

Tobacco smoking and its effect on the periodontium

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Mini review

Dentistry has long been aware of the effects of tobacco on the soft tissues of the oral cavity and the implications of this in clinical practice. Over the past 20 years, there has been an increasing awareness of the role of tobacco use on the prevalence and severity of periodontal diseases.¹ Periodontitis is the result of complex interrelationships between infectious agents and host factors. Environmental, acquired, and genetic risk factors modify the expression of disease and may, therefore, affect the onset or progression of periodontitis. Among the various life style risk factors, tobacco use has been considered to be most important in the progression of the periodontal disease. There has been increasing awareness of the role of tobacco use in the prevalence and severity of periodontal diseases and subsequent tooth loss.¹⁻⁴ Smoking appears to be one of the most significant risk factors in the development and progression of periodontal disease.^{5,6} An association between smoking and acute ulcerative gingivitis (ANUG) has been demonstrated.^{7,8} Results from the first United States National Health and Nutrition Examination Survey (NHANES I) has demonstrated that although current smokers had more plaque and periodontal destruction than former or never smokers.⁹ It has also been demonstrated that smokers have more deeper pockets,^{10,11} greater attachment loss,⁵⁻¹² and more alveolar bone loss.^{6,13} An overwhelming body of data from multiple cross-sectional and longitudinal studies conducted have demonstrated pocket depth and clinical attachment loss were more prevalent and severe in patients who smoke compared with non-smokers.¹⁴ A relationship has also been found between the prevalence of moderate to severe periodontal disease and number of cigarettes smoked per day¹⁻⁶ and to the number of years that the patient had smoked.⁷⁻¹⁵ Use of tobacco in non-smoking form (chewing form) has also shown to increase the incidence of periodontal diseases. Localized gingival recession can occur 25-30 times more frequently among smokeless tobacco users as compared to a normal individual of same age and gender. This attachment loss is more prevalent adjacent to the mandibular buccal areas where smokeless tobacco products are commonly placed¹⁶ several hypotheses have been postulated regarding mechanism of progression of periodontal disease in tobacco users. It has been shown that nicotine and other components in tobacco are produced causing local and systemic effects like decreased tissue perfusion and cellular proliferation due to cytotoxic and vasoactive substances in tobacco,^{17,18} impaired serum antibody response to periodontal pathogens,¹⁹ altered polymorph nuclear leukocyte function resulting in decreased chemotactic migration and phagocytic activity,²⁰ reduction in skeletal bone mineral content²¹ and interference with the fibroblast attachment.²² While smoking cessation does not reverse the past effects of smoking, there is abundant evidence to indicate the rate of bone and attachment loss slows after patients quit smoking and the severity of their disease is intermediate compared to current and non-smokers.²³ The periodontal status of former smokers ranks between that of never smokers and current smokers, which suggests that smoking causes some irreversible changes in the periodontium but that the deterioration does not continue after cessation. It is

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encouraging to note that former smokers respond to periodontal therapy in a manner similar to non-smokers.²⁴

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Conflicts of interest

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