



The Role of Smoking in Periodontal Disease, Current Research

Commander R. Scott Thompson, DC, USN, and Commander Matthew B. Miller, DC, USN

Purpose

Dentists have been aware of the increased risk of periodontal disease in cigarette smokers for well over 30 years. Research in the last 15 years has focused primarily on the effects of smoking on the host immune response and the resulting pathogenesis of periodontal attachment loss. The purpose of this clinical update is to report on these findings of the role of tobacco smoking in periodontitis.

Tobacco is a risk factor for periodontitis.

Tobacco is known to increase the risk of developing periodontitis. In a literature review of longitudinal studies by Heitz-Mayfield, cigarette smokers were shown to be at higher risk for progression of the disease based on increased number of periodontally diseased sites, have more loss of alveolar bone and attachment, and have higher rates of tooth loss compared to non-smokers.¹ Thomson reported on a group of 810 thirty-two year olds he had followed since age 16.² Long-term smokers had higher odds of periodontitis compared with never-smokers. However, those who quit were not statistically different from never-smokers in any category. This study demonstrated that long-term smoking is detrimental to periodontal health while quitting tobacco use can successfully reduce risk of periodontitis. Okamoto conducted a 4 year longitudinal study of 1332 Japanese males who were periodontitis-free at baseline.³ A dose-response association was found between smoking and tooth loss with those smoking >20 cigarettes per day showing significantly higher incidence of periodontitis for all age groups. Again, there was no significant difference between former smokers and non-smokers. Environmental, or second-hand, tobacco smoke has also been shown to increase the incidence and progression of periodontitis. Arbes examined data from the Third National Health and Nutrition Examination Survey (NHANES III), assessing 6611 persons 18 years or older and found exposure to environmental tobacco smoke increased the odds of having periodontitis by 1.6 x.⁴

Mechanisms: Microbial complexes in smokers

The oral biofilm in smokers tends to favor the colonization by periodontal pathogens. Gomes used real-time polymerase chain reaction to determine that periodontal pathogens *M. micros* and *D. pneumosintes* were significantly higher in smokers.⁵ Kumar found that periodontal pathogens colonize the early oral biofilms in smokers, suggesting that smoking favors early acquisition and colonization of pathogenic bacteria.⁶ Van der Velden, et al., cultured periodontal pathogens from periodontitis patients before and after periodontal therapy and noted that smokers remained culture positive for periodontal pathogens even after treatment compared to non-smokers.⁷

Mechanisms: Immunologic changes

Tobacco has been shown to cause changes in the immunologic response to bacteria that favor the progression of periodontitis and loss of attachment. Graswinckel showed that subjects with periodontitis had higher levels of IgG1 and IgG2 compared to healthy subjects.⁸ However, periodontitis patients who were smokers did

not show this increase in antibodies despite the increase of several periodontal pathogens. This smoking cohort also demonstrated more severe bone loss. The conclusion of the study was that smoker periodontitis patients fail to mount an adequate antibody response to periodontal pathogens and this might explain the increased disease severity found in this population.

Smoke exposure promotes an accumulation of neutrophils in the tissue, impairs phagocytosis, and stimulates the release of destructive substances such as hydrogen peroxide and superoxide. The neutrophils themselves do not effectively remove bacteria; rather they continuously release enzymes and inflammatory mediators leading to localized destruction. Smoking tips the balance from the protective functions of neutrophils to one that is unchecked and destructive.⁹

Mechanisms: Cytokine profile differences

Smokers have been shown to possess cytokine profiles that favor the activation of osteoclasts and bone resorption. Receptor activator of nuclear factor kappa- β (RANK) is a receptor found on the surface of osteoclasts. Combination of RANK with RANK ligand (RANKL), found on various tissues including osteoblasts, activates osteoclasts and initiates bone resorption. However, combination of RANK with osteoprotegerin (OPG) serves to block this receptor and inhibits activation of osteoclasts. Buduneli determined that smokers demonstrate higher RANKL and lower OPG concentrations than non-smokers.¹⁰ This increased RANKL:OPG ratio suggests an increased osteoclast activity which could be a mechanism for the increased bone loss seen in smokers.

Most studies seem to report that periodontitis smokers have lower levels of crevicular pro-inflammatory cytokines such as interleukin-1 α , interleukin-6, and interleukin-12 compared to non-smokers with periodontitis.¹¹ However, serum cytokine levels in cases such as tumor necrosis factor α and interleukin-1 β are higher in smokers. As of this time, the findings within the literature are inconclusive regarding the overall net effect of these differences in cytokine levels.

Treatment outcomes

Tobacco has been clearly shown to adversely affect healing outcomes of periodontal therapy.⁹ Current studies show that cigarette smokers in long term periodontal maintenance experience less success maintaining treated teeth. Miller proposed a new evidence-based scoring index to determine the prognosis of periodontally involved molars.¹² In a retrospective study of 800 recall patients receiving periodontal maintenance for over 15 years, smoking was the greatest negative prognostic factor for tooth loss. Based on Miller's model, smokers with periodontal disease had a 264% greater chance of losing teeth compared to non-smokers. A 2010 study by Chambrone examined the effect of risk factors on tooth loss during long-term periodontal maintenance.¹³ Although long-term maintenance decreased the overall rate of tooth loss for everyone, smokers still demonstrated a relative increased risk of losing teeth.

Airila-Mansson published findings in 2005 on a 17 year longitudinal study of bone height changes in individuals with periodontal disease.¹⁴ After 17 years, current smokers had more marginal bone loss com-

pared with never-smokers and former smokers with periodontitis, especially on the lower anterior teeth.

Smoking affects the success of mucogingival surgery. Early followed 17 patients divided into cohorts of smokers and non-smokers for 6 months after connective tissue graft procedures to correct recession defects.¹⁵ Smokers experienced a lower percentage of root coverage and fewer sites yielding 100% coverage.

Guided tissue regeneration efforts to treat class II furcation defects are hampered in smokers. Bowers observed that smokers had a significantly higher proportion of residual class II defects than non-smokers after treatment.¹⁶ A 2012 systematic review clearly indicated that smoking has a negative effect on periodontal bone regeneration, showing that smoking resulted in smaller bone gains in the treatment of intrabony defects.¹⁷

Periodontal tissues associated with dental implants have been shown to experience more problems in smokers. Roos-Jansaker reported on 218 patients followed for 9-14 years after dental implant placement.¹⁸ The long-term study determined that smokers had more peri-implant mucositis, bone loss, and peri-implantitis.

The effects of smoking are reversible. Smokers who successfully quit are found to be similar to non-smokers with respect to incidence of continued attachment loss and response to treatment. Therefore, dentists should encourage cessation and educate our patients. According to a 2005 study by Preshaw, patients with a desire to quit were able to do so 20% of the time.¹⁹ Navy dentists can improve the periodontal health of many service members by taking an active role in tobacco cessation. Instructions on comprehensive tobacco control for Navy healthcare providers are delineated in BUMEDINST 6200.12A.

Conclusion

Smokers are more likely to have higher rates of periodontitis and a more severe course of the disease. Modern research clearly outlines tobacco's role in altering the host immune response to bacteria. Local effects of cigarette smoking's toxic products and heat cause damage directly to tissues. However, smoking also results in systemic effects such as:

- Impaired and altered neutrophil activity
- Increased/earlier colonization of periopathogens
- Decreased levels of immunoglobulins
- Disruptive balance of reactive oxygen species
- Increased release of proteases such as collagenase and elastase
- Impaired fibroblast attachment and decreased collagen synthesis
- Altered release of cytokines
- Increased RANKL/OPG ratio.

Researchers continue to identify and define the exact mechanisms by which tobacco contributes to the progression of periodontitis or impairs the response to treatment. The good news is that these effects are reversible. Ultimately, the best course for our patients is to quit. As dentists we are uniquely positioned to facilitate tobacco cessation because of our regular periodic contact with our patients. Therefore, we should always be on the lookout to help our patients stop tobacco use and improve their oral and overall systemic health.

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Commander Thompson is a resident in the Periodontics Department. Commander Miller is a faculty member in the Periodontics Department at the Naval Postgraduate Dental School.

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