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MEDICAL PRACTICE OF PREVENTIVE NUTRITION

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Throughoutout medical history, there has generally been appreciation that there is value in the use of food, or more recently nutrients, in therapeutics, and there has been acceptance that, since food is vital to life, its wise use prevents health problems. A more systematic examination of how particular nutrients, foods or food patterns might contribute to major disease problems, and therefore how changes in usage might allow prevention, began with the identification of nutrient deficiency disorders, but in the last 20 - 30 years, diet has become the focus of attention in relation to the major non-communicable diseases of people in industrialized and industrially emerging nations. The suggested opportunities for improved quality of life where there is less illness and greater sense of well-being, and which may be possible with changes in nutritional status, are themselves a stimulus for preventive nutrition.

Preventability of the disease need not be absolute or complete avoidance of the disease. It may be the delay in onset of disease until a later age. The severity of the disease may also be reduced. Prevention also has to do with the maintenance or enhancement of well-being. It is important to identify and take opportunities for prevention in primary (where no disease exists), seondary (where the basic disease process has manifested itself) and tertiary (where complications of the disease in question have arisen) settings. Reversability of disease processes is also another dimension of prevention.

How does the approach of using nutritional means to prevent disease fit into medical practice? What are the limitations of this approach and what needs to be known before embarking on it seriously? After assessing these questions, the bulk of the book considers the application of the preventive approach to nutrition in a range of systemic diseases. A critical approach is designed to elicit greater awareness of the various facets of prevention and avoids prescribing facile 'answers'. The book will serve as a practical reference manual to guide practitioners and support teachers of the subjects involved in developing their own, appropriate measures of successful preventive nutrition practice.

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VOBECKY

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This manual is conceived and developed by the IUNS Committee on Medical Education under the Chairmanship of Professor Jitka Vobecky

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INTRODUCTION

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Acceptance of prevention through nutrition

Throughout medical history, there has generally been an appreciation that there is value in the use of food, or more recently nutrients, in therapeutics, and there has been an acceptance that, since food is vital to life, its wise use prevents health problems. A more systematic examination of how particular nutrients, foods, or food patterns might contribute to major disease problems, and therefore how changes in usage might allow prevention, began with the identification of nutrient deficiency disorders, but, in the last 20–30 years diet has become the focus of attention in relation to the major non-communicable diseases of people in industrialized and industrially-emerging nations. Indeed, one of the greatest challenges may now be the approach that should be taken where nutritional status is transitional between protein and specific nutrient deficiency on the one hand and energy imbalance with consequent overfatness on the other.

The suggested opportunities for improved quality of life where there is less illness and greater sense of well-being and which may be possible with changes in nutritional status are themselves a stimulus for preventive nutrition. Once day-to-day survival ceases to be a problem for the poor and hungry, the quality of food supply and healthy food choices can be contemplated. Even so, one is often quite culturally-bound in the food one chooses – and there are historical examples of how people have died where food was available, but not acceptable. The Caucasian Australian explorers, Burke and Wills, perished, in an arid area, with no residual food supplies, but where Aboriginal Australians lived on local bush food. Interestingly, recent work by Ono in Japan indicates that there is specific neuronal memory for food[1,2]. The ability to distinguish between food and non-food is found in the amygdalal part of the brain, so that neurophysiological factors (amongst others) may need to be taken into account, or taken advantage of, in preventive medicine. Furthermore, equity in food distribution, competent food production, storage and processing, sufficient food knowledge and safe food behaviours generally need to be in place before quality of life and long-term health can become personal food choice issues.

In difficult economic times, governments and individuals are driven increasingly

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by considerations of cost-effectiveness. Health departments and agencies see that if chronic, disabling diseases like stroke, diabetes and osteoporosis with fractures can be prevented by nutritional means, then costs to the community will be saved and life-long productivity increased.

Nature of preventability

Preventability of disease need not be absolute or complete avoidance of disease. It may be the delay in onset of disease until a later age. If, for example, the menopause is partly nutritionally determined[3], and it is delayed or modified, then significant osteoporosis and related fracture may be delayed. Again, if biological aging occurs at an older chronological age, as appears to have happened in the last few decades in Sweden and presumably elsewhere, (See chapter 9 and[4]) then this could be an important way in which eating patterns might contribute to prevention.

The severity of disease may also be reduced. For example, if coronary stenosis due to atherosclerosis is lessened, the chances of thrombotic occlusion reduced, or the likelihood of arrhythmia with reduced myocardial blood flow decreased, and each of these is separately influenced by nutritional factors, the severity of ischaemic heart disease is less and, in measure, preventable[5]. Prevention also has to do with the maintenance or enhancement of well-being.

It is important to identify and take opportunities for prevention in *primary* (where no disease exists), *secondary* (where the basic disease process has manifested itself) and *tertiary* (where complications of the disease in question have arisen) settings. *Reversibility* of disease processes is also another dimension of prevention.

Assumptions in prevention strategies

A judgement is required about present evidence for prevention and the risks and benefits of action. The assumptions underlying preventive nutrition include the following:

1. That the disease process is modifiable.
2. That, in a multifactorially determined disease process, changing a particular factor can be critical or, if not, that several factors may require change.
3. That the theoretical basis for prevention is often incomplete and, usually, as with other biomedical approaches, based on composite lines of evidence, with, on occasion, extrapolation to the situation of interest.
4. That the way we eat has a significant role in the disease process in question.
5. That there are certain preferred ways of eating, and limits to the way we can eat, if optimal health and maximal life span are sought.
6. That what we do at one age may be biologically consequential for a later age, or that it may be worth doing in later life.
7. That what is necessary to change is capable of definition, and that the process of change is sufficiently straightforward and realistic to stand a chance of being carried through, taking account of cultural and other constraints.

References

1 Ono T, Tamura R, Nishijo H, Nakamura K, Tabuchi E. Contribution of amygdalar and lateral hypothalamic neurons to visual information processing of food and non-food in monkey. Physiol Behav 1989; 45 (2):411–21.

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2 Nishijo, H, Ono, T. Food memory: involvement in food recognition. Asia Pacific J Clin Nutr 1992; 1:3–12.

3 Wilcox G, Wahlqvist, ML Burger, HG, Medley G. Oestrogenic effects of plant-derived foods in postmenopausal women. Brit Med J 1990; 301, 905–6.

4 Svanborg, A. Aspects of aging and health in the age interval 70–85. In: Health and aging. E JJF Schroots, JE Birren, A Svanborg. Springer Swets Publishing Service, Lisse, 1988, pp 133–141.

5 Wahlqvist ML International trends in cardiovascular disease in relation to dietary fat intake: interpopulation studies, pp 539–543. In Proceedings XIII International Congress of Nutrition, Brighton, UK, August 1985, ed TG Taylor and NK Jenkins, London: John Libbey.

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ALCOHOL

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Strategies for the prevention of alcohol abuse should focus on prevention policies which ideally operate at the level of the individual and the community. At both levels, an understanding of alcohol as a food/beverage and as a drug/chemical, with its early effects on psychology, physiology and social environment, is very important to enable decision-making on drinking habits.

Rapid early screening of alcohol consumption should be performed on every individual coming for a routine health check-up at the primary health care level, admitted to a hospital ward, or during an out-patient visit, to identify those at high risk of alcohol abuse.

Introduction

The word *alcohol* comes from the Arabic 'alcohol', the finely powdered antimony oxide used as black eye paint. Then came spanish, '*alcoholar*', to turn black, and *alcoholado*, a beast with black circles around the eyes. In Europe the word alcohol was first used in conjunction with eye shadow cosmetics. In the sixteenth century, distilled wine, which was previously known as the *spirit of wine*, was called *alcohol vine*, the quintessence of vine, later abridged to *alcohol*[1].

Today, many different substances are chemically classified as alcohols. Most of these substances are toxic, and are non-consumable by humans. An exceptional substance is ethyl alcohol (*ethanol*), which has a chemical formula of C_2H_5-OH. Pure ethanol is colourless and inflammable.

Sources of alcohol

Ethanol is produced by the activities of certain yeasts, contained on the outside of grapes, or added to the brewing process, which ferment sugars to form ethyl alcohol and CO_2. The fermentation process finishes when all the sugars have been converted into ethanol, or when the concentration of ethanol reach a level where the yeast can no longer survive. A higher ethanol concentration can be made by distilling the liquid and allowing the production of a much more potent beverage.

The major components of alcoholic beverages are water and ethyl alcohol. Other minor components, which are called congeners, include ethyl acetate, iso-amyl

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alcohol, various sugars, minerals and B-group vitamins and are present in some alcoholic beverages. Some of these substances may be involved in the 'hang-over' syndrome and other toxic consequences of alcohol.[1]

At an international level, many indigenous alcoholic beverages contain significant amounts of protein and other micronutrients; it is characteristic of Western alcoholic drinks that they are clarified and purified – the processes involved remove nearly all of these nutrients.

As a solvent, absolute ethanol (>95%) is also widely used in industry. Various additional chemical substances are added to this industrial form of alcohol which render the product undrinkable.

Mechanisms of alcohol addiction

The modern theories of the mechanisms involved in alcohol addiction are based on findings that the aldehyde metabolites of central nervous system biogenic monoamines and the aldehyde metabolites of alcohol form a condensation product which structurally resembles the morphine-like alkaloids, and is called tetrahydroisoquinolone (TIQ)[2].

It is thought that TIQ acts as a false neurotransitter. A number of findings support this theory. An increased ethanol preference in rats can be induced by the infusion of TIQ into the lateral ventricles of the brain. In human alcohol abusers, higher levels of TIQ were found in urine and cerebrospinal fluid than in non-drinkers[2]. Alcohol has been reported to inhibit binding of endogenous opioids to s opiate receptors, whilst TIQ has been reported to bind opiate receptors. Naloxone, an opiate antagonist, prevents withdrawal convulsions in ethanol-dependent mice and blocks subcortical seizure activity produced by ethanol in monkeys. In several studies, naloxone reduced alcohol-induced seizures in man and prevented psychomotor impairment induced by a small amount of alcohol in normal volunteers. The genetic control of CNS opioid production is uncertain, and may be different in some alcohol abusers.

Clinical features of the development of alcohol dependence can be subdivided into four states, the preaddiction, the prodromal, the crucial, and the chronic state. The progression from prealcoholic to chronic state is sufficiently insidious and gradual to make it impossible to distinguish between the states.

The preaddiction symptoms begin when drinking is no longer social but rather a means to escape from anxiety and tensions. The habit progresses to a definite pattern. The drinker begins to drink more heavily, and temporary losses of memory (blackouts) can be experienced in this state. With the progression of drinking habit, blackouts occur more frequently. When a chronic hangover is frequent, the drinker has reached the chronic state of alcohol abuse.

At-risk groups

In Western countries, male sex and increasing age are risk factors for alcohol abuse. The proportion of males who drink alcohol is generally higher than the proportion of females at all levels of consumption and across all age groups. In both sexes, the greatest proportion of heavy drinkers tends to be in the third and fourth decades of life. Oriental women also have a lower rate of alcohol abuse than men.

A family history of alcohol abuse increases the risk for abuse. There is evidence

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that children from an alcoholic family become alcohol abusers, even when reared by a non-drinking family[3].

Alcohol consumption patterns differ internationally. In many Western countries, most adults consume some alcohol, and approximately one in every seven persons is a heavy drinker. The incidence of alcohol abuse in oriental countries is less[4].

Genetically inherited metabolic differences in population groups may account for some of these findings. A non-specific form of alcohol dehydrogenase, found in most oriental populations, may cause flushing, palpitations and other unpleasant effects after ingestion of a small amount of alcohol. This reaction is a powerful incentive in preventing such populations from drinking a large amount of alcohol. This reaction can be hindered by some antihistamine drugs.

There is evidence that some of the manifestations of alcohol excess, such as predisposition to Wernicke's encephalopathy, may be genetically based. The thiamine-dependent enzyme transketolase may be relatively inactive in these individuals.

Cultural factors which have been identified include a childhood history of a lack of attention, and various occupational groups. These include bartenders and brewery workers, members of the armed forces, and people in isolated occupations.

About one in every seven patients admitted to general medical wards in hospitals for various reasons not related to alcohol has been identified as a problem drinker.

Metabolism

After consumption, alcohol is absorbed mainly from the stomach and upper small intestine. The blood alcohol rises to a maximum during the absorption phase. After equilibration within tissues, the blood alcohol level decreases linearly with time. The rate of absorption varies amongst individuals, and within an individual at different times, but is mainly dependent on the alcoholic concentration of the beverage consumed, and on the emptying time of the stomach. It increases with concentrations up to 50%, and above that concentration, alcohol will irritate the gastric wall and induce pyloric spasm. Any condition that causes rapid emptying of the stomach will cause the rate of alcohol absorption to increase, as does the fasting state[5].

Alcohol is distributed in the body water. Since women generally have a smaller proportion of body water and greater proportion of body fat than men, the distribution volume is smaller for the same weight, and thus the concentration of alcohol achieved in tissues is proportionally greater.

Between 90% and 98% of the alcohol that is consumed, is oxidized to acetaldehyde, which is further oxidized to acetate and carbon dioxide in the liver[6]. The products of this oxidation may be used for the synthesis of fatty acids, amino acids or carbohydrate. The process supplies energy to the body of approximately 7 kcal per gram of alcohol. The remainder is excreted as carbon dioxide through the lungs and kidneys.

Blood alcohol concentration

The degree of intoxication and its potential impact are generally estimated by measuring the blood alcohol concentration (BAC). It is usually expressed as milligrams of alcohol per 100 ml sample of blood. The body contains, approximately

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40 litres of water of which 5 litres (5000 ml) comprise the blood volume, so that at a BAC of 0.05 the total amount of alcohol present in the blood would be 2.5 g, approximately one eighth of the total body volume.

The quantity of alcohol excreted through the lung depends directly on its concentration in the blood. The relation between BAC and breath alcohol concentrations (BrAC) can be defined as the blood:breath ratio. Although a small variation exists, it is generally accepted for medico-legal purposes to be between 2100:1 and 2300:1.

Effects of excess alcohol consumption on food intake

The effect of alcohol on food intake is difficult to distinguish from its effects on socioeconomic status and psychological status (see below), as well as its effect on nutritional status.

The effects of alcohol on the sensation of hunger have long been studied. Scott *et al.*[7] showed that an increased desire for food was developed within 5 minutes of consumption of 200 ml of 20% alcohol. Beazell & Ivy[8] thought that the increase in appetite was due to the stimulation of taste and other oral sensations increasing the general sense of well-being, and so the desire for food. They noted that high alcohol intakes may depress hunger. Habits such as coffee drinking and cigarette smoking have been reported to be associated with a decrease in the desire for food among heavy drinkers[9], whilst studies on non-abusing alcohol drinkers do not suggest that their drinking practices reduce food intake. There still appear to be few explanations as to why or when alcohol abusers are likely to eat normally or in excess, or when their appetite and food intake are diminished.

The place and the people with whom alcohol consumption takes place are likely to influence food intake and quality, depending on whether, for example, alcohol is taken in public drinking houses or at the home, with friends or alone.

Effects of excess alcohol consumption on nutritional state

Epidemiological evidence exists that in Australia, alcohol accounts for about 6% of consumed dietary energy[10]. This figure increases as alcohol consumption in an individual increases. Chronic alcohol consumption, while providing an energy source, may impair nutritional status by a number of different mechanisms. Apart from alterations in food intake, there may impairment of gastrointestinal absorption through effects on the gut mucosa, liver, and pancreas.

Heavy alcohol intake can induce metabolic pathways within the liver which directly oxidize ethanol to carbon dioxide and water, and hence bypass the dehydrogenase pathways. These so-called MEOS (mitochondrial ethanol oxidizing system) pathways may provide an explanation for some of the wasting seen in alcohol abuse, since not all of the energy derived from oxidation is trapped and transferred to ATP (Figure 1). If food intake is poor and alcohol intake is high, the breakdown of adipose tissue and muscle tissue associated with gluconeogenesis is another explanation for weight loss (Figure 2).

The effect of alcohol intake on energy stores can also be assessed by determining body composition. Alcohol abusers have been considered to be at risk of obesity if their food intake remains normal, and at risk of wasting if their food intake is reduced. D'Alonzo & Pell[11] found that more alcohol abusers were below ideal

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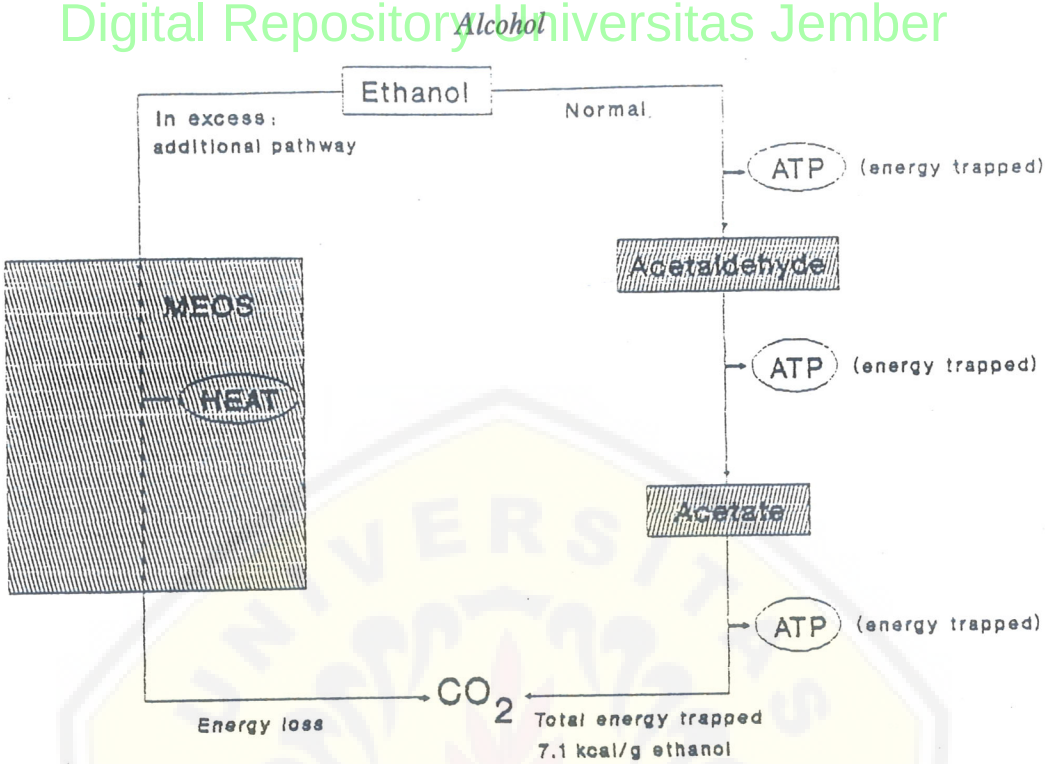

Figure 1 Pathways for alcohol metabolism, a possible explanation for wasting in alcohol abuse.

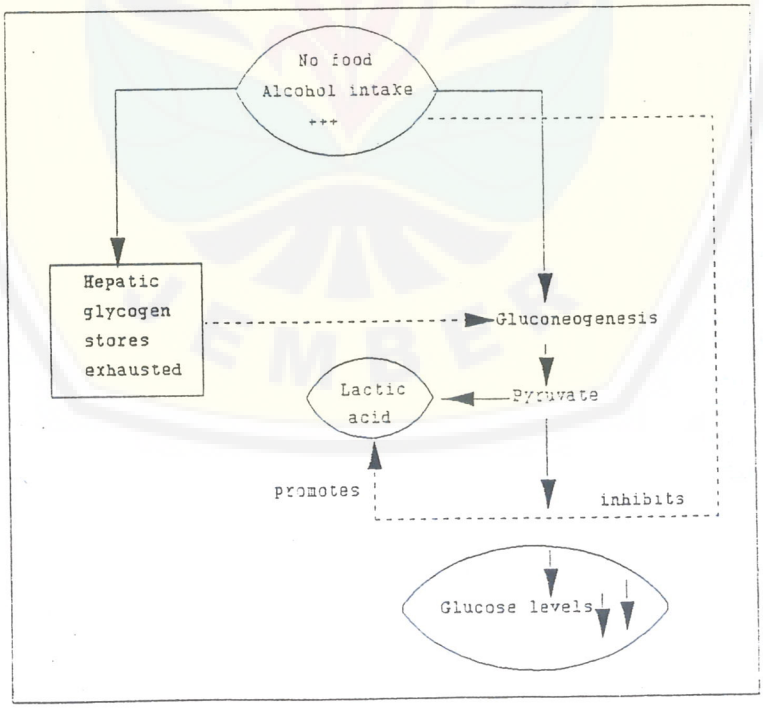

Figure 2. Hypoglycaemia in acute alcohol consumption.

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weight than non-drinkers. Other biological effects may confound this picture. Myrhed[12] studied monozygous and dizygous twins, in whom the drinking habits of one twin were different from those of the other. No significant correlation was found between alcohol consumption and measures of body weight. There is some evidence that heavy alcohol intake increases the waist-to-hip ratio, and hence increases the risk for cardiovascular diseases[13,14].

Most of the studies involving the effect of alcohol on body composition were performed on heavy alcohol intake. Unpublished data (D. Prijatmoko and BJG Strauss) suggest that regular light-to-moderate alcohol consumption will not affect fat mass and fat distribution. However, when other confounding factors such as smoking and income level are included in the analysis, increases in both lean mass and fat mass are associated with relatively small alcohol intakes.

Simko *et al.*[15] compared groups of drinking alcoholics with or without liver disease, which may impair protein synthesis. General wasting was observed in the group with liver disease. Height-to-weight ratio was adversely affected in alcohol drinkers in comparison with abstainers. In Simko's study, where the average dietary intake among the drinking alcoholics was adequate, the possibility that the direct toxicity of alcohol was the major factor in weight loss amongst the alcoholics was raised.

Increased urinary nitrogen excretion was observed when alcohol was fed to normal subjects during a controlled metabolic study. This may explain some of the observed muscle wasting, although alcohol may also damage skeletal muscle, producing lesions characterized by acute or chronic myopathy. Urbano-Marquez and co-workers[16] studied 495 subjects with chronic alcoholism and showed that alcohol is toxic to striated muscle in a dose-dependent manner.

Among the more common vitamin deficiencies among alcohol abusers are deficiencies of folate, thiamin and pyridoxine. Wernicke's encephalopathy, Korsakoff's psychosis, peripheral neuropathy, and wet beri-beri are the frequent accompaniments of thiamin deficiency. Nearly 50% of individuals with folate deficiency have megalobiastic anemia[17]. If the folate deficiency leads to a megaloblastic intestinal mucosa, further malabsorption of micronutrients may be expected. Other vitamin deficiencies can be expected to occur in individuals whose food consumption is restricted in amount and variety. Mean circulating blood levels of biotin, vitamin B-6 and ascorbic acid tend to be lower in alcoholic patients than in controls.

Alcohol disturbs the absorption, transportation and utilization of nutrients required for bone formation. Some studies reported a reduction in vertebral bone density and in the amount of iliac cancellous bone in alcoholic subjects. This reduction can reach 58% of normal[18]. Marked reduction in active bone turnover was observed without evidence of osteomalacia. Normal serum levels of calcium, magnesium, phosphorus, calciferol, calcitriol, parathyroid hormone and cyclic adenosine monophosphate suggest that bone disorders in alcoholics are not due to inhibition of parathyroid hormone secretion or abnormal vitamin D metabolism, but to an inhibition of bone remodelling by mechanisms independent of the calciotropic hormones. Fracture risk is increased in alcohol abuse.

Effects of excess alcohol consumption on socioeconomic state

The effects of excess alcohol consumption involve more than purely biological

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effects. A range of family and personal factors can be altered, depending on the amount of drinking involved. These include isolation from family and community activities, financial problems, legal problems, and an increased probability of driving accidents.

Work performance can be significantly altered; there is an increased frequency of absenteeism, impaired job performance, and an increased probability of industrial accidents.

Effects of excess alcohol consumption on neuropsychological state

Alcohol is a CNS depressant, and its effect on the CNS are related to its concentration in the blood. The classic syndrome includes loss of balance and poor coordination of the eyes and limbs. Vision and the ability to judge distance and speed are also impaired.

Alcohol-related dependency problems can be elicited by the CAGE questionnaire or the MAST questionnaire (see Appendix), which covers a greater range of indicators of problem drinking.

Biochemical markers

Biochemical and haematological abnormalities can be found in the blood during alcohol abuse. Some of these abnormalities have been proposed as indicators of alcohol abuse. Glutamyl transpeptidase, a hepatic cytosolic enzyme (GGT), is used as a quantitative marker of alcohol consumption. In some studies, it was found that GGT is increased in 80–88% of subjects who consume more than 50 g alcohol per day[19-22]. However, increased GGT activity is also found in subjects taking barbiturates or other enzyme-inducing agents, in non-alcohol-related liver disease, in other gastrointestinal disease, and in diabetes mellitus.

Erythrocyte mean corpuscular volume (MCV) increases in alcohol abuse but only 30% of affected individuals can be detected using this measurement. Increases in MCV also occur in other conditions such as thyroid disease, folate deficiency and liver disease from other causes.

The combination of increased GGT and increased MCV is a more powerful discriminant for the detection of alcohol abuse.

More recent findings suggest the use of isoforms of serum transferrin as a biomarker for alcohol abuse. The most constant abnormality in the alcohol abuser is the elevation of transferrin with pI 5.7, and in some cases those with pI 5.8 and 5.9[23]. This biomarker was reported to have a specificity of 100% and a sensitivity of 91%. Increased levels were reported to occur occasionally in healthy individuals after daily consumption of 60 g of alcohol during a 10 day period[24]. It may be that this compound is more sensitive than the combination of GGT and MCV in detecting alcohol abuse.

As yet, no currently available biochemical marker can reflect alcohol consumption within the usual average intake range.

Clinical features of early detection of excess alcohol consumption

The clinical features of early alcohol excess are protean and are difficult to distinguish from other, often minor, disorders. A history of drink-driving offences, or

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deterioration in job performance may be given. Frequent unexplained diarrhoea is another feature of alcohol abuse, whilst features associated with chronic liver disease and other organ damage indicates advanced disease.

Clinical and biochemical criteria for suspecting alcohol abuse include:

(1) Average consumption of more than four standard drinks daily for men or two standard drinks for women.
(2) History of binge drinking.
(3) GAGE questionnaire −2 or more positive responses; MAST questionnaire −4 or more positive responses.
(4) GGT >50 iu/litre for men, >30 iu/litre for women (in absence of other known causes of raised GGT).
(5) MCV >98 fl (in absence of other known causes of raised MCV).

Methods of reducing alcohol intake

Epidemiological evidence suggests that more new heavy drinkers are appearing than the numbers successfully treated. This is likely to be due to a change in drinking habits of young people, since the prevalence of heavy drinking increases with age until the 6th decade[25]. The aim of primary prevention is to stop the statistical progression from light social drinking to regular heavy drinking.

There is evidence that some of the manifestations of alcohol excess, such as predisposition to Wernicke's encephalopathy, may be genetically based (the thaimin-dependent enzyme transketolase may be relatively inactive in these individuals). As yet, markers to identify such individuals are not yet available, but when they are, it will be important to counsel such individuals early in their life with respect to the increased risk associated with alcohol consumption and the possible transmission of this risk to future generations.

A greater potential for preventive strategies is likely to come from identifying and influencing those social, environmental, and behavioural factors that lead to the development of alcohol abuse.

Apart from countries where alcohol consumption is banned for religious reasons, earlier strategies to reduce community alcohol consumption have included taxation on consumption, defining age limits for legal purchase, and defining times during which alchohol may be purchased and consumed in public places, or even, as manifest during the 1930s in the USA, by prohibition of alcohol consumption entirely. Modified forms of 'prohibition' are still in operation today in some Scandinavian countries.

In countries where alcohol consumption is legal, more recent strategies have emphasized changing community and individual attitudes concerning alcohol consumption by increasing the surveillance of behaviours such as driving which are influenced by alcohol consumption and making lower-alcohol-containing beverages more socially and financially desirable. It has also been possible to limit advertising of different beverages publicly.

The approaches of the above strategies to reducing alcohol intake need not be in conflict. Environmental controls by government include increases in alcohol prices through increased taxation, extensive public education programs to inform and change the public attitudes and behaviour concerning alcohol, and punitive

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measures to deter socially costly complications of alcohol, such as drinking and driving. These controls can be complementary to providing the individual with methods for assuming the responsibility in dealing with and using alcohol for social needs whilst taking account of the health profile[26,27], including the psychological environment.

This will help people to reduce the stress which may act as the trigger for alcohol abuse, and yet still enable them to enjoy the social benefits of alcohol consumption in the context of an awareness of the importance of healthy food intake patterns and physical fitness.

Alcohol reduction in the individual

Early education of the younger members of the community who are more likely to be light drinkers or non-drinkers but may, at the same time, be at risk for becoming heavy drinkers, can occur at home, at school, at the time of obtaining driving licences[28], and on those occasions when they come into contact with health professionals.

If it becomes possible to identify those at genetic risk for the consequences of alcohol abuse, then screening of such individuals and providing genetic counselling become important additional roles for medical practitioners.

The workplace is also an important arena in which education programme can take place and individuals at risk can be identified, either through management strategies or through trade-union-based initiatives.

In a hospital study, a group of those admitted to a general medical ward who were identified as being at risk for becoming problem drinkers on the basis of a family history of alcohol abuse received individual counselling, whilst a control at-risk group did not. After one year, a reduction of alcohol intake occurred in both groups, but a greater reduction was found in the group which was counselled[29].

Alcohol reduction in the community

The growing concern of the society to reduce the consumption of alcohol in the community is reflected in changes in laws involving the drinking age, excise taxes, and product advertisements. Other approaches including warning labels, lists of ingredients, and positive advertising can also be used in the community. The role of community-wide biochemical screening for markers of alcohol abuse, when such sensitive and specific markers become available, in a manner analogous to blood pressure or cholesterol screening is more controversial.

Easier availability of reliable self-testing breath analysers or the requirement that cars be fitted with devices which prevent driving unless certain skilled tasks, likely to be affected by alcohol, can be passed are also future legislative possibilities. Establishing community physicians with career interests in the prevention and treatment of alcohol disorders is an important part of the development of a community network of resources.

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Appendix 1

CAGE Test (Mayfield *et al.*, 1974[30]).

1. Have you ever felt you should **cut** down on your drinking?
2. Have people **annoyed** you by criticising your drinking?
3. Have you ever felt bad or **guilty** about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hang-over?
   (Eye-opener)?

Two or three positive responses suggest alcohol dependence.

Appendix 2

Brief MAST (Pokorny *et al.*, 1972[31]).

	circle correct answer	
Do you feel you are a normal drinker?	Yes	No (2pts)
Do friends or relatives think you are a normal drinker?	Yes	No (2pts)
Have you ever attended a meeting of Alcoholics Anonymous	Yes (5pts)	No
Have you ever lost friends or girlfriends or boyfriends because of drinking?	Yes (2pts)	No
Have you ever got into trouble at work because of drinking?	Yes (2pts)	No
Have ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?	Yes (2pts)	No
Have you ever had delirium tremens (DTs), severe shaking, heard voices or seen things that were not there after heavy drinking?	Yes (5pts)	No
Have you ever gone to anyone for help about your drinking?	Yes (5pts)	No
Have you ever been in a hospital because of drinking?	Yes (5pts)	No
Have you ever been arrested for drunken driving or driving after drinking?	Yes (2pts)	No
	Total score:	

Five or more points said to be diagnostic.

References

1 Gastineau CF, Darby WJ, Turner TB. Fermented food beverages in nutrition. The nutrition foundation, Academic Press, New York, 1979.
2 Kaplan HI, Sadock B. Comprehensive text book of psychiatry/IV, Vol 1, 4 ed. Williams and Wilkins, Baltimore London, 1983; 1020–1028.
3 Cadoret RJ, O'Gorman TW, Troughton E, Haywood E. Alcoholism and antisocial personality: Interrelationships, genetic and environmental factors. Arch Gen Psychiat 1985; 42:161–167.

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4 Akutsu PD, Sue S, Zane NWS, Nakamura CY. Ethnic differences in alcohol consumption among Asians and Caucasians in United States: An investigation of cultural and physiological factors. J of Studies on Alcohol 1989; 50(3):261–267.
5 Lieber CS. The Metabolism of Alcohol. Sci Am 1976; 234:25–30.
6 Rognstad R, Grunnet N. Enzymatic pathways of ethanol metabolism. In: Biochemistry and pharmacology of ethanol. Majchrowicz E, Noble EP, eds. Vol. 1, New York: Plenum; 1979:65–85.
7 Scott CC, Scott WW, Luckhardt AB. Effect of alcohol on hunger sense. Am J Physiol 1938; 123:248–255.
8 Beazell JM, Ivy AC. The influence of alcohol on the digestive tract. Quart J Stud Alc 1940; 1:45–73.
9 Friedman GD, Sieglaub AB, Seltzer CC. Cigarettes, alcohol, coffee and peptic ulcer. New Eng J Med 1970; 290:469–473.
10 Alcohol in Australia. A summary of related statistics. Australia Government Publishing Service, Canberra 1988.
11 D'Alonzo CA, Pell S. Cardiovascular disease among problem drinkers. J Occup Med 1968; 7:344.
12 Myrhed M. Act Med Scand 1974: Supp 567:8–92.
13 Patterrson P, Hallgren P, Sjoberg C, Sjostrom L, Bjorntorp P. Alcohol and abdominal distribution of fat. Int J Obesity 1990; 14(2):80.
14 Laeson B, Bengtsson C, Bjorntorp P, Lapidus L, Sjostrom L, Svardsudd K, Tibblin G, Welin L, Whihelmsen L. Is abdominal body fat distribution a main explanation for the male and female difference in the risk of myocardiac infarction? Int J Obesity 1990; 14(2):80.
15 Simko V, Connel AM, Banks B. Nutritional status in alcoholics with and without liver disease. Am J Clin Nutr 1982; 35:197–203.
16 Urbano-Marquez A, Estruch R, Navarro-Lopez F, Grau JM, Month L, Rubin E. The effect of alcoholism on skeletal and cardiac muscle. N Eng J Med 1989; 320:409–415.
17 Pietrzik K. Folate deficiency: morphological and functional consequences. In: Somogy JC Hydas eds. Nutrition in the prevention of disease, bb1 Nutr Dieta Basel. Karger, 1989: No 44, pp 123–130.
18 Bikle DD, Genant HK, Cann C, Recker PR, Halloran BP, Strewler GJ. Bone disease in alcohol abuse. Annals of Int Med 1985; 103:42–48.
19 Rollason J, Pincherly G, Robinson D. Serum GGT in relation to alcohol consumption. Clin Chim Acta 1972; 39:75–80.
20 Rosalki S, Rau D. Serum GGT activity in alcoholism. Clin Chim Acta 1972; 39:41–47.
21 See G, Heffelfinger R, Childers D. GGT in the alcoholic patient's response to abstinence. Ann Clin Lab Sci 1976; 6:349.
22 Morgan M. Markers for detecting alcoholism and monitoring for continued abuse. Pharmacol Biochem Behav 1975; 252(13 suppl):297–306.
23 van Eijk H, van Noort W, Dubelaar M-L, van der Heul C. The microheterogeneity of serum transferrin in Biological Fluids. Clin Chim Acta (183); 132:167–171.
24 Stibler H, Borg S, Joustra M. Micro anion exchange chromatography of carbohydrate-deficient transferrin in serum in relation to alcohol consumption (Swedish Patent 8400587–5). Alcohol Clin Exp Res 1986; 10:535–544.
25 National Heart Foundation of Australia. Risk factor prevalence study, No 2, 1986.
26 Terris M. The epidemiology revolution: National Health Insurance and the role of the Health Department. Am J Publ Health 1976; 66:1156–58.
27 Vladech BC, Weis RJ. Policy alternatives for alcohol control. Am J Publ Health 1975; 65:1340.
28 O'Hagan J, Whiteside E, Bieder L. Hand book on alcoholism for medical practitioners. Wellington: Alcoholic Liquor Advisory Council; 1982:23–45.
29 Chick J, Lloyd G, Crombie E. Counselling problem drinkers in medical wards: a controlled study. BMJ 1985; 292:1703–1708.
30 Mayfield D, McLeod G, Hale P. The Cage questionnaire. Am J Psych 1974; 131:1121–1123.
31 Pokorny AD, Miller BA, Kaplan HB. The brief MAST: a shortened version of the Michigan Alcoholism Screening Test. Am J Psych 1972; 129:342–345.