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# Effect of Smoking on Periodontium and Tobacco Cessation – A review of literature

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**Abstract**

Compared to other behavioural risk factors, the usage of tobacco is the leading cause of death and illness worldwide. Many significant aspects of the immunological and inflammatory responses are chronically affected by smoking.Research on histology has demonstrated that smokers' periodontial tissues' vasculature has changed. Smoking blocks neutrophil transmigration through the periodontal microvasculature and significantly increases systemic neutrophilia.One possible major mechanism in tissue degradation is the release of proteases from neutrophils.It has been discovered that tobacco smoke affects humoral immunity as well as cell-mediated immunity. Studies conducted on the gingival crevicular fluid have demonstrated lower levels of enzymes, cytokines, and even polymorphonuclear cells among smokers. This review gives a general overview of a few tobacco and nicotine products, focusing on how they may affect dental treatment and the risk of periodontal disease.Dental professionals might make use of the cessation and counselling services for tobacco use that are offered in their dental practise.

Keywords: Smoking, Periodontitis, Periodontal therapy,Nicotine replacement therapy, Smoking cessation

## Introduction

The greatest risk factor for mortality is tobacco use, which accounts for 15% of all deaths worldwide each year and over 9 million deaths. It is also the most prevalent behavioural risk factor, with high systolic blood pressure coming in second (1).Tobacco smoke not only negatively impacts almost all organ systems in the body but also serves as the main contributor to cancer, heart disease, and non-cancerous respiratory illnesses (2). The health concerns associated with tobacco use extend to others who are unintentionally exposed to smoke, often known as secondhand smoking (3). It has been determined that the main environmental risk factor for periodontitis is tobacco use, primarily from cigarettes (4). According to Bergeron

(1859), tobacco use plays a significant role in ulceromembranous gingivitis. The statement he made was not that smoking had a direct role in the onset of the disease, but rather that excessive tobacco use may have a predisposing influence(5).It's important to realise that tobacco smoke is made up of thousands of different compounds, even though the main psychoactive component is nicotine, which also causes some people to develop an addiction to it when they consume large amounts of it over time (4). (table 1)

| Table 1; Particulate phase | Gas phase |
|---|---|
| Nicotine<br>‘‘Tar’’ (composed<br>of many chemicals)<br>Benzene<br>Benzo(a)pyrene | Carbon monoxide<br>Ammonia<br>Dimethylnitrosamine<br>Formaldehyde<br>Hydrogen cyanide<br>Acrolein |

Classification of smokers

The Centres for Disease Control and Prevention (CDC) categorises smokers as follows:

i. Current smokers; those that had smoked more than or equal to 100 cigarettes over their lifetime.
ii. Former smokers; those that had smoked more than or equal to 100 cigarettes over their lifetime but were not currently smoking.
iii. Nonsmokers; those that had not smoked more than or equal to 100 cigarettes over their lifetime.

Depending on how many cigarettes they smoke each day, smokers are categorised as;

i. Heavy smokers; those that had smoked more than or equal to 20 cigarettes per day.
ii. Light smokers; those that had smoked less than or equal to 19 cigarettes per day.

**Smoking and periodontitis**

Smoking as a risk factor for periodontitis

One of the most powerful modifiable risk factors for periodontitis is cigarette smoking, which is second only to bacterial plaque in terms of reported risk. Depending on the severity of the condition and the amount of smoke inhaled, smoking is linked to a two- to eight-fold increased risk of bone loss and/or periodontal attachment (6).

Clinical and radiographic parameters in smokers

The mean probing depth is higher in smokers (7) and additional areas with deep probing depths (8). Furthermore, compared to nonsmokers, smokers have more gingival recession (9). Interestingly, the maxillary lingual sites and mandibular anterior teeth show the most differences in attachment levels and probing depths between smokers and nonsmokers (10).In addition, smoking increases the number of teeth with furcation involvement two to four times, the frequency of missing molar teeth (12), the incidence of vertical defects (11) and the loss of alveolar bone height (7). Compared to nonsmokers, smokers exhibit a decreased gingival inflammatory response, as shown by the fibrotic appearance of the tissues and fewer bleeding spots upon probing (13). Comparing smokers in the NHANES III group to never-smokers and periodontal patients, smokers had a roughly six-fold increased risk of bleeding at sites with supra- or subgingival calculus and 4 mm or greater probing depths (13).

Smoking and microflora

Porphyromonas gingivalis, Tannerella forsythia, and Actinobacillus actinomycetemcomitans were found in greater proportions in smokers than in nonsmokers in the Erie County research

(14).The basis for this was immunofluorescence. Treponema denticola was shown to be more frequently present in periodontal pockets among current smokers, according to bacterial culture experiments. (15).Haffajee and Socransky (16) found that smokers had much greater percentages of areas colonised with Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola. This was established by employing checkerboard DNA–DNA hybridization.

Mechanisms of Periodontal Disease Progression in Smokers

Because smoking is associated with a higher incidence and severity of periodontal damage, it is possible that alterations in the host-bacterial interactions characteristic of chronic periodontitis cause the periodontal tissue to break down more aggressively (17).Probing reveals decreased GCF flow and bleeding in addition to increased inflammation. (18). The etiological factors and impact of smoking listed in table no. 2

Table 2: Smoking-related microbiological, immunological, and physiological responses

| Etiology factor | Impact of smoking |
|---|---|
| Microbiology | No impact on the plaque buildup rate<br>An increase in periodontal pathogen colonisation of shallow periodontal pockets<br>Deep periodontal pockets show an increase in periodontal bacteria (19). |
| Immunology | alterations in phagocytosis, oxidative burst, and neutrophil chemotaxis<br>elevated PGE2 and TNF-α in GCF<br>elevated elastase and collagenase levels in neutrophils in GCF<br>PGE2 synthesis by monocytes is elevated in response to LPS (19). |
| Physiology | Reduced blood vessels in the gingival tissue and elevated inflammation<br>reduced GCF flow, bleeding while probing, and elevated inflammation<br>Diminished subgingival temperature<br>prolonged time required to recover from LA (19). |

**Effects of Smoking on the Response to Periodontal Therapy**

Compared to nonsmokers or former smokers, smokers do not react to periodontal therapy as well, according to a number of studies (20).

Therapy

• Nonsurgical:

At increasing levels of plaque control, the negative effects of smoking, the clinical response to scaling and root planning, the reduction of pocket depth, and the clinical attachment gain all become less significant. (20).

•Surgery and implants:

Reduced gain on clinical attachment levels; decreased pocket depth reduction following surgery; increased furcation degradation following surgery; and decreased bone fill and increased recession (20).

Parameters related to smoking mentioned in table no.3

| Table 3: overview Of Findings Regarding Smoking And Periodontal Disease Characteristics | |
|---|---|
| Specifications | Smoking |
| Gingival bleeding | reduced gingival bleeding and an increased percentage of small blood vessels |
| Alveolar bone loss | increased alveolar bone and periodontal attachment loss |
| Smoking as a potential cause of periodontitis | Disruption of wound healingis one of the main risk factors for the onset of periodontal disease. |
| Management for smokers: including nonsurgical and surgical methods | decreased healing response, decreased response to nonsurgical and surgical treatment |
| Nicotine's effects | May affect cells involved in periodontal Repairs |
| Effect of smoke | might have an impact on periodontal regeneration cells |

**Mechanism of action of Nicotine on polymorphoneutrophills (PMN'S)**

The protective immune response is significantly impacted by smoking, and the neutrophil plays a significant role in the host response. It is crucial for phagocytosis, chemotaxis, and killing through both oxidative and nonoxidative mechanisms. Smokers' oral cavity neutrophils or those exposed to nicotine in vitro have been demonstrated to have functional changes in phagocytosis, oxidative burst, and chemotaxis. Protease inhibitor synthesis, integrin expression, and superoxide and hydrogen peroxide formation are also impacted (20).

Research has indicated that exposure to nicotine causes monocytes to secrete more prostaglandins in response to lipopolysaccharides, which leads to increased periodontal damage. Additionally, there is a decrease in antibody formation, which is necessary for phagocytosis and additionally, immunoglobulin G2 levels are lowered. Nicotine prevents neutrophils from releasing reactive oxygen species (ROS), which causes oxidative stress-induced tissue damage and impedes the clearance of periodontal pathogens. (20).

It has been shown that nicotine increases degranulation as measured by neutrophil elastase release but suppresses phagocytic activity in neutrophils without influencing superoxide generation (21).

**Tobacco cessation**

Patients who are not ready to stop should receive extra attention from the 5 A's and 5 R's approach, according to the clinical practise guidelines for treating tobacco addiction and dependence. (20).

a. Ask about the tobacco use
b. Advice all users to quit
c. Assess willingness to make a quit attempt
d. Assist the patient to quit

e. Arrange follow-up contact.
The 5 R's:
a. Relevance
b. Risk
c. Rewards
d. Road blocks
e. Repetition.

**Medications for smoking cessation**

The FDA in the United States has approved several drugs for smoking cessation, including bupropion and varenicline, as well as NRT transdermal patches, gum, nasal sprays, inhalers, and lozenges (25). Despite not having FDA approval, nortriptyline and clonidine have been shown to be clinically beneficial for quitting smoking. (26) NRT and sustained release (SR) bupropion are the first-line treatments for quitting smoking. Other drugs used as second line therapy include varenicline, nortriptyline, and clonidine.

First line agents;

The FDA has currently approved five items to assist individuals in quitting smoking:there are four forms of nicotine replacement therapy: inhalers, nasal sprays, transdermal patches, and gum and sustained-release bupropion hydrochloride (22).

**Nicotine gum**; Use as needed to reduce cravings and on a regularly scheduled basis (e.g., initially every 1-2 hours when awake).The right way to chew slowly chew each piece.After around 15 to 30 chews, or when a peppery, minty, or citrus taste or tingling sensation appears, stop eating.Park between the cheek and the gums to facilitate nicotine absorption through the buccal mucosa.When the taste or sensation goes away, resume chewing.Continue the chew-park method until the majority of the nicotine is gone (about 30 minutes) and thetaste or tingling disappears. Park in various mouth regions (to avoid irritating the mucosa).Nothing to eat, and no acidic drinks (wine, juice, coffee, soft drinks)—15 minutes prior to and during use (may reduce nicotine absorption) (22).

**Nicotine patch**; Apply the patch to the upper limb or upper outer arm in a spotless, hairless area of skin.Avoid applying patches to burned, irritated, or inflamed skin since this may change how well the nicotine is absorbed.To ensure a sufficient seal, apply the patch to the skin and press firmly with the palm of your hand for 10 seconds.Shortly after applying a patch, wash your hands; Nicotine residue on hands can hurt or turn red when it gets in the nose or eyes.If used correctly, water from bathing, swimming, showering, or exercise won't damage the nicotine patch.Mild burning, tingling, or itching may happen shortly after the nicotine patch is applied; this should go away in one hour. The 16-hour patches should be taken off before bed.For the next 24 hours following the removal of the patch, the skin may seem red.Stop using the patch if a rash appears, if the area beneath it swells up, or if it irritates the skin for more than four days.To reduce the risk of skin responses, rotate the locations where patches are applied every day and give yourself at least a week's notice before applying another patch there (22).

**Nicotine nasal spray**; 2 sprays (1 in each nostril) from 1 dosage In the beginning, utilise at least 8 doses per day for optimal effects. If the spray is unclear, blow your nose before applying it. Lean back your head a little; place bottle tip as far into nostril as comfortable Using your mouth, inhale and spray once in each nostril. During administration, avoid sniffing, swallowing, or inhaling through your nose since this will intensify the unpleasant effects of the spray. Smell carefully to maintain the spray in your nose if it runs. Don't blow your nose for at least two or three minutes.Stay away from touch with your lips, eyes, and skin as these areas can absorb

nicotine. Sneezing, coughing, watery eyes, runny nose, and a burning, peppery feeling in the back of the throat or nose could happen. Using the nasal spray on a daily basis for the first week will help you get used to its annoying effects. Aim no more than five sprays each hour or forty sprays per day.In 3-6 months, gradually cut back on usage.

**Nicotine inhaler**; Use a minimum of 6 cartridges daily at first, and up to 16 cartridges daily if necessary.Inhale fumes at the back of your throat or take quick puffs; do not inhale into your lungs like you would with a cigarette; instead, "puff" as though you were lighting a pipe; Inhaling deeply into the lungs can cause symptoms of excessive nicotine intake, such as nausea, vomiting, and dizziness.Avoid eating or drinking anything acidic, such as juice, coffee, wine, or soft drinks, for fifteen minutes before and during the inhaler's use, as this may reduce the amount of nicotine absorbed.The best effects are obtained after 20 minutes of steady, frequent puffing.After 20 minutes of continuous puffing, the nicotine in the cartridge runs out.To manage your cravings for nicotine, try a titrate therapy: For a total of 20 minutes of continuous puffing on each cartridge, use the inhaler for a few minutes, put it down, and then use it once more later.

A cartridge that is open keeps its efficacy for 24 hours; beyond that time, it should be replaced, whether it has been used to its full capacity or not.When using the inhaler for the first time, a slight cough or irritation of the mouth or throat may occur.Throughout the first week, consistent use will aid in acclimating to the inhaler's irritating effects.In extremely cold temperatures, the inhaler's effectiveness may be reduced (for example, the distribution of nicotine vapour is hampered at temperatures below 15°C [59°F]) (22).

**SECOND-LINE AGENTS**

The FDA has not approved clonidine hydrochloride and nortriptyline hydrochloride as drugs specifically for smoking cessation; nonetheless, they are suggested as second-line treatments (23).The centrally acting 2-agonist antihypertensive drug clonidine roughly doubles the rate of cessation. The first recommended dosages are 0.1 mg taken twice a day or administered as a weekly 0.1 mg/day patch.

For three to ten weeks, the effective doses have varied between 0.15 and 0.75 mg taken orally and 0.1 to 0.2 mg applied topically. When compared to a placebo, the chance of quitting increases approximately thrice when using the tricyclic antidepressant drug nortriptyline. For 12 weeks, a goal dose of 75 to 100 mg per day should be gradually increased from the suggested starting dose of 25 mg at bedtime. These medications are currently unable to be classified as first-line due to their undesirable adverse effect profiles and lack of an FDA-approved indication for smoking cessation (23).

**Other pharmacological therapies**

Numerous neurohormonal and other physiological responses are produced during the administration and withdrawal of nicotine, and these effects influence the activities of tobacco. Treating these aspects is typically thought to improve treatment strategies because many of these consequences contribute to both relapse and continued cigarette smoking. These tactics are also a crucial component of many generic drug addiction therapies. Additional pharmaceutical treatments (24).

The patient starts bupropion treatment while they are still smoking, and within two weeks of starting therapy, they select a goal date to quit. This prolongs the time it takes for bupropion plasma levels to stabilise and, consequently, for the medication to begin acting as intended. 150 mg should be taken daily for three days as the starting dose, and then twice a day for the next

two months (a maximum of nine to twelve weeks of treatment is recommended). Some medical professionals do, however, advise up to six months of treatment (27).

Numerous clinical investigations have demonstrated the efficacy of nortriptyline, a tricyclic antidepressant, in aiding individuals in quitting smoking (29). It is recommended to be used in the dosage range of 75–100 mg daily for a period of 8–12 weeks as a second-line treatment for quitting smoking (29).

Initial approval for clonidine was for blood pressure reduction. It is an anti-hypertensive and central sympathetic activity-lowering alfa-2-adrenergic receptor agonist. It affects the central nervous system and might lessen the signs and symptoms of withdrawal from smoking. Before using clonidine as a supplemental smoking cessation medication, it is important to consider its possibly dangerous side effects. (24).

Vannicline, the drug used to treat nicotine addiction, does not contain nicotine. Starting one to two weeks before the scheduled stop date is the recommended course of treatment. 0.5 mg is administered daily for the first three days, then twice a day for the fourth and seventh days. The seventh day sees an increase in the dosage to 1 mg twice a day, which stays that way for the whole 12-week course. One should follow a 12-week course of therapy with 1 mg twice a day to prevent relapse. (30).

**Conclusion**

There are various systemic effects associated with tobacco smoking, some of which could explain why some people are more prone to periodontitis and have inferior treatment outcomes.One element of the environment that interacts with both the bacterial challenge and the host is smoking. It is important to consider how the host's genetic makeup interacts with environmental influences. Considering that people are more likely to visit their dentist than their doctor. Dentists are well-positioned to assist people in quitting smoking. Many options for patient education, intervention techniques, and abstinence reinforcement throughout active and ongoing maintenance therapy are provided by periodontal therapy.

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