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# The Role of the Dental Team in Preventing and Diagnosing Cancer:

## 1. Cancer in General

**Abstract:** Prevention and early detection are integral components of the Health Agenda and areas taking on increasing importance and emphasis. Members of the dental profession have an important role to play and, indeed, a duty to advise on the prevention of, not only oral diseases and notably oral cancer, but also on other potentially malignant conditions and their prevention. They are in a position to advise and help protect their patients, staff, colleagues, families and acquaintances. Apart from prevention, early detection and prompt referral for diagnosis offer the best hope to the patient with cancer, providing the best chance of a cure. The signs and symptoms of cancer often resemble less serious conditions and prompt referral to an appropriate specialist usually allows for the best management.

**Clinical Relevance:** Dental staff have the opportunity to make an impact on preventing, and on improving the poor survival rate and morbidity of cancer.

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Cancer is remarkably common, and of major public health importance, since it is increasingly important in an ageing population and half the people who develop cancer die from their disease. In high-resource countries such as the United Kingdom (UK), about one in four of all deaths is caused by cancer.<sup>1</sup>

It is estimated that, world-wide, there are 10 million new cancer cases

diagnosed each year and 6 million deaths and there are currently some 24 million people alive with cancer (<http://www-dep.iarc.fr/>). In the UK, there are about a quarter of a million new cases of cancer diagnosed each year, a figure which would soar if cases of non-melanoma skin tumours were to be included.<sup>2</sup>

### Incidences of cancer

The International Agency for Research on Cancer (IARC) publishes a compendium of statistics of incidences of cancer from all population-based cancer registries world-wide (<http://www-dep.iarc.fr/>).<sup>3</sup> In the USA, the cancer incidence data from the Centers for Disease Control and Prevention (CDC) and the National Cancer Institute (NCI), the North American Association of Central Cancer Registries (NAACCR), National Program of Cancer Registries (NPCR) and NCI's Surveillance,

Epidemiology, and End Results (SEER) program are reported for 66 selected primary cancer sites and subsites for men, 70 selected primary cancer sites and subsites for women, and for all cancer sites combined. These data are assembled into tables and figures that provide specific information with regard to geographic area, race, sex and age (<http://www.cdc.gov/cancer/>).<sup>4</sup>

### Carcinogenesis

Cancer is disordered growth, maturation and proliferation of a tissue, which arises as a consequence of multiple molecular events causing genetic damage affecting many chromosomes and genes, and leading to DNA changes. The accumulation of genetic changes leads to dysregulation of cell signalling and thus to disordered growth, maturation and proliferation.

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The accumulation of genetic changes eventually leads to cell dysregulation to the extent that growth becomes autonomous and invasive mechanisms develop. In the case of carcinomas, the neoplastic process manifests first near the epithelial basement membrane as a focal, clonal overgrowth of altered keratinocyte stem cells (intraepithelial neoplasia), which expand upward and laterally, replacing normal epithelium. One of the main features identifiable histopathologically, and which appears to precede the onset of frank malignancy, is epithelial dysplasia, a histological term describing the combination of disorderly cell maturation and disturbed proliferation. However, well before these histopathological changes become observable, there are cellular and molecular changes, an increasing number of which are identifiable with appropriate modern technology. Indeed, the number of molecules which may be tumour markers increases almost weekly.

Dysplasia varies in severity, and it is the more severe grades that appear to be associated with higher malignant potential. After some time, invasion of the epithelial basement membrane signifies the start of invasive cancer.

However, the path to carcinoma is not necessarily straight or unimpeded; protective and reparative genetic and immune mechanisms may exert some control. Such mechanisms include systems to metabolize carcinogens and to repair damaged DNA, as well as immune defences (largely T lymphocyte-mediated) which recognize and attack abnormal cells.

### Causes of, liability to, and protection from, cancer

Lifestyle factors are of major importance in tumorigenesis. Over 20 years ago, Doll and Peto<sup>5</sup> estimated that one-third of all cancer deaths in countries such as the USA or the UK were caused by tobacco, and these estimates are still relevant today, despite efforts from the industry to deny this (Figure 1). The American Cancer Society (ACS) estimates that one-third of the 500,000 annual cancer deaths in the USA are from cigarette smoking. The ACS estimate another third are due to poor diet and lack of exercise that leads to excess

## The '50 years of lies over smoking risk'

**Figure 1.** Industry denial of cancer risk through the media.

weight and obesity.

The predisposition to cancer has been attributed mainly to specific risk factors – mainly carcinogens - which cause DNA damage, ie they act as mutagens. The list of known chemical carcinogens is long but many of the most important are aromatic amines (including arylamines and heterocyclic amines) found in tobacco smoke and over-cooked foods (Table 1). Tobacco smoke, for example, is a complex mixture of at least 50 chemical compounds including polycyclic aromatic hydrocarbons (PAHs), nitrosamines, aldehydes and aromatic amines. Potential carcinogens may be inhaled or ingested with full carcinogenic potential (eg tobacco and tobacco-contained nitrosamines, or nitrosamines in products such as Toombak) or sometimes they are pro-carcinogens (carcinogens may be produced during their

Nitrosamines
Polycyclic aromatic hydrocarbons
Arylamines
Alkyl halides
Ethanol
Urethane

**Table 1.** Some recognized carcinogens.

Enzyme	Abbreviation	Main activities of some genotypes
Alcohol dehydrogenases	ADH	Activate ethanol
Cytochrome p450	CYP	Activate ethanol, benzpyrene and nitrosamines
Glutathione S-transferases	GST	Metabolize benzpyrene, alkyl halides, epoxides and lipid peroxides
N-acetyl transferases	NAT	Acetylate arylamines

**Table 2.** Main xenobiotic-metabolizing enzymes.

metabolism, eg acetaldehyde produced from alcohol by alcohol dehydrogenase).

Polycyclic aromatic hydrocarbons include chemicals such as benzo(a)pyrene. Tobacco-specific nitrosamines (TSNAs) include a range of chemicals of complex structure (eg N-nitrosornicotine (NNN) and 4-[methylnitrosoamino]-1-[3-pyridyl]-1-1 butanonone (NNK)). Both PAH and TSNAs are found in tobacco smoke and the ambient environment, but there are variations in their levels even within the same brand of cigarette sold world-wide,<sup>6</sup> and some evidence that these levels are falling.<sup>7</sup> Thus carcinogens are found not only in tobacco and in cigarettes, but also in exhaled (passive) smoke. The environment may therefore contain chemical carcinogens from passive smoke mutagens and also include some from the combustion of fossil fuels. Ionizing radiation is also mutagenic, as are some micro-organisms.

However, not all people exposed to mutagens from lifestyle habits or the environment develop cancer and, equally, nor do all patients with cancer appear to have been so exposed and, therefore, other factors must be involved.

Genetic predispositions and genetically-mediated defences, as well as dietary protective factors such as anti-oxidants, may play a part. Genes which are either defective congenitally or damaged later in life, play crucial roles in carcinogenesis. Such genes include mainly those involved in cell growth, tumour suppressor genes (TSGs) and genes involved in the metabolism and breakdown of carcinogens and/or in the repair of DNA damaged by mutagens. Individual susceptibility to cancer may sometimes be explained by a genotype that results in increased carcinogen exposure

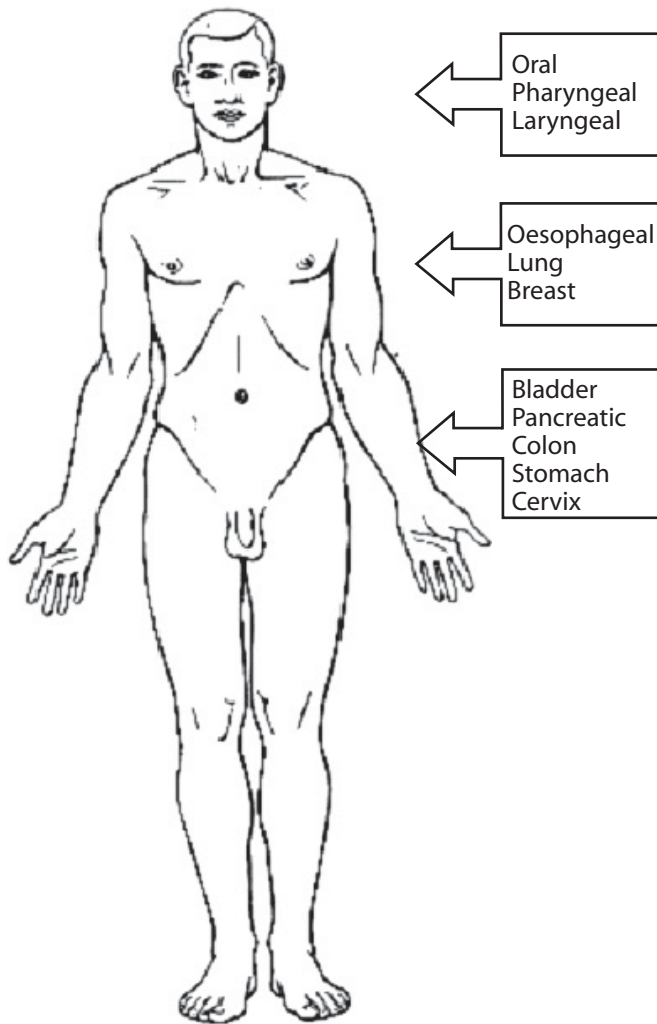


Figure 2. Cancers related to tobacco use.

either acting singly or jointly with alcohol, is also responsible for up to 90% of cancers in the oesophagus, larynx and oral cavity, including oral squamous cell carcinoma (OSCC). Tobacco is also linked to a large proportion of cancers of the bladder and pancreas and a smaller proportion of cancers of the kidney, stomach, breast, cervix and nose, and myeloid leukaemia (Figure 2). Indeed, over 30% of all cancer deaths in developed countries are tobacco-related.

Although the greatest cancer hazard is caused by cigarette smoking, cigars can cause similar hazards if their smoke is inhaled, and both cigar and pipe smoking cause comparable hazards of cancers of the oral cavity, pharynx, larynx and oesophagus. On stopping smoking, the cancer risk rapidly falls, and the lessening of the risk is progressively more marked with time.

Environmental tobacco smoke (ETS) or second-hand smoke is a mixture of two forms of smoke from burning tobacco products:

- Sidestream smoke: smoke that comes from a lighted cigarette, pipe, or cigar;
- Mainstream smoke: smoke that is exhaled by a smoker.

When non-smokers are exposed to second-hand smoke it is called involuntary smoking or passive smoking. Non-smokers exposed to second-hand smoke absorb nicotine and other compounds just as smokers do and, the greater the exposure to second-hand smoke, the greater the level of these harmful compounds in the body. ETS is particularly harmful to children.

That ETS is now recognized as a cancer risk is shown by the fact that the US Environmental Protection Agency (EPA) has classified second-hand smoke as a Group A carcinogen – meaning there is sufficient evidence that it causes cancer in humans. Also, the US National Toxicology Program has classified environmental tobacco smoke as a ‘known human carcinogen’. Inhalation of ETS by so-called ‘passive smoking’ causes a small increase in the risk of lung cancer, but in addition has several deleterious effects on people who inhale it, with an increase in the risk of heart disease and respiratory disease.

Tobacco-chewing in people from parts of Asia, along with a variety of ingredients in a ‘betel quid’ (betel vine leaf, betel (areca) nut, catechu, and slaked

as a consequence of his/her carcinogen or pro-carcinogen metabolism, or that results in impaired DNA repair. Various enzymes found especially in the liver are involved in the activation or degradation of carcinogens/pro-carcinogens; they are termed xenobiotic-metabolizing enzymes (XME), and several of these are shown in (Table 2).

The classic example of impaired DNA repair is the rare condition *xeroderma pigmentosum*, where the individual has to be protected from the sun in order to avoid multiple cancers of the skin and lip. Patients who inherit various immune defects may also be liable to develop various malignant neoplasms.

### Cancer prevention

Cancer can thus be seen as the consequence of the interaction between lifestyle, environmental and genetic factors. The major way to prevent cancer, therefore, is to avoid the adverse lifestyle habits, since other environmental and genetic factors are difficult, if not impossible, to change.

### Avoiding tobacco

This topic will be discussed at greater length in article 4 in this series.

Cigarette smoking is responsible for over 85% of lung cancer and a one-pack-a-day smoker has a ten-fold risk of lung cancer over a non-smoker. Tobacco,

lime together with tobacco) appears to predispose to OSCC and oesophageal cancer, particularly when the habit is started early in life and is used frequently and for prolonged periods. Snuff-dipping, for example in women in south-east USA, who place snuff in the buccal sulcus, can predispose to OSCC close to where the snuff is placed.

Stopping tobacco use reduces the risk of cancer. Clearly, cessation of tobacco use could prevent a huge number of cancers and other diseases.

### Avoiding alcohol

Alcohol drinking increases the risk of upper digestive and respiratory tract neoplasms, especially squamous cell cancers of the oral cavity, pharynx, larynx and oesophagus.

The risks from alcohol tend to increase with the amount of ethanol drunk, and alcohol drinking and tobacco smoking together greatly increase the risk of upper aerodigestive tract cancer.

A direct carcinogenic effect of acetaldehyde, the main ethanol metabolite, and of other agents present in alcoholic beverages is possible. Alcoholic beverages may also contain other carcinogens or pro-carcinogens, such as nitrosamines and urethane. A diet poor in fruits and vegetables, typical of heavy drinkers, is also likely to play an important role. Another likely carcinogenic mechanism of alcohol is by facilitating the carcinogenic effect of tobacco, and possibly of other carcinogens, to which the upper digestive and respiratory tract are exposed.

Alcohol drinking is also strongly associated with liver damage, and thus a reduced ability to metabolize carcinogens, and impaired immunity. Alcoholism is associated also with a risk of primary liver cancer; particularly among smokers and among people chronically infected with hepatitis C virus, and may be associated with an increased risk of colo-rectal and breast cancer. Indeed, more cases of breast cancer than of any other cancer are attributable to alcohol drinking among European women. It is possible that alcohol acts on hormonal factors involved in breast carcinogenesis, although the mechanism of action is not yet clearly understood.

There seems to be little

difference in the effects of beer, wine or spirits on cancer risk; rather the total amount of ethanol ingested appears to be the key factor in determining the increase in cancer risk.

Stopping alcohol drinking reduces the risk of oesophageal cancer by 60% ten years or more after drinking cessation. Recommended limits of alcohol consumption for men should be 20 g of ethanol per day (ie approximately two drinks of either beer, wine or spirit each day) and as low as 10 g per day for women.

### Weight reduction

Obesity is associated with an increased risk of cancer of the colon, breast (post-menopausal), endometrium, kidney and oesophagus (adenocarcinoma). Therefore, avoid becoming overweight and maintain a body mass index of below 25 kg/m<sup>2</sup>.

### Increasing physical activity

Regular physical activity is associated with a 40% reduction in the risk of colo-rectal cancer, a 20–40% reduction in the risk of breast and oesophageal cancer, and some reduction in risk of prostate cancer. Therefore, undertake regular physical activity of at least one hour per day.

### Increasing intake of fruit and vegetables

Vegetables and fruits contain a large number of potentially anticarcinogenic agents, particularly those with antioxidant activity. People who eat a balanced, low-fat diet daily with enough fruits, vegetables, whole grains and beans can reduce their chances of colon cancer by 75%, breast cancer by 50% and lung cancer by 30% and there may be a protective effect against other cancers, in particular oral, oesophagus, stomach, rectum and pancreas.

'Five-a-day' (minimum 400 g/day, ie 2 pieces of fruit and 200 g of vegetables) is advocated to lead to a reduction in cancer risk.

### Avoiding sun exposure

Skin and lip cancers are predominantly seen in white-skinned

people, and caused by the sun's ultraviolet rays. The recipients of transplanted organs are at particular risk, as a result of the combined effects of immunosuppression, human papillomaviruses (HPV) and sun exposure.

Fair people are also more susceptible to skin melanoma, particularly those with red hair, facial freckles and a tendency to burn in the sun – characteristics which are genetically determined at least partially by the MC1R gene which codes for the melanocyte stimulating hormone receptor. The strongest phenotypic risk factor for melanoma, however, is the presence of large numbers of moles or melanocytic naevi which may be normal in appearance but are also usually accompanied by so-called atypical moles – the atypical mole syndrome phenotype (AMS) – which is present in about 2% of the Caucasian population and is associated with approximately a ten times increased risk of melanoma.

The best protection from the summer sun is to stay out of it as much as possible. If outdoors, keep out of the sun between 11 am and 3 pm, wear a hat and close weave heavy cotton which affords good protection, and use sunscreens with high SPF. Do not rely upon sunscreens alone for protection, however useful they may be to protect sites such as the ears and lips.

### Avoiding occupational exposures

The more common occupational exposures likely to be mutagenic include solar radiation, passive smoking, crystalline silica, diesel exhaust, radon, wood dust, benzene, asbestos, formaldehyde, polycyclic aromatic hydrocarbons, chromium, cadmium and nickel compounds. There is no reliable evidence which has yet emerged regarding a cancer risk from power plants or power lines. However, chemical carcinogens can be common in some workplaces; the rubber industry and painters are examples and lung cancer is increased in dental technicians – though possibly from smoking.

Up to 29 chemical or physical agents, groups of agents or mixtures that occur predominantly in the workplace have been classified as human carcinogens. Indeed, IARC has classified more than

200 agents, groups of agents or exposure circumstances that are classified as 'possibly carcinogenic' to humans, largely on the basis of carcinogenicity data from animal experiments. Many chemicals are known or suspected carcinogens and many of the agents in this group are still widely used, for example, 1-3 butadiene, formaldehyde. Thirty-five agents or industrial processes are classified as probably carcinogenic. The effect of specific occupational carcinogens, such as aromatic amines or PAHs, is also mediated by genetic factors such as genetic polymorphisms of the NAT2 or the GSTMI genes (Table 2).

The cancers that have most frequently been associated with occupational exposures include the following:

- Lung (including mesothelioma);
- Urinary bladder;
- Larynx;
- Bone marrow (leukaemia);
- Liver (angiosarcoma);
- Nose, nasal cavity and sinuses; and
- Skin (non-melanoma).

Several other neoplasms which have also been associated with occupational exposures, albeit on weaker evidence, include soft-tissue sarcoma, lymphomas and multiple myeloma, and cancers of the oral cavity, nasopharynx, oesophagus, stomach, colon and rectum, pancreas, breast, testis, kidney, prostate, brain, and bones.

Prevention of workplace exposures can be effective: for example, occupational bladder cancer has been substantially reduced after the ban on the use of beta-naphthylamine in the rubber and chemical industries.

## Avoiding environmental exposures

Environmental exposures usually refer to exposures of the general population that cannot be directly controlled by the individual and include:

- Air-pollution;
- Drinking water contaminants;
- Passive smoking;
- Radon gas in buildings;
- Exposure to solar radiation and to low frequency electromagnetic fields;
- Food contaminants such as pesticide residues, dioxins or environmental oestrogens;

■ Chemicals from industrial emissions, ionizing radiation and waste from nuclear processes, and others. These exposures have been associated with a variety of neoplasms including cancers of the lung, urinary bladder, leukaemia and skin. The impact of several exposures that are widespread, such as disinfection by-products in drinking water, is still inconclusive.

Agents in the general environment to which a large number of subjects are exposed for long periods, such as passive smoking or air-pollution, although increasing only modestly the relative risk for certain cancers, may be the origin of a sizeable number of cases.

## Achieving prevention of cancer

### Primary prevention

Primary prevention is the approach that focuses on removing risk factors in order to prevent the cancer. The crucial point to note is that the causes of several cancers are so similar that primary prevention, such as cessation of tobacco use, can avoid or minimize the chances of developing a wide range of cancers.

However, changes in lifestyle habits can be surprisingly difficult to achieve and maintain ([http://www.cancer.org/docroot/NWS/content/NWS\\_2\\_1x\\_American\\_Communities\\_Can\\_Change\\_Lifestyle\\_Habits\\_to\\_Prevent\\_Cancer.asp](http://www.cancer.org/docroot/NWS/content/NWS_2_1x_American_Communities_Can_Change_Lifestyle_Habits_to_Prevent_Cancer.asp)). National, federal, state and local governments, along with private and public organizations, should all work together to create a social and physical environment that will make it easier for people to gain the individual willpower to be active, eat correct diets, and avoid or stop adverse lifestyle habits which will, in turn, help reduce the likelihood of cancer development.

Healthcare professionals can have an important role in primary prevention, and this applies to dental as well as to medical and other personnel.<sup>8-11</sup> The US Clinical Practice Guideline on Treating Tobacco Use and Dependence encourages clinicians to:

- Ask about tobacco use;
- Advise patients to stop;
- Assess willingness to quit;
- Assist in treatment; and
- Arrange for follow-up.

Applying these five 'As' to the dental team by implementing a system to collect the necessary information, while providing multiple and reinforcing messages, may help tobacco-using patients progress toward abstinence.<sup>12</sup> However, the belief that members of the dental team should engage in delivering smoking cessation interventions is not held by all parties, and dental students have a low perception of their effectiveness of smoking cessation counselling and the adequacies of the current evidence base.<sup>13</sup> Advice from nurses does appear to increase the rate of quitting, but prior training has little added benefit.<sup>14</sup> More dentists believe that they should offer smoking cessation support than actually do provide it, and stated reasons for not providing it include:

- Time and reimbursement issues;
- The need for further training; and
- Poor co-ordination of dental and smoking cessation services.<sup>15</sup>

This is discussed more fully in Article 4 of this series.

### Secondary prevention

Secondary prevention is the detection of early cancer or precancer, and is the basis for screening. This is a complex area with a number of disadvantages.

## Screening for cancer and potentially malignant lesions

Screening to identify cancers and potentially malignant lesions would appear attractive. The following 10 criteria need to be followed:

- The cancer should be an important public health problem;
- Treatment should confer a significant survival advantage;
- Facilities for further diagnosis and treatment should be available;
- There should be an identifiable latent stage;
- The screening test should be effective with few false positive results;
- The screening test should be well accepted;
- The natural history of the cancer should be completely defined;
- The strategy for treatment should be well defined;
- The cost should be affordable;

■ Regular observation of potentially malignant lesions should result in early detection of malignant change.

There are surprisingly few cancers for which screening programmes meet these criteria, but there is adequate evidence of the efficacy of screening for:

- Bowel cancer: faecal occult blood tests (FOBT);
- Breast cancer: mammography;
- Cervical cancer: cervical (Papanicolaou: 'Pap') smear examination.

The correct screening tests and the frequency recommended depend on the person's age, gender, medical history, family history and lifestyle. For example, a patient with a strong family history of breast cancer may well merit more extensive and frequent screening. Women who have had breast cancer face a greater risk of getting breast cancer in the other breast. A woman's risk for developing breast cancer rises if her mother, sister or daughter has had breast cancer, especially at a young age. Having a diagnosis of atypical hyperplasia or lobular carcinoma *in situ* (LCIS) or changes in certain genes (BRCA1, BRCA2, and others) also increase the risk of breast cancer.

## The European Code Against Cancer

The European Code Against Cancer has been introduced to be a series of recommendations based mainly on primary prevention which, if followed, could lead in many instances to a reduction in cancer incidence and also to reductions in cancer mortality. The first version of the European Code Against Cancer was published in 1995,<sup>16</sup> and the latest in 2003 (Figure 3).<sup>2</sup>

The European Code Against Cancer is as follows:

*Many aspects of general health can be improved and many cancer deaths prevented, if we adopt healthier lifestyles:*

1. Do not smoke; if you smoke, stop doing so. If you fail to stop, do not smoke in the presence of non-smokers.
2. Avoid obesity.
3. Undertake some brisk, physical activity every day.
4. Eat a variety of vegetables and fruits every day: eat at least five portions daily. Limit your intake of foods containing fats from animal sources.
5. If you drink alcohol, whether beer, wine or spirits, moderate your consumption to two drinks per day if you are a man or one drink per day if you are a woman.
6. Care must be taken to avoid excessive sun exposure. It is specifically important to protect children and adolescents. For individuals who have a tendency to burn in the sun active protective measures must be taken throughout life.
7. Comply strictly with regulations aimed at preventing occupational or environmental exposure to known cancer-causing substances. Follow advice of National Radiation Protection Offices. There are Public Health programmes which include secondary prevention that could prevent cancers developing, or increase the probability that a cancer may be cured:
8. Women from 25 years of age should participate in cervical screening. This should be within programmes with quality control procedures in compliance with EU Guidelines for Quality Assurance in Cervical Screening.
9. Women from 50 years of age should participate in breast screening. This should be within programmes with quality control procedures in compliance with EU Guidelines

for Quality Assurance in Mammography Screening.

10. Men and women from 50 years of age should participate in colorectal screening. This should be within programmes with built-in quality control procedures.

11. Participate in vaccination programmes against Hepatitis B Virus infection (confers protection against liver cancer).

### Tertiary prevention

Tertiary prevention is designed to prevent the recurrence of the cancer. Sadly, lifestyles can be very difficult to change, even in a patient who has reaped the consequences.

### Summary

Dental staff should be aware of the European Code Against Cancer, and advise colleagues, family and patients to adhere to it.

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## Book Review

### The Scientific Basis of Oral Health Education.

By RS Levine and CR Stillman-Lowe. BDJ Books, London, 2004 (88pp., £19.95p/b). ISBN 0-904588-84-X. (Available from BDA Shop, BDA, 64 Wimpole Street, London W1G8YS: tel. 020 7563 4555; fax 020 7563 4556; email bdashop@bda.org; website www.bdjbooks.com)

*The Scientific Basis of Oral Health Education* by Levine and Stillman-Lowe is the 5th edition of this document but the first to be published by BDJ Books. *The Scientific Basis of Dental Health Education* was published in 1976 by the

Health Education Council and in its many incarnations provided a 'scientific basis' for health education in dentistry. The change in name from 'Dental' in previous editions to 'Oral' in the present edition represents a shift in focus. In the 1976 and first edition the focus was strictly tooth-related, whereas the 2004 and 5th edition is orally focused. Thus the 5th edition includes additional chapters on the removal of plaque, erosion, oral cancer, oral ulceration, oral candidal infections, preventive advice for denture wearers and dentate older people. An additional and equally important change in the 2004 edition is the inclusion of a classification of evidence-based research to support the key messages of diet, toothbrushing, water fluoridation and dental attendance. The classification of research evidence is based upon the work of Shekelle *et al.*<sup>1</sup> and divides the evidence base into levels in accordance with the strength of the evidence. This ranges from very strong evidence (meta-analysis or systematic literature reviews) classified as Evidence A, to statements supported by the majority of relevant research studies, classified as Evidence B, and finally statements that cannot be supported by a substantial body of research evidence, but where there is a consensus of scientific and professional opinion to support the statement, classified as Evidence C.

The content of the book provides the scientific basis of oral health education on the topics itemized previously.

It provides in-depth and evidence-based material on the scientific basis of, for example, oral cancer advice for children under 5 and frequency of oral examinations. In addition, it also furnishes the reader with information on strategies as to how to change one's knowledge into action to provide oral health education for patients and clients. An appendix provides the reader with a list of key points and the strength of supporting evidence, references and further reading. This book may be obtained as a volume in its own right or part of *A Guide to Prevention in Dentistry* edited by E.J. Kay. With regard to the value of the 5th edition, now entitled *The Scientific Basis of Oral Health Education*, it is essential for all those health practitioners involved in developing oral health education strategies for their patients and clients. It provides a useful reference and key document for all members of the dental team and undergraduate students who wish to provide evidence-based and scientifically grounded oral health education.

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#### Erratum

The book review of *Human Disease for Dentistry*, published in the January/February 2005 issue, was incorrectly credited to Blackwell Publishing with an ISBN of 0-63206-453-6. It is actually published by Oxford University Press and the ISBN is 0-19-263163-2.

