We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

5

Effects of Tobacco Smoking on Chronic Periodontitis and Periodontal Treatment

Nurcan Buduneli
Department of Periodontology, School of Dentistry, Ege University, İzmir, Turkey

1. Introduction

Periodontitis is one of the most common oral diseases and is characterised by gingival inflammation and alveolar bone resorption (Savage et al. 2009). Periodontitis is a multifactorial irreversible and cumulative condition, initiated and propagated by bacteria and host factors (Kinane 2001). More than 500 different bacterial species are able to colonise the oral biofilm and up to 150 different species of bacteria are possible in any individual's subgingival plaque. There are two forms of periodontitis; chronic and aggressive periodontitis which differ from each other not only in clinical findings but also age of onset and rate of progression. Chronic periodontitis progresses more slowly compared to the aggressive forms of periodontitis and responds much better to periodontal treatment. According to a report by the World Health Organisation, severe chronic periodontitis leading to tooth loss was found in 5% to 15% of most populations worldwide (Armitage 2004). Hence, it can be considered among the prevalent and important global health problems in terms of quality of life.

The multifactorial nature of periodontitis is based on the complex interactions between microorganisms in the microbial dental plaque, namely dental biofilm, host response mechanisms and environmental factors. Smoking is well known to be the leading environmental factor that is closely related not only with the risk but also the prognosis of periodontitis. Indeed, the harmful effects of smoking on numerous organs have been well-documented for years now. Cigarette smoking is recognised as the major preventable cause of death in the United States (Centres for Disease Control 2002 (CDC 2002)). Smoking is the second strongest modifiable risk factor for periodontal disease after the first one which is the microbial dental plaque. Smokers are more likely to harbour a higher prevalence of potential periodontal pathogens, and smoking impairs various aspects of innate and acquired immune responses. Numerous molecules in the oral fluids namely; gingival crevicular fluid (GCF) and saliva, as well as molecules in blood circulation; serum or plasma have been investigated so far in an attempt to provide a sensitive and specific marker for periodontal tissue destruction (Buduneli & Kinane 2011). The aim of the present literature review is to provide an overview of the evidence for smoking as a risk factor for chronic periodontitis and to discuss the possible mechanisms of action and finally the negative effects of smoking on the outcomes of periodontal treatment in patients with chronic periodontitis.

Smokers are accepted to be more susceptible to advanced and aggressive forms of periodontitis than non-smokers (Haber et al. 1993, Calsina et al. 2002) and former smokers are at decreased disease risk than current smokers (Bostrom et al. 2001). Tobacco smoking modifies the periodontal response to microbial challenge (Barbour et al. 1997, Palmer et al. 2005). Although, smoker and non-smoker patients exhibit more or less the same periodontal pathogens (Preber et al. 1992, Buduneli et al. 2005a) smokers also tend to respond less favourably to periodontal treatment (Ah et al. 1994, Renvert et al. 1998). Smoking was suggested to influence host cytokine levels (Boström et al. 1999, Buduneli et al. 2005b, Buduneli et al. 2006). Furthermore, smoking was reported to reduce salivary osteoprotegerin concentrations in untreated and also treated chronic periodontitis patients (Buduneli et al. 2008). Chronic periodontitis has been associated with various systemic diseases and/or conditions such as cardiovascular diseases and preterm low birth weight. Over the last decade there have been a number of reviews that have considered the biological mechanisms underlying susceptibility to periodontitis in smokers (Barbour et al. 1997, Johnson & Hill 2004, Kinane & Chestnutt 2000, Mullally 2004, Palmer et al. 2005, Scott et al. 2001). Despite that a clear dose-response relationship between chronic periodontitis and smoking was reported (Martinez-Canut et al. 1995) the mechanisms by which smoking contributes to the pathogenesis of periodontitis are not yet clearly understood.

The fundamental mechanisms that lead to the development of chronic periodontitis are closely related to the dynamics of the host immune and inflammatory responses to periodontal pathogens present in the dental biofilm (Gemmell & Seymour 2004). The immune and inflammatory responses are critical to understanding the pathogenesis of periodontal diseases and they are orchestrated by a number of host-related factors, either intrinsic or induced (Taubman et al. 2005). Under normal circumstances, there is a balance between microbial virulence factors and host response. Tissue homeostasis is maintained as long as this balance is preserved. In periodontitis, this balance between the microbial virulence factors and host response is impaired in favour of microbial challenge. Smoking as an environmental factor has been suggested to interact with host cells and affect inflammatory responses to this microbial challenge (Palmer et al. 2005). It is also likely that the toxic components of tobacco smoke, mainly nicotine, may directly or indirectly deteriorate periodontal tissues. Cigarette smoking represents a risk factor for progression of periodontitis, the effect of which may be dose related. Heavy smokers should be considered as high-risk individuals for progression of periodontitis. The clinical implications for this are that smokers should be identified during patient examination and efforts should be made to modify this behavioural risk factor. Furthermore, smoking or molecules related to smoking such as blood cotinine induced by smoking should be considered as important risk markers of periodontal disease that are relevant to the assessment of prognosis (Calsina et al. 2002, Tang et al. 2009).

Smokers are almost four times more likely to have severe periodontitis than non-smokers (Haber et al. 1993). Nicotine and lipopolysaccharide (LPS) effects on the expression of macrophage colony-stimulating factor (M-CSF), osteoprotegerin (OPG), and prostaglandin E_2 (PGE_2) have been evaluated by Tanaka et al. (2006) in osteoblasts and osteoclast-like cells. OPG expression was increased in the initial stages of culture with nicotine and LPS but decreased in the later stages of culture. Apatzidou et al. (2005) stated that smokers with periodontal disease have a suppressed inflammatory response, a significantly less favourable clinical outcome and

seem to have an altered host antibody response to antigenic challenge than non-smokers although; the subgingival microflora of smokers appears similar to that of non-smokers. Lappin et al. (2007) reported decreased serum OPG levels and greater soluble receptor activator of nuclear- factor kappa B ligand (sRANKL) sRANKL/OPG ratios in smoker patients in the maintenance program than the non-smoker counterparts. Negative correlation between pack-years and total OPG amount in peri-implant crevicular fluid was detected in clinically healthy implants (Arıkan et al. 2008). Our recent finding of higher sRANKL, lower OPG concentrations in saliva of untreated/treated smokers than non-smokers (Buduneli et al. 2008) confirmed these findings. Furthermore, our saliva data (Buduneli et al. 2008) indicated that treated non-smokers had lower sRANKL levels than untreated non-smokers possibly indicating a more active inflammatory process in untreated patients. Although the exact GCF concentrations of RANKL and OPG varied from study to study, overall RANKL/OPG ratio showed a trend to increase in periodontitis compared to healthy controls (Mogi et al. 2004, Lu et al. 2006, Bostancı et al. 2007, Crotti et al. 2003). This may either be explained by an increase in RANKL or a decrease in OPG levels or both.

Significantly lower plasma OPG concentrations were detected in smoker chronic periodontitis patients than the smoker healthy controls without any significant differences between the smoker and non-smoker study groups (Özçaka et al. 2010). That finding suggests that smoking alone may not be effective on plasma levels of RANKL/OPG system in periodontal disease but when it is coupled with periodontal inflammation the disturbing effect on bone homeostasis may become detectable. Moreover, the significant positive correlations found in the smoker groups between plasma OPG concentrations and probing depth may indicate a tendency towards increased bone metabolism which aims to compensate the increased susceptibility to alveolar bone resorption (Özçaka et al. 2010). Neither the clinical periodontal measurements nor the laboratory data obtained in plasma samples showed significant differences between the smoker and non-smoker chronic periodontitis patients (Özçaka et al. 2010). Indeed, the similarity in clinical periodontal findings between the smoker and non-smoker chronic periodontitis patient groups may explain the lack of significant differences in plasma levels of RANKL and OPG. Darby et al. (2005) suggested that the inferior improvement in probing depth following scaling and root planing in smoker chronic periodontitis patients may reflect the systemic effects of smoking on the host response, the healing process, and the poorer clearance of the microorganisms. The plasma data reported by Özçaka et al. (2010) are in line with the recent study by Tang et al. (2009) reporting similar sRANKL and OPG levels in GCF samples of never smokers, former smokers and current smokers. In that study the only significant difference could be found in GCF OPG levels of the high pack-years group and never smokers.

It has been shown that not only the number of leukocytes is increased but also leukocytes particularly polymorphonuclear neutrophils (PMNs) are significantly activated in smokers, suggesting a systemic inflammatory state. Nicotine has been shown to activate PMNs. Pathogens in microbial dental plaque are capable of stimulating host cells to increase their matrix metalloproteinase (MMP) release which is considered among the indirect mechanisms of tissue destruction seen during periodontitis (Sorsa et al. 2006). Periodontal tissues are infiltrated mainly by neutrophilic granulocytes and PMN which play an important role in the development of inflammatory injury. Tobacco-induced degranulation events in neutrophils, tobacco-induced alterations to the microbial flora, and tobacco-

induced increases in pro-inflammatory cytokine burden could each, theoretically, influence MMP-8 levels in the periodontal tissues of smokers. In a recent study (Özçaka et al. 2011), it was hypothesised that smoking may affect MMPs and neutrophil degranulation products in the systemic level eventually leading more severe periodontal tissue destruction and systemic inflammation predisposing to cardiovascular diseases (Pussinen et al. 2007). In that exploratory study, the serum concentrations of MMP-8, MMP-9, TIMP-1, NE, and MPO were evaluated comparatively in smoker versus non-smoker patients with chronic periodontitis as well as periodontally healthy subjects. The clinical periodontal measurements were recorded and serum samples were analyzed in 55 patients with the clinical diagnosis of chronic periodontitis (16 smoker and 39 non-smoker) and 56 periodontally healthy subjects (17 smoker and 39 non-smoker). The findings of significantly elevated serum MMP-9, MPO, NE together with decreased TIMP-1 in smoker patients with chronic periodontitis than non-smoker counterparts support the idea that smoking together with periodontal destruction may expose/predispose to cardiovascular diseases.

MMP-8 activity has been found to be modified in various organs and body fluids in tobacco smokers. Knuutinen et al. (2002) noted an increased MMP-8 concentration in the resulting fluid infiltrate in smokers compared to that in the non-smokers following the induction of suction blisters on the upper arm. Furthermore, Betsuyaku et al. (1999) have shown increased MMP-8 and MMP-9 activity in the bronchial alveolar lavage fluids of smokers with emphysema compared to those without emphysema. A significant correlation between increased MMP-8 levels and periodontal disease severity has been suggested. Liu et al. (2006) have reported increased local MMP-8 expression in the periodontal tissues of smokers compared to the non-smokers and a slight increase in the serum MMP-8 concentration, although the difference between the smoker and non-smoker groups did not reach the level of significance.

On the other hand, Söder et al. (2002) found a positive correlation between elastase complexed to α1-antitrypsin and MMP-8 concentrations in the gingival crevicular fluid (GCF) of smokers in individuals with various persistent periodontal diseases. However, they did not observe any difference in GCF MMP-8 levels between smokers and non-smokers. Persson et al. (2003) reported that GCF MMP-8 levels remained unchanged in the smokers following surgical treatment for periodontitis, whereas decreased levels were observed in the non-smokers, suggesting a tobacco-induced MMP-8 burden. Liede et al. (1999) examined salivary MMP-8 concentrations in 327 smokers and 82 quitters and found lower MMP-8 levels in the current smokers than the ex-smokers. According to our recent data (Özçaka et al. 2011), serum MMP-8 concentrations did not differ significantly between the smokers and non-smokers. It should be kept in mind that self-reports of non-smoking people can sometimes be unreliable (Buduneli et al. 2006). Therefore, confirmation of the smoking status by serum and/or salivary cotinine analysis may result in changing the accurate group of individual subjects which may eventually affect the results. Therefore, apart from the relatively small numbers of smokers in some of the studies, lack of cotinine analysis may explain the discrepancies between the findings of different clinical studies.

MPO was suggested as an early marker of systemic inflammation in smokers without severe airway symptoms in a study aiming to relate smoking and chronic obstructive pulmonary disease (Andelid et al. 2007). The authors reported significant increases in serum MPO concentrations in smokers than never smokers at 6th year of follow-up. Enhanced serum

levels of MPO indicate increased degranulation of specific granules of neutrophils (Rautelin et al. 2009). Accordingly, our recent data indicated significant increases in serum concentrations of MPO in smoker chronic periodontitis patients, although the clinical periodontal measurements did not differ from those of the non-smoker counterparts (Özçaka et al. 2011a). This increase in serum MPO concentration may be regarded as an indicator of increased risk for local and systemic inflammation such as periodontal tissue destruction or an early sign of atherosclerosis.

NE, which is a serine protease, can also accelerate MMP-cascades by activating latent proMMPs and inactivating TIMP-1 (Sorsa et al. 2006). NE was suggested to be involved in the degradation of non-collagenous protein-covered collagen fibrils in the early destructive stages of periodontal diseases (Ujiie et al. 2007). To our best of knowledge, serum NE levels as a systemic inflammatory parameter reflecting effects of smoking on chronic periodontitis has been evaluated only in our recent study (Özçaka et al. 2011) which revealed significantly higher serum NE concentrations in smoker chronic periodontitis patients than those of non-smokers.

Significantly increased serum concentrations of MMP-9 together with significant decreases in TIMP-1 concentrations in smoker chronic periodontitis patients deserve further investigation and suggest that chronic periodontitis together with smoking can predispose the development of cardiovascular diseases (Pussinen et al. 2007). Persistent smoking and periodontal inflammation predispose the patients for enhanced systemic inflammation in addition to enhanced periodontal destruction.

Carboxyterminal-telopeptide pyridinoline cross-links of type I collagen (ICTP) is released into the periodontal tissues as a consequence of collagen degradation and alveolar bone resorption (Seibel 2003). Type I collagen composes 90 % of the organic matrix of bone and is the most abundant collagen in osseous tissue (Narayanan & Page 1983). Studies assessing the role of ICTP levels in GCF or peri-implant crevicular fluid as a diagnostic marker of periodontal disease activity have reported promising results so far (Oringer et al. 1998, 2002). ICTP was suggested to predict future bone loss, to correlate with clinical parameters and putative periodontal pathogens and also to reduce following periodontal therapy (Giannobile 1999). Apart from the direct cigarette smoke-mediated effects, tissue damage mediated by impaired balance of bone turnover markers originating from tobacco smoke and tobacco-induced inflammation may be a potential mechanism.

Osteocalcin (OC) is a calcium-binding protein of bone and the most abundant non-collagenous protein of the mineralized tissue (Lian & Gundberg 1988). Serum level of OC is considered as a marker of bone formation (Christenson 1997). Serum levels of OC were reported to be lower in periodontitis patients compared with healthy subjects suggesting lower osteoblastic activity and bone formation ability (Shi et al. 1996).

In a recent study, we investigated possible effects of smoking on saliva ICTP and OC concentrations comparatively in patients with chronic periodontitis and periodontally healthy subjects (Özçaka et al. 2011b). Smoker periodontitis patients revealed similar clinical periodontal index values with non-smoker counterparts, whereas salivary OC levels were lower in smokers than non-smokers. ICTP levels in non-smoker chronic periodontitis patients were higher than non-smoker controls and smoker healthy control group revealed higher ICTP levels than non-smoker counterparts. The data suggested that suppression of

salivary OC level by smoking may at least partly explain the deleterious effects of smoking on periodontal status. In another recent study by our group (Gürlek et al. 2009) similar salivary ICTP levels were detected in smoker, non-smoker and ex-smoker patient groups with similar clinical periodontal findings. Smoking status was confirmed by salivary cotinine analysis but there was no clinically healthy control group in that study and the number of teeth present, average probing depths and attachment levels were all similar in the three study groups. There were no significant differences in saliva ICTP concentrations between the smoker and non-smoker patient groups. It may be suggested that the similarity in clinical periodontal disease parameters may explain the similar salivary ICTP levels obtained in these studies.

Significantly lower salivary OC concentrations in both healthy and diseased smokers than their non-smoker counterparts were reported by Özçaka et al. (2011b). The detrimental effects of smoking may explain these decreases in salivary levels of OC in smokers also indicating a deficiency in tissue response to the injuries in smoker subjects. The differences in patient numbers and/or the possible differences in the disease activity states may explain the differences in findings of the present study and the previous ones. On the other hand, significantly lower salivary OC concentrations in the smoker patients than the non-smokers as well as the ex-smokers may at least partly explain the mechanisms of negative effects of smoking on periodontal health.

Lymphocyte functions including antibody production may also be affected by smoking. However, such affects seem to be complex and some components of cigarette such as nicotine are immunosuppressive (Geng et al. 1996) whereas some others are immunostimulatory such as tobacco glycoprotein and metals (Francus et al. 1988, Brooks et al. 1990). Serum immunoglobulin G (IgG) levels were reduced in smoker patients with periodontitis (Quinn et al. 1998). On the other hand, the number of B lymphocytes seemed to be similar in smokers and non-smokers but their function in peripheral blood was impaired in smokers, as reflected in proliferative response to polyclonal B cell activators and antigens (Sopori et al. 1989).

Interleukin-1 (IL-1), tumour necrosis factor (TNF), and prostaglandin E_2 (PGE_2) are among the major inflammatory mediators that play significant role in alveolar bone resorption. Increased GCF levels of TNF-alpha were detected in current and former smokers (Bostrom et al. 1998). Moreover, it was reported that the expressions of inflammatory mediators such as IL-1, IL-6, IL-8, TNF-α, and cyclooxygenase-2 (COX-2) in response to lipopolysaccharide (LPS) of gram negative bacteria were increased in smokers (Bostrom et al. 1999, Tappia et al. 1995, Kuschner et al. 1996).

Tobacco smoking mostly in the form of cigarette smoking has been accused of impairing microcirculatory system and the relevant changes in vascular formations and functions may have a negative influence on the immune and inflammatory reactions in periodontal tissues. Smokers were reported to have significantly less number of vessels in inflamed gingival tissue compared to non-smokers (Rezavandi et al. 2002). Long-term smoking has an established negative effect on the vasculature of periodontal tissues. Acute exposure to cigarette smoke induces gingival hyperaemia, which is caused by the concomitant increase in blood pressure against a small but significant sympathetically induced vasoconstriction in healthy gingiva (Mavropoulos et al. 2003). Smoking even one cigarette

has been suggested to have the potential to cause a decrease in gingival blood flow (Mavropoulos et al. 2007). Such small but repeated vasoconstrictive attacks and impairment of revascularization due to cigarette smoking may contribute to disruption of immune response and delay in the healing response, leading to an increased risk of periodontal disease (Ojima & Hanioka 2010). Vascular dysfunction may also reduce oxygen delivery to gingival tissue. Pocket oxygen tension was reported to be significantly lower in smokers than non-smokers providing support for the negative effects of smoking on vascular system (Hanioka et al. 2000). Evidence from both human and experimental studies suggests that smoking has a long-term chronic effect, and its effect is not simply a vasoconstriction. Its suppressive effects on the vascular system of gingiva can be observed through less gingival redness, lower bleeding on probing and fewer vessels visible clinically and histologically.

A better understanding of the influence of tobacco smoke on the host response to periodontal infection has been the major concern in numerous studies whereas, limited research has been published aiming to identify the influence of tobacco smoke on the dental biofilm. Tobacco smoke has been shown to cause shifts in the microbial species that comprise dental plaque (Haffajee & Socransky 2001, Kamma et al. 1999, Shiloah et al. 2000, Umeda et al. 1998, van Winkelhoff et al. 2001, Zambon et al. 1996). In a recent study by our group (Buduneli et al. 2011), it was hypothesized that tobacco may induce alterations to the molecular structure of lipid A in a manner consistent with reduced inflammatory potential. Therefore, the ratios of 3-OH fatty acids in smoking and non-smoking chronic periodontitis patients were investigated. The findings suggested that smoking induces specific structural alterations to the lipid A-derived 3-OH fatty acid profile in saliva that are consistent with an oral microflora of reduced inflammatory potential. Such data may explain increased infection with periodontal pathogens but reduced clinical inflammation in smokers. However, further studies are warranted to better clarify this issue.

In a systematic review, Bergstrom (2006) concluded that 100% of 70 cross-sectional studies and 100% of 14 case-control studies indicate an association between smoking and an impaired periodontal health condition, and 95% of 21 cohort studies indicate a greater periodontal health impairment rate in smokers than in non-smokers. Therefore, he suggested that there is good evidence to recommend smoking to be specifically considered in a periodontal health examination. Indeed, in a review by Hilgers & Kinane (2004) it was concluded that smoking cessation is indicated in the promotion of better general health and in the improvement of periodontal health. Considering the existing evidences, the authors suggested that dentists should offer to refer patients for smoking cessation counselling.

In conclusion, smoking is accepted as a strong risk factor for destructive periodontal disease. Gelskey (1999) stated that smoking meets most of the criteria for causation proposed by Hill (1965). This statement is based on the consistency and strength of association between smoking and periodontal disease severity demonstrated by multiple cross-sectional as well as longitudinal studies. Further evidence comes from the dose effect of smoking on periodontal disease severity and a slowing of disease progression in patients who quit smoking. Furthermore, smoking has a negative impact on periodontal treatment outcomes. Smoking is harmful virtually to every tissue in the body. The basis for these deleterious effects is related to the adverse impact of smoking on microbial and host factors.

The proposed mechanisms for the negative effects of smoking on periodontal health were summarised by Johnson & Guthmiller (2007) as follows; decreased immunoglobulin G2 production, chronic reduction in blood flow and vascularity, increased prevalence of potential periodontal pathogens, shift in neutrophil function towards destructive activities, negative effects on cytokine and growth factor production and inhibition of fibroblast growth, attachment and collagen production.

In addition to be accepted as an important risk factor for destructive periodontal disease, smoking has been suggested to interfere with the outcomes of various periodontal therapies. Bostrom (2006) systematically reviewed the intervention studies both in terms of non-surgical and surgical periodontal therapy. It was concluded that the results of the intervention studies suggest an inferior therapeutic outcome in smoker patients compared to non-smoker counterparts. The author reports that in 80% of studies the results were statistically significant. When measured in terms of mean probing depth reduction or mean clinical attachment level gain after a maximum of 9 months, the outcome on the average is less efficient in smokers than in non-smokers (Bostrom 2006). However, none of the non-surgical intervention studies has evaluated the effect of smoking in terms of a successful versus a non-successful outcome following predetermined criteria. Furthermore, it was stated that it is still not known whether or not a negative short-term effect of smoking observed in terms of probing depth or clinical attachment level holds true in the long run as tooth loss. Most of the intervention studies have a rather short follow-up period and therefore unable to provide an answer in terms of the rate of tooth loss. It is quite clear that further intervention studies with larger scales and longer follow-ups are required to better clarify this issue. Smoking more than 10 cigarettes per day is considered as heavy smoking and heavy smokers have a poorer treatment response than non-smokers or ex-smokers (Kaldahl et al. 1996, Norderyd 1998).

A meta-analysis by Labriola et al. (2005) evaluated the impact of smoking on non-surgical periodontal therapy and reported that probing depth reduction in sites where probing depth was initially equal to or more than 5 mm was significantly greater in non-smokers than in smokers in eight studies (Grossi et al. 1997, Mongardini et al. 1999, Palmer et al. 1999, Preber et al. 1995, Pucher et al. 1997, Renvert et al. 1998, Ryder et al. 1999, Williams et al. 2001).

In a recent intervention study (Buduneli et al. 2009), effects of initial periodontal treatment on GCF levels of interleukin-17 (IL-17), sRANKL, and OPG in smoker versus non-smoker patients with chronic periodontitis. All clinical periodontal measurements decreased significantly after the initial periodontal treatment in both the smoker and non-smoker patient groups. There were no significant differences between the smoker and non-smoker patients in regards with the changes in clinical periodontal data following initial periodontal treatment. Data indicated that GCF volume, OPG total amount and concentration decreased in both smokers and non-smokers after scaling and root planning (SRP), whereas IL-17 concentration increased. sRANKL levels did not differ between groups or with SRP. Significant correlations were found between baseline IL-17 and RANKL levels, baseline papilla bleeding on probing and OPG levels. According to the findings, it was suggested that neither smoking nor periodontal inflammation appears to influence GCF RANKL levels in systemically healthy patients with chronic periodontitis. Smoker and non-smoker patients with chronic periodontitis seem to be affected indifferently by the initial periodontal

treatment in regards with GCF IL-17 and OPG concentrations. Smoking seems to suppress OPG synthesis and might be contributory to increased bone destruction often seen in smokers.

Surgical periodontal therapy aims to eliminate periodontal pockets and obtain physiological contours in both soft and hard periodontal tissues. As with the non-surgical intervention studies, the primary outcome measures in surgical intervention studies are probing depth and clinical attachment level. On the basis of a systematic review, Bostrom (2006) reported that 91% of 10 non-surgical and 93% of 14 surgical therapy intervention studies indicate an untoward effect of smoking on the therapeutic outcome. The author concluded that there is limited but consistent evidence to suggest that smoking negatively interferes with the therapy outcomes of nonsurgical as well as surgical periodontal interventions.

Haesman et al. (2006) also reviewed the clinical evidence for the relative clinical responses to periodontal treatment in smokers, non-smokers and ex-smokers. The authors concluded that data from epidemiological, cross-sectional and case-control studies strongly suggest that quitting smoking is beneficial to patients following periodontal treatments. The response of ex-smokers to periodontal treatment suggests that quitting smoking helps to improve the clinical periodontal status but there are only limited data from long-term longitudinal clinical trials to demonstrate unequivocally the periodontal benefit of quitting smoking. It is clear that long-term, longitudinal clinical trial that monitors over several years the response to treatment in a cohort of smokers with chronic periodontitis will provide valuable data on the detrimental effects of smoking. However, such a study is definitely not easy to conduct both from the practical and financial points of view. At present, the dental profession can rely on the strong evidence base of the epidemiological, cross-sectional, and case-control studies concluding that quitting smoking is likely to be beneficial to oral and in particular periodontal health.

Johnson & Guthmiller (2007) suggested that smoking cessation cannot reverse the negative past effects of smoking; however, the rate of bone and attachment loss slows after patients quit smoking. Periodontal disease severity in former smokers falls between that of current smokers and never smokers (Bergstrom et al. 2000, Jansson & Lavstedt 2002, Tomar & Asma 2000, Torrunggruang et al. 2005). Preshaw et al. (2005) evaluated the effect of smoking cessation on non-surgical periodontal treatment in 49 smokers who wanted to quit smoking. Therapy included individualised cessation interventions, scaling and root planning and oral hygiene instructions followed by maintenance phase of periodontal treatment. Only 26 patients completed the study; 10 successfully stopped smoking, 10 continued smoking, and six stopped but relapsed during the study period. The authors reported that the patients who successfully quit smoking had the best response to therapy; the “oscillators” had an intermediate response to the non-smokers and quitters. These results underscore the challenges of cessation of tobacco smoking. Counselling on the benefits of smoking cessation by dentists may encourage the patients to seek periodontal therapy and eventually lead to improved periodontal health. Likewise, Borrell & Papapanou (2005) suggested that smoking cessation is a key in the prevention and control of periodontal disease because it affects both the bacterial and host etiological components in the disease process.

In conclusion, the data from both cross-sectional and epidemiological studies over the past 15 years evaluating the relationship between smoking and periodontal diseases suggest that there is strong evidence of a positive association between smoking and clinical and biochemical signs of periodontitis, as well as an increased risk of periodontitis in smokers. Furthermore, the evidence for smoking having a deleterious effect on periodontal treatment outcomes is quite convincing but still needs more data from longitudinal intervention studies.

2. Source of funding

The author declares no conflict of interest.

3. References

Ah MK, Johnson GK, Kaldahl WB, Patil KD, Kalkwarf KL. (1994) The effect of smoking on the response to periodontal therapy. *Journal of Clinical Periodontology* 21:91-97.

Al-Ghamdi HS, Anıl S. (2007) Serum antibody levels in smoker and non-smoker Saudi subjects with chronic periodontitis. *Journal of Periodontology* 78: 1043-1050.

Andelid K, Bake B, Rak S, Lindén A, Rosengren A, Ekberg-Jansson A. (2007) Myeloperoxidase as a marker of increasing systemic inflammation in smokers without severe airway symptoms. *Respiratory Medicine* 101:888-895.

Apatzidou DA, Riggio MP, Kinane DF. (2005) Impact of smoking on the clinical, microbiological and immunological parameters of adult patients with periodontitis. *Journal of Clinical Periodontology* 32:973-983.

Arıkan F, Buduneli N, Kütükçüler N. (2008) Osteoprotegerin levels in peri-implant crevicular fluid. *Clinical Oral Implants Research* 19:283-288.

Armitage GC. (2004) Analysis of gingival crevice fluid and risk of progression of periodontitis. *Periodontology 2000* 34: 109-119.

Barbour SE, Nakashima K, Zhang JB, Tangada S, Hahn CL, Schenkein HA, Tew JG. (1997) Tobacco and smoking: environmental factors that modify the host response (immune system) and have an impact on periodontal health. *Critical Reviews in Oral Biology and Medicine* 8:437-460.

Betsuyaku T, Nishimura M, Takeyabu K, et al. (1999) Neutrophil granule proteins in bronchoalveolar lavage fluid from subjects with subclinical emphysema. *American Journal of Respiratory and Critical Care Medicine* 159:1985-1991.

Bergstrom J, Eliasson S, Dock J. (2000) A 10-year prospective study of tobacco smoking and periodontal health. *Journal of Periodontology* 71:1338-1347.

Bergstrom J. (2006) Periodontitis and smoking: An evidence-based appraisal. *Journal of Evidence Based Dental Practice* 6:33-41.

Borrell LN, Papapanou PN. (2005) Analytical epidemiology of periodontitis. *Journal of Clinical Periodontology* 32 (Suppl.6): 132-158.

Bostanci N, İlgenli T, Emingil G, Afacan B, Han B, Töz H, Atilla G, Hughes FJ, Belibasakis GN. (2007) Gingival crevicular fluid levels of RANKL and OPG in periodontal diseases: Implications of their relative ratio. *Journal of Clinical Periodontology* 34: 370-376.

Boström L, Linder LE, Bergstrom J. (1999) Smoking and crevicular fluid levels of IL-6 and TNF-alpha in periodontal disease. *Journal of Clinical Periodontology* 26:352-357.

Boström L, Bergström J, Dahlen G, Linder LE. (2001) Smoking and subgingival microflora in periodontal disease. *Journal of Clinical Periodontology* 28: 212-219.

Brooks SM, Baker DB, Gann PH, et al. (1990) Cold air challenge and platinum skin reactivity in platinum refinery workers. Bronchial reactivity precedes skin prick response. *Chest* 97:1401-1407.

Buduneli N, Baylas H, Buduneli E, Türkoğlu O, Dahlen G. (2005a) Evaluation of the relationship between smoking during pregnancy and subgingival microbiota. *Journal of Clinical Periodontology* 32: 68-74.

Buduneli N, Buduneli E, Kardeşler L, Lappin D, Kinane DF. (2005b) Plasminogen activator system in smokers and non-smokers with and without periodontal disease. *Journal of Clinical Periodontology* 32: 417-424.

Buduneli N, Kardeşler L, Işik H, Willis CS 3rd, Hawkins SI, Kinane DF, Scott DA. (2006) Effects of smoking and gingival inflammation on salivary antioxidant capacity. *Journal of Clinical Periodontology* 33:159-164.

Buduneli N, Bıyıkoğlu B, Sherrabeh S, Lappin D. (2008) Saliva concentrations of RANKL and osteprotegerin in smoker versus non-smoker chronic periodontitis patients. *Journal of Clinical Periodontology* 35: 846-852.

Buduneli N, Buduneli E, Kütükçüler N. (2009) Interleukin-17, RANKL, osteoprotegerin levels in gingival crevicular fluid from smoker versus non-smoker chronic periodontitis patients during initial periodontal treatment. *Journal of Periodontology* 80: 1274-1280.

Buduneli N, Kinane DF. (2011) Host-derived diagnostic markers related to soft tissue destruction and bone degradation in periodontitis. *Journal of Clinical Periodontology* Mar;38 Suppl 11:85-105.

Buduneli N, Larsson L, Biyikoglu B, Renaud DE, Bagaitkar J, Scott DA. (2011) Fatty acid profiles in smokers with chronic periodontitis. *Journal of Dental Research* 90:47-52.

Calsina G, Ramon JM, Echeverria JJ. (2002) Effects of smoking on periodontal tissues. *Journal of Clinical Periodontology* 29: 771-776.

Centres for Disease Control and Prevention. (2002) Annual smoking- attributable mortality, years of potential life lost, and economic costs-United States, 1995-1999. *Morbidity and Mortality Weekly Reports* 51:300-303.

Christenson RH. (1997) Biochemical markers of bone metabolism: an overview. *Clinical Biochemistry* 30: 573-593.

Crotti T, Smith MD, Hirsch R, Soukoulis S, Weedon H, Capone M, Ahern MJ, Haynes D. (2003) Receptor activator NF kappaB ligand (RANKL) and osteoprotegerin (OPG) protein expression in periodontitis. *Journal of Periodontal Research* 38: 380-387.

Darby IB, Hodge PJ, Riggio MP, Kinane DF. (2005) Clinical and microbiological effect of scaling and root planing in smoker and non-smoker chronic and aggressive periodontitis patients. *Journal of Clinical Periodontology* 32:200-206.

Francus T, Klein RF, Staiano-Coico L, Becker CG, Siskind GW. (1988) Effects of tobacco glycoprotein (TGP) on the immune system. II.TGP stimulates the proliferation of human T cells and the differentiation of human B cells into Ig secreting cells. *Journal of Immunology* 140:1823-1829.

Fredriksson MI, Figueredo CM, Gustafsson A, Bergström KG, Asman BE. (1999) Effect of periodontitis and smoking on blood leukocytes and acute-phase proteins. *Journal of Periodontology* 70: 1355-1360.

Gelskey SC. (1999) Cigarette smoking and periodontitis: methodology to assess the strength of evidence in support of a causal association. *Community Dentistry and Oral Epidemiology* 27:16-24.

Gemmell E, Seymour GJ. (2004) Immunoregulatory control of Th1/Th2 cytokine profiles in periodontal disease. *Periodontology 2000* 35:21-41.

Geng Y, Savage SM, Razani-Boroujerdi S, Sopori ML. (1996) Effects of nicotine on the immune response.II.Chronic nicotine treatment induces T cell anergy. *Journal of Immunology* 156:2384-2390.

Giannobile WV. (1999) C-telopeptide pyridinoline cross-links. Sensitive indicators of periodontal tissue destruction. *Annals of New York Academy of Sciences* 878, 404-412.

Grossi SG, Zambon J, Machtei EE, Schifferle R, Andreana S, Genco RJ, Cummins D, Harrap G. (1997) Effects of smoking and smoking cessation on healing after mechanical periodontal therapy. *Journal of American Dental Association* 128:599-607.

Gürlek Ö, Lappin DF, Buduneli N. (2009) Effects of smoking on salivary C-telopeptide pyridinoline cross-links of type I collagen and osteocalcin levels. *Archives of Oral Biology* 54, 1099-1104.

Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. (1993) Evidence for cigarette smoking as a major risk factor periodontitis. *Journal of Periodontology* 64:16-23.

Haffajee AD, Socransky SS. (2001) Relationship of cigarette smoking to the subgingival microbiota. *Journal of Clinical Periodontology* 28: 377-388.

Haesman L, Stacey F, Preshaw PM, McCracken GI, Hepburn S, Haesman PA. (2006) The effect of smoking on periodontal treatment response: a review of clinical evidence. *Journal of Clinical Periodontology* 33:241-253.

Hanioka T, Tanaka M, Ojima M, Takaya K, Matsumori Y, Shizukuishi S. (2000) Oxygen sufficiency in the gingival of smokers and non-smokers with periodontal disease. *Journal of Periodontology* 71:1846-1851.

Hilgers KK, Kinane DF. (2004) Smoking, periodontal disease and the role of the dental profession. *International Journal of Dental Hygiene* 2:56-63.

Hill AB. (1965) The environment and disease: association or causation? *Proceedings of the Royal Society of Medicine* 58:295-300.

Jansson L, Lavstedt S. (2002) Influence of smoking on marginal bone loss and tooth loss-a prospective study over 20 years. *Journal of Clinical Periodontology* 29:75-756.

Johnson GK, Hill M. (2004) Cigarette smoking and the periodontal patient. *Journal of Periodontology* 75:196-209.

Johnson GK, Guthmiller JM. (2007) The impact of cigarette smoking on periodontal disease and treatment. *Periodontology 2000* 44:178-194.

Kaldahl WB, Johnson GK, Patil KD, Kalkwarf KL. (1996) Levels of cigarette consumption and response to periodontal therapy. *Journal of Periodontology* 67:675-681.

Kamma JJ, Nakou M, Baehni PC. (1999) Clinical and microbiological characteristics of smokers with early onset periodontitis. *Journal of Periodontal Research* 34: 25-33.

Kinane DF. (2001) Causation and pathogenesis of periodontal disease. *Periodontology 2000* 25, 8-20.

Kinane DF, Chestnutt IG. (2000) Smoking and periodontal disease. *Critical Reviews in Oral Biology and Medicine* 11, 356-365.

Knuutinen A, Kokkonen N, Risteli J, et al. (2002) Smoking affects collagen synthesis and extracellular matrix turnover in human skin. *British Journal of Dermatology* 146:588-594.

Labriola A, Needleman I, Moles DR. (2005) Systematic review of the effect of smoking on non-surgical periodontal therapy. *Periodontology 2000* 37:124-137.

Lappin DF, Sherrabeh S, Jenkins WM, Macpherson LM. (2007) Effect of smoking on serum RANKL and OPG in sex, age and clinically matched supportive-therapy periodontitis patients. *Journal of Clinical Periodontology* 34:271-277.

Lian JB, Gundberg CM. (1988) Osteocalcin: biochemical considerations and clinical applications. *Clinical Orthopedia Related Research* 226: 267-291.

Liede KE, Haukka JK, Hietanen JH, Mattila MH, Ronka H, Sorsa T. (1999) The association between smoking cessation and periodontal status and salivary proteinase levels. *Journal of Periodontology* 70:1361-1368.

Liu KZ, Hynes A, Man A, Alsagheer A, Singer DL, Scott DA. (2006) Increased local matrix metalloproteinase-8 expression in the periodontal connective tissues of smokers with periodontal disease. *Biochim Biophys Acta* 1762:775-780.

Lu HK, Chen YL, Chang HC, Li CL, Kuo MY. (2006) Identification of the osteoprotegerin/receptor activator of nuclear factor-kappa B ligand system in gingival crevicular fluid and tissue of patients with chronic periodontitis. *Journal of Periodontal Research* 41: 354-360.

Martinez-Canut P, Lorca A, Magan R. (1995) Smoking and periodontal disease severity. *Journal of Clinical Periodontology* 22: 743-749.

Mavropoulos A, Aars H, Brodin P. (2003) Hyperaemic response to cigarette smoking in healthy gingiva. *Journal of Clinical Periodontology* 30:214-221.

Mavropoulos A, Brodin P, Rosing CK, Aass AM, Aars H. (2007) Gingival blood flow in periodontitis patients before and after periodontal surgery assessed in smokers and non-smokers. *Journal of Periodontology* 78:1774-1782.

Mogi M, Otogoto J, Ota N, Togari A. (2004) Difefrential expression of RANKL and osteoprotegerin in gingival crevicular fluid of patients with periodontitis. *Journal of Dental Research* 83: 166-169.

Mongardini C, van Steenberghe D, Dekeyser C, Quirynen M. (1999) One stage full- versus partial-mouth disinfection in the treatment of chronic adult or generalised early-onset periodontitis I. Long-term clinical observations. *Journal of Periodontology* 70:632-645.

Mullally BH. (2004) The influence of tobacco smoking on the onset of periodontitis in young persons. *Tobacco Induced Diseases* 2:53-65.

Narayanan AS, Page RC. (1983) Connective tissues of the periodontium: A summary of current work. *Collagen Related Research* 3: 33-64.

Norderyd O. (1998) Risk for periodontal disease in a Swedish adult population. Cross-sectional and longitudinal studies over two decades. *Swedish Dental Journal* Suppl. 132:1-67.

Offenbacher S, Odle BM, Van Dyke TE. (1984) The use of crevicular fluid prostaglandin E2 levels as a predictor of periodontal attachment loss. *Journal of Periodontal Research* 21: 101-112.

Ojima M, Hanioka T. (2010) Destructive effects of smoking on molecular and genetic factors of periodontal disease. *Tobacco Induced Diseases* 8:4.

Oringer RJ, Palys MD, Iranmanesh A, Fiorellini JP, Haffajee AD, Socransky SS, Giannobile WV. (1998) C-telopeptide pyridinoline cross-links (ICTP) and periodontal pathogens associated with endosseous oral implants. *Clinical Oral Implants Research* 9: 365-373.

Oringer RJ, Al-Shammari KF, Aldredge WA, Iacono VJ, Eber RM, Wang HL, Berwald B, Nejat R, Giannobile WV. (2002) Effect of locally delivered minocycline microspheres on markers of bone resorption. *Journal of Periodontology* 73: 835-842.

Özçaka Ö, Nalbantsoy A, Köse T, Buduneli N. (2010) Plasma osteoprotegerin levels are decreased in smoker chronic periodontitis patients. *Australian Dental Journal* 55:405-410.

Özçaka Ö, Bıçakçı N, Pussinen P, Sorsa T, Köse T, Buduneli N. (2011a) Smoking and matrix metalloproteinases, neutrophil elastase, and myeloperoxidase in chronic periodontitis. *Oral Diseases* 17:68-76.

Özçaka Ö, Nalbantsoy A, Buduneli N. (2011b) Salivary osteocalcin levels are decreased in smoker chronic periodontitis patients. *Oral Diseases* 17:200-205.

Palmer RM, Matthews JP, Wilson RF. (1999) Non-surgical periodontal treatment with and without adjunctive metronidazole in smokers and non-smokers. *Journal of Clinical Periodontology* 26:158-163.

Palmer RM, Wilson RF, Hasan AS, Scott DA. (2005) Mechanisms of action of environmental factor-tobacco smoking. *Journal of Clinical Periodontology* 32:180-195

Persson L, Bergstrom J, Gustafsson A. (2003) Effect of tobacco smoking on neutrophil activity following periodontal surgery. *Journal of Periodontology* 74:1475-1482.

Preber H, Bergstrom J, Linder LE. (1992) Occurrence of periopathogens in smoker and non-smoker patients. *Journal of Clinical Periodontology* 19:667-671.

Preber H, Linder L, Bergström J. (1995) Periodontal healing and periopathogenic microflora in smokers and non-smokers. *Journal of Clinical Periodontology* 22: 946-952.

Preshaw PM, Haesman L, Stacey F, Steen N, McCracken GI, Haesman PA. (2005) The effect of quitting smoking on chronic periodontitis. *Journal of Clinical Periodontology* 32:869-879.

Pucher JJ, Shibley O, Dentino AR, Ciancio SG. (1997) Results of limited initial periodontal therapy in smokers and non-smokers. *Journal of Periodontology* 68:851-856.

Pussinen PJ, Paju S, Mantyla P, Sorsa T. (2007) Serum microbial- and host-derived markers of periodontal diseases: A review. *Current Medical Chemistry* 14, 2407-2412.

Rautelin HI, Oksanen AM, Veijola LI, et al. (2009) Enhanced systemic matrix metalloproteinase response in Helicobacter pylori gastritis. *Annals of Medicine* 41:208-215.

Renvert S, Dahlen G, Wikstrom M. (1998) The clinical and microbiological effects of non-surgical periodontal therapy in smokers and non-smokers. *Journal of Clinical Periodontology* 25:153-157.

Rezavandi K, Palmer RM, Odell EW, Scott DA, Wilson RF. (2002) Expression of ICAM-1 and E-selectin in gingival tissues of smokers and non-smokers with periodontitis. *Journal of Oral Pathology and Medicine* 31:59-64.

Ryder MI, Pons B, Adams D, Beiswanger B, Blanco V, Bogle G, Donly K, Hallmon W, Hancock EB, Hanes P, Hawley C, Johnson L, Wang HL, Wolinsky L, Yukna R,

Polson A, Carron G, Garrett S. (1999) Effects of smoking on local delivery of controlled-release doxycycline as compared to scaling and root planning. *Journal of Clinical Periodontology* 26:683-691.

Savage A, Eaton KA, Moles DR, Needleman I. (2009) A systematic review of definitions of periodontitis and methods that have been used to identify this disease. *Journal of Clinical Periodontology* 36:458-467.

Scott DA, Palmer RM, Stapleton JA. Validation of smoking status in clinical research into inflammatory periodontal disease. *Journal of Clinical Periodontology* 2001; 28:715-722.

Seibel MJ. (2003) Biochemical markers of bone metabolism in the assessment of osteoporosis: useful or not? *Journal of Endocrinology Investment* 26: 464-471.

Shi F, Yu S, Xu L. (1996) Analysis of serum osteocalcin of patients with periodontitis. *Zhonghua Kou Qiang Yi Xue Za Zhi* 31: 300-302. (Article in Chinese)

Shiloah J, Patters MR, Waring MB. (2000) The prevalence of pathogenic periodontal microflora in healthy young adult smokers. *Journal of Periodontology* 71:562-567.

Sopori ML, Cherian S, Chilukuri R, Shopp GM. (1989) Cigarette smoke causes inhibition of the immune response to intratracheally administered antigens. *Toxicology Applications in Pharmacology* 97:489-499.

Sorsa T, Tjaderhane L, Konttinen YT, Lauhio A, Salo T, Lee H-M, Golub LM, Brown DL, Mantyla P. (2006) Matrix metalloproteinases: Contribution to pathogenesis, diagnosis and treatment of periodontal inflammation. *Annals of Medicine* 38: 306-321.

Söder B, Jin LJ, Wickholm S. (2002) Granulocyte elastase, matrix metalloproteinase-8 and prostaglandin E2 in gingival crevicular fluid in matched clinical sites in smokers and non-smokers with persistent periodontitis. *Journal of Clinical Periodontology* 29:384-391.

Tanaka H, Tanabe N, Shoji M, et al. (2006) Nicotine and lipopolysaccharide stimulate the formation of osteoclast-like cells by increasing macrophage colony-stimulating factor and prostaglandin E2 production by osteoblasts. *Life Sciences* 78:1733-1740.

Tang TH, Fitzsimmons TR, Bartold M. (2009) Effect of smoking on concentrations of receptor activator of nuclear factor κB ligand and osteoprotegerin in human gingival crevicular fluid. *Journal of Clinical Periodontology* 36: 713-718.

Tappia PS, Troughton KL, Langley-Evans SC, Grimble RF. (1995) Cigarette smoking influences cytokine production and antioxidant defences. *Clinical Sciences* 88:485-489.

Taubman MA, Valverde P, Han X, Kawai T. (2005) Immune response: The key to bone resorption in periodontal disease. *Journal of Periodontology* 76 Suppl.: 2033-2041.

Tomar SL, Asma S. (2000) Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *Journal of Periodontology* 71:743-751.

Torrungruang K, Nisapakultorn K, Sutdhibhisal S, Tamsailom S, Rojanasomsith K, Vanichjakvong O, Prapakamol S, Premsirinirund T, Pusiri T, Jaratkulangkoon O, Kusump S, Rajatanavin R. (2005) The effect of cigarette smoking on the severity of periodontal disease among older Thai adults. *Journal of Periodontology* 76:566-572.

Quinn SM, Zhang JB, Gunsolley JC, Schenkein HA, Tew JG. (1998) The influence of smoking and race on adult periodontitis and serum IgG2 levels. *Journal of Periodontology* 69:171-177.

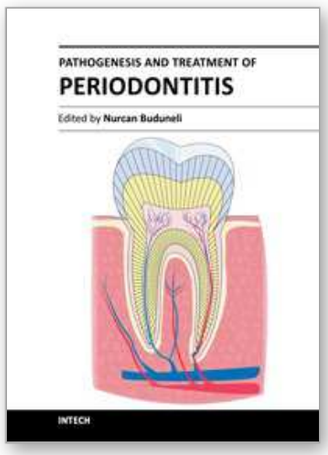
Ujiie Y, Oida S, Gomi K, Arai T, Fukae M. (2007) Neutrophil elastase is involved in the initial destruction of human periodontal ligament. *Journal of Periodontal Research* 42:325-330.

Umeda M, Chen C, Bakker I, Contreras A, Morrison J, Slots J. (1998) Risk indicators for harbouring periodontal pathogens. Journal of Periodontology 69:1111-1118.

van Winkelhoff AJ, Bosch-Tijhof CJ, Winkel EG, van der Reijden WA. (2001) Smoking affects the subgingival microflora in periodontitis. *Journal of Periodontology* 72: 666-671.

Williams RC, Paquette DW, Offenbacher S, Adams DF, Armitage GC, Bray K, Caton J, Cochran DL, Drisko CH, Fiorellini JP, Giannobile WV, Grossi S, Guerrero DM, Johnson GK, Lamster IB, Magnusson I, Oringer RJ, Persson GR, Van Dyke TE, Wolff LF, Santucci EA, Rodda BE, Lessem J. (2001) Treatment of periodontitis by local administration of minocycline microspheres: a controlled trial. *Journal of Periodontology* 72:1535-1544.

Zambon JJ, Grossi SG, Machtei EE, Ho AW, Dunford R, Genco RJ. (1996) Cigarette smoking increases the risk for subgingival infection with periodontal pathogens. *Journal of Periodontology* 67: 1050-1054.



Pathogenesis and Treatment of Periodontitis

Edited by Prof. Nurcan Buduneli

ISBN 978-953-307-924-0
Hard cover, 200 pages
Publisher InTech
Published online 20, January, 2012
Published in print edition January, 2012

Pathogenesis and Treatment of Periodontitis includes comprehensive reviews on etiopathogenic factors of periodontal tissue destruction related to microbial dental plaque and also host response components. Adjunctive treatment modalities are also addressed in the book. Topics covered range from microbial pathogenic factors of P. gingivalis to the relationship between metabolic syndrome and periodontal disease, and from management of open gingival embrasures to laser application in periodontal treatment.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Nurcan Buduneli (2012). Effects of Tobacco Smoking on Chronic Periodontitis and Periodontal Treatment, Pathogenesis and Treatment of Periodontitis, Prof. Nurcan Buduneli (Ed.), ISBN: 978-953-307-924-0, InTech, Available from: http://www.intechopen.com/books/pathogenesis-and-treatment-of-periodontitis/effects-of-tobacco-smoking-on-chronic-periodontitis-and-periodontal-treatment

INTECH
open science | open minds

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

IntechOpen