

## Chapter 6

# Periodontal Disease Risk Management: Smoking, The Patient Controlled Modifiable Risk

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### Introduction

In many ways we define periodontal diseases as being the body's response to plaque, yet work in the last two decades has emphasised that while dental plaque is necessary for the disease process to begin, it is not by itself sufficient to cause disease in all subjects. Periodontal diseases require a combination of pathogenic plaque at an appropriate environmental location (e.g. the dento-gingival junction) in a susceptible host. It could be said that most oral diseases have these requirements, however the clinical entity of disease only occurs in the rare situation that all these factors occur together at the same time. An understanding of these fundamentals have allowed Page and Kornman (Page & Kornman 1997) to describe the currently accepted paradigm of periodontal pathogenesis which combines the knowledge that the interactions between the host, the environment and the aetiology are not just in one direction, but are at least two way, if not more complex exchanges.

### Diagnosis and management of periodontal disease

The complexities of the pathogenesis of the periodontal diseases has helped make sense

of the ironic diagnostic feature that while our traditional diagnostic tools (probing, radiographs etc.) are good at measuring the extent of disease that has occurred in the past they provide little, if any, information about the current level of disease activity (i.e. actual loss of connective tissue attachment) or predicting future disease progression (Hirschfield & Wasserman 1978). The disease process (i.e. activity) is a rare event even in susceptible individuals, and as with the treatment of many chronic diseases, there has been a move towards managing the risk of disease progress as well as dealing with the presenting acute exacerbations. Lang and Tonetti (Lang & Tonetti 1996, Lang & Tonetti 2003) have settled on 6 objective parameters of risk that have been validated for use in determining the level of SPT required for periodontal patients. The resulting *spider web* diagram currently encompasses the percentage of sites with bleeding on probing (BOP), the absolute number of sites with probing depths of 5mm, the number of teeth lost, the ratio of percentage bone loss to age, the presence of systemic/genetic factors (eg diabetes) and the level of smoking. It is quite possible that with further knowledge, more factors will come to be added to this mix, however the current evidence supports only these factors at present.

Clinicians are well aware that that

successful management of periodontal disease relies not only on their own skill as clinicians but also on the ability of the patient to participate in appropriate home care measures or failing this, frequent professional care. There is only one parameter of risk identified by Lang and Tonetti (Lang & Tonetti 2003) which is modifiable by the patient. While the measurement of percentage of sites with BOP relies on effective oral hygiene to allow the assessment to be made *in the absence of marginal gingivitis*, the actual BOP score relates to the effectiveness of the subgingival debridement. Thus it is only smoking that can be truly altered by the patient to make a positive outcome towards management of their disease progression risk.

### **Effects of smoking on periodontal disease**

The actual mechanisms of the deleterious effects of smoking on periodontal disease are yet to be fully elucidated however many aspects are understood. Smoking can be thought to act on the host via effects on the vasculature, cells and the repair potential and possibly also on the flora (although this effect may be opportunistic due to host changes). The principal vascular effect is peripheral vasoconstriction (Clark *et al* 1981, Danielsen *et al* 1990). Smoking also has deleterious effects on fibroblasts (Raulin *et al* 1988) and in particular PMNs (Bridges *et al* 1977, Kenney *et al* 1977, Kinane & Chestnutt 2000), to say little of the detrimental inflammatory effect on immunoglobulin and cytokine production. (For reviews see Johnston 1999, Kinane & Chestnutt 2000.)

There have been many studies linking smoking to increased prevalence of periodontal disease. Early studies were hampered by poor design and control of other factors such as plaque control however more recent work has clearly shown a consistent

association with smoking leading to a 200-600% increased risk of having periodontitis depending on the study population (Kinane & Chestnutt 2000). Further close analysis suggests smokers have greater attachment loss, furcation involvements, and deep pockets and more calculus than non-smokers. There is also some evidence to suggest a dose response effect such that smoking more cigarettes per day and for more years results in greater periodontal destruction. Young adults who smoke seem to be particularly at risk of showing periodontal disease early in life.

### **Smokers versus non-smokers**

Periodontal treatment is not as effective in smokers as non-smokers. This is not to say that treatment is completely ineffective but rather than it is not as effective (Ah *et al* 1994, Machtei *et al* 1998, Preber *et al* 1995). This is true of both surgical and non-surgical approaches. Of considerable concern however is that of patients considered refractory to usual treatment, approximately 90% are reported to be smokers (MacFarlane *et al* 1992). In summary, smokers have more periodontal disease which is of greater severity than non-smokers and do not respond as well to treatment. There is some indication that the level of smoking is influential on the level of risk involved. It should be noted that smoking in and of itself does not cause periodontitis but it does have significant detrimental effects in patients who are otherwise susceptible to developing periodontitis.

### **Cessation of smoking**

The best news for patients is that smoking cessation appears to be quite beneficial (Haber 1994), and because of the cumulative nature of periodontal disease destruction ultimately ex-smokers will present with similar disease prevalence to never smokers (Kraal *et al*

1997). Smoking cessation is never easy however members of the dental team are well suited to assisting patients in quitting their smoking habits. It is well known that the longer the face to face interaction with a patient the greater the impact on cessation. Members of the dental team often have more time and more follow-up visits (particularly in periodontal patients) than in medical settings to discuss smoking cessation. The 4-A's of ask, advise, assist and arrange are often advised for use in the health care setting and are based largely on observational studies suggesting these methods are effective in helping patients to stop smoking. (To date it appears that only pharmacologic agents have been fully evaluated in controlled trials). There have been few published reports of the use of this method in the dental setting although anecdotal evidence suggests that it is helpful. Our own group is evaluating a modified version of this method of helping patients in a dental setting, however we have yet to evaluate the results.

### **Smoking education and prevention campaigns**

The problem of smoking in the Asia-Pacific region is very large with smoking rates ranging from almost 40% (in places like Bangladesh, Cambodia and China) to about 15% (eg. Singapore). In many countries of the region smoking is a largely male activity (e.g. in China, Korea, Tonga more than 60% of adult males smoke) although there is significant country variation. Some significant increases in female smoking rates are also seen in some cases (e.g. Nauru, New Zealand, Australia and Japan) (<http://nationmaster.com> 2004). Of the World Health Organisation (WHO) health regions in the world, the Western Pacific has the most rapid uptake of smoking, with one in three of all the cigarettes smoked in the world being smoked in China

and at the current rate one in five tobacco related deaths in the world occurs in the WHO Western Pacific region. In the last 2 decades the Western Pacific and Southeast Asian Regions have experienced increases in tobacco consumption, and of particular concern is the increased rate of uptake amongst children and young adults (David 2000). Individual country summaries are available online from the WHO as are the results of the Global Youth Tobacco Survey (<http://tfi.wpro.who.int/gyts.asp> 2004) which highlight the disturbing trend of smoking uptake in adolescents, offers of free cigarettes from tobacco companies and the large proportion of youth smokers who are trying to stop smoking.

While health education campaigns are not without their worth, the evidence to date would suggest that on a population basis, effective methods of reducing smoking prevalence are to provide no government support for the tobacco industry and to enforce comprehensive tobacco control laws (e.g. preventing sale to children and banning advertising and sponsorship). However the most effective smoking prevention programme appears to be raising the cost of cigarettes via taxes (David 2000). To date, both in Australia and Malaysia, tobacco company sponsored youth prevention programs have been instituted however these appear to be largely public relations exercises to ingratiate the companies to governments and the public to limit or reduce taxes and advertising restrictions.

The WHO holds "World No Tobacco Day" each year on May 31, in an effort to focus attention on tobacco issues in health and in particular the Tobacco Free Initiative (<http://www.who.int/tobacco/en/> 2004). Each year focuses on a theme such as second-hand, passive smoking or the 2003 theme of tobacco free film and fashion (<http://www.who.int/tobacco/areas/communications/events/wntd/en> 2004). Much of the understanding of the

link between tobacco use and product placement in Hollywood produced movies has been compiled by Stanton Glantz at the University of California (Glantz 2004). Recent findings have indicated that in adolescents of non-smoking parents, 52.2% of smoking initiation could be attributed to exposure to smoking in movies (Dalton *et al* 2003). The 2004 theme relates to poverty and tobacco use, highlighting the high costs of tobacco products particularly for those of little means, which is particularly applicable in many parts of the Asia Pacific region.

## Conclusion

Clinicians have a number of roles to play. Firstly there is need to act as non-smoking role models within communities and act as true healthcare professionals. We need to acknowledge that smoking is an additive health issue and not just a social choice. It is the responsibility of oral healthcare workers to ask patients about their smoking, discuss the detrimental effects it has on their oral health and to encourage them to quit their habit. There is a need to have appropriate resources or referral lines for patients who express an interest in smoking cessation and finally as individuals and as part of professional groups we should reinforce the need for effective tobacco control with governments.

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