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Smoking, periodontal disease and the role of the dental profession

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Abstract: Epidemiological investigations support a firm relationship between smoking and periodontal disease. The likely benefits of smoking cessation programmes are considerable for periodontal disease, cancers and nearly all chronic systemic diseases. The mechanisms by which smoking may influence the development and progression of periodontal disease are as yet unclear, but may include changes in the vasculature, the immune and inflammatory systems, tissue oxygenation and the healing processes. Unfortunately, although dental professionals have more opportunities to encourage smokers to quit (most people visit their dentist more frequently than their doctor), dentists claim that they are not well informed on this subject. The purpose of this review is to describe the evidence for a link between smoking and periodontal disease, the possible pathology induced by smoking on the periodontal tissues and its impact on therapy, and to outline the smoking cessation techniques that are currently available.

Key words: smoking; periodontitis risk factors; smoking cessation

Introduction

The concept that smoking tobacco may be prejudicial to periodontal health is not new. In fact, an association between acute necrotising ulcerative gingivitis and smoking was observed nearly 60 years ago (1). Since then, numerous investigations of the relationship between smoking and periodontal disease have resulted in many publications on which this review is based. The role of the dentist in maintaining oral health is a vital one and is not limited to dental treatment. Most people see their dentist/dental hygienist more frequently than their physicians, and thus, oral health care providers should be properly educated in counselling patients on the adverse effects of smoking and the available techniques for smoking cessation.

The aim of this review is to discuss the reported association between smoking and periodontal disease, the pathogenic effects

of smoking on the periodontal tissues, the impact of smoking on periodontal therapy and the smoking techniques that are currently available.

Smoking and periodontal disease

More than 46 million (23%) American adults smoke. This includes 25% of males and 21% of females. There are more than 400 000 smoking-related or one in five deaths per year in the United States. In fact, over $45 billion is spent per year on treatment, and on average, smoking is thought to cause a 10-year life reduction. Unfortunately, although mortality related to cancer has declined 39% between 1970 and 1990, there has been no decline in lung cancer (2, 3).

The relative risk of contracting a disease is often reported as an Odds ratio. A larger Odds ratio means the factor is more likely to be a true risk for the development of the disease. Several cross-sectional studies have reported the effect of smoking on periodontal health to have an Odds ratio of between 2 and 6, depending on frequency of smoking. In 1995, a study of 1361 subjects aged 25–74 years showed an increased risk of severe bone loss in smokers when compared to non-smokers. The Odds ratio ranged from 3.25 in light smokers to 7.28 in heavy smokers (4). The reported risk of alveolar bone loss was also reported as 2.5 for smokers in a Swedish study of 155 patients when compared to the population at large (5). When controlling for other behavioural and socio-demographic risk factors, attachment loss was still significantly associated with smoking (6).

Longitudinal studies also report smoking as a significant risk factor for alveolar bone loss. In a 10-year study, smoking was a significant predictor for future bone loss (7). Another 10 years prospective study found significantly more periodontal bone loss in smokers compared to non-smokers (8). Furthermore, a 5-year study on the periodontal health of 800 community dwelling adults also suggested that smoking increased attachment loss (9).

In a dose–response study, Grossi *et al.* (10) demonstrated that the Odds ratio for more severe attachment loss was 2.05 in light smokers to 4.75 in heavy smokers, when compared to non-smokers. Tomar and Asma (11) found that those who smoked less than 10 cigarettes per day had an Odds ratio of 2.8, while those who smoked more than 30 cigarettes per day had an Odds ratio of 6.9. When considering 'pack years' (i.e. packs of cigarettes smoked per day multiplied by the number of years the subject has smoked), Alpagot *et al.* (12) reported a significant correlation for probing depth. The effect of smoking, however, may require a certain level of tobacco use before disease severity is increased (13, 14).

Studies evaluating the prevalence of moderate and severe periodontitis have found that cigar and pipe smoking have similar adverse effects on periodontal health as cigarette smoking. Results showed over 25% of current smokers, 20% of formerly heavy smokers, nearly 18% of cigar/pipe smokers and only 13% of non-smokers had moderate-to-severe periodontal bone loss (15).

There are some inherent difficulties when evaluating smoking as a risk factor. When measuring a subject's tobacco exposure over time, researchers most often rely on interviews or questionnaires; all retrospective studies are subject to recall bias. Many studies also quantify lifetime exposure in pack years, where current cigarette use may not reflect past use. To address this, more recent studies have measured cotinine levels to provide a quantitative measure of smoking status. Cotinine is the principle metabolite of nicotine and has been shown to correlate directly with periodontal breakdown (16).

Pathogenic effects of smoking

Several theories exist regarding the pathogenic effects of smoking on periodontal tissues. A brief discussion of each is included in this review. The toxins to which tissues are exposed during smoking may induce direct damage to the tissues.

Additionally, smoking may cause an altered microbial composition of plaque and an altered host response to the plaque. Smoking may also reduce blood flow, thus reducing the host's inflammatory/immune response, and impairing healing.

Cigarette smoke contains at least 500 potentially toxic substances, including hydrogen cyanide, carbon monoxide (resulting in carboxyhaemoglobin), free radicals, nicotine, nitrosamines (potent carcinogens) and a variety of oxidant gases (causing platelet activation and endothelial dysfunction). Nicotine itself can cause both psychological and physical dependency. Nicotine causes physical effects similar to that of caffeine; it is a stimulant and may cause a rise in blood pressure and vasoconstriction. Because nicotine is not carcinogenic itself, it can be used in other forms, such as patches and gum, to aid in smoking cessation. Nicotine and the noxious substances in tobacco smoke provide a dangerous combination in that nicotine whilst not obviously dangerous, addicts the user to smoke more and frequently. Thus, smokers expose themselves to more noxious agents that predispose to a variety of diseases.

Although some studies have shown conflicting results (17), smokers may have a more 'pathogenic' microbial flora. Grossi *et al.* (4) found an increased prevalence of specific Gram-negative bacteria in smokers. *Actinobacillus actinomycetemcomitans* (*Aa*) and *Tannerella forsythensis* (*Tf*) were harboured subgingivally more in smokers than in non-smokers, even after adjustments for periodontal disease and age; *Porphyromonas gingivalis* (*Pg*) counts, although not statistically significant, were higher in smokers than in non-smokers (18). In a study by Umeda *et al.* (19), current

smokers displayed an increased risk for harbouring *Treponema denticola* (*Td*) after molecular analysis techniques were used to examine six putative periodontal pathogens. Recently, Vander Velden *et al.* (20) found that among smokers, more patients remained culture positive for *Aa, Pg, Prevotella intermedia* (*Pi*), *Tf, Fusobacterium nucleatum* (*Fn*) and *Peptostreptococcus micros* (*Pm*).

Although currently a controversial theory, nicotine may cause vasoconstriction in the peripheral blood vessels (21), which may then reduce the clinical signs of gingivitis. Bergstrom (22) compared the compliance of smokers and non-smokers with an oral hygiene intervention programme, thus controlling for oral hygiene, and demonstrated significantly less gingival bleeding in smokers than in non-smokers even with the same plaque indices. Thus, chronic inflammation and clinical expression of gingivitis are suppressed in smokers.

Albandar *et al.* (23) looked at the prevalence of periodontitis and gingivitis among the general population and smokers. Advanced periodontitis increased from 3% in the general population to almost 5% in smokers; however, extensive gingivitis actually decreased from nearly 11% in the general population to only 5% in smokers, and limited gingivitis decreased from 22% in the general population to 13% in smokers. Thus, smokers may have a suppressed inflammatory response to plaque, which reduces the signs of gingivitis.

Kinane and Radvar (24) reported that gingival crevicular fluid (GCF) volumes were significantly lower among smokers than among non-smokers with the same pocket depths. Overall, a reduced GCF flow reduces the quantity of antibodies and other defence molecules derived from the serum, microbial nutrients and flushing of the gingival crevice, all of which may influence the microflora at these sites.

Macrophages are important in both cell-mediated and humoral immunity as antigen-presenting cells. Alveolar macrophages from smokers exhibit reduced expression of class II major histocompatibility complex (MHC; 25, 26), which may eventually lead to a reduction in the humoral immune response to invading organisms. Smokers had significantly higher peripheral blood levels of tumour necrosis factor alpha (TNF-α), interleukin-6 (IL-6) and the acute phase protein α2-macroglobulin (27). Smokers also had significantly higher TNF-α levels in their GCF than non-smokers in untreated and treated periodontitis patients (28). A dose-dependent effect of smoking on bronchial lavage levels of IL-1, IL-6, IL-8 and monocyte chemotactic protein (MCP)-1 levels was also reported (29). Current theories suggest that cytokine overproduction may be a detrimental host response, which predisposes an individual to periodontitis (30).

In general, the concentration of serum IgG is decreased in smokers (31–36). Smokers also have reduced titres of serum IgG to *Pi* and *Fn* (37). The level of IgG_2 against *Aa* is lower among early onset periodontitis (EOP) patients who smoke (38). Importantly, the IgG_2 isotype is associated with the humoral immune response against several antigens associated with oral pathogens (39).

Smoking decreases tissue oxygenation from 65 ± 7 to 44 ± 3 mmHg (40). When tissue oxygenation reaches levels of 40–50 mmHg, the tissues are predisposed to infection (41). With 'pack-a-day' smokers being hypoxic most of the time, smoking is therefore likely to increase the risk of surgical wound infection. In 1996, a study of smoking and infection after general operative surgery substantiated this. It was found that postsurgical infections increased from 7% ($n = 148$) of non-smokers to 22% ($n = 76$) of smokers (42).

The multifactorial nature of periodontal disease complicates the evaluation of risk factors for such a disease. Genetic, social and environmental factors not only affect the development and progression of disease, but may also interact with each other to contribute to the disease. For example, Meisel *et al.* (43) in 2003 found that the risk for periodontal disease increased 2.5 times ($n = 537$; $P = 0.013$) in IL-1 genotype positive smokers adjusting for age, gender, education and plaque.

Smoking and periodontal treatment

Smoking influences clinical assessment. Prior to treatment, smokers have underestimated probing depths and attachment loss. This occurs because the gingiva is less swollen and more resistant to probing (tighter or improved tissue tone) in smokers with an altered inflammatory response than in non-smokers (44). In addition, smokers often have more extensive recession than non-smokers (13, 45). After treatment, a larger change in probing depth with improved probing resistance can be expected in non-smokers.

Healing involves the following activities: inflammation, capillary budding, fibroblast proliferation, collagen production, revascularisation, epithelial attachment and periodontal ligament restoration. Following conventional scaling and root planing, healing is demonstrated clinically by reduction in pocket depth. It is a result of a reduction in inflammation, which leads to tissue shrinkage and reduced inflammatory swelling. The improved tissue tone is more resistant to pocket probing forces, detected clinically as an increase in clinical attachment. Combined with tissue shrinkage and recession, probing depths are reduced.

As already discussed, smokers have reduced inflammation. Therefore, in smokers, there may be a lack of inflammatory tissue swelling prior to treatment, which would have contributed to the post-treatment pocket depth reduction (24).

Healing from 3 months and 1 year may differ for smokers because of the cellular and tissue differences. Fibroblast function may be impaired in smokers, which may be because of fibroblasts binding and internalising nicotine (46). Important to periodontal healing, fibroblasts may not produce collagen fibres as efficiently in smokers. Thus, gingival tissue support and adaptation can be impaired or slowed down, resulting in poor tissue form and greater microbial plaque retention around teeth.

Non-surgical treatment in smokers, such as scaling and root planing, has not been as effective as in non-smokers or in previous smokers. Overall, there was less gingivitis resolution, less probing depth reduction and less attachment gain (47, 48). Subgingival antimicrobial therapy was also deemed less successful in smokers (24). After 6 weeks, pocket depth was reduced 1.14 mm in non-smokers and only 0.76 mm in smokers; a greater degree of recession also occurred among the non-smokers compared with the smokers.

Several studies have demonstrated a poorer response to periodontal treatment in smokers compared with non-smokers in terms of pocket depth reduction (48–50). In fact, studies have reported that approximately 90% of patients whose conventional periodontal therapy failed were smokers (51, 52).

As previously discussed, smokers are predisposed to postoperative infection. In addition, suture removal after general operative procedures was found to be delayed from 10 ± 2 days in non-smokers ($n = 76$) to 11 ± 4 days in smokers ($P < 0.04$). Hospital duration was also prolonged from 13 days in non-smokers to 15 days in smokers (42).

Following guided tissue regeneration, smokers are significantly more likely than non-smokers to have a reduced probing attachment level gain (53–56). Other investigators have also found smoking detrimental to healing with regenerative procedures using allografts (57, 58), and that the use of anti-infective therapy was necessary within the treatment protocol for smokers in order to achieve positive results (59).

Implants placed in smokers have a greater risk of developing peri-implantitis than in non-smokers (60). With similar mean plaque indices in smokers and non-smokers (maxilla and mandible), smokers had higher bleeding indices, mean peri-implant pocket depth and peri-implant mucosal inflammation, mesial and distal to the implant, especially in the maxilla. Implant failures were also more than two times as prevalent in smokers (11%) when compared to non-smokers (5%; 61).

Smoking cessation

Periodontal effects after cessation

Smoking cessation is beneficial to periodontal health (37, 62). When evaluating the presence of moderate or advanced periodontitis (after controlling for age and sex), the Odds ratio for those who currently smoked was 3.3 compared to those who never smoked, while that for former smokers were 2.1 compared to those who never smoked (63). After a 6-year prospective study, Krall *et al.* (64) concluded that smoking cessation significantly increased an individual's likelihood of tooth retention, but that it may take decades for the individual to return to the rate of tooth loss observed in non-smokers.

A dose–response relationship among current smokers between the number of cigarettes smoked per day and the odds of developing periodontitis ($P < 0.000001$) was demonstrated by Tomar and Asma (11) in 2000. Smokers who reduced the number of cigarettes smoked from over 30 per day to less than 10 subsequently reduced their likelihood of developing periodontitis (OR = 5.88 to 2.79, respectively). In patients who had stopped smoking for more than 10 years, the risk of periodontitis was even reduced to that of non-smokers. The response to periodontal treatment was also similar to that of non-smokers (65).

The clinical benefits of smoking cessation in the delivery of implants are also evident. Implant failure resulted in 39% of smokers, but only in 6% of non-smokers and 12% of former smokers (66).

The dentist's role

According to the American Dental Association, 'dentists have a key role to play in providing smoking cessation advice.' Patients often visit their dentist more often than their physician, which provides them with the opportunity to receive more frequent counselling and support from their dentists and/or their dental hygienists. Additionally, because smoking impacts the periodontal tissues, as well as the patient's overall health, dentists should be equipped to help patients to quit smoking. Dentists are health care providers, which is not limited to the oral cavity. When 696 dentists were asked if they thought that dentists should encourage their patients to stop smoking, 89% responded 'yes'; however, only 42% believed that dentists were effective in this area. Only 48% routinely recorded their patients' smoking status and less than 27% discussed the habit with smokers. Unfortunately, fewer than 10% reported that they felt they had adequate knowledge of smoking cessation techniques and half of them requested more training on the subject (67).

Techniques for habit cessation

Transtheoretical model

Although many dentists in the past have stated that they did not have the adequate materials or training to provide smoking cessation counselling (67, 68), dentists and dental hygienists

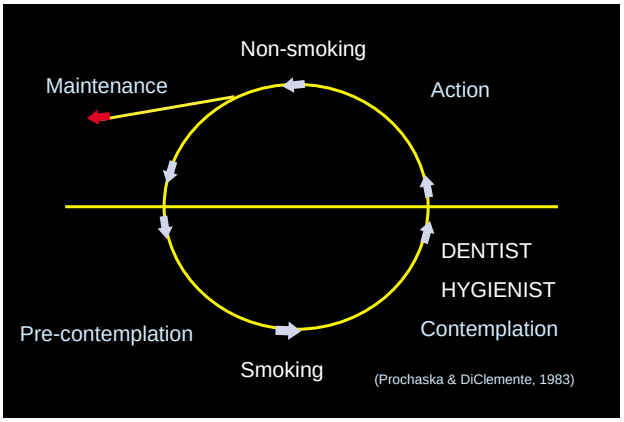

Fig 1. Transtheoretical model.

should be well versed in the techniques available for habit cessation. Currently, the transtheoretical model is the most widely used cognitive framework, which describes progression through five stages (69; see Fig. 1). This model is helpful in helping health care providers to understand at what stage the patient is in and what type of support is indicated for success.

- *Precontemplation:* The patient is not aware that the habit is the cause of his/her problem and has no intention of quitting. The goal is to increase the awareness of the problem and to guide them into the contemplation stage.
- *Contemplation:* The patient is aware that the habit is the cause of his/her problem and expresses an intention to stop within 6 months. The aim is to reinforce and encourage the patient to follow through with their intentions.
- *Preparation:* The patient intends to stop the habit within less than 1 month. Often, patients at this stage have previously attempted to quit, but without success. Patients are most responsive. Adjunctive therapies should be offered.
- *Action:* The patient stops the habit and maintains early abstinence. Significant effort should be made to control withdrawal symptoms at this stage.
- *Maintenance:* The patient has stopped the habit for six continuous months. Continued efforts should be made to prevent relapse.

Cessation advice

Other guidelines for health care providers when giving smoking cessation advice include the 'Five A's' (Fig. 2).

- *Ask* at each appointment about current and past smoking status.
- *Advise* all smokers to quit.
- *Assess* the smoker's current stage in the transtheoretical model.
- *Assist* the patient in the preparation stage including nicotine replacement therapy (NRT).
- *Arrange* for proper follow-up, and refer the patient to a smoking cessation clinic, if necessary.

ASK	Ask about past/current smoking status (incl: quit attempts NRT use).
ADVISE	Educate on the risks of smoking and the benefits of cessation.
ASSESS	Identify the current stage on the trans-theoretical model.
ASSIST	Set a quit date. Offer NRT or bupropion.
ARRANGE	Follow up in 1 week. Consider a referral to a cessation clinic.

Fig 2. The five A's of habit cessation.

Patients should not be recommended to gradually reduce the number of cigarettes per day. There is no evidence that 'cutting back' or switching to low tar/nicotine cigarettes contributes to success (70). Rather, patients attempting to quit should set a date to stop smoking completely. Studies have shown that when people reduce the number of cigarettes, they smoke more efficiently, subconsciously compensating for the increased fluctuation in blood nicotine levels (71). Some physicians actually encourage patients to 'over-smoke' in the hope that the patient will remember the unpleasant effects of agitation and nausea while refraining from the habit.

Smoking cessation is a difficult process for many people. In fact, most patients require three to four quit attempts before reaching success (71). Because nicotine is nearly as addictive as heroin and cocaine (72), every effort should be made to help reduce the symptoms of withdrawal. Practitioners should be well versed in the available adjunctive therapies. Additionally, patients should plan to quit during a time when home, family and work are most conducive to success. If other members of the household smoke and want to quit, every effort should be made to co-ordinate the cessation together.

Nicotine replacement therapy

Nicotine replacement therapy should be offered to all patients in the preparation stage in order to reduce the withdrawal effects of the action phase of cessation. No current NRT formulation matches the nicotine delivery to the brain that cigarette smoking provides. Nevertheless, NRT is more effective than placebo when achieving smoking cessation success (73). NRT is safe when delivered for a short term (matter of weeks; 74) and has not been shown to cause an excess of cardiovascular events (75).

No specific NRT appears to be superior to the others. Patches are simple to use, but nicotine gum, nasal spray and inhalers offer more controlled dose and delivery speed. The patch is available in 16- and 24-h preparations and in a variety of doses (76). The level of dependence should determine the dose of the patch used. It is applied in the morning, and the strength can be gradually reduced or stopped abruptly.

Nicotine gum is recommended for heavy smokers (>20 cigarettes a day) and is available in 2 and 4 mg. Approximately, 12–15 pieces are required per day initially, reducing the amount over

Table 1. **Contraindications and side-effects of nicotine replacement products and bupropion**

Treatment	Contraindication	Side-effects
Trans-dermal patch		Localised skin rash Insomnia with 24-h patch
Gum		Throat irritation Indigestion
Tablet/lozenge	Severe cardiovascular disease Recent myocardial infarction Recent cerebrovascular event	Throat irritation Indigestion Aphthous ulceration
Inhaler		Throat irritation Indigestion Cough Dry mouth
Nasal spray		Throat irritation Indigestion Nasal irritation Epistaxis
Bupropion	History of seizures Eating disorder CNS tumour Acute alcohol withdrawal Acute benzodiazepine withdrawal	Dry mouth Insomnia Seizures (1 : 1000)

Hobbs and Bradbury (71).

2–3 months. The gum is chewed until the flavour is strong, at which time it should be held in the cheek for absorption through the buccal mucosa. The nicotine inhaler is useful for those who miss the hand-to-mouth movement of smoking (oral fixation). It resembles a cigarette, and the nicotine is instead absorbed by the buccal membrane rather than by the lungs. Nicotine nasal spray is absorbed more rapidly than other forms of NRT, but it can cause nasal and throat irritation. Combination therapy is sometimes employed, but should be reserved for those who are unsuccessful with a single mode of treatment. Side-effects of each therapy are listed in Table 1.

Pharmacotherapy and other treatment

Bupropion (Zyban) is an antidepressant that is often helpful in smoking cessation. Its positive effects in smoking cessation are thought to be because of its inhibition of noradrenaline and dopamine reuptake rather than its antidepressant effects (77). Smokers in the preparation stage should take 150 mg daily for 6 days and then increase to the same dose two times a day. The quit date should be delayed 1–2 weeks to allow for the drug effect, and the patient should continue the prescription for six more weeks. Strong support and encouragement should accompany drug therapy. Other NRTs may be used in combination with bupropion. However, patients with a history of seizures should not use bupropion, as it has been associated with seizures and other negative side-effects (71).

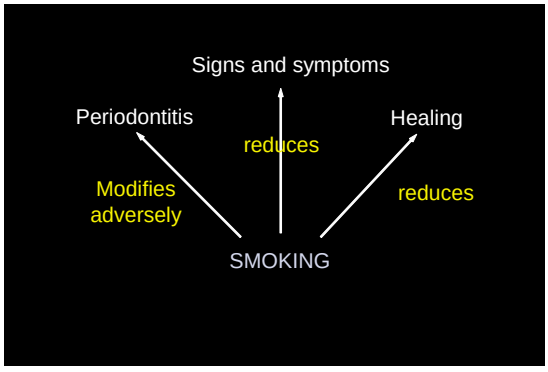

Fig 3. The effect of smoking on the periodontium.

Specialist smoking clinics are available to provide added support to patients attempting to quit smoking. Such clinics have reported higher success rates when compared to brief interventions, but it is unknown whether patient selection and motivation added bias to such results.

Other interventions are also available, such as acupuncture, acupressure, hypnotherapy, aversive smoking and antidepressants. Unfortunately, such techniques have shown little promise for success (78–81).

Conclusions

1 Epidemiological evidence indicates that smoking is strongly associated with periodontal disease.

2 There are numerous mechanisms by which smoking may adversely affect the periodontal tissues, but we are as yet unclear as to the most significant pathogenic mechanisms (Fig. 3).

3 Smoking has a demonstrable adverse affect on periodontal treatment outcomes and is likely to be because of a

combination of an actual reduction in healing, differences in the pathological processes and clinical differences in tissue morphology between smokers and non-smokers.

4 Smoking cessation is indicated in the promotion of better general health and in the improvement of periodontal health.

5 Dentists should offer to refer patients for smoking cessation counselling.

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