

Cigarette Smoking – A Risk Factor

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INTRODUCTION

Periodontitis, one of the most common diseases among adults, is caused by oral bacteria and leads to irreversible bone loss and eventual tooth loss.¹ Although the direct cause for periodontitis is oral bacterial infection, its progression and severity depends upon a number of genetic and environmental factors. Both environmental and genetic factors contribute to individual variations in the risk for periodontal disease.² Several studies were performed to elucidate the inter-individual variations of severity and progression of periodontitis.³⁻⁶ Several epidemiological studies in different population demonstrate a relationship between smoking and periodontal disease.^{7,8}

Tobacco smoking is a risk factor that acts independently and may also be in a synergistic way with other risk factors. Tobacco smoking may affect periodontal tissues directly causing a local irritation and indirectly, as a systematic risk with a negative effect on microcirculation in periodontal tissues and the alveolar bone mass. Smoking is detrimental to periodontal health as it worsens oral hygiene status and depresses the host defense posture.

Smoking increases the risk of periodontal disease between two and six times. For example, Calsina et al, 2002 in a casecontrol study showed that smokers had 2.7 times and former smokers 2.3 times greater probabilities to have established periodontal disease compared with nonsmokers.⁹

Linder and Mullally (1994)¹⁰ found the odds ratio for periodontal disease to be as high as 14.1 in young smokers where as Hyman examined data from the National health and nutrition examination survey III and reported an odds ratio of 18.6 for ≥ 3 mm attachment loss among 20 - 49 smokers compared with nonsmokers. Among those over 50 years of age, the odds ratio increased to 25.6 for loss of attachment ≥ 4 mm.¹¹

Evidence of risk factor status is strengthened by the ability to demonstrate a dose- response, and years of exposure to tobacco products is a statistically significant risk factor for periodontal disease.^{12,13} It remains challenging, however, to determine the strength of smoking as a risk factor owing to inherent problems in measuring accurately a subject's exposure to tobacco.¹⁴⁻¹⁶

In general, therefore, there is a substantial body of evidence to support the observation that the more a patient smokes, the greater the degree of periodontal disease.

It is also important to note that although nonsmokers universally respond better to periodontal treatment than do smokers, there is nevertheless substantial evidence of clinical improvement in smokers after treatment, indicating that smoking as a risk factor will compromise rather than prevent tissue healing.

Besides acting as a risk factor for disease, tobacco smoking also seems to decrease the functional capacity, for instance bone mineral content, lung capacity, body mass and the concentration of several blood components. These findings indicate that tobacco smoking has a negative influence on functional state similar to that of ageing itself.

SYSTEMIC EFFECTS OF SMOKING

Forty years have passed since the landmark U.S. Surgeon general's reports in 1964 warned that smoking played a causative role in lung cancer and was associated with cardiovascular diseases since then a number of smoking related health effects have grown.

Here are some of the ways that smoking tobacco affects our body:

1. **Brain:** Nicotine activates the "pleasure centers" in the brain, which causes pleasure and alertness. Initially nicotine stimulates the brain and regulates well-being, mood and memory. About 20-40 minutes after smoking a cigarette, a person begins to experience withdrawal symptoms. When experiencing these withdrawal symptoms, a person's mood changes, they become irritable, anxious and uncomfortable. These feelings often make people to crave more cigarettes in order to combat the symptoms of withdrawal thus the addictive nature of nicotine.
2. **Throat:** Smoking causes cancer of the larynx and esophagus. It also irritates the membranes of the throat causing it to become sore.
3. **Heart:** Nicotine causes a person's heart rate and blood pressure to increase. Carbon monoxide from the cigarette smoke decreases the flow of oxygen to the heart, which increases a person's risk of heart attacks and strokes. Peripheral vascular diseases and aortic aneurysm have also been established. Smoking also causes the heart to weaken, making it harder to pump blood through the body, which leads to death.
4. **Liver:** Smoking is linked to cirrhosis of the liver.
5. **Adrenal Glands:** Smoking causes the adrenal glands to speed up the production of adrenaline, which causes the heart and blood pressure to increase.
6. **Vertebrae:** There is an increased risk of vertebral cancer among smokers.
7. **Reproductive Systems:** Smoking reduces sex drive and increases risk of impotence in men. In women, there is an increased risk of cervical cancer. Women smokers are also less fertile, experience more miscarriages and pregnancy complications. Smoking while pregnant may interfere with the baby's growth and development. Women smokers on average reach menopause 1-2 years earlier than normal.
8. **Bones:** There is an increased risk of early onset osteoporosis.
9. **Bladder:** Smoking causes bladder cancer.
10. **Blood Vessels:** Nicotine causes blood vessels to constrict, which increases a person's blood pressure and risk of heart attacks.
11. **Kidneys:** Smoking reduces the kidney's ability to process fluids and waste, which interferes with urination and can lead to cancer.
12. **Stomach and Duodenum:** Smoking causes ulcers to develop, which can be very painful.
13. **Lungs:** Smoking progressively limits the amount of air flow into and out of the lungs. This damages and destroys the tiny air sacs in the lungs, which reduces the ability to bring oxygen into the body and remove carbon dioxide. Smoking also causes the bronchial tubes to become inflamed, thickened and mucus-filled, resulting in smaller air passages and emphysema. Tar and other chemicals from cigarettes settle in the bronchial tubes causing lung cancer. Many components of cigarette smoke have also been characterized as ciliotoxic materials that irritate the lining of respiratory system resulting in increased bronchial mucus secretion and chronic diseases in pulmonary and mucociliary function.

Other changes:

Generalized redness of soft palate and fauces, burns and keratotic patches on lips the site particularly where it is held. Frictional keratosis, coated tongue, discoloration of teeth due to tarry deposits.

SMOKING AND SYSTEMIC HEALTH STATUS

The combination of smoking with other systemic factors further enhances the risk of periodontal destruction.

- In the Erie County study, diabetics were approximately twice as likely to exhibit periodontal attachment loss compared to non-diabetics and the combination of diabetes and heavy smoking in an individual over the age of 45 years who harbored *Porphyromonas gingivalis* or *Tannerella forsythensis* (formerly *Bacteroides forsythus*) resulted in an odds ratio of attachment loss that was 30 times that of person lacking these risk factors.
- Smoking also increases the risk of attachment and/or bone loss in postmenopausal women and AIDS and HIV seropositive patients.
- In a retrospective private practice study, heavy smoking and interleukin (IL) - 1 genotype individually increased the risk of tooth loss over a 14 year period by 2.9 and 2.7, respectively, but the combination of these two factors increased the risk of tooth loss by 7.7.
- Because one of the risk factors for periodontitis is tobacco use, susceptibility to this disease may be linked to polymorphism in genes coding for enzymes metabolizing tobacco-derived substances.¹⁷

Cytochrome P450 (CYP) enzymes, CYP1A1 and CYP2E1, are considered to play important roles in the activation of xenobiotics, especially tobacco derived substances such as polycyclic aromatic hydrocarbons¹⁸ and nitrosamines.¹⁹ On the other hand, Glutathione S-transferase (GST) M1 and N-acetyltransferase (NAT1 and NAT2) are involved in the detoxification of these activated metabolites. Polymorphism of CYP1A1 and CYP2E1 are associated with enhanced catalytic activities of these enzymes. In addition, the null GSTM1 genotype and the mutation in the NAT gene result in the inability to efficiently detoxify xenobiotics. Recently, it has been reported that the slow acetylator genotype of NAT2 is associated with a higher risk of periodontitis, particularly in smokers^{20,21}. The genetic polymorphisms of these enzymes have been linked with an increased risk for tobacco-related disease such as oral cavity cancer.²²⁻²⁴

Collectively, these studies suggest that smoking interacts with various systemic conditions; the end results are not purely additive, but can be synergistic, resulting in greater disease severity than either factor alone.

CIGARETTE SMOKING AND TOOTH LOSS

Smokers have fewer teeth, higher prevalence of edentulism and greater incidence of tooth loss than nonsmokers. Longitudinal studies have demonstrated that young individuals smoking more than 15 cigarettes per day showed the highest risk for tooth loss. Periodontal maintenance patients who smoke are reported to be twice as likely to lose teeth over a 5 year period as compared with periodontal maintenance patients who do not smoke.

Although no direct relationship has been established between tooth loss and cigarette smoking. Strong associations have been established between cigarette smoking on one side and cervical caries, coronal caries and periodontitis on the other.

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