

Tobacco and oral disease

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There is overwhelming evidence that tobacco usage produces harmful effects in the mouth. Among the various health staff groups, dentists are those most frequently in contact with the population. This fact renders dentists a favourable position in connection with tobacco intervention. Evidence shows that clinical interventions during dental care are as effective as in other healthcare settings.¹

In this document the harmful effects of tobacco usage are briefly reviewed. The document is a revised version of a previously published report from the 'EU Working Group on Tobacco and Oral Health'² in which a more detailed discussion and further references can be found. The Working Group is currently developing practical guidelines on how to help patients stop smoking.

Tobacco and general diseases

It appears that: about 30% of all cancer diseases and deaths; about 90% of all lung cancers; 30% of all cases of ischaemic heart disease and strokes; about 80% of myocardial infarctions before the age of 50 years; and 70% of chronic lung diseases (smoker's lung), are caused by tobacco smoking.³⁻⁵

The British doctors' study has shown that smokers are reducing their life length by an average of 7.5 years, irrespective of the type of tobacco smoked and the amount of daily smoking.⁶ The proportion of smokers reaching the age of 70 years was 59% compared with 80%

among non-smokers. Thus, a very large proportion of smokers are dying before pensionable age.

The tar in tobacco is responsible for the development of cancer. The most important malignancy related to smoking is lung cancer. The 5-year survival rate is only between 5 and 10%. A substantial fraction of cancers in the mouth, throat, oesophagus, urinary bladder and pancreas are also caused by tobacco smoking.³⁻⁵

The most disabling chronic lung disease related to smoking is chronic obstructive lung disease (COLD, 'smokers lung') comprising emphysema and chronic bronchitis. The terminal stage of COLD is increasing respiratory insufficiency and death. Smoking also increases the risk of developing asthma.

The risk of developing myocardial infarction is doubled in smokers compared with non-smokers. The risk increases with increasing consumption of tobacco and with other cardiovascular risk factors (cholesterol, hypertension, overweight). Arteriosclerosis is increased among smokers in other parts of the arterial tree and about 75-80% of all cases of intermittent claudication and aortic aneurysms are caused by smoking. The risk for developing myocardial infarction and stroke decreases rapidly after smoking cessation, and after 5-10 years ex-smokers have reached the same risk as never-smokers.

Several meta-analyses have shown that passive smoking can cause lung cancer as well as ischaemic heart disease. The risk related to passive smoking is, however, much lower than the risk related to active smoking.

Passive smoking can cause increased allergic reactions against specific allergens, reduced lung function, and increased asthmatic attacks in patients with asthma.

In brief

The EU-Working Group on Tobacco and Oral Health found:

- Smoking often results in discolourations of teeth and dental restorations.
- Halitosis, diminished taste and smell acuity are common side effects of smoking.
- Periodontal disease is increased both in prevalence and severity in smokers. Smoking cessation may halt disease progression and improve the outcome of periodontal treatment.
- Oral cancer and precancer occurs much more frequently in smokers than in non-smokers. Smoking cessation significantly decreases the increased risk of oral cancer within 5-10 years.
- Dental implant failure rates are significantly higher in smokers than in non-smokers.
- The entire dental team should be aware of the relationship between smoking and dental problems and should convey the message that non-smoking is the norm.
- Smoking counselling should be a fundamental part of the dental curriculum and any practice prevention programme.

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Smoking during pregnancy may have serious consequences for the foetus as well as during the first years of life. Asthmatic bronchitis and infectious diseases in the upper respiratory tract among small children are to a great extent explained by the mother's smoking habits during pregnancy and/or after birth.

Tobacco and oral disease

Aesthetics

Smoking causes discolouration of teeth, dental restorations and dentures.^{7,8} The effect of smoking is more severe than that of the consumption of coffee and tea.⁹

Saliva

In the short term smoking increases the flow rate of the parotid gland.^{10,11} However, the data on long-term effects on salivary flow rates show no difference between smokers and non-smokers.¹²⁻¹⁴

The pH of saliva rises during smoking.¹⁵ Over longer time periods smokers have a lower pH in stimulated whole saliva,¹³ however another report showed no difference.¹⁶ Buffer capacity was found to be lower in smokers^{12,17} although this was not confirmed in another study.¹⁴

The concentration of thiocyanate, a product present in tobacco smoke and in normal saliva, is increased in the saliva of smokers.¹⁸

Dental caries

Although smoking is a factor which has often been included in the analysis of caries rates there is insufficient evidence of any aetiological relationship. From the evidence mentioned above on the relation between tobacco usage and saliva composition this is not surprising. If thiocyanate concentrations are higher one might predict less dental caries. However, a possible lower salivary pH and buffering power, and the fact that there is a shift of the bacterial population towards lactobacillus and the cariogenic streptococci in smokers,^{12,13,19} might all argue for increased dental caries.

There are three recent surveys which link tobacco smoking with dental caries, one of different age strata²⁰ and one of older subjects which identified tobacco smoking as a significant risk factor for tooth loss and

dental caries,²¹ and one of teenagers which found all caries epidemiological data to be higher among tobacco users.²² However, the latter paper points out the danger of assuming that correlations necessarily imply a causative role.

Smell and taste

Many studies have shown that taste and smell acuity are affected by smoking.^{23,24} It should be mentioned here, that smoking is a common cause of halitosis.

Wound healing

Several studies have shown that tobacco influences wound healing in the mouth, eg after periodontal scaling and curettage, periodontal surgery or tooth extraction.²⁵⁻²⁷ The mechanism of impaired healing is likely associated with increased plasma levels of adrenaline and noradrenaline after smoking, leading to peripheral vasoconstriction. Several studies also show impaired PMN function in smokers compared with non-smokers.²⁸

Periodontal diseases:

The role of smoking in periodontal diseases has been extensively studied for many years. An increasing amount of scientific data have demonstrated a clear association between smoking and the prevalence and severity of periodontal diseases, suggesting smoking as an important risk factor for periodontal disease.²⁹⁻³¹

Microflora and host response

The exact mechanisms by which smoking affects the periodontal tissues are not known. Many epidemiological and clinical studies have reported smokers to harbour more supragingival plaque than non-smokers. However, clinical studies have not reported any differences in plaque accumulation rate between smokers and non-smokers³² thereby indicating smoker's excess amount of plaque is probably caused by an inferior oral hygiene. One recent study reported that smokers harboured significantly higher levels of *B. forsythus* than non-smokers, although other studies on patients with periodontal disease have not reported any differences

in the composition of the subgingival periopathogenic microflora.³³⁻³⁵

The greater prevalence and severity of disease in smokers might be explained by an altered host response. Smokers have demonstrated disturbances in immunoglobulin and cytokine levels, lymphocyte counts and impairment of the function of oral neutrophils.^{28,36,37}

Besides possible specific effects from the periopathogenic bacteria and by an altered host response, there might be cytotoxic effects from nicotine on fibroblast function.³⁸

Gingivitis

Many earlier studies on smoking and chronic gingivitis have reported both more gingival inflammation and more dental plaque and calculus in smokers. However, in recent studies, when the plaque level has been controlled for, smokers have demonstrated less gingival inflammation and less gingival bleeding when compared with non-smokers, indicating a suppressed gingival inflammation.^{32,39} These results, which suggest a lower bleeding propensity for smokers, are not surprising given the well known effect of nicotine exerting local vasoconstriction on peripheral circulation.

Acute necrotizing ulcerative gingivitis (ANUG)

Many studies have reported smokers to have a higher prevalence of ANUG than non-smokers.⁴⁰ Recently, a similar relationship has been reported between smoking and ANUG-like lesions in HIV infected individuals.⁴¹ The results from these studies show a clear association between smoking and ANUG.

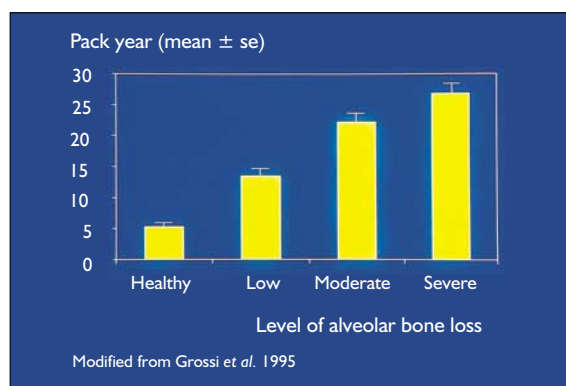
Periodontitis

The association between tobacco smoking and adult periodontitis has been studied during the past 20 years in well controlled studies on large groups of populations.³¹ The results from these studies suggest smokers to have an increased prevalence and severity of periodontitis, as reported by greater marginal bone loss, deeper periodontal pockets, more severe attachment loss and more teeth with furcation involvements (Fig. 1).

Fig. 1 Smokers periodontitis. A 54-year-old female heavy smoker with severe adult periodontitis. Great gingival recession and discoloured root surfaces. Low gingival inflammation in spite of heavy plaque accumulation



Fig. 2 Relationship between alveolar bone loss and amount of smoking (from ref. 48)



In many of the studies multivariate analyses suggested smoking as an independent risk factor for periodontal disease; after oral hygiene, plaque, calculus, and socio-economic factors were controlled for. Risk assessments suggest the smoking-attributable relative risk for a smoker to be 2.5 to 6.0 the risk of a non-smoker.^{29,42,43}

Periodontal therapy

Clinical studies have demonstrated that smoking adversely affects the outcome of various modalities of periodontal therapy. Studies on surgical as well as non-surgical therapy report inferior results in smoking patients.^{44,45} Smokers have also been reported to have poorer success rates following periodontal regenerative surgery.⁴⁶ Furthermore, refractory periodontitis cases, ie patients who do not respond as expected following therapy exhibiting continuing progression of disease, are heavily overrepresented in smokers.⁴⁷

Effects of smoking cessation

At present there are no studies on the effect of smoking cessation on periodontal disease. However, several studies report a lower prevalence and severity of disease in former smokers as compared with current smokers, and a relationship has also been demonstrated between the severity of disease and the amount of smoking (Fig. 2).⁴⁸

Furthermore, in a recent longitudinal

study, 44 subjects who gave up smoking during a 10-year period, had significantly less marginal bone loss as compared with those 139 who smoked regularly during the period.⁴⁹

The observations of these studies imply that smoking cessation may slow down or halt the progression of periodontal disease.

Dental implants

There is increasing evidence that tobacco is detrimental to both the initial and longterm success of dental implants, and that smoking cessation can be beneficial in improving implant success rates.

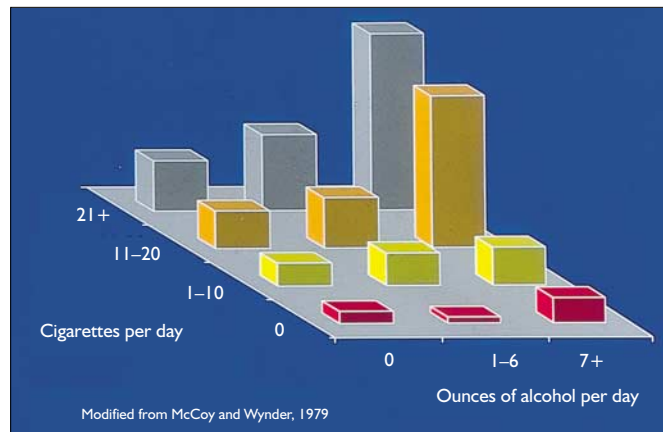
Smoking was found to be by far the most significant factor predisposing to implant failure.⁵⁰ Failure rates were 4.8% in non-smokers and 11.3% in smokers. When the maxilla was assessed separately failure rates in smokers exceeded 17.8% and were greatest with shorter implants. In a later study comparing implant failures prior to loading in the maxilla of smokers with non-smokers failure rates were 9% and 2% respectively.⁵¹

A recent study has also suggested an ongoing detrimental effect around the successfully integrated maxillary implants of smokers, with significantly greater bleeding index, mean peri-implant pocket depth, peri-implant inflammation and radiographically discernible mesial and distal bone loss.⁵² The authors suggest that these findings will lead to higher future failure rates in integrated implants in smokers. This position is supported by a 15-year prospective study of mandibular fixed implant prostheses in which it was found that smoking correlated more strongly with

Fig. 3 Carcinoma of the floor of the mouth in a heavy smoker



Fig. 4 Relative risks of oral cavity cancer by daily consumption of alcohol and cigarettes for men (from ref. 58)



marginal bone loss around implants than even poorer oral hygiene.⁵³

Smoker cessation

Following a protocol of complete cessation for one week before and eight weeks after initial implant placement surgery, a significant reduction in implant failure in the group who stopped smoking was found when compared with smokers who continued the habit.⁵⁴ The group who followed the cessation protocol had no significant difference in their failure rate when compared with non-smokers over the same period.

Oral mucosal diseases

Tobacco use is associated with several changes in the oral mucous membranes ranging from innocent and reversible lesions, such as smoker's palate, to oral cancer. The innocent lesions offer an excellent opportunity to initiate a discussion on tobacco.

Oral cancer

Tobacco smoke has a direct carcinogenic effect on the epithelial cells of the oral mucous membranes. It has been well demonstrated that there is a dose-response relationship for tobacco use and the risk of the development of oral cancer (Fig. 3). From earlier studies it appeared that those who smoked pipe or cigars probably experienced a risk of oral cancer lower than that of cigarette smokers.⁵⁵ However, it has been concluded recently that the risks of oral cancer are similar for cigar smokers and cigarette smokers.⁵⁶ Cessation of cigarette smoking

eliminates the increased risk of development of oral cancer within 5–10 years.⁵⁷

Smokers who do not use alcohol have a two- to four-fold risk of developing oral cancer than tobacco and alcohol abstainers; the oral cancer risk of smokers who are heavy drinkers is 6–15 times greater than that of non-smokers/non-drinkers (Fig. 4).^{55,57–59} Alcohol increases the permeability of the oral mucous membranes, thereby

probably enhancing the carcinogenic effect of tobacco products.⁶⁰

Oral precancer

Of the potentially malignant lesions of the oral mucous membranes, the so-called leukoplakia is the most common (Figs 5 and 6). Leukoplakia occurs six times more frequently in smokers than in non-smokers.⁶¹ There is a dose-response relationship between tobacco usage and the prevalence of oral leukoplakia. Reducing or cessation of tobacco use may result in the regression or disappearance of oral leukoplakia.^{62,63}

Smoker's palate

Heavy smokers, especially pipe-smokers, frequently develop white changes in the hard palate, often combined with multiple red dots sometimes located centrally in small elevated nodules (Fig. 7).⁶⁴ Smoker's palate is asymptomatic and it disappears shortly after cessation of the smoking habit (Fig. 8). It is not premalignant. The palatal keratosis associated with reverse smoking, as it can be seen in some



Fig. 5 Leukoplakia of the commissure in a heavy smoker



Fig. 6 Malignant transformation of a leukoplakia of the tongue in a smoker

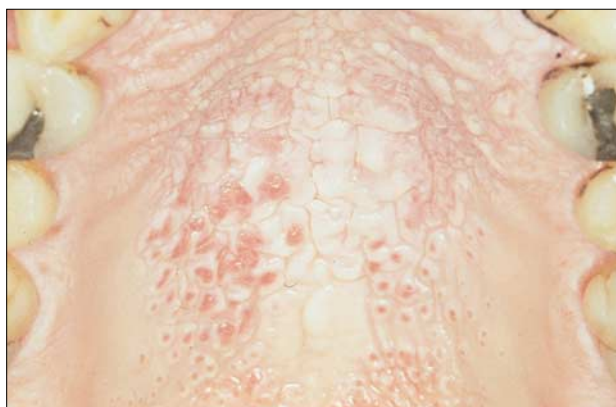


Fig. 7 Smoker's palate in a pipe smoker



Fig. 8 The same patient 2 months after smoking cessation

parts of the world, is a premalignant lesion.⁶⁵

Smoker's melanosis

Melanin pigmentation of the oral mucous membranes is normally seen in coloured races. In north European Caucasians, however, it is far less prevalent (about 10%) and normally with a subtle appearance. Heavy cigarette smokers show a pigmentation prevalence of about 30%⁶⁶ thus giving rise to the designation smoker's melanosis⁶⁷ which is most prevalent on the attached gingiva (Fig. 9). There are no symptoms, the change is not premalignant, and the pigmentation is reversible although it usually takes a year or more after cessation of the smoking habit.^{66,68}

Oral candidosis

Several factors predispose to oral candidosis. Whether tobacco smoking is one of them has been discussed for years, but during the past two decades a number of studies

have found that smoking, either alone or in combination with other factors, appears to be an important predisposing factor for oral candidosis although the exact pathogenic influence of smoking is not revealed.⁶⁹⁻⁷¹

Patients continuing the smoking habit after cessation of antimycotic therapy had relapses of the candidal infection in all

cases.⁶⁹ Furthermore, it is a clinical experience that some candida infections disappear following smoking cessation alone.

Oral smokeless tobacco-induced changes

The use of smokeless tobacco is well known to induce wrinkled changes in the oral mucosa at the site where the quid is placed.⁷²⁻⁷⁵ The changes are usually discreet with a colour similar to the surrounding oral mucous membranes or whitish-yellowish to brown. The changes seem to be reversible.⁷⁶ Gingival recessions may also be seen in users of smokeless tobacco at the site where the quid is placed.⁷²

The relationship between the use of oral smokeless tobacco and cancer development has been amply discussed. Snuff use as practiced in Scandinavia carry none or very low risks for development of oral cancer.⁷³ Differences in habits and between products around the world are probably important when evaluating the harmful effects of oral smokeless tobacco, so that a general statement on this subject cannot be made. However, many forms of oral smokeless tobacco in common use in Asia, the Middle East, and North Africa are carcinogenic.

Tobacco intervention and the dentist

Helping patients to stop using tobacco may be the single most important service dentists can provide for their patients' general health.⁷⁷

Dentists probably have the highest access to 'healthy' smokers in the healthcare sys-



Fig. 9 Smoker's melanosis in a heavy cigarette smoker

tem and a direct professional interest because of the harmful effects of smoking in the oral cavity. The term 'tobacco intervention' includes at least three different strategies:⁷⁸

- To reduce recruitment of new tobacco users — mainly young people
- To support and assist tobacco users who wish to quit
- To avoid that non-smokers — including the foetus — suffer because of other people's smoking.

Preferably, dentists should be competent in all three areas. The phrase 'Helping patients and the public to be tobacco free' encompasses these components.

The FDI has adopted a Position Statement on Tobacco in 1996 dealing with:⁷⁹

- Tobacco in daily practice
- Tobacco in all education
- Protect the children
- Prevent the initiation.

Tobacco in daily practice

The use of tobacco is harmful to general health, as it is a common cause of addiction, preventable illness, disability, and death. The use of tobacco also causes an increased risk for oral cancer, periodontal diseases and other deleterious oral conditions, and it adversely affects the outcome of oral care.

The FDI urges its Member Associations and all oral health professionals to take decisive actions to reduce tobacco use and nicotine addiction among the general public.

The FDI also urges all oral health professionals to integrate tobacco use prevention and cessation services into their routine and daily practice.

All National Dental Associations should be encouraged to adopt tobacco-free policies which could be based on the FDI Position Statement. The British Dental Association concurs.

The entire dental team should be aware of the relationship between smoking and dental problems. The clinic staff should be encouraged to actively participate in tobacco intervention routines and to convey the message that non-smoking is the norm.⁸⁰

Thus, current facts on tobacco and oral

health and smoking counselling should be a fundamental part of the dental curriculum and any practice prevention programme.

The goal is to ensure that all patients who smoke are routinely identified, monitored, and appropriately taken care of. The EU Working Group on Tobacco and Oral Health is currently developing practical guidelines on how to help patients stop smoking, based on that recently published by the UK Health Education Authority.⁸¹

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