Oral Diseases (1998) 4, 48–67

MEETING REPORT

EU Working Group on Tobacco and Oral Health

The first meeting of the 'EU-Working Group on Tobacco and Oral Health' was held in Copenhagen from 23 to 26 October 1997. The Working Group is supported by a grant from the EU-Commission (the 'Europe Against Cancer' Programme) and has the following objectives:

(1) to incorporate tobacco prevention in dental curricula;
(2) to make private practitioners and dentists in the public sector aware of their roles in the management and prevention of oral tobacco-related lesions; and
(3) to train dentists in incorporating oral tobacco-related lesions and the prevention of these in the examination of patients of all ages.

In an attempt to ensure both the scientific content of the work and at the same time the support of the National Dental Associations in conveying the message to dentists in each of the EU-countries, the Working Group consists of two members from each EU-country, one representing the Dental Schools and one representing the National Dental Associations.

Tobacco use is a modifiable risk factor for oral and general disease, and dentists could play a major role in its modification. Among the various health staff groups, dentists are those most frequently in contact with the population—even in the absence of symptoms, i.e. 'healthy patients'. The clinical manifestations of several oral lesions known to be caused in whole or in part by tobacco are well known. Further, even in the absence of tobacco-related diseases in the mouth the dentist will easily recognize the patient's smoking status. These facts place dentists in a favourable position in connection with the prevention of tobacco-related diseases.

Studies from several European countries have shown a need for improvement of dental curricula and postgraduate training in relation to anti-smoking counselling and harmful effects of tobacco in the mouth. In the US the American Dental Association and the National Cancer Institute/National Institute of Dental Research have successfully prepared instructional material and training programmes for the dental profession and patients. The initiation of a broad training and information effort towards dentists in Europe would be a natural consequence of these facts.

The first meeting of the Working Group was arranged as a consensus meeting focusing on the possible effects of tobacco in the mouth. Experts were invited to review the current knowledge in the field followed by group work and panel discussions. The following summaries were prepared by the speakers at the meeting.

In order to know what material is needed and to be able to measure the effect of the efforts in the field it was decided to prepare a questionnaire on the EU-dentists' knowledge, attitude and behaviour on the subject of tobacco and oral health. Further, on the basis of the reviews and the discussions at the meeting, written material will be produced and reviewed by communication expert(s) before the next meeting in the Working Group in June 1998.

It is our hope that this initiative, together with the actions of numerous other bodies, will make dentists aware of their role in tobacco prevention throughout Europe.

Jahn Legarth
Jesper Reibel
Working Group Coordinators

EU-Working Group on Tobacco and Oral Health Consensus meeting Copenhagen: 23–26 October 1997

Programme

Friday 24 October

9.00 Introduction
Welcome remarks
Background for the Working Group
Objectives for the Working Group
Jahn Legarth
Copenhagen, Denmark

9.15 Objectives for the Consensus Meeting
Jesper Reibel
University of Copenhagen, Denmark

9.30 Tobacco and general health
PE Nielsen
Danish Council on Smoking and Health, Denmark

10.00 Tobacco and oral cancer and precancer
Isaac van der Waal
University Hospital Vrije Universiteit, Amsterdam

10.30 Tobacco and oral candidosis
Palle Holmstrup
University of Copenhagen, Denmark

10.50 Coffee/tea

11.20 Tobacco and other oral mucosal diseases
Tony Axéll
Oslo, Norway

11.40 Smokeless tobacco and oral health—the Swedish experience
Tony Axéll
Oslo, Norway

12.00 Smokeless tobacco and oral health—the Irish/EU experience
Bernard McCartan
Trinity College, Dublin, Ireland

12.30 Lunch

14.00 Tobacco and periodontal disease
Hans Preber
Karolinska Institutet, Stockholm, Sweden

14.40 Tobacco and saliva/dental caries
David B Ferguson
University of Manchester, UK

15.10 Tobacco and dental implants: tobacco and wound healing
Crawford Bain
Glasgow Dental Hospital and School, Scotland

15.30 Coffee/tea

16.00 Tobacco prevention and the health personnel
A Hirsch
Hôpital Saint-Louis, France

16.45 Tobacco prevention and dentists—the experience from USA
Robert Mecklenburg
National Cancer Institute, Bethesda, USA

17.30 Tobacco prevention in dental practice: Strategies, guidelines and goals
Ö Åkerberg
Centre for Public Health, Mariestad, Sweden

18.00 End of Meeting

HEALTH CONSEQUENCES OF SMOKING

Poul Ebbe Nielsen, Chief Physician, Medical Department, Frederikssund Hospital, and Chairman of Danish Council on Smoking and Health, Denmark

Smoking of tobacco is responsible for an extremely high proportion of deaths and chronic diseases in the western world and to increasing illness in the developing countries. It can be calculated that about 3 million people are dying every year due to smoking. Of these, 2 million deaths are occurring in the industrialized countries. By the year 2025 about 10 million people will be killed every year by smoking.

In the EU-region about half-a-million people are dying every year due to smoking. Total calculated deaths in Denmark explained by smoking were 11446 in 1995. Of these 5179 were before the age of 75.

It can be concluded that tobacco smoking is an epidemic, and the most important preventable epidemic, in the western world. In spite of this the amount of money spent in the different countries in order to prevent tobacco-induced diseases is extremely low compared to the taxes coming in from the sale of tobacco, and also compared to the money spent on advertising in order to recruit new smokers and increase sales.

Proportion of the diseases caused by smoking

From several cohort studies as well as case control studies the proportion of the different diseases caused by tobacco smoking can be calculated (aetiological fraction).

It can be concluded that about 30% of all cancer diseases and deaths, 90% of all lung cancer, 30% of all cases of ischaemic heart disease and cerebral strokes, and 70% of chronic lung diseases (smokers lung) are directly caused by tobacco smoking. That means, that if smoking does not exist, then the above-mentioned proportions do not exist.

The aetiological fraction can vary from country to country, but the above-mentioned fractions are the best estimates for North European countries. For younger people suffering from ischaemic heart disease, the aetiological fraction seems to be much higher. In subjects suffering from myocardial infarction before the age of 50 years about 80% of the cases are explained by tobacco smoking.

Life expectancy in relation to smoking

The famous British Doctors Study, where about 40 000 medical doctors have been followed since 1955, 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. The proportion of smokers reaching the age of 70 was in that study 59% compared to 80% among non-smokers.

The proportions reaching the age of 85 were 12% and 33% respectively. Similar results have been found in Danish cohort studies. It is clear from these analyses that a very large proportion of smokers are dying before pensionable age, which is very different from what people normally think about the problem. The study of British doctors also showed that smoking cessation before the age of 35 years gave a life expectancy similar to non-smokers, and that cessation even in the older age groups resulted in a greater proportion reaching pensionable age with a decreased proportion in relation to age of smoking cessation.

Smoking and cancer

As already mentioned about 30% of all cancer cases and deaths are explained by smoking. It seems clear that the tar in tobacco is responsible for cancer development. The lower the tar content, the lower the risk. The EU has, during the last years, regulated the tar content which, from 1998, must not exceed 12 mg per cigarette.

It can be calculated that 90% of lung cancer in males and about 70% in females, about 60% of cancers of the mouth, throat and oesophagus, about 60% of cancer in the urinary bladder in males and 25% in females, and about 25% of cancers of the pancreas are caused directly by tobacco smoking.

The most important malignancy related to smoking is lung cancer. Total deaths in the EU-region are now about 90 000 per year. In Denmark it is about 3000 annually. There is only a very small difference between incidence data and death calculations, explained by the very poor long-term prognosis of this disease: the 5-year survival rate is only between 5 and 10% with some small interesting differences between different countries, probably explained by differences in the different histological subgroups of lung cancer, and differences in the primary health examination in people with initial symptoms related to the disease.

Smoking and lung diseases

Smoking causes an increased risk of developing asthma in genetically disposed subjects and progression of asthma in subjects with the disease. The most important chronic lung disease related to smoking is chronic obstructive lung disease, also called 'smokers lung' or COLD. COLD is divided into emphysema and chronic bronchitis. In nearly all subjects both diseases can be shown. Some of them have asthma too. Normally lung function is decreasing with age. In smokers this decrease is much higher than among non-smokers, especially in a subgroup of about 20% of the smokers. It is not yet possible by screening young smokers using lung function tests to find this subgroup. The end stage of COLD is increasing respiratory insufficiency and death. In Denmark about 2900 annual deaths are occurring due to respiratory insufficiency.

Smoking and the cardiovascular system

The immediate effect of smoking on the cardiovascular system is increasing heart rate, peripheral vasoconstriction, and release of catecholamines from suprarenal glands. The long term effects are very complex. Smokers normally have a little lower blood pressure than non-smokers, although heavy smoking can cause high blood pressure. The risk of developing myocardial infarction is doubled in smokers compared to non-smokers. On average, smokers are developing myocardial infarction 10 years before non-smokers. There is no difference between different tobacco products apart from smokeless tobacco. This is contrary to cancer risk, which is much higher for cigarette smokers compared to smokers of other types of tobacco.

Smokers who inhale have a much higher risk than those who do not. The risk is increased with the increasing

amount of tobacco used (daily consumption as well as total life consumption (pack-years) i.e. calculated number of years with a daily consumption of 20 g tobacco = 20 cigarettes). There is a curvilinear relationship between amount of tobacco and risk, i.e. a bigger difference between non-smokers and 'light' smokers (daily consumption up to 5 g) than between light and heavy smokers. The risk is increasing with other cardiovascular risk factors (cholesterol, hypertension, overweight). Before the age of 50 there exists an interesting sex difference with a much higher risk among males compared to females, probably explained by a vasodilating effect of estrogens. Sex differences disappear with increasing age.

The reason for developing myocardial infarction to a greater extent at a younger age among smokers is not more pronounced coronary sclerosis, but increased platelet aggregability, increased P-fibrinogen and decreased fibrinolytic activity. One can say that thrombosis of the coronary vessels among smokers is developed in arteries with a lesser degree of arteriosclerosis than among non-smokers. This, together with the above mentioned age maxima explain why hospital mortality after myocardial infarction is lower among smokers than among non-smokers.

Arteriosclerosis (i.e. plaque-development) is increased among smokers in other parts of the arterial tree - like carotid arteries, aorta and leg arteries. About 75–85% of all cases of intermittent claudication and aortic aneurysms are due to smoking.

One hundred per cent of Bürger Disease (thrombangiitis obliterans) is due to smoking. Therapy of this disease is abstaining from smoking.

Effect of smoking cessation

The risk for developing myocardial infarction and stroke decreases rapidly after smoking cessation, but first after 5–10 years cessation ex-smokers have reached the same risk as never-smokers. The risk for cancer development is decreasing much slower after cessation, and nearly all studies show an increased risk of ex-smokers compared to never-smokers. Patients with myocardial infarction who stopped smoking after infarction halved their risk of infarction re-occurring as well as death compared to patients continuing to smoke after AMI. Similarly patients with intermittent claudication halved their risk of amputation as well as of reconstructive surgery.

New symptoms after bypass surgery and balloon dilatation are much more common in patients continuing to smoke after intervention.

The most important therapy in patients with chronic lung disease (COLD) is smoking cessation. Several of these subjects are unnecessarily treated with asthma-medication and accepted continuous smoking.

Recently it has been discussed whether nicotine supplementation, which has a scientifically-based place in smoking cessation programmes, could give rise to increased risk of platelet aggregation and, secondary to that, an increased risk of myocardial infarction. Until now no proof exists, but further studies are required.

Passive smoking and health consequences

It is well-known from epidemiological studies that a very high proportion of the population are complaining of discomfort from passive smoking. Recent Danish studies have shown about 45% among non-smokers and about 25% among smokers. Workplace analyses have shown that about 40% of smokers and 20% of non-smokers are exposed to passive smoking at their work site.

Passive smoking can cause increased allergic reactions against specific allergens, reduced lung function and increased asthmatic attacks in patients with asthma, while allergy against tobacco smoke is questionable.

Several meta-analyses have shown that passive smoking can cause lung cancer as well as ischaemic heart disease (RR about 1.30). These calculations are based on cohort studies comparing non-smoking women married with smoking compared to non-smoking males. It should be stressed that the risk related to passive smoking is much lower than the risk related to active smoking. From the meta-analyses we have an example calculated for Denmark, that maximal about 65 of the 3200 annual cases of lung cancer are explained by passive smoking.

The risk of developing serious diseases due to passive smoking is the reason why passive smoking is a serious workplace problem, which can only be solved by introducing a smoking policy, where the goal should be that nobody should be exposed to passive smoking against their will.

The health consequences for the foetus exposed to mothers smoking lies outside this overview. It should be stated that smoking during pregnancy might have serious consequences for the foetus as well as during the first life years. Asthmatic bronchitis among small children is to a great extent explained by the mother's smoking both before and after pregnancy. Infectious diseases in the upper respiratory tract are also much more common among children exposed to passive smoking than non-exposed children.

Other health consequences of smoking

Smokers are developing increased osteoporosis and fractures due to weakening of the bones. Smokers have an increased risk of developing discus degeneration and prolapse in the lumbar region. Several new studies have shown that smokers have decreased wound healing after operation and increased postoperative wound infections. Some studies have shown decreased fertility and decreased semen quality among smokers. This knowledge has until now only been used sporadically in the advice to parents with decreased fertility. Skin elasticity seems to be decreased in smokers giving rise to the characteristic changes in the face of smokers.

One disease, colitis ulcerosa, is less common among smokers than non-smokers. The reason is probably a direct effect of nicotine on the surface cells of the intestinal tract, as it has been shown that nicotine supplementation reduces the symptoms from this chronic disease. It has been claimed, but there is until now no firm scientific data, that smokers have a decreased risk for Alzheimers disease and Parkinson's disease.

Smoking policy in the EU-region

As already mentioned about half-a-million deaths are occurring in the EU-region per year due to tobacco smoking. In the same region taxes from tobacco sales amount to 36 billion ECU (216 milliard Danish crowns), but only about 15 million ECU are used on health promotion (in the Europe against Cancer Project). Subsidies for tobacco production in the southern countries of the EU has an amount of 993 million ECU. The very low budget for health promotion is the reason why remarks are made about the double moral standards of the EU. A milestone in the EU-region seems to be the ban on tobacco advertising, which was passed in December 1997.

In Denmark, tobacco taxes are about 1.4 billion ECU. The total amount spent on health promotion related to smoking is about 2.5 million ECU. The annual budget of the Danish Council on Smoking and Health is about 1.8 million ECU.

Health professionals and their organisations have a great responsibility for the future health promotion against the epidemic of smoking.

TOBACCO AND ORAL CANCER AND PRE-CANCER

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Introduction

Of all cancers that may occur in the body some 2% are located in the oral cavity. The majority of these oral cancers consists of squamous cell carcinomas that arise from the mucosal surface. Oral cancer mainly affects middle-aged and elderly people and is more common in men than in women. The incidence figures, representing the number of newly diagnosed oral cancer patients per year per 100 000 population vary worldwide, even between the European countries (Table 1) (Parkin *et al*, 1992). Both the gender preference and the geographical differences can largely be explained by differences in lifestyle, of which the use of tobacco, alcohol and an unbalanced diet with regard to fruit and vegetables are important components (La Vecchia *et al*, 1997).

Tobacco and oral cancer

Tobacco smoke has a direct carcinogenic effect on the epithelial cells of the oral mucosa. It has been well demonstrated that there is a dose-response relationship for tobacco use and the risk of the development of oral cancer. Those who smoke pipes or cigars probably experience a risk of oral cancer lower than that of cigarette smokers (Wynder *et al*, 1977). Cessation of cigarette smoking eliminates the increased risk of development of oral cancer within 5–10 years (Blot *et al*, 1988).

Smokers who do not use alcohol, have a two- to four-fold risk of developing oral cancer as do 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 (Wynder *et al*, 1977; McCoy and Wynder, 1979; Blot *et al*, 1988; Kato and Nomura, 1994).

Table 1 Incidence rate of oral cancer (ICD-0, C00, C02-C05*), age standardized, per year per 100 000 population in certain European countries in the period 1983–1987 (Parkin *et al*, 1992)

Country (Region)	*Women*	*Men*
Denmark	1.8	5.9
Finland	1.6	6.2
France (Bas-Rhin)	2.0	24.0
Germany (Federal Republic)	0.8	4.9
Hungary (County Vas)	3.0	16.1
Iceland	2.6	5.3
Ireland (Southern)	1.1	5.5
Italy (Torino)	1.9	7.4
Netherlands (Eindhoven)	1.7	4.2
Norway	1.8	6.0
Poland (Warsaw City)	1.3	5.7
Portugal	2.3	8.4
Romania (City Cluj)	1.7	7.9
Russia (St. Petersburg)	1.0	8.9
Spain (Granada)	1.3	17.5
Sweden	1.6	4.7
Switzerland (Geneva)	2.0	10.6
UK, England and Wales	1.0	2.9

* Topography code of the WHO International Classification of Diseases for Oncology (ICD-O), 2nd edn (C Perry, W van Holten, C Muir), WHO, Geneva (1990)

Alcohol increases the permeability of the oral mucosa, thereby enhancing the carcinogenic effect of tobacco products (Squier *et al*, 1986; Lesch *et al*, 1989; Hsu *et al*, 1991). The effect of dentition and oral hygiene on the incidence of oral cancer has been shown to be modest, if at all, particularly when compared with that of cigarette smoking and alcohol consumption (Marshall *et al*, 1992).

In a group of 690 patients it was shown that oral cancer in the retromolar area and in the floor of the mouth was significantly related to the use of tobacco than cancers at other oral sites, such as the tongue and the cheek (Jovanovic *et al*, 1993). In another series of 359 male patients, tobacco smoking was more strongly associated with soft-palate lesions than in lesions in more anterior sites; patients with cancer of the floor of the mouth and tongue had higher odds ratios for alcohol drinking than subjects with cancer of other sites (Boffetta *et al*, 1992).

Tobacco and oral precancer

Of the potentially malignant lesions of the oral mucosa, the so-called leukoplakia is the most common. The reported prevalence varies from 0.2% to 4.9% (Axéll, 1976; Gupta *et al*, 1980). Incidence figures, only available from studies in India, range from 0.2 to 2.4 per 1000 population per year (Gupta *et al*, 1980). There is a peak incidence above the age of 50 years. The gender distribution varies in different parts of the world.

Leukoplakia occurs six times more frequently in smokers than in non-smokers (Baric *et al*, 1982). 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 (Roed-Petersen, 1981; Gupta *et al*, 1995). On the other hand, disappearance of leukoplakia has occasionally been reported in patients who continued to smoke (Silverman and Rozen, 1968).

The malignant transformation rate of leukoplakia varies widely in the numerous reports on this subject, ranging from almost 0% to more than 20%. The differences can be partly explained by differences in the population sample and the observation time. Paradoxically, patients with oral leukoplakia who are non-smokers are at a higher risk of such malignant transformation (Silverman, 1990).

Conclusion

There is overwhelming evidence of the adverse effect of tobacco usage with regard to the development of oral cancer and precancer. The dentist should be aware of this adverse effect, including the synergistic effect of alcohol.

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TOBACCO AND ORAL CANDIDOSIS

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Candida albicans is the most common infecting yeast of the oral cavity. However, the organism is in fact not very pathogenic. Being a commensal which is commonly isolated from the oral cavity, it only infects the host if it gets the chance to do so. This means that oral candidosis is a disorder which occurs in patients with inherent predisposing factors. Several general and local factors have been identified so far. Among general factors some of the most important are drugs and diseases affecting the inflammatory defence system, and among local factors are drugs, radiation therapy, oral hygiene and lesions of the oral mucosa (Samaranayake, 1990). Whether tobacco smoking is one of the predisposing factors has been discussed for years, but during the past two decades a number of studies have supported that tobacco smoking, either alone or in combination with other factors, appears to be an important predisposing factor for the establishment of oral candidosis although the exact pathogenic influence of smoking is not yet revealed.

Among 32 patients with chronic oral multifocal candidosis all were tobacco smokers. Furthermore, patients who continued tobacco smoking after cessation of antimycotic therapy had relapses of the candidal infection in all cases (Holmstrup and Bessermann, 1983). Significantly more of patients (85%) with median rhomboid glossitis were tobacco smokers as compared with the corresponding proportion (41%) of the healthy age and sex-matched control individuals (Arendorf and Walker, 1984). An investigation of the effect of cannabis smoking combined with methaqualone and tobacco on candidal prevalence, density and oral candidosis compared similar findings in age and sex-matched tobacco smoking and non-smoking controls. The study demonstrated an increased prevalence and density of *Candida albicans* in cannabis users while there was no apparent difference in the prevalence of candidosis (Darling *et al*, 1990). A study of the oral presence of *Candida*-strains in healthy dentate adults and in patients with oral leukoplakia and erythematous oral candidosis also supported that tobacco smoking may be a predisposing factor for candidal infection (Rindum *et al*, 1994).

Several studies on the significance of tobacco smoking for the occurrence of candidal infection have been conducted among HIV-infected individuals. Among 14 HIV-seropositive homosexual men 13 (93%) had oral candidosis whereas this disorder was found in 26 (50%) out of 52 seronegative homosexual men. In the latter group oral candidosis was correlated to cigarette smoking (Syrjanen *et al*, 1988). Tobacco use was related to increased occurrence of virus and yeast infections of the oral mucosa and to periodontal diseases among 230 military personnel who were HIV-infected (Swango *et al*, 1991). Among HIV-seroposi-

tive homosexual men, presence of white lesions on the tongue, oral candidosis and gingival erythematous banding was significantly associated with decreased levels of CD4, and positive associations were seen with current smoking, antiviral and antibiotic drug use (Lamster *et al*, 1994). In another study of oral manifestations of HIV-infection, smoking was identified as a strong aetiological factor (Palmer *et al*, 1996). An investigation of the response to systemic antimycotic treatment with ketoconazole in HIV-infected patients showed that non-smokers were more likely to respond to the treatment than smokers (Silverman *et al*, 1996). Further, cigarette smoking HIV-infected individuals developed oral candidosis more quickly than did non-smokers (Conley *et al*, 1996).

Clinical and laboratory investigations of oral candidosis in psychiatric patients under treatment with psychotropic drugs and who were wearing complete upper dentures compared with non-psychiatric controls revealed *Candida* among 64.7% compared to 33.3% in the controls. Cigarette smoking, sugar consumption and a poor standard of denture hygiene were more common in the psychiatric patients than in the controls (Lucas, 1993). Smoking and alcohol were also identified as risk factors for oral colonization by *Candida* during radiation therapy for malignant conditions of the head and neck (Epstein *et al*, 1993). Finally, full-denture-wearing individuals who were tobacco smokers had a significantly greater incidence of erythematous candidosis than the controls (Crockett *et al*, 1992).

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TOBACCO AND OTHER ORAL MUCOSAL DISEASES

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Tobacco use, especially in the form of smoked tobacco, is associated with several different changes of the oral mucosa, from 'physiological changes' such as leukoedema and smoker's melanosis to modification of clinical appearance of lesions, as is the case for oral lichen and recurrent aphthous ulcers.

Leukoedema

This 'physiological' condition comprises highly prevalent velvet-like diffuse veils most often seen in the buccal mucosa. It may also appear in the lip and vestibular mucosa, in the floor of the mouth and on the tongue. Clinical grading of severity has been published making it possible to relate severity to trauma, e.g. to tobacco habits (Axéll and Henricsson, 1981). The prevalence in non-tobacco users is 37% and in daily smokers 60%. Highest prevalence figures in tobacco users are found in heavy cigarette smokers—69%, and the lowest ones in snuff users—53%. Highest prevalence of moderate/severe changes is found in pipe smokers—8% as opposed to 1% in non-tobacco users.

Smoker's melanosis

Orally visible melanin pigmentation appears in about 100% in coloured races. In North European Caucasians it is far less prevalent—about 10%, but then normally with a subtle appearance. It has been stated that 'physiological' pigmentation only counts for about 3% and that tobacco smoking causes a considerable increase of prevalence. Heavy cigarette smokers show a pigmentation prevalence of about 30% while snuff users show almost no rise of prevalence—5% (Axéll and Hedin, 1982). This is a reason why the pigmentation, which is most prevalent on the attached gingiva, has been called smoker's melanosis (Hedin, 1977). The change is reversible and, thus, it disappears about 2 years after cessation of habit (Axéll and Hedin, 1982; Hedin *et al*, 1993).

Smoker's palate

The hard palate of heavy smokers, especially pipe and cigar/cheroot smokers, frequently shows an apparently whitish mucosa. Sometimes it is combined with wrinkling and/or the appearance of multiple nodules with red, centric dots. Depending on which criteria have been used, preva-

lence varies from 1–6% in Scandinavia (Axéll, 1976; Saietz, 1975). Smoker's palate is reversible and non-precancerous as opposed to smoker's palatal keratosis encountered in subjects practising reverse smoking and most frequently found in Indian women (Mehta *et al*, 1977), but also in the Caribbean islands and among men in Sardinia. Tobacco habits, especially smoking habits, cause modification(s) of the clinical appearance/spectrum of several oral mucosal lesions.

Lichen planus

The prevalence of oral lichen planus is about 1–2% (Kleinman *et al*, 1991). The oral manifestations have been subgrouped into white and red forms. For all of them, there is a negative statistical relationship to tobacco smoking, for all except one—the plaque form of lichen (Neuman-Jensen *et al*, 1977; Axéll and Rundquist, 1987). Thus, differential diagnosis between tobacco-associated leukoplakia and plaque-lichen may offer a diagnostic challenge.

Recurrent aphthous ulcers

RAU may appear in many different clinical forms and at highly varying frequencies in one and the same individual. Typically, it is almost exclusively appearing on the non-keratinized mucosa (except on the tongue). Tobacco smoking seems to change the keratinization process also affecting the keratin filaments in the cells of the clinically non-keratinized mucosa. It has been shown that the prevalence of RAU is considerably lower among tobacco smokers than among non-tobacco users, the prevalence of episodes within a 2-year period being 14% and 22%, respectively, and for pipe smokers only 7% (Shapiro *et al*, 1970; Axéll and Henricsson, 1985).

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SMOKELESS TOBACCO AND ORAL HEALTH: THE SWEDISH EXPERIENCE

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The Swedish consumption figures for wet snuff are the highest in the world, about 0.7 kg per capita. In the age groups 15–75 years about 18% of the men and 2% of the women are habitual users of snuff and consuming at an average 19 g of loose snuff and 10 g of portion-bag-packed snuff.

The clinical characteristics of lesions associated with Swedish wet snuff have been carefully described in a series of papers since the mid-1970s, for reviews and details see Andersson (1991) and Axéll (1993). At the place where the quid is inserted almost invariably a clinically visible change appears. A 4-degree scale has been suggested for describing varying degrees of clinical 'severity' (Axéll *et al*, 1976), a scale that later has been been modified by American scientists to a 3-degree scale. Based on the clinical grading it has been shown that lesions become most pronounced after a high daily intake rather than after many years of consumption and also after use of loose snuff as compared to use of portion-bag-packed snuff. The snuff-induced changes also seem to be reversible.

For chewing tobacco there are very low sales figures in Scandinavia. Only about 15 tons are sold in Sweden as compared to about 5000 tons for snuff. Changes of the oral mucosa are very discrete and the histopathological picture similar to the one of a leukoedema (Axéll *et al*, 1992).

The question of carcinogenicity of snuff has been vividly discussed in Scandinavia. The IARC statement of 1985 that there is sufficient evidence that snuff causes cancer (IARC, 1992) has to some extent been supported by some Swedish scientists, and questioned by others. The statement has led to a law which orders the manufacturers to label the snuff product with a the text 'Causes cancer'. In the light of previous research and recent Swedish studies the validity of this label has been seriously questioned at a symposium at the Swedish Board of Health and Welfare in September 1996.

One of those recent studies was a case control study on oral cancer in Northern Sweden. The number of cases were 418. Relative risk (RR) for developing oral/pharyngeal cancer in present snuff users was 0.7 (95% CI 0.4–4.1) and for previous snuff users 1.5 (95% CI 0.8–2.9) (Schildt *et al*, 1997). Another case control study comprised 128 cases of oral cancer. RR for present snuff users was 1.0 (95% CI 0.7–1.6) and for previous snuff users 1.2 (95% CI 0.6–1.9) (Lewin *et al*, 1997).

Also the risk for circulatory and heart diseases have been evaluated in recent Swedish studies. One case control study comprised 585 cases with 10% snuff users and 589 controls with 15% snuff users. RR for myocardial infarction in snuff users was 0.89 (95% CI 0.62–1.29) as compared to 1.87 (95% CI 1.40–2.48) for tobacco smokers (Huhtasaari *et al*, 1992). In another study data from health screenings of 135 036 construction workers (6297 snuff users) were analysed. The RR for habitual snuff users to die of cardio-

vascular disease was 1.4 (95% CI 1.2–1.6) as compared to the RR for smokers 1.9 (95% CI 1.7–2.2) (Bolinder *et al*, 1994).

No increased risk among snuff users who had never smoked was registered in a recent case control study among men with the inflammatory bowel diseases Crohn's disease and ulcerative colitis (Persson *et al*, 1993).

Thus, it appears that snuff habits as they appear in Scandinavia carry very low risks for contributing to serious health hazards including oral cancer. However, this does not mean that use of snuff should be encouraged—on the contrary. The fact that no serious local or general health hazards have been detected in Scandinavian epidemiological studies does not guarantee an absence of such risks. Further, it is an unequivocal fact that using snuff almost always causes lesions of the oral mucosa, damages salivary glands and also in some cases causes gingival recessions.

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SMOKELESS TOBACCO: THE IRISH AND EU EXPERIENCE

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Smokeless tobaccos were used in Ireland within living memory, especially in rural areas. A small number of men chewed tobacco and there was the occasional use of nasal stuff by both men and women. These practices have fallen into disuse and would no longer be socially acceptable. There does not appear to be any history in Ireland of moist snuff use or snuff dipping. Indeed, during oral oncology courses, the practice of snuff dipping has to be explained to our students.

Ireland and the UK have shared a common trade area going back to before our membership of the European Union. The Irish Health Department (Ministry of Health) became concerned, therefore, when it learnt in 1985 that a UK government agency had provided a development grant to enable the United States Tobacco Company to build a factory in Scotland for the manufacture of portion snuff to be sold as 'Skoal Bandits'. Later that year the Department of Health received a query from a potential importer asking if the product was covered by existing legislation on tobacco advertising, if the Minister felt that the product was harmful and if the Minister intended to introduce legislation to regulate the sale of portion snuffs. The Minister and his officials sought urgent assistance from the UK Health Department, the United States of America National Institutes for Health and the Massachusetts Department of Public Health. Following that advice it was decided to ban the importation and sale of portion snuffs using the only suitable existing legislation, the Health Act of 1947. Section 66 of the act applies to: "*any instrument, appliance or apparatus . . . the use of which by the general public . . . involves a risk of serious injury to health or body . . .*" and makes it an offence, "*unless authorised by the Minister, to import, manufacture, sell or otherwise dispose of or offer to keep for sale or other disposal, or advertise a restricted article . . .*" with a penalty of a £100 fine or 6 months imprisonment or both. Under this law the Minister issued the following statutory order: "*Whereas the Minister for Health is of the opinion that tobacco in the form of finely-cut, moist tobacco contained in sachets or pouches and intended for use by being placed in the mouth, is likely, when accessible to the general public, to be used for purposes involving risk of serious injury to health or body; now therefore the Minister for Health hereby orders as follows: tobacco in the form of finely-cut, moist tobacco contained in sachets or pouches and intended for use by being placed in the mouth, shall be a restricted article for the purposes of Section 66 of the Health Act 1947.*' (Health (Restricted Article) Order, 1995)

In 1987 United States Tobacco sought to overturn the ban in the High Court (US Tobacco Int. Inc, 1986). The President of the High Court ruled that as the Health Act 1947 covered "*medical and toilet preparations and certain other articles*", smokeless tobacco was too remote from that definition for parliament to have intended that it should be covered by the Act.

As the Irish parliament was then considering legislation to restrict the use of tobacco in places of public access, an amendment was urgently drafted to restore the *status quo ante*. This became the Tobacco (Health Promotion and Protection) Act 1988. Section 6 contains a complete ban on oral smokeless tobaccos: "*any person who imports, manufactures, sells or otherwise disposes of, or offers for sale or other disposal, or advertises, an oral smokeless tobacco product shall be guilty of an offence and shall be liable . . . on conviction . . . to a fine not exceeding £10 000.*

. . . In this section 'oral smokeless tobacco product' means any product or substance, made wholly or partly from tobacco, which is intended for use, unlit, by being placed in the mouth and kept there for a period, or by being placed in the mouth and sucked or chewed." This ban is more comprehensive than that under the 1947 Act which covered only portion snuffs.

Again United States Tobacco sought to challenge the law. Two principal reasons were advanced; one based on EU law and the balance between health protection and free movement of goods and the other based on a perceived imbalance between the risk to health and the remedy imposed by the Irish government *viz.* a complete ban rather than health warnings on packaging as with other tobacco products.

The case was heard in the High Court 1990 (US Tobacco (Ireland) Ltd, 1990). The case was regarded by the government as an important public health matter and a panel of expert witnesses was assembled from Ireland and the USA. The two sides were represented by very senior lawyers, each of whom was later to serve as Attorney General. Witnesses for the company were called from the UK, the USA and Sweden. Expert witnesses for the company argued that oral smokeless tobaccos were not carcinogenic and that while there would be other experts who would hold that they were carcinogenic, this was a matter of scientific controversy. There was conflict of evidence between expert witnesses for the company on the carcinogenic effects of nitrosamines in animal experiments and one expert witness for the company stated thtat he did not believe that smoking causes lung cancer. A dental expert called for the company argued that there was no significant evidence in Scandinavia for a carcinogenic effect from moist snuff but that other soft tissue changes were seen; he agreed that the ban was justified on those grounds. Evidence was heard that no tax had been paid on the manufacture or importation of chewing tobacco for many years. It was argued, therefore, that smokeless tobaccos were new products on the Irish market and could therefore be regulated differently from existing forms of tobacco. Judgment in favour of the government position was given in early 1991. The court ruled that the ban was justified both in Irish and in EU law and that individual EU member states had the right to decide on health and safety where the scientific evidence is uncertain. The judge rejected the contention that there was scientific controversy on the carcinogenic effects of smokeless tobacco. He ruled that printed warnings on packages would be insufficient for protection of the health of the public. Following this decision, United States Tobacco Ltd appealed against the finding to the Supreme Court.

While the case was in progress, the EU had been considering a ban on smokeless tobaccos and officials followed the progress of the Irish case with interest. An EU directive of 1989 had regulated the advertising of tobacco products (EU Directive, 1989). A new directive (EU Directive, 1994) amended the title of the 1989 directive to include the words "*and the prohibition of the marketing of certain types of tobacco for oral use,*' and a new Article 2(4) was added as follows: "'*Tobacco for oral use', for the purposes of Article 8a, means all products for oral use, except those intended to be smoked or chewed, made wholly or partly from tobacco, in powder or particulate form or in any combination of these forms—particularly those presented in sachet portions or porous sachets—or in a form resembling a food product.*" Article 8a states: "*Member states shall prohibit the placing on the market of tobacco for oral use as defined in Article 2(4).*"

In 1995, possibly in the light of the European ban, United States Tobacco Ltd withdrew its Supreme Court appeal against the Irish ban.

With the accession of Sweden to the EU in 1995, there arose a problem because of the traditional use of moist snuffs. Sweden was granted an exemption from the directive but the Swedish government was ordered to prohibit the export of smokeless tobacco products to other EU states.

The EU ban covers moist snuffs including portion snuffs, but exempts chewing tobaccos. The Irish ban covers all smokeless tobaccos. How should we now view those bans? Traditional Swedish use of moist snuffs does not appear to be associated with any significant increase in oral cancer incidence (Axéll *et al*, 1978). Chewing tobaccos, however, must remain suspect as the older evidence for carcinogenic effects has never been rebutted. Thus the EU ban may well have had the effect of prohibiting a relatively less harmful form of smokeless tobacco while exempting the harmful form.

If the ban on moist snuffs cannot be justified on grounds of carcinogenesis, can the ban be justified on other grounds? There are arguments both for and against. Certainly, if tobacco were a new product it would be banned on the grounds of the risk to health; all tobacco products cause some damage. Products marketed at teenagers are problematic; we do not know if use of smokeless products in teenagers leads to smoking later or if smokeless tobacco is a useful way to wean smokers off cigarettes, or, indeed, if smokeless tobacco is neutral in the initiation of smoking. Knowledge of these factors would have a considerable bearing on any study of the validity of the EU ban.

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TOBACCO AND PERIODONTAL DISEASE

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The role of tobacco, particularly tobacco smoking, in periodontal disease has been extensively studied for many years. An increasing amount of scientific data from recent years has demonstrated a clear association between smoking and the prevalence and severity of periodontal disease, suggesting smoking as an important risk factor for periodontal disease (Haber *et al*, 1993; Bergström and Preber, 1994; Zambon *et al*, 1996; Salvi *et al*, 1997).

Gingivitis

Many earlier studies on smoking and chronic gingivitis have reported both more gingival inflammation and more dental plaque and calculus in smokers (Arno *et al*, 1958; Alexander, 1970; Preber *et al*, 1973). However in recent studies, when the plaque level has been controlled, smokers have demonstrated less gingival inflammation and less bleeding on probing when compared to non-smokers, indicating a suppressed gingival inflammation (Bergström and Floderus-Myrhed, 1983; Bergström and Preber, 1986; Danielsen *et al*, 1990). The results from studies on smoking and acute necrotizing gingivitis (ANUG) are conclusive, suggesting an association between smoking and ANUG (Pindborg, 1947; Kardachi and Clarke, 1974).

Periodontitis

During the last 15 years a number of well controlled studies on larger groups of populations has unanimously demonstrated a clear relationship between smoking and periodontal disease (Ismail *et al*, 1983; Feldman *et al*, 1987; Goultschin *et al*, 1990; Grossi *et al*, 1995). The results from these studies suggest smokers 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.

Furthermore, risk assessments based on an increasing body of investigations over the past few years, suggest that the smoking attributable relative risk is considerable; 2.5 to 6.0 the risk of non-smokers (Bergström, 1989; Haber and Kent, 1992).

Microflora

Many clinical and epidemiological studies have reported smokers to harbour more supragingival plaque than non-smokers. Clinical studies have reported a similar plaque accumulation rate in smokers and non-smokers (Bastian and Waite, 1978; Bergström and Preber, 1986), thereby suggesting smokers' excess amount of plaque depending on inferior oral hygiene (Macgregor, 1984). Recent studies on patients with periodontal disease have not reported any differences in the composition of the subgingival periopathogenic microflora between smoking groups (Preber *et al*, 1992; Stoltenberg *et al*, 1993). However, in one recent study including a sample of the general population, microbiological analysis demonstrated that smokers harboured significantly higher levels of *B. forsythus* than non-smokers (Zambon *et al*, 1996).

Host response

Smoking has important effects on the immune system. Smokers have demonstrated decreased immunoglobulin levels of IgG, IgA and IgM, but increased levels of IgE. Lower T-suppressor lymphocyte counts and higher counts of other lymphocytes and lymphocyte DNA adducts have been reported for smokers. Smoking impairs the viability, chemotaxis and phagocytosis of oral neutrophils. Smokers have reported higher levels of TNF-α (Holt and Keast, 1977; Kenney *et al*, 1977; Johnson *et al*, 1990; Boström *et al*, 1998).

The exact mechanisms by which smoking affects the periodontal tissues are not known. Besides possible specific effects from the periopathogenic bacteria and by immunosuppression, there might also be cytotoxic effects from nicotine on the fibroblast function (Tipton and Dabbous, 1995).

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 periodontal therapy report inferior results in patients who smoke (Preber and Bergström, 1990; Kaldahl *et al*, 1996). Smokers have also been reported to have a poorer success rate with different kinds of periodontal regenerative surgery (Trombelli and Scabbia, 1997). Furthermore refractory periodontitis cases, i.e. patients who do not respond as expected following periodontal therapy, are heavily overrepresented in smokers (MacFarlane *et al*, 1992).

Smoking cessation

At present there are no studies on the effect of smoking cessation on periodontal disease. However, several studies report a greater prevalence and severity of disease in current smokers as compared to former smokers; and a relationship has also been demonstrated between the prevalence of disease and the number of cigarettes smoked per day and the number of smoking years; results that might indicate beneficial effects of smoking cessation (Bergström *et al*, 1991: Grossi *et al*, 1995). Furthermore in a recent longitudinal study (Bolin *et al*, 1993), 44 subjects gave up smoking during a 10-year period, demonstrating significantly less marginal bone loss as compared to the 139 who smoked regularly during the period.

Smokeless tobacco

Smokeless tobacco products and their effects on the periodontal tissues have not been extensively studied. Smokeless tobacco and snuff have long been associated with oral leukoplakia and local gingival recession at site of placement. However, data are currently insufficient to support an association between the use of smokeless tobacco and generalized or severe periodontal disease (Robertson *et al*, 1990).

Conclusion

- Tobacco smoking is an important risk factor for periodontal disease.

- The smoking attributable risk for periodontal disease is of the order of 2.5 to 6.0 or even greater.
- Tobacco smoking affects the prevalence and progression of adult periodontal disease and refractory periodontal disease.
- Tobacco smoking affects the prevalence of ANUG.
- Tobacco smoking suppresses symptoms of gingival inflammation.
- Smokers do not respond as favourably as non-smokers to periodontal therapy.
- Smokeless tobacco induces local gingival recession at site of placement.

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THE EFFECTS OF TOBACCO USAGE ON SALIVA FLOW RATE AND COMPOSITION AND ON SUSCEPTIBILITY TO DENTAL CARIES

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Abstract

The effects of tobacco usage on salivary flow rates, composition, and bacterial population are reviewed together with the evidence on caries incidence. The identity and effects of tobacco-derived substances dissolved in saliva are considered. Despite the immediate stimulation of saliva production by tobacco usage, there is no long term effect on stimulated or unstimulated saliva flow rates. Salivary pH and buffering power are slightly reduced in smokers and tobacco users have lower salivary lysozyme and lactoferrin concentrations. Tobacco users have higher salivary IgA concentrations but poorer binding to secretory component.

Lactobacillus and Streptococcus mutans counts are higher in tobacco users. Thiocyanate, nicotine and cotinine appear in the saliva of tobacco users and their concentrations can be used to monitor usage. These components and the soluble tobacco-specific nitrosamines can be genotoxic; they also have a deleterious effect on bone metabolism. Smokers often have a higher incidence of dental caries although there is no evidence of any aetiological relationship and the dental caries experience of smokeless tobacco users is similar to that of the non-tobacco-using population.

The effects of tobacco on saliva may be considered under headings of salivary flow, salivary composition, solution of tobacco components in saliva and their effects, and the effect on dental caries.

Tobacco usage and saliva

Effects of tobacco on saliva flow

Tobacco might affect salivary flow in the short term by stimulating or depressing flow and it might have a longer term effect on the glands.

In the short term, effects are those of direct stimulation of oral receptors since the nicotine concentrations that might reach parasympathetic ganglia are too low to act directly at the synapses. As long ago as 1776 (Murray, 1776) an increase in salivary flow was observed during smoking tobacco. More recent observations demonstrate that smoking increases the flow rate of both parotid (Barylko-Pikielna *et al*, 1968; Pangborn and Sharon, 1971), and whole saliva (Winsor, 1932). The observations of Heintze (1984) that smoking a cigarette did not increase the flow rate of paraffin-stimulated whole saliva does not contradict reports on a direct effect.

The data on long-term effects on salivary flow rates shows no difference between smokers and non-smokers in unstimulated whole saliva (Heintze, 1984; Lopez Jornet and Bermejo Fenoll, 1995) paraffin-stimulated whole saliva (Heintze, 1984; Parvinen, 1984; Olsen *et al*, 1985), unstimulated (Chandler *et al*, 1974) or stimulated parotid saliva (Chandler *et al*, 1974; Baum, 1981).

The short term stimulatory effects of chewed snuff and nicotine-containing chewing gums have also been reported on: chewing snuff increases whole saliva flow rates by at least twice (Magnusson, 1991), but nicotine-containing chewing gum is no more effective in stimulating flow than is non-nicotine-containing gum (Duner-Engstrom *et al*, 1986).

Intrinsic changes in salivary composition

Salivary composition has been studied in smokers and smokeless tobacco users primarily to see whether any changes might affect the progress of dental caries.

As both the pH and buffering power of saliva show a positive correlation with flow rate, one would anticipate that they should increase during stimulation by tobacco products but otherwise be similar in tobacco users and non-users.

The pH of saliva rises during smoking (Kirsch, 1963; Kenney *et al*, 1975), probably as a result of the increased salivary flow rate. Tobacco smoke itself is said to be acid in solution. Over longer time periods smokers have a lower pH in paraffin stimulated whole saliva and the pH is negatively correlated with saliva nicotine concentration (Parvinen, 1984). This confirmed the earlier observation that, despite the higher pH during smoking, salivary pH in smokers was normally lower (Kenney *et al*, 1975). There is also, however, a report that no difference was observed (Courant, 1967).

Buffer capacity of paraffin-stimulated whole saliva was found to be lower in smokers: 4.6 *vs* 6.2 as final pH (Heintze, 1984) (*cf.* pancreatic HCO3 reduction in smokers). This was not confirmed in a later study with approximately an equal number of subjects (Olsen *et al*, 1985), but more recently in a comparison of smokers and snuff chewers, the former but not the latter were found to have reduced buffering capacity (Wikner and Soder, 1994).

The total protein concentration in non-stimulated whole saliva was similar in smokers and non-smokers as was the amylase activity (Nagaya and Okuno, 1993). However there are consistent reports that IgA and IgA2 and J chain concentrations are higher in smokeless tobacco users and passive smokers (Wagner *et al*, 1987; Gregory *et al*, 1990, 1991) and lysozyme and lactoferrin concentrations are lower (Gregory *et al*, 1991). It is also reported that the binding of the secretory component to the IgA/J complex is reduced in smokeless tobacco users (Gregory *et al*, 1990).

There is a report from Russia that the activity of inhibitors in saliva of proteolytic enzymes is lowered by tobacco smoke (Danielvskii and Ischenko, 1990).

Micro-organisms in saliva

The changes in salivary flow rate and composition observed in tobacco users might be expected to affect the oral flora. In particular, changes in pH, in antibacterial factors and in specific tobacco products present in saliva might cause changes in oral ecology reflected in the organisms which can be grown from salivary samples.

Saliva lactobacillus and acidogenic bacteria counts have been used as indicators of caries risk. Lactobacillus counts are higher in smokers (Heintze, 1984; Parvinen, 1984; Sakki and Knuuttila, 1996). Streptococcus mutans counts have also been found to be higher by many workers

(Heintze, 1984; Sakki and Knuuttila, 1996). Gregory *et al* (1991) found only slight differences in antibody titre towards Strep. mutans in oral tobacco users. Oral tobacco users do appear to have changes in bacterial flora: changes in the balance of different streptococci have been reported (Fujimori *et al*, 1995) and aqeuous extracts of tobacco are reported to favour growth of Strep. mutans, salivarius and sanguis (Falkler *et al*, 1987).

Extrinsic changes in salivary composition

(a) Changes that can be used diagnostically since they are directly proportional to exposure The thiocyanate ion (CNS) is an important component of normal saliva since it is a cofactor with peroxidase in the generation of the bacteriocidal agent, hypothiocyanate: It has been known for many years that thiocyanate concentrations are increased in the saliva of smokers (e.g. Dacre and Tabershaw, 1970; Tenovuo and Makinen, 1976) and this is still used as a marker of smoking activity (e.g. Brauer *et al*, 1996).

As thiocyanate is a product present in tobacco smoke it also measures exposure to passive smoking. Other cyano products are also present in tobacco smoke and may react with saliva components (Tu *et al*, 1988).

Nicotine and its metabolite, cotinine, both appear in the saliva of tobacco users and can be assayed to provide an estimate of exposure. Cotinine is the more readily measured and is more commonly used (e.g. Ogden *et al*, 1997).

(b) Tobacco components extractable in saliva The tobacco-specific nitrosamines are a group of potentially carcinogenic molecules extractable from tobacco in aqueous solutions. They have been identified in saliva obtained from users of many different tobacco preparations, including betel nut quids (Brunnemann *et al*, 1987; Bhide *et al*, 1987; Nair *et al*, 1987; Osterdahl and Slorach, 1988; Osterdahl, 1990; Idris *et al*, 1992, 1994; Prokopczyk *et al*, 1992; Stich *et al*, 1992; Brunnemann *et al*, 1996) and even tobacco-containing toothpastes although the amounts from the latter are very small (Stich *et al*, 1992).

(c) Modification of saliva components by tobacco products Inhibition of monoamine oxidase activity in saliva was observed after smoking (Yu and Boulton, 1987).

Saliva after betel chewing has been found to contain more o- and m-tyrosine suggesting that (HO.) radical is formed and may cause genetic damage to epithelial cells (Nair *et al*, 1995) although previously it had been found that betel nut quid components but not tobacco generated superoxide in aqueous extracts (Nair *et al*, 1987).

Effects of metabolites on tissues

Nicotine at saliva levels is genotoxic by Chinese hamster ovary tests (Trivedi *et al*, 1990). In bacterial luminescence tests nicotine was not itself genotoxic but did enhance the activity of other genotoxic agents, whilst cotinine was shown to be of low genotoxicity (Yim and Hee. 1995).

Nicotine in concentrations similar to those in smokeless tobacco users inhibits osteoblast synthesis of alkaline phosphatase and collagen but doubles DNA synthesis (Ramp *et al*, 1991). Smokeless tobacco extracts, however. also contained other unidentified agents which inhibited bone collagen synthesis and osteoblast mitochondrial activity (Galvin *et al*, 1988).

The observation that secretory component, lactoferrin, and lysozyme were significantly lower in concentration in the saliva of smokeless tobacco users, although IgA concentrations were higher, stimulated research into the effect of various smokeless tobacco components on a secretory cell line. Aqueous extract, nicotine and cotinine all decreased synthesis and secretion of secretory component, lactoferrin and lysozyme (Gregory and Gfell, 1996).

Tobacco usage and dental caries

Although cigarette smoking is a factor which has often been included in analysis of caries rates there is little evidence of a direct link. From the evidence relating to tobacco usage and saliva composition this is not surprising. If thiocyanate concentrations are higher one might predict less dental caries. However, the lower salivary pH, the lower buffering power, the shift of bacterial population towards lactobacillus and the cariogenic streptococci, might all argue for increased dental caries. There are two recent surveys which link tobacco smoking with dental caries, one of 1156 older (over 70 years) subjects which identified tobacco smoking as a significant risk factor for tooth loss, coronal and root caries (Jette *et al*, 1993), and one of 2145 teenagers which found all caries epidemiological data to be higher among tobacco users and found a positive correlation between number of cigarettes smoked per day and the number of decayed, missing and filled teeth and initially decayed proximal surfaces (Hirsch *et al*, 1991). The latter paper points out the danger of assuming that correlations necessarily imply a causative role.

The position over smokeless tobacco usage and dental caries is much clearer. In 1987 Weintraub and Burt surveyed the literature on this topic and concluded that there was insufficient evidence to support any association between smokeless tobacco use and dental caries. Two surveys of professional baseball players (Ernster *et al*, 1990, Sinusas *et al*, 1992) concluded that in the 1109 and 206 players surveyed, dental caries incidence did not differ between users and non-users. A survey of 247 Bangladeshi women who chewed betel quids found that in the low levels of dental caries in this population there was no correlation between coronal dental caries and the frequency of betel quid chewing (Williams *et al*, 1996).

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TOBACCO AND DENTAL IMPLANTS: TOBACCO AND WOUND HEALING

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There is increasing evidence that tobacco is detrimental to both the initial and long-term success of dental implants, and that smoking cessation can be beneficial in improving implant success rates. Wound healing within the oral cavity has also been shown to be compromised by tobacco both in oral and periodontal surgery. This review paper discusses the current literature in these areas and makes recommendations to clinicians for the management of smokers.

Tobacco and dental implants

For well over 15 years titanium endosseous implants have been increasingly used in various edentulous situations. While a generally high success rate has been achieved in many reports, several authors have described a few unfortunate patients who lose disproportionately high numbers of implants (Engquist *et al*, 1988; Kirsch and Ackmeran, 1989). This led Bain and Moy (1993) to assess the various factors which predisposed to implant failure in a group of 540 patients who received 2194 Branemark implants. They found that smoking was by far the most significant factor predisposing to implant failure. Failure rates were 4.76% in non-smokers and 11.28% in smokers ($P < 0.001$). When the maxilla was assessed separately failure rates in smokers exceeded 17.8% and were greatest with shorter implants. In a later study De Bruyn and Collaert (1994) compared implant failures prior to loading, in the maxilla of smokers with non-smokers, and found failure rates of 9% and 2% respectively. They demonstrated at least one failure in one out of three smokers compared with only one out of 25 non-smokers.

Recently Haas *et al* (1996) have 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. They suggest that these findings will lead to higher future failure rates in integrated implants in smokers. This position is supported by Lindquist *et al* (1996) in a 15-year prospective study of mandibular fixed implant prostheses in which they found that smoking correlated more strongly with marginal bone loss around implants than even poor oral hygiene.

The effects of smoking cessation have recently been investigated in smoking implant patients. Following a protocol of complete cessation for 1 week before and 8 weeks after initial implant placement surgery, Bain (1996) showed a significant reduction in implant failure in the group who stopped smoking when compared with smokers who continued the habit. The group who followed the cessation protocol had no significant difference in their failure rate when compared with failures in non-smokers over the same period. Jaffin and Berman (1991) have shown that patients with type 4 bone had a much higher implant failure rate than all other patients, and recently Bain and Moy (1994) showed that there is almost twice the rate of type 4 bone in moderate and heavy smokers (over 10 cigarettes per day), compared with light and non-smokers.

It has been suggested that implants should only be placed if a likely success rate of 80% or better can be predicted. Based on our research this would exclude sites in the maxilla of smokers where it is anticipated that implants of 10 mm or less can be placed. It is probably more important that the patient is fully informed of risks and that informed consent has been obtained.

Tobacco and wound healing

Several dental studies have shown that tobacco influences wound healing. Preber (1986) showed significantly less reduction in probing depth in smokers than non-smokers after scaling and curettage. Miller (1988) describes poorer healing after mucogingival surgery in smokers. Mechan *et al* (1988) from Newcastle in England showed significant reductions in postextraction socket filling with blood ($P < 0.01$) and more painful sockets ($P < 0.02$) in tobacco users.

In the medical literature smoking has been shown to be a significant risk factor in both non-healing and recurrence of gastric ulcers; impaired healing of hand wounds; unsatisfactory scarring after gynaecological surgery and increased deterioration of arterial grafts. Several plastic surgery studies show impaired healing and increased flap sloughing in tobacco smokers.

The mechanism of impaired healing is probably associated with increased plasma levels of adrenaline and noradrenaline after smoking, leading to peripheral vasoconstriction. A single cigarette has been shown to reduce the velocity of peripheral blood flow by 40% for 1 h. A local vasoconstriction in oral mucosa has also been shown in smokers. Several studies also show impaired PMN function in smokers compared to non-smokers (Kenney *et al*, 1977). Since the oral cavity is likely to be exposed to both local and systemic influences of tobacco byproducts, it is unclear whether smoking cessation aids such as the nicotine patch will in fact reduce oral wound healing problems.

Recommendations

(1) All health histories should include a question on the patient's smoking habits. The medical literature uses the measure of 'Pack Years' to quantify the habit. It is

especially important to identify former heavy smokers who have recently stopped.

(2) All smoking patients should be advised of the increased risk of poor healing after various dental procedures. The role of the dental hygienist is particularly important in this area and all hygienists should be familiar with current facts and should see smoking counselling as much part of their role as plaque control and dietary advice.

(3) Smokers who are considering implant treatment should be advised of the poorer prognosis of implants in smokers, especially on the maxilla. At the very least such patients should be encouraged to follow a smoking cessation protocol and better still to stop indefinitely. It is for the individual dentist to decide if implant treatment should be undertaken in high risk situations in the smoker's maxilla. Informed consent is, however, essential.

(4) The entire dental health team should be aware of the relationship between smoking and dental problems. Smoking counselling should be a fundamental part of the dental curriculum and any practice prvention programme. Interested practitioners should pursue more formal training in smoking cessation counselling, so that these can be offered in practice in a professional manner.

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TOBACCO PREVENTION AND DENTISTS: THE USA EXPERIENCE

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There is overwhelming scientific evidence that tobacco use is a leading cause of preventable disease, disability and death. Globally, it is a rising public health problem. The World Health Organization estimates that the current 3 million deaths annually from smoking will exceed 10 million per year by the year 2025 (Peto *et al*, 1994, 1995). Tobacco use is responsible for a variety of adverse oral diseases and conditions and adversely affects many dental treatments and prognoses (Christen *et al*, 1991; Mecklenburg *et al*, 1994; Research, Science and Therapy Committee, 1996). Also, the oral health team can prevent more premature death and better protect health and well-being by providing brief, simple tobacco intervention services than by any other oral health service (US DHSS, 1990).

Most individuals who use tobacco want to quit and make multiple attempts (Henningfield, 1991). Nicotine addiction is a progressive, chronic, relapsing disease (Fiester, 1996). Thus, self-help quitting methods have low success rates (US DHSS, 1988). The developing understanding of substance abuse on brain functions is helping scientists and clinicians understand that internal, nicotine-driven behavior is an overlay to social-cultural incentives to use tobacco (Balfour, 1994; Rubboli, 1994; Nisell, 1995; Balfour, 1996).

Scientifically sound clinical cessation treatment methods exist. Evidence shows that clinical interventions during dental care are as effective as in other health care settings. Currently, the strongest evidence is found in the 1996 Agency for Health Care Policy and Research's *Clinical Practice Guideline No. 18: Smoking Cessation* (Fiore *et al*, 1996a, b). An increasingly sophisticated array of pharmaceutical agents for smoking cessation are available to support behavioral interventions (Henningfield, 1995; Leischow, 1996; Ostrowski, 1996; Schneider, 1996; Hurd, 1997). Evidence-based methods are described and resource documents offered.

The term 'tobacco intervention' includes at least three components; cessation, prevention and public policy development (US DHHS, 1991). Dentists must be competent in all three areas so that youth are protected and ex-users sustained, users determined to quit, and the public supportive of the profession's commitment to protecting and promoting health. The phrase 'Helping patients and the public to be tobacco-free' encompasses these components.

The process of establishing tobacco intervention services as a clinical practice norm requires a sustained effort to educate clinicians and ensure that the health care system supports such services. Dental practice surveys have identified a variety of misperceptions that need to be overcome before clinical tobacco intervention services will become widely practised (Tomar, 1996; Dolan, 1997; Hayes, 1997). Dental schools have potential as a primary change agent within dentistry since faculties are uniquely positioned to

open frontiers of knowledge, serve as opinion leaders in dental organizational affairs, and develop innovative means for resolving health-related problems in society.

All materials developed under the auspices of the US federal government are in the public domain. Thus, they are freely available to national dental organizations and educational institutions as they develop materials and programs that meet their unique professional objectives and cultural conditions (Mecklenburg, 1993; US DHHS, 1996; Mecklenburg, 1997).

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TOBACCO PREVENTION IN DENTAL PRACTICE: STRATEGIES, GUIDELINES AND GOALS

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Principally there are three different strategies in tobacco preventive work:

(1) to reduce recruitment of new tobacco users—mainly young people;
(2) to support and assist tobacco users who wish to quit;
(3) to avoid non-smokers suffering from other people's smoking—including the foetus.

In this paper, we are mostly dealing with the above-mentioned items (1) and (2) and thus reduce the negative effects in item (3) (Guidelines, 1996; Pellmer and Wramner, 1997).

Preventive measures of lifestyle chances have several

advantages which many treatment methods lack. There is another important difference between prevention and treatment. Prevention affects the *risk* of being ill. When treating a disease, you can only affect the *consequences* of the disease. The essential thing is, not to add years to life, but add life to years, i.e. to add years free of disease.

Strategies

A health professional's approach to tobacco prevention (and cessation) should be a longitudinal strategy involving a multitiered approach that includes the society we live in, the office, schools, civic organisations, and the mass media, in addition to the more traditional individual patient encounter (Gregorio, 1994a; Richards *et al*, 1994). A national tobacco legislation, if there is any, is often a basis for our work (Magnusson and Nordgren, 1994).

What is the opinion of our National Dental Associations? Do they have the same view, as for example, expressed in the FDI Position Statement on Tobacco. To have support from the National Dental Association is almost an absolute necessity if we are going to reach our goals.

The international network and association for dentists, Dental Associations and Dental Organisations is the FDI World Dental Federation. At the annual World Dental Congress in Hong Kong 1995, the FDI section World Dentistry Against Tobacco (WDAT) was formed and an Agenda for Action was adopted.

The WDAT suggested a FDI Position Statement on Tobacco which was adopted at the annual World Dental Congress 1996. This is a unique FDI document as it involves 'all oral health professionals in the world', not only dentists (Akerberg, 1996).

The position statement deals with:

- tobacco in daily practice;
- tobacco in all education;
- protect the children;
- prevent the initiation;
- FDI smoke-free.

The National Dental Associations should be encouraged to adopt a non-tobacco policy, based on the FDI Position Statement.

Involvement in community-based smoking control activities poses a different, but no less important challenge. Medical professionals' responsibility for health of the patients cannot be limited solely to those procedures performed in an office practice.

To work with tobacco prevention and cessation services is a matter of quality (Humphris and Lennon, 1995). Most patients expect today to be informed about tobacco at their annual visits. Many dentists around the world use the American 4 A-program in their tobacco counselling (Mecklenburg *et al*, 1991). That means:

- ASK patients about smoking at every opportunity;
- ADVISE all smokers to quit;
- ASSIST patients with stopping;
- ARRANGE follow-up visits.

Due to different circumstances, dentists in some European countries Ask. Advise and then refer the patient to a smoking cessation specialist or the family doctor.

The office should convey the message that non-smoking is the norm. The obvious and perhaps most important factor is the tobacco habits of the staff. The most professional and elegant tobacco counselling loses of course all its power if the patient can rest his/her eyes on a pack of cigarettes in the breast pocket of the dentist/hygienist/assistant. Then, let us not forget positive arguments for tobacco cessation. It is also positive to join a professional network against tobacco use.

Guidelines

Tobacco counselling is for many colleagues a new and unexplored field of work, something nobody told us at the School of Dentistry. The first and perhaps most important guideline should come at the medical and dental schools via education (Richmond *et al*, 1996).

Appropriate guidelines can be directed to both patients and to dental staff, often produced nationally in co-operation with dental associations, national cancer organisations and other public health authorities but also very often by pharmaceutical companies (McIllvain *et al*, 1997). What could be very useful but is still lacking is some kind of international survey of guidelines, what is available and produced by whom.

Goals

The goal for Europe by WHO in 'Action Plan for a Tobacco Free Europe Year 2000', target 17 is that 80% of the European population should be non-smokers (Tobacco-Free Europe, 1994). Unfortunately, this will not be fulfilled by most countries. There are interesting data from several studies regarding dentists willingness to work with tobacco counselling (Gregorio, 1994b; Chestnutt and Binnie, 1995; John *et al*, 1997). Results from studies on tobacco cessation and prevention in dental practice help us frame goals for our work with tobacco cessation and prevention.

As a basis for discussion, the following goals for EU Countries for year 2002 (5 years from now) are framed.

- 75% of all dentists practising shall work with tobacco counselling in daily practice;
- 85% of all dental hygienists practising shall work with tobacco counselling in daily practice;
- 10% of all patients participating in smoking cessation activities shall be smoke-free 1 year after quit date;

Tobacco counselling shall include accurate statistics according to WHO (or similar European standard) and be reported to the National Dental Association once a year.

By discussing and adopting strategies, guidelines and goals in Europe, we can considerably reduce the number of tobacco-related oral cancer and other tobacco-related disease and undesired effects on dental treatment.

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Appendix

FDI POSITION STATEMENT ON TOBACCO

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 disease and other deleterious oral conditions and it adversely affects the outcome of oral health care.

The FDI urges its Member Associations and all oral health professionals to take decisive action 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.

Tobacco in all education

Brief interactions, for example, by identifying tobacco users, giving direct advice, supportive material and follow up, all have a significant impact on the patients use of tobacco products.

The FDI urges all oral health institutions and all continuing education providers to integrate tobacco-related subjects into their programme.

Protect the children

The adverse consequences of environmental tobacco smoke are particularly severe for children—and life long.

The FDI strongly endorses and promotes public and professional education and policies, that prevent and/or reduce the exposure to tobacco smoke for infants, children and young people.

Prevent the initiation

More than eighty percent of adults who use tobacco, started their use of tobacco before the age of eighteen. Use of tobacco among children and youths easily produces a nicotine dependency, the risk of which is vastly underestimated by the young people themselves.

The FDI vigorously supports all measures that endeavour to prevent the initiation of tobacco use.

FDI smoke free

The hazards of environmental tobacco smoke are well documented.

The FDI's Congresses, educational programme, business meetings and Head Office are all smoke free.