

COLIN S. RICHMAN, D.M.D., P.C.  
D.M.D., B.D.S., L.D.S., R.C.S., H.D.D.  
PERIODONTICS, ORAL MEDICINE  
AND DENTAL IMPLANTS  
SUITE 104  
1305 HEMBREE ROAD,  
ROSWELL, GEORGIA 30076-3810  
TELEPHONE (770) 442-1010  
FAX: (770) 475-7658

DIPLOMATE, AMERICAN BOARD OF PERIODONTOLOGY

### **SMOKING: A REVIEW OF PERIODONTAL LITERATURE**

A review of the literature documenting the role of smoking in periodontal disease progression was published in the June Issue of the Compendium of Continuing Education. Twenty-nine references on the subject were cited. Periodontal destruction involves specific pathogens, breakdown in the physical defense mechanisms, and alterations in the immune response. In this literature review, the effects of smoking are related to these aspects of periodontal destruction.

First, high levels of anaerobic pathogens have been measured on the tongues of smokers, suggesting an anaerobic condition in the mouth. One of the bacteria identified was bacteroides, which is implicated in periodontal destruction.

Physical defense mechanisms are affected by both the nicotine and heat of smoking. The oral cavity temperature of smokers reaches 42 degrees Centigrade, resulting in alterations in the epithelial tissue. Nicotine causes vasoconstriction, reducing nutrition to the area, and accounting for fewer white blood cells reaching the area to fight infection. Reduced blood flow can last up to 3 hours after a single cigarette. Nicotine will initially stimulate saliva flow, followed by a decrease in secretions, which directly affects the microflora of the mouth. This alteration may also be responsible for the higher levels of calculus found in smokers. Nicotine metabolics remain in the saliva and crevicular fluid much longer than nicotine, continuing the active properties of nicotine. Nicotine has also been related to significant levels of bone loss.

Looking at the immune system, polymorphonuclear leukocytes (PMNs) are the first line of defense in an inflammatory response. Smokers have PMNs with reduced phagocytic capability, compared to nonsmokers. The effect on PMNs is even more pronounced when a cigarette is smoked one hour before testing. These effects will compromise the immune response to periodontal infection, as well as the healing following periodontal therapy.

The authors point out the need for researchers to take this information into consideration when conducting studies which include smokers. Plaque, calculus, bleeding and gingival indices will be altered by smoking. Perhaps, newer diagnostic tests will identify the effects of smoking on a cellular level, which will show clinicians the correlation of smoking to periodontal disease.

Akef, J., Weine, F., Weissman, D.: "The Role of Smoking in the Progression of Periodontal Diseases: A Literature Review". The Compendium of Continuing Education in Dentistry 13: 526, 1992.

P.S.: Similar effects associated with chewing tobacco or smokeless tobacco have also been noted and documented. In addition, local physical traumatic defects occur, especially associated with severe gingival recession and decay. These lesions usually occur adjacent to the wad of tobacco in the labial pouch.