

**SMOKING AND PERIODONTAL DISEASE**Grover Harpreet Singh<sup>1</sup>, Bhardwaj Amit<sup>2\*</sup>, Singh Yaswin<sup>3</sup><sup>1</sup>MDS, Professor and Head, Department of Periodontics, SGT Dental College, Budhera, Gurgaon, Haryana.<sup>2</sup>MDS, Reader, Department of Periodontics, SGT Dental College, Budhera, Gurgaon, Haryana.<sup>3</sup>BDS, Post Graduate Student, Department of Periodontics, SGT Dental College, Budhera, Gurgaon, Haryana.Email: [amitmds1980@rediffmail.com](mailto:amitmds1980@rediffmail.com)

DOI: 10.7897/2277-4572.02202

Published by Moksha Publishing House. Website [www.mokshaph.com](http://www.mokshaph.com)

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Received on: 15/01/13 Revised on: 22/03/13 Accepted on: 05/04/13

**ABSTRACT**

Periodontitis is the result of complex interrelationships between infectious agents and host factors. Environmental, acquired, and genetic risk factors modify the expression of disease and may, therefore, affect the onset or progression of periodontitis. Numerous studies of the potential mechanisms whereby smoking tobacco may predispose to periodontal disease have been conducted, and it appears that smoking may affect the vasculature, the humoral immune system, and the cellular immune and inflammatory systems, and have effects throughout the cytokine and adhesion molecule network. The aim of present review is to consider the association between smoking and periodontal diseases.

**Key words:** Smoking, cessation, nicotine, immunoglobulins, periodontal diseases

**INTRODUCTION**

Periodontal diseases are a group of conditions affecting the supporting structures of the dentition.<sup>1</sup> Progression and severity of the disease depends on the complex interactions between several risk factors such as microbial, immunological, environmental and genetic factors, as well as

age, sex and race. Tobacco smoking is a significant risk factor for periodontal disease.<sup>2</sup> Tobacco smoking is an addictive habit first introduced into Europe. Smoking is now recognized as the most important cause of preventable death and disease.<sup>1</sup> Smokers are three times more likely to get acute periodontitis than non smokers.<sup>3</sup>

**Table 1: Evidence for smoking as an etiologic factor for periodontitis<sup>4</sup>**

Criterion	Evidence
Strength of association	Cross-sectional and case-control studies demonstrate a moderate to strong association between smoking and periodontitis.
Consistency	Multiple studies of various designs (cross sectional, case-control, and longitudinal) and in various populations have demonstrated an association between smoking and periodontal attachment loss.
Specificity	Disease progression slows in patients who quit smoking as compared to those who continue to smoke.
Temporality	Longitudinal studies show that smokers do not respond as well to periodontal therapy as non-smokers.
Biologic gradient	There is a dose-response effect in that heavy smokers have increased disease severity compared to light smokers.
Biologic plausibility	The biologic plausibility of the explanation of the relationship between smoking and periodontitis is supported by tobacco's adverse impact on microbial and host response parameters.
Coherence	The effects of smoking on periodontitis are consistent with our knowledge of the natural history of periodontal disease.
Analogy	Periodontal effects of smoking are analogous to other adverse smoking-related general health effects.
Experiment	Evidence not currently available.

The utilization of tobacco products has long been linked to periodontal disease. Cigarette smoking alone does not cause periodontal disease. Periodontal disease is caused by bacterial plaque that gets under the gingival tissues and initiates a process by which the infection initiates bone loss around the teeth. In smokers, once periodontal disease starts, smokers lose bone at a faster rate than nonsmokers. This is because of the effect of nicotine on the circulatory system, which in turn cut down on the blood supply and also reduces the intake of oxygen by hemoglobin, thus in turn retards the body's ability to fight the infection and bone resorption.<sup>5</sup>

WHO estimates that there are about 1100 million regular smokers in the world today. About 300 million (200 million males and 100 million females) are in the developed countries and nearly three times as many (800 million: 700 million males and 100 million females) in developing countries.<sup>5</sup>

Tobacco companies are producing cigarettes at the rate of five and a half trillion a year. Nearly 1000 cigarettes for every man, woman and child on the planet. Asia, Australia and the Far East are by far the largest consumers (2,715

billion cigarettes), followed by the Americans (745 billion), Eastern Europe and Former soviet Economies (631 billion) and western Europe (606 billion).<sup>5</sup>

Dr. David Kessler, former Commissioner of the Food and Drug Administration (FDA), has referred to smoking as a "paediatric disease" because the average smoker begins by age 15 years and is a daily smoker by age 18 years. According to Michelle Bloch of the American Medical Woman's Association "Half of all long-term smokers, especially who begin in their teenage years, are most likely to be killed by tobacco & half among them in early middle age"<sup>6</sup>

**Historical Background**

Christopher Columbus reported a gift of strange dry leaves from a native of San Salvador. The word tobacco is said to have come from sources after the island of 'Tobago' in the West Indies. Friar Roman Paine, a monk who had accompanied Columbus in his 2<sup>nd</sup> voyage (1493) is reported to have carried a supply of tobacco back to Portugal and the practice of sniffing started to spread. In the medieval era, in

Europe, it was believed that tobacco protected and cured a long list of disorders including toothache. Hence it gained social acceptance and respectability.<sup>5</sup>

In 1604, King James I of England issued the first official condemnation of tobacco, "A Counterblast to Tobacco" in which he warned his subjects that "the habit of smoking tobacco is disgusting to sight, repulsive to smell, dangerous to the brain and noxious to the lung."<sup>7</sup>

The first commercial plantation of tobacco was in Virginia (USA) in 1612. Pierre Lorillard established the first snuff mill in America in printed American advertisement.<sup>5</sup>

Tobacco is derived from the species of the plant of genus *Nicotiana* of the potato family. Carl Linnaeus in 1753 had named the genus of the tobacco plant '*Nicotiana*' after the French ambassador to Portugal, Jean Nicot.<sup>5</sup>

One of the first published medical reports on the effects of tobacco appeared in 1859. It was based on a study of 68 patients in a hospital in Montpellier, France, who had cancer of the lips, tongue, tonsils and other part of the mouth. The authors noted that all of the patients used tobacco and that 66 of them smoked short stemmed clay pipes in typical fashion, rather than give up tobacco, smokers just switched to long-stemmed pipes.<sup>7</sup>

In January 1964, U.S. Surgeon General Luther L. Terry issued the landmark report on health hazards of cigarette smoking based on an analysis and cigarette smoking of 1200 studies. The following year federal legislation was passed requiring all cigarette package to carry health hazards warning labels.<sup>7</sup>

In 1986, the requirement for warning labels was extended to smokeless tobacco products.<sup>7</sup>

### Constituents of Tobacco

Tobacco smoke is estimated to contain over 4000 compounds many of which are pharmacologically active, toxic, mutagenic and carcinogenic. There are 43 known carcinogens in tobacco smoke. NNN (N-nitrosornicotine), a volatile N-nitroso compound, was the first organic carcinogen isolated from smokeless tobacco.<sup>5</sup>

Tobacco smoke consists of a gaseous phase which is composed of carbon monoxide, nitrogen, oxygen, and carbon dioxide as well as particulate phase containing nicotine, water and polycyclic aromatic hydrocarbons.<sup>8</sup>

The following are the constituents of tobacco and the adverse effects caused by each one of them:

#### I. Nicotine

The IUPAC name of nicotine is 3-[(2S)-1-methylpyrrolidin-2-yl] pyridine. Its chemical formula is  $C_{10}H_{14}N_2$ . Its bioavailability 20 to 45% having the hepatic metabolism and half life of 2 hours.<sup>9</sup> The average lethal dose for an adult human is estimated to be between 30-60 milligrams (mg). Nicotine is the pharmacological agent in the tobacco smoke that causes addiction among smokers.<sup>5</sup> It is metabolized in the body into two pharmacologically inactive metabolites-cotinine and nicotine-N- oxide.<sup>8</sup> Cotinine has a longer half life of 20 hours than nicotine and has been used to estimate intake of nicotine by its measurement in plasma, urine or saliva of cigarette smokers.<sup>10</sup>

The addictive effect of nicotine is linked to its capacity to trigger the release of dopamine - a chemical in the brain that is associated with the feeling of pleasure.<sup>9</sup> A positive correlation has been found between serum cotinine levels and the severity of periodontal attachment loss in cigarette

smokers.<sup>9</sup> Cotinine has been identified in samples of saliva and gingival crevicular fluid from smokers.<sup>9</sup>

#### 2. Tar

It describes the particulate matter inhaled when the smoker draws on a lighted cigarette. In its condense form, tar a sticky brown substance which can stain smoker's fingers and teeth yellow brown. It also stains the lung tissue.<sup>5</sup>

#### 3. Carbon monoxide

The amount of oxygen carried by the blood may be severely deprived in heavy smokers due to the effects of carbon monoxide. Oxygen levels may be reduced by as much as 15%. Carbon monoxide also restricts the oxygen available to the foetus, contributing to the low weight of babies born to women who smoke. The baby in the womb cannot grow normally if deprived of oxygen.<sup>5</sup>

#### 4. Nitrogen oxide

#### 5. Hydrogen cyanide and other ciliotoxic agents

#### 6. Metals

Thirty metals have been detected in tobacco smoke, including nickel, arsenic, cadmium, chromium and lead.<sup>5</sup>

#### 7. Radioactive compounds

The radioactive compounds found in highest concentration in cigarette smoke are polonium-210 and potassium-40. Radioactive compounds are well established as carcinogens.<sup>5</sup>

#### Oral and Systemic Effects of Nicotine

In the gingival crevice fluid of smokers, nicotine concentrations are nearly 300 times than found in plasma (20 ng / ml) It is logical to assume that the vasoconstrictive properties of nicotine impair gingival blood flow; however, gingival blood flow in humans during active smoking has been shown to be unaltered or increased as assessed by laser Doppler flowmetry.<sup>11</sup>

Nicotine binds to the root surface in smokers. This can alter gingival and periodontal ligament fibroblast attachment and proliferation in vitro. Tipton and Dabbous also demonstrated reduced collagen production as well as increased collagenase activity in gingival fibroblast cultures exposed to nicotine.<sup>11</sup>

Oral polymorphonuclear leukocytes (PMNs) harvested from smokers were less vital and less able to phagocytose particles than PMNs from non-smokers.<sup>12</sup>

Exposure to tobacco smoke causes decreased lymphocyte viability; reduced antibody production; cell degranulation; decreased protein, RNA and DNA synthesis in macrophages, decreased macrophage culture viability and increased viability and DNA synthesis in fibroblasts.<sup>4</sup>

#### Smoking as Risk Factor for Periodontal Disease

Cigarette smoking is a well-established risk factor for periodontitis and second to bacterial plaque, is the strongest of the modifiable risk factors. Smoking is associated with a two to eight-fold increased risk for periodontal attachment and or bone loss, depending on the definition of disease severity and smoking dose.<sup>11</sup>

#### Effects of Smoking on Prevalence and Severity of Periodontal disease

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.<sup>13</sup>

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 and to the number of years that the patient has smoked. 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. 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. 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.<sup>13</sup>

### Effects of Smoking on Tooth Deposits

Smoking is known to increase the rate of salivary flow. Although there is a initial drop in calcium concentration on stimulation of salivary flow, after a few minutes of stimulation at a constant flow rate, the calcium concentration rises; the proportion of saliva from the parotid gland increases, thus tending to raise the average calcium concentration in the mouth.<sup>14</sup>

Prevalence as well as severity of supragingival calculus were found to be greater in smokers than in individuals currently not smoking tobacco. The association with smoking was quite evident, a current smoker running 3-fold increased risk of exhibiting supragingival calculus compared to a non-smoker.<sup>15</sup>

The metabolism of carbohydrate by plaque microbes leads to the formation of acid, the accumulation of which can lead to dissolution of the underlying tooth surface. On the other hand, the metabolism of nitrogenous substrates results in the formation of base and a high pH level, which can lead to deposition and accumulation of calcium phosphate as calculus within the plaque. It has been shown that higher pH levels are found not only in plaques located in regions of a greater flow of saliva but also in plaques of individuals with higher flow rates of resting saliva.<sup>16</sup>

### Etiopathogenesis of Periodontal Disease Progression in Smokers

#### Microbiology

#### 1) Effect of smoking on plaque development and accumulation

Smokers showed a higher prevalence of dental plaque than non-smokers suggested that more severe periodontal disease in smokers might be because of greater accumulation of plaque.<sup>17</sup>

#### 2) Effect of smoking on the subgingival microflora in periodontitis

The earliest reported evidence of microbiological differences between effects of tobacco smoking on periodontitis 181 smokers and non-smokers was provided by Zawhon et al.<sup>17</sup> The results indicated a higher prevalence of Aa, T.

forsythensis and p. gingivalis in the current or former smokers. In particular, the authors reported that the risk of subgingival infection with T. forsythensis in current smokers was 2.3 times that of non-smokers

### Immunology

#### A) Effect of Smoking on Neutrophils

##### 1) Smoking & neutrophil function

PMNs harvested from the gingival junction of smokers were shown to have reduced phagocytic capacity compared to PMNs from non-smokers.<sup>18</sup>

Neutrophil transmigration across the periodontal microvasculature is impeded in tobacco smokers.<sup>17</sup>

Neutrophils express functional receptors for several components and metabolites of tobacco smoke, such as nicotine, cotinine. The numbers of nicotinic receptors expressed by human Neutrophils are increased in smokers and decline on cessation.<sup>17</sup>

##### 2) Neutrophil derived degradative proteases

Tobacco smoking leads to significant increases in the circulating burden of neutrophil elastase and MMPs in humans. Tobacco smoking has also been shown in human skin to decrease the rate of synthesis of specific collagen types, by increasing the production of collagen-degrading enzymes, and decreasing levels of the major endogenous MMP inhibitor, tissue inhibitors of MMP-1.

##### 3) Neutrophil respiratory burst

The respiratory burst represents the combined oxygen-dependent processes by which neutrophils kill phagocytosed bacterial cells through the generation of multiple reactive oxygen and reactive nitrogen species. A compromised respiratory burst may reduce the capacity of neutrophils to destroy plaque bacteria. Several studies have suggested that cigarette smoke constituents inhibit the respiratory burst of neutrophils.<sup>17</sup>

#### B) Effect of smoking on Lymphocyte Function

The effects of smoking on lymphocyte function and antibody production are very complex, having the potential to cause immunosuppression or stimulation. Particulate phase of cigarette smoke confers immunosuppressive properties. Acute or chronic exposure to hydrocarbons, may stimulate or inhibit the immune response, the net effect being dependent upon the dose and duration of exposure to components of tobacco smoke. The leucocytosis observed in smokers result in increased number of circulating T and B lymphocyte. Smoking appears to affect both B and T cell function, inducing functional unresponsiveness in T-cells.<sup>18</sup>

##### 1) T lymphocytes

Reduced, increased or no change in the number of CD4 T cells. Smokers suffer from respiratory infections more frequently than non-smokers. The total number of lymphocytes is increased in BALF and the CD4 cell subpopulation is reduced producing a reduced CD4/CD8 ratio in smokers compared with non-smokers, while the CD4/CD8 ratio in peripheral blood is similar.<sup>17</sup>

##### 2) B lymphocytes and immunoglobulins

B cells recognize antigen once it has bound to antigen-binding sites of immunoglobulin on the B cell antigen receptor, namely antibody expressed on the B cell surface. In

order to mount successful humoral immune responses, B cells require T helper cell-derived cytokines to proliferate and differentiate into plasma cells (as well as for immunoglobulin class switching). In experiments in animals and humans, tobacco smoke has been found to affect both humoral immunity and cell-mediated immunity. Chronic exposure of rats to nicotine inhibits antibody-forming cell responses and this immunosuppression appears to be the result of impairment of antigen mediated T cell signalling. These findings are supported by reports indicating that serum IgG levels are reduced in smokers and in periodontitis patients; non-smokers have higher levels of IgG2 compared with smokers.<sup>17</sup>

The effects of cigarette smoking on serum IgA and IgM classes are controversial, with some reports indicating suppression of IgM and IgA levels, while other studies indicate no effect of smoking on either class of antibody. IgE is greatly elevated in smokers and not related to enhanced skin reactivity.<sup>17</sup>

### 3) Natural killer (NK) cells

NK cells, components of the innate immune system, are large granular non-T non-B lymphocyte like cells that make up a small proportion of peripheral blood lymphoid cells. Unlike

T and B cells, NK cells do not have antigen specific receptors, but are able to recognize and kill antibody-coated target cells (antibody dependent cellular cyto-toxicity) and this is triggered when antibody bound to the surface of the cell interacts with Fc receptors on the NK cells. The mechanism of attack is analogous to cytotoxic T cells involving the release of granules containing perforins and granzymes.<sup>17</sup>

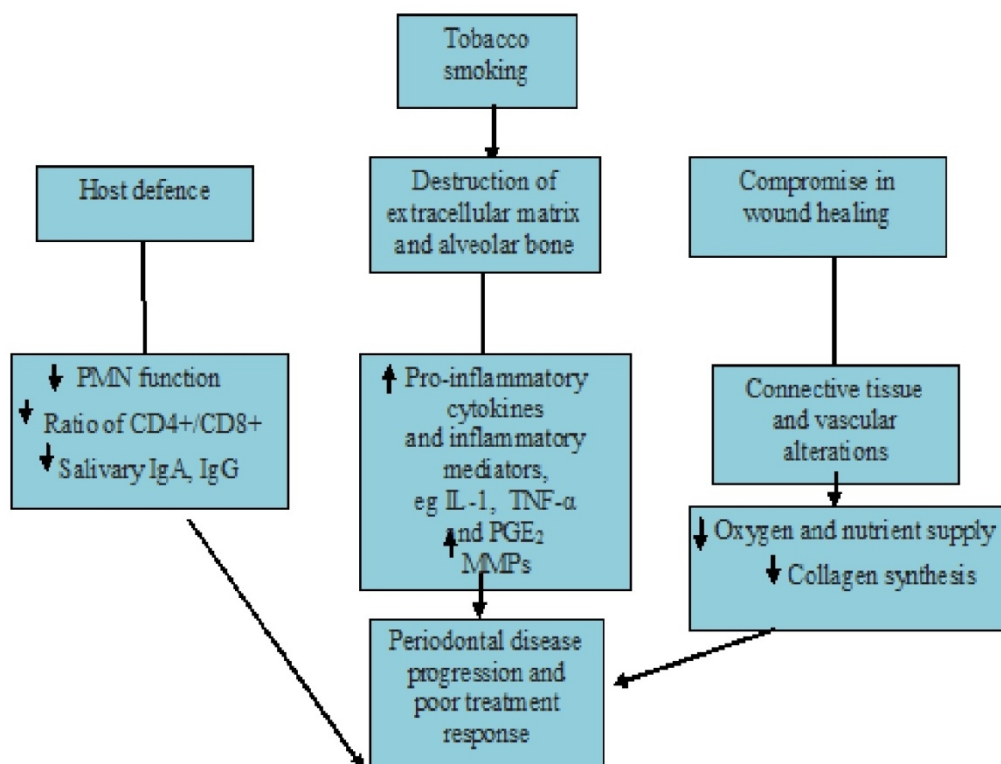
Effect of smoking on NK cells is reversible and cytolytic activity may increase even within a short period of a month of smoking cessation.<sup>17</sup>

### C) Cytokines & Other factors

Higher levels of TNF $\alpha$  in GCF in smokers and former smokers compared with non-smokers, with comparable levels of moderate/ severe periodontitis. No differences were found in levels of IL-6, which were frequently below the detection levels of their ELISA.<sup>17</sup>

It was found that levels of IL-1 and IL-1ra to be significantly lower in GCF from diseased sites in smokers compared with non- smokers, using a 3min. paper strip sample measured with a Periotron 6000 and collected following the discarding of a 30 s sample.<sup>17</sup>

### Effect of Tobacco Smoking On Periodontal Tissues<sup>19</sup>



### C) Smoking and fibroblast function

#### 1) Gingival fibroblasts

There is evidence that gingival fibroblasts from smokers may be less susceptible to the cytotoxic effects of high levels of nicotine possibly because of the development of tolerance. There is reduction in cell viability and disruption to the microtubules, intermediate filaments and actin.<sup>17</sup>

#### 2) PDL fibroblasts

Cell attachment was significantly less on root surfaces obtained from heavy smokers compared with non-smokers and healthy controls.<sup>17</sup>



## Physiology

### 1) Effect of smoking on gingival blood flow

The light smokers responded with a significant increase in blood flow, paralleling the changes but the heavy smokers showed no response, indicating a high level of tolerance.

### 2) Oxygen tension in the gingival tissues

In healthy gingiva, smokers have lower oxygen saturation, determined using tissue reflectance spectrophotometry.<sup>17</sup>

### 3) Gingival inflammation and bleeding

Smokers experienced less gingival bleeding than non-smoker.<sup>17</sup>

Tobacco smoking mask the inflammatory signs of gingivitis and periodontitis, particularly the propensity of the gingiva to bleed on brushing, eating or after periodontal probing.<sup>17</sup>

### 4) Effect on the gingival vasculature

High proportion of small vessels compared with large vessels in smokers than non-smokers, but no difference in the vascular density was found.<sup>17</sup>

### 5) Effect of Smoking on Subgingival Temperature

Temperature was found to be on the higher side in smokers. Subgingival temperature was measured at 6-sites around each of 4 maxillary anterior teeth.

Smokers have higher mean probing depths and more sites with deep probing depths. Gingival recession is greater in smokers compared to non-smokers. Smokers have two to four times more teeth with furcation involvement, are more frequently missing molar teeth, and demonstrate greater loss of alveolar bone height and a higher prevalence of vertical defects. Smoking affect the mineralization rate of calculus.<sup>11</sup> A higher amount of alveolar bone destruction has been seen in smokers and the severity of destruction was also found to be greater in smokers compared to non-smokers. The bone mineral content among smokers was found to be 10-30%

lower as compared to non smokers in longitudinal cohort study.<sup>20</sup>

## Effects of Smoking on Response to Periodontal Therapy

### Non-surgical

Current smokers do not respond as well to periodontal therapy as non- smokers or former smokers. The majority of clinical research supports the observation that reduction in pocket depth is more effective in non- smokers than in smokers after non-surgical periodontal therapy, including oral hygiene instructions, scaling and root planing. Smokers respond less well to non- surgical therapy than non-smokers.<sup>21</sup>

Compared with non-smokers, smokers have less reduction of PD for the dentition as a whole. Compared with non smokers, the reduction in bleeding was less pronounced in smokers inspite of greater reduction of plaque index. Treated infra-bony defects are adversely affected in smokers compared with non-smokers. Smoking adversely affects treatment outcomes.<sup>22</sup>

Smokers have less healing and reduction in subgingival *Tanarella forsythensis* and *Porphyromonas gingivalis* after treatment compared to former and non- smokers.<sup>1</sup>

Increased vasoconstriction of peripheral blood vessels observed in smokers has been related to reduce bleeding and oedema in periodontal patients who smoke, compared to non-smokers.<sup>23</sup>

Smokers have less potential for resolution of inflammation and oedema within marginal tissues and therefore less potential for gingival recession.<sup>23</sup>

Smokers, nonsmokers and ex-smokers did not differ significantly in plaque, BOP, Attachment levels, radiographic, or biochemical regimes. Smokers have a poorer treatment response to scaling and Root planning regardless of the application of either systemic or locally applied adjunctive metronidazole.<sup>22</sup>

Table 2: Impact of smoking on therapeutic outcomes<sup>11</sup>

Treatment	Outcomes in smokers compared to non- smokers
Scaling and root planing; various forms of surgical periodontal therapy.	Smokers exhibit 50-75% as much improvement in clinical parameters.
Antimicrobial therapy	Adjunctive antimicrobial therapy brings the smokers response to that of non-smokers receiving scaling and root planning alone.
Sub- antimicrobial doxycycline therapy	Adjunctive sub – antimicrobial doxycycline therapy brings the smokers response to that of the non- smokers receiving scaling and root planning alone.
Site development and dental implant therapy	Smokers experience approximately twice the failure rate based on a variety of implant designs and surfaces.

## ii) Surgical Therapy

The less favourable response of the periodontal tissues is observed in surgical therapy in smokers.<sup>21</sup>

Smokers showed less percentage of reduction in presurgical pocket depth than non- smokers. Open flap debridement surgery without regenerative or grafting procedures is the most common surgical procedure used for assessing the root and osseous surfaces.<sup>21</sup>

Smokers exhibit a less favourable healing outcome following a surgery in terms of vertical and horizontal attachment gain. Smoking impairs healing of GTR treated infra-bony defect.<sup>22</sup> Smokers respond less favourably to flap debridement surgery in terms of pocket depth reduction and attachment level gains especially in sites with deep pocket depth.<sup>24</sup>

## Smoking and implants

The increased risk of wound healing complications as well as the risk of periimplant bone loss and increased implant failure rates is found in smokers.<sup>25</sup>

Smoking more negatively impacts implants placed in maxillary arch than in mandible.<sup>4</sup> The percentage of maxillary implant failures among smokers (10.9%) was higher than that reported for non smokers or past smokers (6.4%).<sup>11</sup>

In both smokers and nonsmokers, the acid etched implants fared better than machined surface implants in smokers and in nonsmokers.<sup>11</sup>

## Implants and site development

Marginal bone loss around hydroxyapatite-coated and machined titanium surface implants in maxillary sinus grafts in smokers was twice that in non smokers.<sup>11</sup>

### Effects of Smoking Cessation

Nicotine dependence is classified as a chemical addiction by American psychiatric Association in diagnostic and statistical manual of mental disorders 1994.<sup>4</sup>

Smoking cessation is beneficial to pdl treatment outcomes and periodontal health. Periodontal disease progression slows down in individuals who quit smoking. Smoking cessation restore the normal periodontal and microbial healing responses: the healing responses of ex smokers become similar to that of non smokers.<sup>19</sup>

Former smokers who no longer smoke tend to exhibit a periodontal condition in between those of current smokers and non- smokers.<sup>26</sup> Periodontal status of former smokers is intermediate between that of never smokers and current smokers.<sup>13</sup>

Gingiva of treated current smokers exhibit minimal redness and bleeding while brushing presumably because of immune suppression or vascular effect of smoking.<sup>27</sup>

Nicotine exceeds addiction to hard drugs such as heroin or cocaine. A small proportion of smokers do not appear to be addicted to nicotine. Cigarettes are extremely efficient devices for delivering nicotine into the system; it takes less than 8 s for nicotine to reach the brain and 20s for nicotine to reach the furthest parts of the body (from the mouth). Smokers smoke in order to maintain a level of nicotine in their system, and when the nicotine falls below that level, they suffer withdrawal symptoms, including the desire to smoke. Light smokers may smoke for the 'peak' given by the nicotine hit, while more dependent smokers smoke to address their nicotine withdrawal.<sup>28</sup>

The vast majority of smokers want to quit-around 70% say they want to give up - but only around 2% a year will succeed. A large number of smokers (79%) have previously tried to quit.<sup>28</sup>

### Role of Dental Professionals in Smoking Cessation

#### Giving advice on smoking Cessation

There are a number of approaches that can be used to give individual advice to smokers. This can vary from 'very brief advice', where attention is drawn to the smokers' habit, 'brief advice' including five step programme and is a more detailed

advice such as that given by the specialist smoking cessation services.<sup>28</sup>

#### Very brief advice

The aim of this advice was to draw attention to the smokers, habit, and advice to quit is given that lasts no more than 3 min. The main effect of this advice would be to motivate attempts at quitting, rather than to increase cessation rates.<sup>28</sup>

#### Brief advice

Brief advice for the patient to stop smoking may last for around 10 min.

A five-step program recommended by the Agency for Health Care Research and Quality, which uses the five A's -<sup>28</sup>

1. Ask - Identify patients tobacco use status
2. Advise - On association between oral disease and smoking and the benefits of cessation.
3. Assess - Patients interest and readiness to participate in tobacco cessation programs
4. Assist - Use appropriate technique to assist patient in tobacco cessation
5. Arrange - Follow-up contacts with the patient

#### ABC model of smoking cessation

A new simple model - ABC - has recently been integrated into the New Zealand Smoking Cessation Guidelines.

Ask about smoking status;

Brief advice to stop smoking to all smokers

Cessation support for those who wish to stop smoking.<sup>28</sup>

#### Brief intervention

Advice to quit smoking should be given that takes between 5 and 10 min to deliver. These may include one or more of the following.<sup>28</sup>

- Simple opportunistic advice to stop.
- An assessment of the patient's commitment to quit.
- An offer of pharmacotherapy and/ or behavioural support to quit.
- Provision of self-help materials and referral for more intensive support such that delivered by the National Health Service. Referral can include referral oneself if suitably trained.

Table 3: First Line Pharmacotherapies For Smoking Cessation<sup>4</sup>

Pharmacotherapy	Precaution/ Contraindications	Side effects	Dosage	Duration
Bupropion sustained release (It is an antidepressant that act on dopaminergic pathway in CNS) <sup>8</sup>	History of seizure History of eating disorder	Insomnia, dry mouth	150 mg every morning for 3 days, then 150 mg twice daily (begin treatment 1 to 2 weeks precessation)	7 to 12 weeks Maintenance upto 6 months
Nicotine gum	Temporo-mandibular disorders exacerbated by chewing gum	Mouth soreness, dyspepsia	1 to 24 cigs/day-2mg gum (upto 24 pieces/day) 25+ cigs/day-4mg gum (upto 24 pieces/day)	Upto 12 weeks
Nicotine inhaler		Local irritation of mouth and throat	6 to 16 cartridges/day	Upto 6 months
Nicotine patch		Local skin reaction, insomnia	22mg/24 hours 14mg/24 hours 7 mg/24 hours 15mg/16 hours	4 weeks Then 2 weeks Then 2 weeks 8 weeks
Nicotine lozenge		Heartburn, mouth soreness	2 mg lozenge (if 1 <sup>st</sup> cigarette is 30 min. or more after waiting) 4 mg lozenge (if 1 <sup>st</sup> cigarette is 30 min. or less after waking) No more than 20 lozenges per day	Weeks 1 to 6: 1 lozenge every 1 to 2 hours Weeks 7 to 9: 1 lozenge every 2 to 4 hours Weeks 10 to 12: 1 lozenge every 4 to 8 hours

## CONCLUSION

Tobacco smoking has widespread systemic effects, many of which may provide mechanisms for the increased susceptibility to periodontitis and the poorer response to treatment. As an environmental factor, smoking interacts with the host and the bacterial challenge. The host genetic and environmental interaction is of the utmost importance. As knowledge of the genetic susceptibility to periodontitis increases, this will provide further opportunities to explore this relationship with tobacco smoking. It is quite possible that many of the pathogenic mechanisms involved in tissue degradation in periodontitis in tobacco smokers could be quite different from those involved in non-smokers.

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### How to cite this article:

Grover Harpreet Singh, Bhardwaj Amit, Singh Yaswin. Smoking and periodontal disease *J Pharm Sci Innov*. 2013; 2(2): 7-13.