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The Role of the Dental Team in Preventing and Diagnosing Cancer: 5. Alcohol and the Role of the Dentist in Alcohol Cessation

Abstract: Alcohol use contributes to many health disorders and social problems which may affect both the individual and the community, and is a major risk factor for oral cancer and potentially malignant lesions – leukoplakia and erythroplakia. Counselling by doctors and dentists can increase users’ motivation to stop alcohol use but is not often applied in a systematic or frequent manner to people presenting with potentially malignant oral lesions. This paper makes recommendations for interventions by health professionals to encourage and aid cessation of alcohol use as a part of prevention of oral cancer.

Clinical Relevance: Health professionals should encourage and aid cessation of alcohol use as a part of prevention of oral cancer. **Dent Update 2005; 32: 454-462**

Alcoholic beverages are commonly consumed in many cultures. However, there is wide variability among countries in which alcohol is consumed, in terms of per capita average alcohol (ethanol; ethyl alcohol) consumption, prevalence of heavy drinkers, drinking pattern, and preferred type of alcoholic beverage. For example, in Europe, three groups of countries are traditionally identified according to the prevalent drinking culture (wine drinking in

the south, beer drinking in the centre and spirit drinking in the north-east). There is considerable variability within such groups and within countries, and new patterns are evolving such as increasing consumption of wine in northern European countries and increasing prevalence of binge drinking, that is, high ethanol intake in a single drinking occasion, in particular among women.

Alcohol and health

Anyone can fall prey to alcoholism, but occupations in which there is ready access to alcohol (eg people working in the food and drink industry) are particularly liable. Health professionals should also be aware of the possibility of alcohol abuse amongst colleagues. Space precludes discussion of these aspects here.

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Beverage	Amount	Number of AUs
Spirits	70cl bottle	32
Spirits	One measure	1
Fortified wine eg sherry	70cl bottle	16
Red wine (12% alcohol)	75cl bottle	9
White wine (10% alcohol)	75cl bottle	7.5
White wine (10% alcohol)	One glass	1
Beer, cider (8% alcohol)	Pint	4.5
Beer, cider (5% alcohol)	Pint	3
Beer, cider (3.5% alcohol)	Pint	2
Beer, cider (3.5% alcohol)	Half pint	1

Table 1. Number of Alcohol Units (AUs), which approximately corresponds to 12 g of ethanol, in various alcoholic drinks.

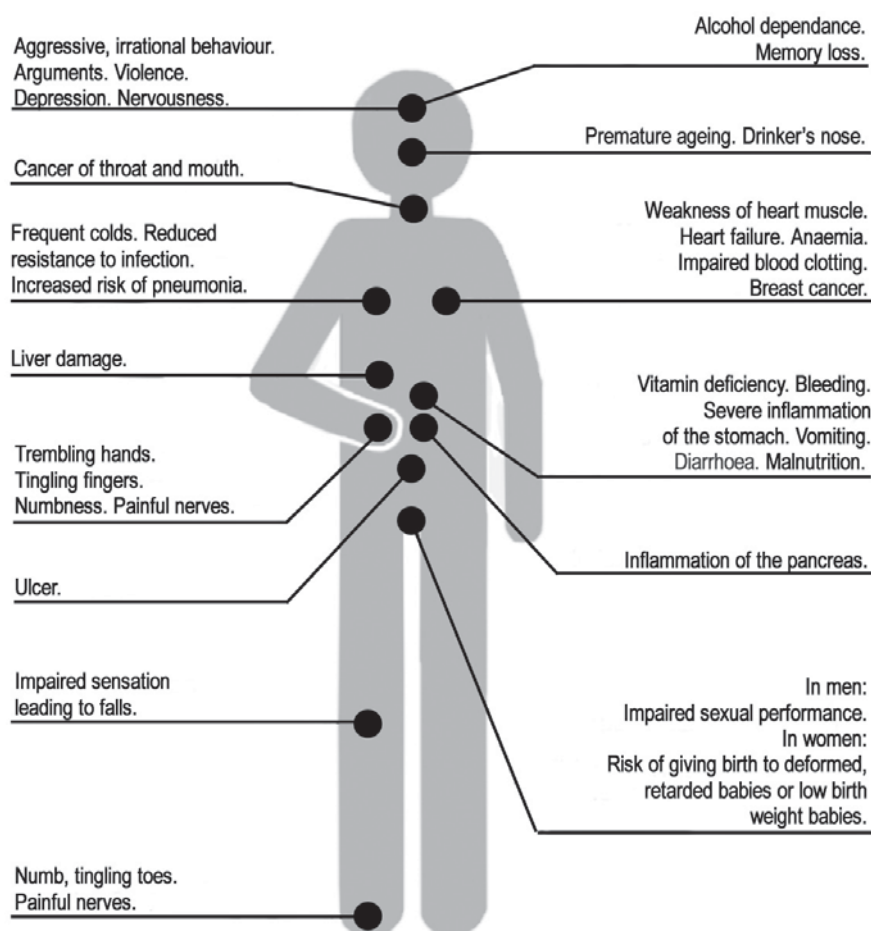


Figure 1. Health effects of heavy drinking.⁴

In reviewing the social and health effects of alcohol consumption, a preliminary distinction must be made between moderate and heavy drinking, including alcoholism, the discriminating limit being generally set at 40 g/day (usually defined as Tolerable Upper Alcohol Intake Level – TUAL),¹ corresponding to 3–4 Alcohol Units/day (the Alcohol Unit – AU, approximately corresponds to 12 g of ethanol, and is usually contained in a can of light beer, a glass of wine, or a small glass of spirits – Table 1).

A loose definition of alcoholism is consumption of alcohol to such a degree as to cause deterioration in social behaviour, or physical illness, and the development of dependence, from which withdrawal is difficult or causes adverse effects. Alcoholism is defined by the American Medical Association as ‘an illness

characterized by significant impairment (a type of drug dependence) that is directly associated with persistent and excessive use of physiological, psychological or social dysfunction’.

Alcohol use is one of the major lifestyle avoidable health hazards, and has been shown to be causally related to more than 60 different medical conditions (Figure 1). The overall mortality rate in alcoholism is significantly raised: over 15 years it may be about 300% above the norm.² Overall, the global burden of disease attributable to alcohol use, that is, the fraction of deaths attributable to alcohol consumption, has been estimated at 4%. Thus, alcohol accounts for about as much of the burden of disease as tobacco (4.1%) and high blood pressure (4.4%) and is exceeded only by the burdens caused by obesity (9.5%) and unsafe sex (6.3%).³

Some consequences of alcohol use are acute, such as suicide, homicide, accidents, eg falls, poisoning, fires or by accidents in motor vehicles (alcohol is an important, if not the main, causal factor in over 25% of road traffic accidents), and convulsions. Other effects, such as cirrhosis, pancreatitis, stroke and various forms of cancer are consequences of chronic alcohol use. Alcohol abuse, through inadequate diet, is also an indirect cause of nutritional defects, leading to peripheral polyneuropathies (burning hands and feet), pellagra, visual defects (amblyopia) and organic brain disorders (Wernicke’s encephalopathy and Korsakoff’s psychosis). Moreover, alcohol drinking during pregnancy has a detrimental effect on foetal development⁴ with ‘foetal alcohol syndrome’.

The alcohol attributable burden of disease is affected not only by the volume of alcohol consumption, but also by the pattern of drinking. In fact, with similar annual alcohol intake levels, drinkers from Eastern Europe generally consume beverages with high ethanol concentration during weekends, whereas traditionally many drinkers from Western, particularly Mediterranean, Europe consume beverages with moderate to low ethanol concentration with meals, daily. This different drinking behaviour leads to an alcohol attributable fraction of overall mortality almost double in Eastern than in Western Europe (12.0% vs 6.8%, respectively).^{5,6}

On the other hand, there is evidence that moderate alcohol consumption may have some beneficial effects for some conditions, such as many cardiovascular diseases and diabetes mellitus.⁷ Therefore, the risk curve of alcohol consumption and total mortality is U-shaped, with a decreased risk in light drinkers compared to non-drinkers and then an increasing risk as alcohol consumption increases.⁸

Alcohol and oral cancer

Alcohol use is associated with various forms of cancer. Systematic reviews report convincing epidemiological evidence that alcoholic beverages increase the risk of squamous cell carcinoma of the oesophagus and of cancers of the pharynx, larynx and oral cavity.^{9,10} Such increased risk

is particularly evident among young adults, where among heavy smoking and drinking subjects an odds ratio for oral cancer of 48 has been estimated with respect to non-smoking, non-drinking peers.¹¹ Ecological studies suggest that the impact of alcohol on oral cancer deaths has increased in recent years and is higher than expected.^{12,13} Globally, the alcohol attributable fraction of overall mortality has been set at 19% (22% among males, 9% among females) for oral and pharyngeal cancers and at 29% (37% among males, 15% among females) for oesophageal cancer.¹⁴

The combination of alcohol drinking with tobacco smoking (which is common) greatly increases the risk of these cancers, each factor approximately multiplying the effect of the other. Compared to never-smokers and non-alcohol drinkers, the relative risk of these malignant neoplasms is increased between 10- and 100-fold in people who drink and smoke heavily. Indeed, in the case of total abstinence from drinking and smoking, the risk of oral, pharyngeal, laryngeal and squamous cell oesophageal cancers in European countries would have been extremely low. Dysplasia of oral mucosa is also increased in people who drink and smoke heavily.^{11,15}

Alcohol is also associated with other cancers. For example, one quarter of liver cancer cases are attributable to alcohol abuse, while an alcohol intake of more than 15 g/day has been associated with colorectal cancer. As for breast cancer, more cases than of any other cancer are attributable to alcohol drinking among European women. It has been suggested that alcohol acts on hormonal factors involved in breast carcinogenesis. A pooled analysis of six cohort studies found a significant dose-response alcohol effect, with consumption of one daily AU increasing the risk for such a condition by 9% and consumption of 3–5 AUs increasing the risk by 41%.¹⁶

Alcohol metabolism and carcinogenicity

Most ethanol, rapidly absorbed through the gastric and duodenal mucosa, is metabolized by the liver, with a small fraction also metabolized by oral and other mucosae of the upper digestive tract.¹⁷

The enzyme alcohol dehydrogenase (ADH) normally catalyses ethanol oxidation to acetaldehyde, which is rapidly oxidized into acetate by the enzyme acetaldehyde dehydrogenase (ALDH). Acetate is oxidized into fatty acids, carbon dioxide and water. It has been demonstrated that the highest rate of ethanol that humans can metabolize does not vary and is not affected by ethanol concentration, by ethanol tolerance, or by persistently heavy drinking. Therefore, a high alcohol intake on a single occasion, such as is the case in binge spirit drinking, leads to acetaldehyde accumulation in the oral mucosa, which promotes the formation of carcinogenic, highly reactive, free radicals. Acetaldehyde seems to be a carcinogenic factor. This mechanism may also help explain the multiplicative carcinogenic effect of heavy smoking and drinking, since acetaldehyde is also produced by tobacco combustion, with a consequent over-accumulation in the oral mucosa. Moreover, ethanol increases membrane permeability of the oral mucosa epithelial cells, thus promoting the penetration of other tobacco carcinogens such as nitrosornicotine.^{18, 19,20}

Alcoholics with the ADH 1C*1 allele seem to be particularly at risk as this allele encodes for an enzyme which rapidly metabolizes ethanol, leading to increased acetaldehyde levels. The allele frequency of the ADH1C*1 allele is significantly increased in heavy drinkers with upper aerodigestive tract cancer compared with age matched alcoholic controls without cancer. Heavy drinkers homozygous for the ADH1C*1 allele have a predisposition to develop upper aerodigestive tract cancer, possibly due to elevated salivary acetaldehyde levels following alcohol consumption.²¹

The ADH1C gene is polymorphic. The ADH1C*1 allele metabolizes ethanol to acetaldehyde at a higher rate than the variant ADH1C*2 allele. The ADH1C*2-2 genotype is associated with susceptibility to smoking and drinking-related squamous cell carcinoma by modifying the biologically effective dose of alcohol.²² Genotype AA of ADH3 may also be a risk factor for upper aerodigestive tract cancer, especially in individuals with low alcohol or tobacco consumption,²³ but others have denied this.²⁴

Alcohol drinking is also strongly associated with the risk of development

of liver cirrhosis, which may result in impaired metabolism of carcinogens, and in impaired immunity. A diet poor in fruits and vegetables, typical of heavy drinkers, is also likely to play an important role in carcinogenesis.^{19,20}

The effects of stopping alcohol use

Few studies have analysed the relationship between stopping alcohol drinking and the risk of cancers of the upper respiratory and digestive tract. There is clear evidence that the risk of oesophageal cancer is reduced by 60% ten years or more after drinking cessation, but the pattern of any risk modification is less clear for oral and laryngeal cancers.

Nevertheless, stopping (or reducing) alcohol drinking, particularly in association with smoking cessation, represents a priority for preventing oesophageal, and possibly oral, cancer.

Diagnosis of alcohol abuse

Whilst it is easy to recognize the effects of acute alcohol intoxication, recognition of the alcoholic patient can be notoriously difficult. Some significant findings suggesting alcohol abuse may be present in the social or medical history. In particular, signs and symptoms that may indicate alcohol misuse include:

- Work absenteeism;
- Unreliability in keeping appointments, meeting deadlines and work performance;
- Marital disharmony;
- Personality change with mood swings;
- Progressive deterioration in personal appearance and hygiene;
- Family history of alcoholism;
- Deteriorating interpersonal relations but person rarely admitting or accepting blame for errors or oversights.

Self-reported questionnaires and structured interviews, such as the CAGE or the Alcohol Use Disorders Identification Test (AUDIT) proposed by WHO, may help.^{25,4}

Laboratory investigations that may be diagnostically helpful include:

- Raised blood levels of alcohol;
- Raised blood levels of gamma-glutamyl transpeptidase and other hepatic enzymes;
- Breath alcohol analysis by infrared light absorption (this has advantages in that it is

non-invasive, fairly hygienic, and a result is obtained almost immediately);

- Macrocytosis and folate deficiency of no obvious cause (macrocytosis alone is one of the earliest signs of alcoholism, later there may be macrocytic anaemia).

- The Blood Breath Ratio (the numeric relationship of alcohol in breath and blood) is often used legally.

The use of saliva makes it easier and more convenient for the dentist to screen for alcoholism, a notable example being *Alco-Screen* (Chematics Inc, North Webster, IN), a rapid test for the estimation of blood alcohol concentration which takes about 2 minutes. Another non-invasive method using saliva is the QED Saliva Alcohol Test.

Treatment

Ninety five per cent of untreated alcoholics die of alcoholism, so treatment is important. The success in quitting alcohol use is strictly dependent on the severity level of the condition and, for the same degree of alcohol dependence, on the appropriateness of the management, as shown in Table 2.

The wide range of interventions that have been developed to deal with alcohol-related problems, can be divided into three categories:

- Brief intervention;
- Specialized treatment programmes;
- Psycho-social treatments.^{26,27}

Brief interventions

Brief interventions, aimed at

providing prophylactic treatment before or soon after the onset of alcohol-related problems, are based on advice about the risk for the consequences of acute alcohol intoxication and the possibility of developing alcohol dependence. The cumulative evidence shows that clinically significant effects on drinking behaviour and related problems can be obtained by this kind of intervention. Nevertheless, the evidence of a true benefit for alcoholics is not clear.

Specialized treatment

This refers to interventions directed at the management of alcohol withdrawal, the prevention of relapse to alcohol dependence and the social and psychological rehabilitation of the drinker. Alcohol withdrawal management is aimed at relieving discomfort, preventing medical complications and preparing the patient for rehabilitation. Subjects with mild or moderate withdrawal features generally require non-pharmacologic or social detoxification, which consists of reassurance, personal attention, vital sign monitoring and general nursing care. For more serious forms of withdrawal, pharmacologic detoxification is more appropriate.

Alcohol rehabilitation is generally provided in residential settings and is costly. Therefore, it is generally restricted to patients who are highly treatment-resistant or have serious medical or psychiatric conditions.

Pharmacotherapy has an evident effect in the treatment of co-morbid

psychiatric disorders in alcoholics and in the rehabilitation of alcohol dependence. However, its impact on treatment of alcohol dependence is debatable. The most frequently used medications are:

- Alcohol-sensitizing drugs, such as disulfiram, which cause an unpleasant reaction (owing to increased acetaldehyde production) when combined with alcohol;
- Opioid antagonists, such as naltrexone, which have been designed to reduce alcohol consumption directly;
- Serotonin, the indoleamine neurotransmitter;
- Acamprosate, an amino acid derivative, which affects GABA and excitatory amino acid (glutamate) neurotransmission.

Psycho-social treatments

Mutual help groups, such as those based on the Twelve Steps of the Alcoholics Anonymous, have been shown to increase abstinence rates and improve the quality of life for many alcoholics, especially when applied in combination with pharmacological treatment.

Alcohol and public health

A recent systematic review on the management of alcohol use disorders from the public health point of view²⁷ has reached the following conclusions:

- Individuals who obtain help for a drinking problem, especially in a timely manner, have better outcomes than those who do not receive help;
- There is little evidence that the type of treatment affects the long-term outcome;
- Treatment intensity and duration are not associated with more pronounced improvements in outcome;
- Medically-based inpatient treatment is more costly but generally no more effective than non-medical outpatient care.

National and community-based approaches have been developed in many countries, with differing levels of success. The first recourse in case of public concern about alcohol-related problems is to implement public information campaigns which, however, are unlikely to be effective in the long term.²⁸

A more effective community-based preventive approach is controlling alcoholic beverage price and availability.

Percentage staying sober for the next 12 months

Quit on their own	4
Go through treatment with use of drugs to reduce dependence	50
Go through treatment with use of drugs to reduce dependence and regularly attend Alcoholics Anonymous meetings	70
Go through treatment with use of drugs to reduce dependence, regularly attend Alcoholics Anonymous meetings, and go to aftercare once a week	90

Table 2. Different success probability in quitting alcohol use.

Tax increases have been shown to affect positively some acute and chronic drinking-related causes of death, such as cirrhosis, driving deaths and violent crime.²⁹ Another public health measure is the reduction of alcohol-related vehicle casualties, by means of countermeasures, such as forbidding driving above a given blood alcohol concentration level.³⁰

Oral implications of anti-alcohol misusing campaigns

The potential benefits, in terms of oral cancer, alcohol-attributable mortality prevented fraction, of an alcohol drinking preventive campaign implemented in, for example, the UK on only heavy drinking individuals (the so-called high-risk approach) can be estimated using the national figures of heavy drinking prevalence among adults (6.0% for males, 2.0% for females),² crude oral cancer mortality (2.4 x 100,000 and 1.5 x 100,000 for males and females, respectively),³¹ and the alcohol attributable fractions of deaths for any cause and for oral cancer (22% and 9% for males and females, respectively).¹⁴ It was supposed that the hypothetical campaign, which was aimed at changing alcohol drinking behaviour, from heavy binge drinking to moderate drinking during the main meals, would be 33% effective. The results are that the estimated prevented oral cancer deaths would be 67 annually (54 males, 13 females), with an estimated prevented fraction of 7.2% and 2.9% for males and females, respectively.

The role of the dentist in anti-alcohol misusing campaigns

The dentist could also play an active role in preventing alcohol-related conditions and deaths, since a large proportion of the adult population attend for dental care. The medical history may reveal some alcoholics and others are detected from the results of investigations.

Intervention in the dental setting, through a secondary or even tertiary prevention measure, could be a useful social contribution in reducing the damage caused by heavy drinking to the alcohol users themselves and to the community.

Mouthwashes

Mouthwashes	% v/v Alcohol
Listermint	13.0
Listerine Mouthwash	26.9
Scope Mouthwash	19.0
Search	15.3
Mentadent Mouthwash	12.0
Oral B Dental Rinse	9.4
Colgate Fluorigard Daily	5.0
Macleans Active Mouthguard	17.5
Safeway ExtraStrength Antiseptic	26.9
Safeway Antiplaque Mouthwash	7.7
Corsodyl	7.0
Pearldrops Smokers 1+1	14.2
Perio Aid	6.5
Colgate Total (Plax) red	8.0–8.3
Colgate Total (Plax) green	5.0–5.2
Plax (Pfizer – USA only)	8.7
Cepacol	14.5
Listerine Coolmint (other flavours also)	21.6
Colgate Fluoriguard	5.0
Oral B Anti-Plaque Dental Rinse Alcohol Free	0.0
RetarDEX Oral Spray and Oral Rinse	0.0
Colgate Rinse Alcohol-free	0.0
TheraBreath	0.0
Dentyl pH	0.0
Macleans Alcohol Free Smoothmint Mouthwash	0.0
Yotuel Whitening Mouthwash	0.0
Frador Coolmint Mouthwash	0.0
Cariax Gingival	0.0
Elmex	0.0
Meridol	0.0
Colgate Fluoriguard	0.0
Perio Aid New Formulation	0.0

NB *Medicina Oral* 2004, Vol. 9 No. 2 pp. 116–123 Table 1 shows different alcohol concentrations for Spanish versions of some of the above mouthwashes, eg Perio Aid 11.6% rather than 6.5%.

Table 3. Alcohol concentration in various mouthwashes.

Alcohol-containing mouthwashes

A potential activity of alcohol-containing mouthwashes on oral cancer has been postulated, since alcohol in mouthwashes can be as high as 27%, equivalent to 54-proof spirits (Table 3). Mouthwashes increase the time the mucosa is in contact with alcohol, and those with a high content of alcohol can cause hyperkeratotic lesions, both in laboratory animals and humans.

Nevertheless, systematic reviews provide no sufficient evidence of

a causal relationship between the use of alcohol-containing mouthwashes and the development of oral cancer.^{32,33}

In the absence of a clear evidence-based positive or negative response, prescription of alcohol-containing mouthwashes should, therefore, be made with prudence, particularly among children and high-risk subjects.

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