found to be statistically significant. In this same study the investigators found smokers were 3 times more likely to harbor *A. actinomycetemcomitans*.

Many authors (47, 48) investigated the relationship between cigarette smoking and the prevalence of periodontal pathogens using polymerase chain reaction techniques. In this study, which included equal numbers of smoking and non-smoking subjects with generalised aggressive periodontitis, the investigators could find no significant differences in the occurrence of any of the pathogenic species which included *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tanarella forsythensis*, *Actinobacillus actinomycetemcomitans* and *Tanarella denticola*.

Mahuca and colleagues (49) evaluated the degree of periodontal disease and its relationship to smoking habits in a population of young healthy male Spanish mili-

tary recruits. They report higher plaque and bleeding indices in non-smokers although probing depths and attachment loss were greater in smokers. Young smokers diagnosed with aggressive forms of periodontitis were shown to have more affected teeth and a higher mean loss of periodontal attachment than non-smokers with these conditions (50).

Cigarette smokers had significantly greater probing depths and bone loss than non-smokers although no difference was found in relation to tooth mobility (14). Bergstrom et al. (51) found smokers not only to have significantly increased probing depths and alveolar bone loss, but also increased tooth mobility. Some studies have also highlighted the dose relationship between the effect of cigarette consumption and periodontal attachment loss (52, 53, 54).

Luzzi et al. (55) previously reported a relationship between alveolar bone loss and tobacco consumption. The findings when they investigated the relationship between cigarette smoking and bone loss in a group of dental hygienists were suggestive of an effect on alveolar bone that was independent of plaque levels. They also reported that this relationship was age-related, suggesting that progression was more significant in younger smokers.

It has been found consistently that smokers suffer more tooth loss than non-smokers (56, 57). Daniell (58), in study of osteoporosis in 208 women, aged 60-9, found that 75% of non-smokers and 67% of smokers had natural teeth remaining at 50 years of age. Bergstrom and Floderus-Myrhed (32) in their study and Feldman (16) and Calsina (59) in their surveys of periodontal disease, found that cigarette smokers had significantly fewer teeth than non-smokers. Osterberg (60) examined a random sample of 1377 patients: toothlessness in men was more common in smokers (48%) and ex-smokers (32%) than in non-smokers.

Smoking and host response

Nicotine metabolites can concentrate in the periodontium and their effects include the promotion of vasoconstriction, and the impairment of the functional activity of polymorphs and macrophages. The numbers of neutrophils in peripheral blood are also increased by tobacco use and their migration through capillary walls.

The polymorphonuclear leukocyte (PMN) is the most abundant phagocyte found at the site of acute inflammation, and probably has an important role in the defence of the marginal periodontal tissues against bacterial invasion. Corberand (1980) found PMN morbidity to be severely depressed by a solution of tobacco-smoke concentrate, although phagocytosis and bactericidal activity were not affected. Smokers have higher blood PMN counts than do non-smokers and chemotaxis of PMN s from smokers was suppressed relative to non-smokers (61).

Alani et al. (62) reported lower levels of both salivary elastase and neutrophils in the oral cavity in smok-

ers with periodontitis. Their study demonstrated that oral elastase and neutrophil counts are lower in smokers compared with nonsmokers with similar levels of periodontal disease. Their results also suggest that these values return to non-smoking levels after smoking cessation.

The passage of fluid through the junctional epithelium into the gingival crevice is markedly increased in gingival inflammation and resembles an inflammatory exudate. It contains leukocytes and plasma proteins, and probably plays an important role in the defence of the gingival tissues against bacterial attack (63).

Smoking appears to reduce the flow of this gingival fluid exudate. Bergstrom and Preber(18) studied 10 smokers and 10 non-smokers over a 4-week period during which the subjects abstained from all oral hygiene measures. They found that the degree of gingival redness, the occurrence of bleeding from gingival margin, and the gingival fluid exudate all increased during the experimental period.

It should also be noted that a significant genetic component has been identified in relation to aggressive periodontitis (64-66) and the combined interaction of cigarette smoking and various genetic polymorphisms might also contribute to disease status in young adults (67, 68).

Smoking and periodontal therapy

Following non-surgical therapy including scaling, root planing and professional tooth cleaning, healing in terms of gingival bleeding reduction and pocket depth reduction was less favorable in smokers (69) as compared to nonsmokers. The clinical results (70-73) showed a statistically significant reduction of pocket depth and number of diseased sites in both smokers and nonsmokers patients. This findings are in agreement with recent long-term results (74, 75) which suggest that tobacco smoking interferes with the healing process following non-surgical periodontal therapy.

A study by Grossi et al. (76) showed that current smokers have less healing and reduction in subgingival *Tanarella forsythensis* and *Porphyromonas gingivalis* after treatment compared to former and non-smokers, suggesting that smoking impair periodontal healing. Ah et al. (77), who reported less probing depth reduction and attachment gain in smokers who had been treated by periodontal surgery, corroborated this finding that smokers were poor candidates for successful periodontal care.

A statistically significant difference was observed in the reduction of probing depth between smokers and non-smokers at 12 month post-surgical follow-up after Widman flap surgery on 4 to 6 mm pockets (78).

James and colleagues (79) investigated the in vitro effect of nicotine on fibroblast activity. They found that it inhibited attachment and growth of periodontal ligament fibroblasts. The results of these studies all indicate that smoking has a deleterious effect on wound healing

and may help to explain why smokers respond less favourably to periodontal therapy.

Conclusion

Epidemiological studies of dental disease have consistently found poorer oral hygiene in tobacco smokers than in non-smokers. All of the surveys have reported increased quantities of calculus in smokers. It has long been known that smoking causes a marked increase in salivary flow rate as a simple reflex effect and this could explain the tendency of smokers to accumulate increased amounts of calculus. There is some evidence that smoking also increases the mineralizing potential of saliva.

All of the studies that measured plaque in smokers and non-smokers have found more in smokers. There is no evidence that smoking increases the rate at which plaque develops, or that it has any material effect on salivary precipitation. It seems likely that the major factor leading to greater plaque accumulation in smokers is inadequate oral hygiene. Toothbrushing habits in smokers tend to be less favourable than in non-smokers.

Smoking does not ordinarily give rise to striking gingival changes. At a clinical level, smoking appears to suppress visible gingival inflammation in response to plaque accumulation and there is mounting evidence that gingival bleeding is reduced in smokers. Gingival bleeding is an important early sign of chronic gingivitis and the masking of this feature may result in failure to recognize the presence of disease.

Smokers have fewer teeth than non-smokers and more severe destructive periodontal disease; they have deeper periodontal pockets and more alveolar bone loss. Smoking does not ordinarily lead to tooth loss, but the prospect of losing teeth from periodontal disease can be a persuasive argument against smoking.

Tobacco smoke has a strong reducing capacity in the mouth and appears to contribute to anaerobiosis, possibly by altering the oxidation-reduction potential in favour of anaerobic microorganisms, and possibly by a selective toxic effect on particular species. This predisposes smokers to oral infection by anaerobes, such that ANUG, which is associated with proliferation of anaerobic bacteria, is rarely seen in non-smokers, and it could contribute to the progress of destructive periodontal disease.

There is good evidence that smoking depresses the activity of oral PMNs, reducing their chemotactic response, mobility, and phagocytic ability. Blood flow in the gingiva and output of crevicular fluid are also reduced, further decreasing cellular and humoral immune components in the region of gingival crevice. It is not surprising that smoking has recently been shown to impair periodontal wound healing, and this includes non-surgical as well as surgical therapy.

It is hoped that the evidence of the harmful effects of smoking presented in this review might serve to stimulate dentists to give informed advice to help their patients who smoke to stop.

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PUŠENJE I PARODONTALNO OBOLJENJE

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Kratak sadržaj: *Uloga pušenja do sada je važila kao jedan od mogućih faktora koji učestvuju u progresiji parodontalnog oboljenja. Danas, veliki broj studija, objavljenih u stomatološkoj literaturi, osvrće se na ovu moguću ulogu. Veliki broj članaka navodi da pušači, koji imaju parodontalno oboljenje, manje reaguju na parodontalni tretman, bio on nehirurški, hirurški ili regenerativni. Ovaj članak obuhvata radove koji su ispitivali efekte pušenja na različite vidove parodontalnog oboljenja i prezentuje objašnjenje za moguću vezu između pušenja i progresije parodontalnog oboljenja.*

Ključne reči: *Pušenje, parodontalno oboljenje*