

Nitzan D, Mamlider A, Levin L, Schwartz-Arad D. Impact of smoking on marginal bone loss. *Int J Oral Maxillofac Implants* 2005; 20: 605-9. 35 (Refs)

Purpose: The purpose of the article was to compare marginal implant bone loss (MBL), survival, and radiographic evidence of success of dental implants among smokers and non-smokers.

Materials and Methods: The study was based on a consecutive cohort of patients who received implants between 1995 and 1998 and consisted of 161 patients (mean age 57 years) with a total of 646 implants, of which 391(61%) were immediately placed implants. There were 102 nonsmokers (375 implants) and 59 smokers (271 implants). Mean follow-up was 42.9 months for smokers and 48.4 months for nonsmokers. The patients were divided into three groups: nonsmokers, moderate smokers (≤ 10 cigarettes per day), and heavy smokers (>10 /day). Smokers were also divided into 2 groups according to tobacco consumption; less than and more than 16 pack-years. Implants were placed by one surgeon and all radiographs were analyzed by 2 examiners. MBL was measured on radiographs using the implant threads as an internal standard. Bone level at the time of implant exposure was compared with that at the most recent follow-up. The number of threads unsupported by bone on both the mesial and distal sides of each implant was counted, and the average number was used to calculate bone loss. The implants were studied by their placement in 1 of 5 areas: anterior mandible, posterior mandible, anterior maxilla (between the canines), middle maxilla (premolar sites), and posterior maxilla (molar sites). Radiographic evidence of implant success was evaluated using a modification of the Albrektsson criteria and an implant was considered successful if bone loss apical to the implant neck was <0.3 mm per year (starting from the first year).

Findings: The smokers had an average of 4.6 implants while nonsmokers had 3.7 implants per person. No difference in MBL was noted in regard to implant system or gender. Location: The average MBL was greater for smokers than nonsmokers (0.153 vs. 0.047). Smoking had a greater effect on MBL in the maxilla than the mandible. In the maxilla, heavy smokers experienced the greater bone loss (0.1897mm), followed by moderate smokers (0.123mm) and nonsmokers (0.046mm). In the mandible, there was no distinction between heavy and moderate smokers; both had greater MBL than nonsmokers. Survival and Success Rates: Of the 646 implants, 8 (1.2%) failed (5 at time of implant exposure and 3 after loading). The 7-year cumulative survival rate was 99.5%. The overall radiographic success rate for all implants was 93.2%. Nonsmokers had a higher radiographic success rate than smokers (97.1% vs. 87.8%).

Conclusions: Greater MBL was found in smokers demonstrating a relationship between MBL and smoking habits. The maxilla was more susceptible to the effects of smoking.

Palmer RM, Wilson RF, Hasan AS, Scott DA. Mechanisms of action of environmental factors – tobacco smoking. J Clin Periodontol 2005; 32(suppl. 6): 180-95. 179 (Refs)

Purpose: The purpose of the article was to review the potential biological mechanisms underlying the effects of tobacco smoking on periodontitis.

Materials and Methods: Literature review.

Findings: Much of the work in the periodontal and medical literature has concentrated on the effects of nicotine, the primary psychoactive component. However, nicotine may be unfairly blamed for the effects of the thousands of different compounds found in the tobacco smoke, many of which are directly noxious to living cells. It is also very important to appreciate that most of the harmful effects of tobacco products will result from systemic exposure through absorption in the lungs rather than topical absorption in the oral cavity. A regular smoker exposes themselves to these compounds many times a day for several minutes at a time. No other drug is administered so frequently or over such a time period. The detrimental effects on the periodontium are derived from long-term chronic exposure.

Effect of Smoking on Plaque: The rate of plaque formation and composition of the oral flora is similar between smokers and non-smokers. Differences have been found when the subgingival microflora was examined. Several studies suggest a trend for smokers to harbor more or greater numbers of potential periodontal pathogens than non-smokers without increasing the amount of plaque.

Effect of Smoking on the Periodontal Tissues: Smoking has a long-term chronic effect impairing the vasculature of the periodontium rather than a single vasoconstrictive effect following a smoking episode. The suppressive effect on the vasculature can be observed through less gingival redness, lower BOP and fewer vessels visible clinically and histologically. This may have relevance to the healing response with impairment of revascularization.

Smoking and Neutrophil Function: Smoking induces a significant systemic neutrophilia, but neutrophil transmigration across the periodontal microvasculature is impeded. The suppression of neutrophil cell spreading, chemokinesis, chemotaxis, and phagocytosis has been described. Neutrophils are critical cells in the maintenance of periodontal health because of their multifaceted roles in the control of plaque bacteria, but they may also contribute to the progression of periodontitis in chronic inflammatory responses. While there are conflicting data, it is clear that tobacco smoking affects multiple functions of neutrophils and may shift the net balance of neutrophil activities into the more destructive direction.

Smoking and Lymphocyte Function: There are inconsistencies and variations in findings reflecting the complex relationship between smoking, race, periodontal diagnosis, and age. There has been very little periodontal-specific research in this area. Additionally, in many periodontal diseases, the important antigens are not known and it is difficult to assess the effect of smoking on antigen-specific responses that are relevant to periodontitis.

Evidence from Studies on GCF: It would seem logical to expect that factors that are associated with tissue destruction should be higher in smokers. This is not the case for many of these factors when assayed in the GCF. For the most part, research has demonstrated that there are lower levels of cytokines, enzymes, and possibly PMN's. This

correlates with the lower levels of inflammation seen clinically and within the tissues. The GCF could be viewed as an end product of the destructive process, and lower levels of factors may simply indicate higher levels of activity within the tissue or effects on gingival fluid flow dynamics.

Smoking and Fibroblast Function: Smoking has a detrimental effect on clinical healing of non-surgical and surgical treatment modalities. The biological basis for this is undoubtedly multifactorial as smoking affects the vasculature and revascularization, the inflammatory response and fibroblast function. It is not possible to estimate the in vivo potential of these effects based on the data from these in vitro experiments, which usually test high levels of nicotine and do not take the other noxious compounds into account. Nevertheless, it is likely that smoke products will affect fibroblast recruitment and adhesion to root surfaces.

Conclusions: Tobacco smoking is the most important environmental risk factor in periodontitis. Smoking has widespread systemic effects, many of which may provide mechanisms for the increased susceptibility to periodontitis and the poorer response to treatment.