

**Tobacco - A slow poison - A Review**

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**Abstract**

Periodontal disease afflicts many people in some unspecified time in the future during existence and is a chief motive of tooth loss in adults. The distributions of Periodontitis (inflammation of the periodontium) within the population suggest that a subset of individuals is exceedingly at risk of this infection, even as closing majority exhibits various ranges of resistance and slight susceptibility. Contemporary data imply that dental plaque is the purpose of periodontitis and cigarette smoking is a recognized hazard element, which adversely affects periodontal fitness. There’s evidence that cigarette smoking exerts both systemic and nearby results, though research had proven an affiliation between the 2, specific mechanisms underlying the consequences of smoking on periodontal reputation and wound recovery continue to be unresolved. Tobacco is used as a psychoactive substance. Tobacco smoke anticipated to incorporate over 4000

compounds lots of which can be pharmacologically active, toxic, mutagen and carcinogenic. Nicotine is one of the most studied additives of tobacco products and the maximum pharmacologically lively compound in tobacco smoke. it has been proven to have numerous temper altering effects on its purchasers. Nicotine is a poisonous alkaloid determined in tobacco smoke and can input the body via absorption thru the oral mucosa and skin or inhalation via the lungs. Nicotine is likewise relatively addictive. Only 2.5 percent of the people who smoke who try and end smoking are successful.

**Keywords:** Periodontal disease, Tobacco, Lungs.

**Introduction**

Periodontal disease afflicts many individuals at some point during life and is a major cause of tooth loss in adults. The distribution of Periodontitis (inflammation of the periodontium) in the population suggest that a subset of individuals is highly susceptible to this infection, while

remaining majority exhibit varying degrees of resistance and moderate susceptibility. Current data indicate that dental plaque is the cause of periodontitis and cigarette smoking is a known risk factor, which adversely influences periodontal health. There is evidence that cigarette smoking exerts both systemic and local effects. Though studies had shown an association between the two, precise mechanisms underlying the effects of smoking on periodontal status and wound healing remain unresolved.

The disease is initially confined to the gingival and is called chronic marginal gingivitis; later the supporting tissues are involved, and are called marginal periodontitis. Gingivitis is a problem frequently occurring in young adults, which often gets unnoticed.<sup>1</sup>

Periodontitis or inflammation of the periodontium result from the extension of the inflammatory process initiated in the gingival to the supporting periodontal tissues in which case there is bone damage and loosening and eventual loss of teeth. The Periodontium is a connective tissue organ, covered by epithelium that attaches the teeth to the bones of the jaws and provides a continually adapting apparatus for support of teeth during function. The periodontium comprise of four connective tissues. They are the cementum, alveolar bone, periodontal ligament and the gingiva. The gingival is a part of the oral mucosa that covers the alveolar process of the jaws and surrounds the neck of the tooth. The periodontal ligament is the connective tissue structure that surrounds the root and connects it to the bone. The cementum is a calcified tissue that forms the outer covering of the anatomical root of the teeth. The alveolar process is the bone that forms and supports the tooth sockets.

#### **Constituents of tobacco**

Tobacco is used as a psychoactive substance. Tobacco smoke estimated to contain over 4000 compounds many

of which are pharmacologically active, toxic, mutagen 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>3</sup> Tobacco smoke consists of a gaseous phase which is composed of carbon monoxide, nitrogen, oxygen, carbon dioxide; tar as well as particulate phase contains nicotine, water and polycyclic aromatic hydrocarbons.<sup>4</sup>

#### **Nicotine**

Its a addictive substance which release dopamine – a chemical in the brain that is associated with the feeling of pleasure.<sup>5</sup> Nicotine is one of the most studied components of tobacco products and the most pharmacologically active compound in tobacco smoke. It has been shown to have various mood altering effects on its consumers. Nicotine is a poisonous alkaloid found in tobacco smoke and can enter the body by absorption through the oral mucosa and skin or inhalation via the lungs. Nicotine is also highly addictive. Only 2.5 percent of the smokers who attempt to quit smoking are successful.<sup>6</sup>

#### **Tar**

Sticky brown substance which stain teeth, finger and lung tissue.

#### **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%. Oxygen level reduced due to carbon monoxide which effect foetus, low weight of babies born to women who smoke. The baby in the womb cannot grow normally due to lack of oxygen.<sup>3</sup>

#### **Nitrogen oxide**

Damage the respiratory airways. When smokers release nitrogen oxide in air they cause many effect to environment.

Breathing high level of nitrogen oxide can cause swelling of tissue in the throat and upper respiratory tract, reduced oxygenation of body tissue. Blood vessels of smokers are reduced.

### **Hydrogen cyanide**

Hydrogen cyanide is poisonous gases which are formed from the combustion of the protein and nitrate compounds existed in tobacco.

They effect neurological, respiratory, cardiovascular and thyroid. They are not carcinogen but effect other health problem. HCN is a poisonous gas which lead to problem of central nervous system. These problems include:- Muscle weakness, headache, dizziness, vomiting.

### **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>3</sup>

7. Metals – Thirty metals have been detected in tobacco smoke including nickel, arsenic, cadmium, chromium and lead.<sup>3</sup>

### **Oral and systemic effects of tobacco**

Tobacco is the most important etiological factor for oral diseases including oral cancer, oral mucosal lesions and periodontal disease. Tobacco affects both local and systemic either it is used in the form of chewing and smoking.

### **Effects of smokeless tobacco (chewers)**

Chewers or person who takes tobacco in the form of snuff and ghutka. Oral effect of Smokeless tobacco is typically seen on the mucosal surface where the product is placed, as well as periodontium. The affected site or lesion can be white or yellow- brown colour and it is known as leukoplakia. Smokeless tobacco contains high levels of sodium which may contribute to elevated blood pressure and heart rate. The most consistently reported periodontal

effects of Smokeless tobacco (Chewers) are mucosal lesions that may also affect the gingival, gingival recession and various form of periodontal diseases.

Smokeless tobacco which provides nicotine along with other substance that is known to cause cancer. While it is not inhaling directly to lungs, it is still being absorbed through the lining of mouth and circulating through bloodstream to all parts of the body, and cause bacteremia and endotoxemia which is responsible for damage of the vascular endothelial integrity, platelet function and blood coagulation. Use of smokeless tobacco has linked to oral cancer, esophageal cancer, and pancreatic cancer. It can also cause heart disease, oral lesion (leukoplakia) and gum disease (periodontitis).

### **Effect of smokers**

For many years, smoking has been linked with lung disease, cancer, cardiovascular disease and poor pregnancy out comes, such as miscarriage and low birth weight.<sup>7</sup> Over the past decades, it has also been recognized that smoking is associated with periodontal disease. As early as the 1940s, Pindborg noted that acute necrotizing ulcerative was associated with smoking in the Danish Royal Marines.<sup>8</sup>

### **Tobacco as a risk factor for periodontal disease**

(Effect of tobacco with periodontal disease)

An individuals' periodontal health has been identified to be affected by tobacco chewing and smoking. Tobacco intake is related with increasing rate of multiple health complications which also includes loss of periodontal bone and the loss of the periodontal attachment and bone formation. In addition, tobacco smoking and chewing has been connected with the masking of the gingival swelling symptoms. Gingivitis is a disease that has been related with smoking for a long time.<sup>9</sup> Studies have revealed that increased rate of tobacco smoking and gingival bleeding on probing are the early symptoms of gingivitis, a subject

that is not dependent on significant signs of gingivitis and is now broadly applied towards identifying lesions in periodontal complications.<sup>10</sup> Gingival bleeding is higher in non-smokers than in smokers.<sup>11</sup> This is attributed to the fact that there is vasoconstriction of the gingival cells or there is an occurrence of the keratinisation of the gingivae among the smokers.<sup>12</sup>

Periodontal diseases are a group of conditions affecting the supporting structures of the dentition. Progression and severity of the disease depends on the complex interactions between the host defence mechanism and risk factor such as microbial growth, genetic and environmental factor and oral hygiene as well as age, sex and race. Tobacco smoking is a significant risk factor for periodontal disease.<sup>13</sup> Tobacco smoking is an addictive habit first introduced into Europe. Smokers are three times more likely to get acute periodontitis than non-smokers.<sup>14</sup>

Periodontitis start from gingivitis (gum disease) where specific bacteria present in plaque play important role to cause gingivitis. Then gingivitis or bacteria resulting in progressive destruction of periodontal ligament and alveolar bone with pocket formation, and in periodontal pocket anaerobic bacteria grow and enter in blood stream and cause bacteraemia and endotoxemia.

#### **Prevalence of plaque development in smokers**

Cigarette smoking alone does not cause periodontitis disease. Periodontal disease is caused by bacterial plaque that gets under the gingival tissue and initiates bone loss around the teeth. Smokers show 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 which play important role to cause periodontal disease.

#### **Prevalence of probing depth in smokers**

On the basis of CPITN

The difference between smokers and non-smokers in probing depth could also be an explanation as smokers have deeper periodontal pockets than non-smokers, which might confound the effect of smoking on periodontal pathogens (Kigure et al., 1995) Stoltenberg et al.<sup>24</sup> reported that the odds ratio for having a mean probing depth  $\geq 3.5$  mm was 5.3 times greater in smokers. Stoltenberg et al.<sup>24</sup> found that smoking was the strongest risk indicator for increased pocket depth.

#### **Early onset of inflammation in smokers**

Smokers also had an earlier onset of clinically visible inflammation compared to non-smokers, which was attributed to the early pathogenic colonization, leading to sustained pathogen enrichment with periodontal pathogens. This is consistent with other findings in smokers, indicating that gingivitis is preceded by a decrease in the abundance of early colonizers, such as the genera *Streptococcus* and *Veillonella*, and an increase in the abundance of periodontopathogens, such as the genera *Treponema* and *Selenomonas* (Matthews et al., 2013; Peruzzo et al., 2016).<sup>15</sup>

#### **Smoking and microorganism**

Haffajee and Socransky<sup>16</sup> investigated the relationship between cigarette smoking and subgingival microbiota using checkerboard DNA hybridization. They concluded that the major difference between smokers and non-smokers was in the prevalence of species (i.e., periodontal pathogens colonized a larger proportion of sites) rather than counts or proportions. Kamma and colleagues<sup>17</sup> investigated the effects of smoking in subjects with aggressive periodontitis. They reported that smokers harboured greater number of strict anaerobes, especially *Porphyromonas gingivalis*, *Bacteroides forsythus*

(*Tanarella forsythus*) and *Eikenella corrodens* compared to non-smoking subjects.<sup>2</sup>

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 forsythus* than non-smokers.<sup>18</sup> Adjusting for disease severity, *Porphyromonas gingivalis* was also more likely to subgingivally infect smokers than non-smokers.<sup>19</sup>

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 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*.<sup>20,21</sup>

Many authors 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 forsythus*, *Actinobacillus actinomyces comitans* and *T.denticola*.<sup>22, 23</sup>

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