Alcohol, nutrition and health maintenance: selected aspects

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In view of the developments in health care relating to the increased prevalence and incidence of chronic diseases and the continuing increase in health-care expenditure, more attention should be paid to health maintenance and disease prevention. Any strategy that can influence health maintenance is of interest, especially lifestyle factors such as nutrition, exercise or stress control. Alcohol has an important place in the daily life of many healthy as well as sick individuals. Alcohol has three major characteristics; it is a nutrient (energy source), a psycho-active drug and a toxin. Each consumer has the choice of which of the characteristics of alcohol he/she wants to utilise. Thus, alcohol represents one of the most important self-implemented disease modifiers in our modern society. The major determinants of the health effects of alcohol are the absolute amount consumed, the consumption frequency, associated lifestyle factors (e.g. smoking, nutrient intake, substrate composition, physical activity pattern) and last, but not least, the genetic background. There are few known disease conditions that have not already been associated positively or negatively with alcohol consumption. The list of diseases includes atherosclerosis, dementia, diabetes, obesity and conditions relating to Zn metabolism. Obesity represents the most important disease modifier in the world and the prevalence rates are increasing rapidly. Evidence suggests that alcohol represents a risk factor for overweight and obesity as a result of specific effects on energy metabolism and substrate metabolism. The potential role of alcohol as an important modulator for the postprandial lipidaemia and its role in the pathogenesis of modern diseases will be discussed.

Alcohol: Health maintenance: Disease prevention

The importance of alcohol as a disease modifier is well established. Despite the well-known negative as well as positive aspects of its role in the pathogenesis of different chronic diseases such as coronary artery disease (CAD) or cancer (Leppala *et al*. 1999; MacDonald, 1999; Ruf, 1999; Suter, 2001*a*; Mukamal *et al*. 2003), the controversy about the 'ideal' intake of alcohol for health maintenance continues.

In view of the recent developments in the health-care system, with a continuing increase in health-care costs, disease prevention is gaining more attention. Any successful strategy for disease prevention is of interest, as long as the side effects remain in an acceptable range, which for alcohol is not always the case. However, if consumers can avoid excessive intake, alcohol can be beneficial. Compared with other disease risk factors that have a strong foundation of evidence relating to their pathogenesis as well as treatment, the role of alcohol as a preventive agent is less well established and more controversial. The role of alcohol as a preventive agent for CVD is accepted by most patients and by many physicians, which is, in view of the high potential for negative effects, rather astonishing. The reason for the acceptance of a substance with considerable side effects is related to the different characteristics of alcohol (psycho-active drug) as well as the fact that it is much easier and less demanding to have a drink than to walk or jog 1 km or to reduce energy intake. Accordingly, in a recent survey of patterns of health behaviour in US adults the adherence status for different health recommendations was comparatively good for the recommendation to consume daily one drink or less for women and two drinks or less for men (Berrigan *et al*. 2003). Of the population surveyed >70% adhered to the latter recommendation. However, only 40% reported adherence to the exercise recommendation and 35% adhered to the recommendation to increase intake of fruit and vegetables. Despite the controversy about the role of alcohol in disease prevention, alcohol cannot be disregarded in modern society and there should be comprehensive guidelines for sensible drinking and educational means should be found to avoid the growth of excessive drinking (Inter-Departmental Working Group, 1995).

Abbreviation: CAD, coronary artery disease.

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There is no chronic disease that is not affected by alcohol, and indeed light to moderate alcohol intake may both positively and negatively modulate the disease burden in most societies. The healthy life expectancy in the UK is 69·6 years and the expected loss of healthy years at birth is about 9 years for men and women (i.e. about 8–11 % of the total life expectancy is lost: World Health Organization, 2001). At higher intakes alcohol can have an effect on all the top ten causes of burden of disease (World Health Organization, 2002). However, small intakes of alcohol may have favourable effects on CVD, which still represents the top cause of burden of disease.

In the present short review selected aspects of the role of alcohol in health maintenance and disease prevention are discussed. Special attention is given to its role in CAD, hypertension and as a modulator of postprandial metabolism.

Alcohol: an important health modulator

Alcohol is positively and/or negatively associated with the major determinants of health. Excessive alcohol intake is negatively associated with most socio-economic determinants of health (e.g. poverty, lower educational status, selected psychological factors, unemployment, etc). In addition, alcohol is associated with most modifiable lifestyle risk factors (physical inactivity, smoking, malnutrition, stress, etc). Thus, alcohol consumption in combination with these risk factors leads to a disproportional increase in risk (Gallus *et al*. 2003; Salaspuro, 2003). According to the WHO European Health Report (World Health Organization, 2002) 55 000 young Europeans die each year from the effects of alcohol abuse and one in four deaths in European men aged 15–29 years is related to alcohol. Furthermore, 40–60 % of all deaths from injuries are attributable to alcohol. The total cost to society caused by the uncontrolled intake of alcohol corresponds to 1–3 % of the gross domestic product (World Health Organization, 2002). The major alcohol-related diseases are liver cirrhosis, hypertension, alcoholic heart disease, stroke, pancreatitis and different cancers (oro-pharynx, larynx, oesophagus, stomach, liver and rectum). In view of the many negative effects of alcohol, the cardio-protective effects seem to be overrated; however, it should be remembered that >20 % of the burden of disease is caused by CVD. Thus, the potential positive effects of alcohol could become relevant in a society in which alcohol consumption is not excessive. In a recent systematic review even a warning for 'insufficient' alcohol intake has been formulated (White *et al*. 2002).

Alcohol and coronary artery disease

CAD represents the principal burden of disease, and any means available to reduce the morbidity and mortality of CAD would be welcomed. Alcohol has been consistently associated with a reduced risk of CAD (Agarwal, 2002). Most prospective and retrospective studies have reported an alcohol-induced reduction in cardiovascular risk (Boffetta & Garfinkel, 1990; Wannamethee & Sharper, 1998; Leppala *et al*. 1999; White *et al*. 2002). During the last 20 years the level of alcohol that has been shown to be associated with a reduced cardiovascular risk has declined from moderate amounts (20–30 g/d) to small amounts (5–15 g/d). It seems that even very small amounts of alcohol can reduce the risk of CVD (Klatsky *et al*. 1986*a*); the rate of hospital admission as a result of myocardial infarction was significantly ($P < 0{\cdot}03$) reduced in individuals who consumed alcohol less than once per week but more than once per month. So far, no single major mechanism for the cardio-protective effect of alcohol has been identified. Different putative mechanisms, such as effects on the plasma lipid profile, thrombogenesis or hormones, have been identified (Agarwal, 2002). The protective effects of alcohol are more pronounced in consumers of advanced age and in individuals with established cardiovascular risk factors (Boffetta & Garfinkel, 1990), which suggests that alcohol may favourably modulate existing cardiovascular risk factors. The evidence for this role is that alcohol may aggravate certain cardiovascular risk factors, such as blood pressure or plasma lipoprotein concentrations (see pp. 84–85), but nevertheless elicits protection. This apparent conflict represents one of the central 'paradoxes' surrounding the health effects of alcohol.

The alcohol-related reduction in CAD risk despite an unhealthy diet rich in saturated fat corresponds to the 'French Paradox' (Renaud & de Lorgeril, 1992; Renaud & Gueguen, 1998), and also provides further support for the role of alcohol as a risk factor modulator (Boffetta & Garfinkel, 1990). The effect of alcohol as a cardiovascular risk factor modulator implies that under an optimal control of the conventional risk factors, alcohol would add only a small additional amount of cardio-protection. Unfortunately, there are few data relating to this issue; nevertheless, the hypothesis is supported by the data set of the American Cancer Society Study, which showed that the cardio-protective effects of alcohol were considerably smaller in subjects without existing cardiovascular risk factors (Boffetta & Garfinkel, 1990). Furthermore, a recent systematic review has concluded that the lowest level of alcohol intake for optimal cardiovascular prevention will decline in future years as a result of better risk factor control (White *et al*. 2002).

Another paradox associated with the effects of alcohol on CVD risk concerns the effect of alcohol on plasma homocysteine concentration. Alcohol leads to an increase in plasma homocysteine levels, which is regarded as an independent cardiovascular risk factor (i.e. the 'homocysteine paradox of alcohol'; Refsum *et al*. 1998; Mangoni & Jackson, 2002). Most of the classical risk factors of atherosclerosis (age, hypertension, obesity and lifestyle factors such as physical activity, smoking, high coffee consumption and alcohol) influence plasma homocysteine levels (Nygard *et al*. 1998). The increase in plasma homocysteine may even counterbalance the protective effect of moderate alcohol consumption (Bleich *et al*. 2001; Halstred, 2001). The cardio-protective effect despite elevated homocysteine levels could be the result of modulation of the effects of homocysteine by alcohol. The negative effect of alcohol on homocysteine can be overcome by increasing the intake of fruit and vegetables,

i.e. the major source of folate, which is the most important factor in the reduction of plasma homocysteine concentration (Suter, 2001*b*). In alcohol consumers increased fruit and vegetable intake can lead to a reduction in the plasma homocysteine levels, even in individuals with a genetic polymorphism that gives rise to elevated plasma homocysteine levels (Folsom *et al*. 1998). This observation is important because it indicates that a genetic disadvantage can be overcome by improved nutrition. Fruit and vegetables represent the most important nutritional disease modifier (Ziegler, 1991; Ness & Powles, 1997; Joshipura *et al*. 1999, 2001; Suter, 2001*b*). Unfortunately, in most populations fruit and vegetable intake is rather low and there is an inverse relationship between the consumption of fruits and vegetables and alcohol intake (Johnsen *et al*. 2003). Furthermore, it is easier to follow the recommendation 'to drink moderately but not too much' than to follow the recommendation 'to increase fruit and vegetable consumption' (Berrigan *et al*. 2003).

Another 'alcohol paradox' is the effect of alcohol on insulin resistance, i.e. acute alcohol dosage leads to a decrease in insulin sensitivity. However, several cross-sectional studies have shown that chronic moderate alcohol intake is associated with improved insulin sensitivity (decreased insulin resistance; Zilkens & Puddey, 2003). Several prospective studies have shown a decreased risk of diabetes in regular light to moderate alcohol consumers (Wannamethee *et al*. 2003; Zilkens & Puddey, 2003). A recent study found a linear inverse relationship between alcohol intake and the risk of type 2 diabetes with ≤30 g alcohol, but above this level the risk increased compared with light and moderate intake (Wannamethee *et al*. 2003). The same study reported a protective effect on type 2 diabetes that was independent of the frequency of consumption, but found slightly stronger protection with increasing frequency (i.e. 4–7 d/week *v*. one to three times per week) when the same amount of alcohol was consumed (5·0–29·9 g/d). A recent study of the relationship between alcohol intake and CAD confirmed the previous observation that non-daily alcohol intake is also associated with a reduced cardiovascular risk (Mukamal *et al*. 2003). In this study alcohol intake on ≥3 d/week was associated with a reduced risk of CAD. There is growing evidence that the consumption of more than one drink every 2 d offers little incremental increase in benefit for cardiovascular risk (Maclure, 1993; Mukamal *et al*. 2003).

Very small amounts of alcohol (≥5 g/d) appear to be effective in chronic disease prevention, as is non-daily consumption. Daily consumption is highly correlated with the absolute amount of alcohol consumed, and also carries a higher risk of dependency. However, the question of whether the protective effect is related to the drink or the drinker has been the subject of a recent editorial (Klatsky, 1999). It is likely that the characteristics of the drinker are usually underestimated in the discussion of the protective effect of alcohol on chronic disease risk. The level of alcohol intake as well as the type of alcoholic beverage is associated with a variety of characteristics (socio-demographic, lifestyle such as diet, physical activity pattern, smoking) that all have a high potential to modulate alcohol effects (Mortensen *et al*. 2001; Burger *et al*. 2003). Thus, in the range of light to moderate alcohol intake the characteristics of the drinker (including genetics) might be as important as the effects of alcohol (Klatsky *et al*. 1990; Hines *et al*. 2001).

Hypertension

Excessive alcohol consumption has been identified as one of the most important factors leading to increased blood pressure and frank hypertension (Suter & Vetter, 2000). Although the pathogenesis of hypertension is multifactorial, alcohol does play an important role in the pathogenesis of essential hypertension. Most studies examining the relationship between alcohol and blood pressure have found a curvilinear (i.e. 'J'- or 'U'-shaped) relationship. In several studies a threshold effect has been described. In the classical study from the Kaiser-Permanente group, Klatsky and coworkers (Klatsky *et al*. 1977; Klatsky & Friedman, 1984) found a progressive relationship between alcohol intake and blood pressure that was independent of gender, age, body weight, socio-economic status, smoking habits or ethnic background. In several studies no significant relationship was found in non-daily drinkers, whereas in daily consumers blood pressure increased linearly across all consumption categories (Donahue *et al*. 1986; Potter & Beevers, 1991; Russell *et al*. 1991; Suter *et al*. 1995). Although there is a strong relationship between the frequency of drinking and the amount of alcohol consumed (Bulpitt *et al*. 1987), the pathophysiological potential of a fixed amount of alcohol may vary according to the frequency of consumption. The effect of the consumption frequency on blood pressure is controversial. In the Kaiser-Permanente Study the amount of alcohol ingested and the level of blood pressure were only weakly related (Klatsky *et al*. 1986*b*), and other investigators have described similar findings (Russell *et al*. 1991; Ueshima *et al*. 1992). In most studies less-frequent alcohol intake is usually associated with lower alcohol intake and lower blood pressure (Russell *et al*. 1991). Using a hierarchical regression analysis it was observed that a more frequent intake of alcohol was associated with higher blood pressure independent of the amount of alcohol consumed (Russell *et al*. 1991). Thus, an individual consuming alcohol on a daily basis would have a systolic blood pressure that is 6·6 mmHg higher than that of an individual consuming the same amount of alcohol on a weekly basis. These findings provide further support for a non-daily alcohol intake.

In the present context it should be mentioned that occasional alcohol intake could also have favourable effects on Na balance and thus blood pressure. The effects of alcohol on suppression of the secretion of vasopressin (antidiuretic hormone) have been known for many years (Kleemen *et al*. 1955; Leppaluoto *et al*. 1992). In a recent study (P Suter and A Schibli, unpublished results) Na and NaCl excretion after a parenteral 5 g NaCl load with and without alcohol (0·5 g/kg body weight) were measured during a 7 h period. The ingestion of alcohol led to increased excretion of water and NaCl during the first few hours, for as long as the alcohol remained in the circulation (over the 7 h period the difference in NaCl excretion was

>1 g; $P < 0{\cdot}05$). The experiment was carried out under an optimal hydration state. This finding suggests that when a meal rich in NaCl is ingested a high level of (non-alcoholic) fluid intake (Choukroun *et al*. 1997) and a moderate amount of alcohol could promote the elimination of the Na load. In view of the potential importance of NaCl in hypertension (He & MacGregor, 2002) even a modest improvement in the Na balance may be of clinical importance. Nevertheless, in the clinical setting of hypertension and Na sensitivity the control of NaCl intake as well as avoidance of excessive alcohol consumption should have priority.

Alcohol and postprandial metabolism

It was >20 years ago that Zilversmit (1979, 1995) postulated that atherosclerosis may represent a postprandial phenomenon. This hypothesis has gained considerable support during the last few years. It has been shown that during the postprandial phase there are many proatherogenic events and, often, cardiovascular events (Silveira, 2001; Servoss *et al*. 2002). The large changes in lipid concentration that persist during the postprandial phase may enhance the development of atherosclerosis. A typical feature of alcohol consumption is fasting, as well as postprandial, hypertriacylglycerolaemia (Taskinen & Nikkilä, 1977; Taskinen *et al*. 1987; Pownall *et al*. 1999). The lipid changes during the postprandial phase, such as the increased concentration of chylomicrons and triacylglycerols, may have a causal role in the development and progression of atherosclerosis (Austin, 1999; Ginsberg, 2002; Segrest, 2002). Alcohol has cardio-protective effects, despite its negative effect on postprandial lipidaemia, which can be considered to be another paradox. Considering these findings it is possible that alcohol may affect the characteristics of lipid particles in the postprandial phase (e.g. modulate particle size and/or composition) so that they become less atherogenic. In general, any factor that reduces postprandial lipidaemia may be considered to be cardio-protective (Parks, 2001; Petitt & Cureton, 2003).

Exercise represents one of the central strategies for disease prevention (Thompson *et al*. 2003). Thus, a study of the postprandial lipid response to an oral fat load with and without alcohol was carried out recently in trained healthy young men after an exercise session and in untrained healthy men who did not undertake the exercise session (Suter *et al*. 2001). All subjects were of normal weight (BMI (kg/m^2): trained subjects 21·6 (SE 1·5), untrained 23·2 (SE 1·8)) and received 1 g fat/kg body weight with and without alcohol (0·5 g/kg body weight). The trained subjects performed a jogging session of 5·4 km (running speed 10–13 km/h) before ingestion of the meal. In the trained subjects the ingestion of alcohol led to an increase in the triacylglycerol area under the curve from 7·4 (SE 0·04) mmol/l.h on the control day to 11·3 (SE 0·9) mmol/l.h on the experimental day ($P = 0{\cdot}001$; Suter *et al*. 2001). The corresponding areas under the curve in the untrained subjects were 13·4 (SE 2·3) mmol/l.h and 19·4 (SE 3·5) mmol/l.h ($P = 0{\cdot}004$). Alcohol intake and level of physical activity training as well as the pre-meal exercise session were the major determinants of the postprandial lipidaemia. The ingestion of a fat meal in combination with alcohol led to an increase in postprandial lipidaemia that was independent of the level of training. Nevertheless, a higher level of training was associated with a lower postprandial triacylglycerol response (lower peak concentration and shorter postprandial lipidaemia). Since alcohol has cardio-protective effects, the alcohol-induced increase in postprandial lipidaemia appears to have a lower pathogenetic potential than the non-alcohol-related increase in postprandial lipidaemia. The data suggest that the (potentially) unfavourable effect of alcohol and a high-fat diet could be modified by fat restriction or a combination of a pre-meal exercise session and a higher level of physical activity (Suter *et al*. 1993, 1994, 2001).

HDL-cholesterol concentrations, as well as those of other lipid fractions, vary during the day as a function of food intake (Miida *et al*. 2002). In agreement with other studies, during the postprandial phase a decline in the HDL-cholesterol level was observed in alcohol intake studies and there was a return to the initial pre-meal value within approximately 8 h (Suter *et al*. 2001). It is possible that the lower HDL-cholesterol levels in individuals with a high meal frequency could be a result of the longer duration of increased triacylglycerol levels (Lamarche *et al*. 1999). In view of the central role of HDL-cholesterol in atherogenesis (Tulenko & Sumner, 2002) any strategy that avoids, or at least reduces, the postprandial decline in HDL-cholesterol may be beneficial in controlling the risk of atherosclerosis in the long term. As the HDL-cholesterol-raising effect of alcohol is well known (Fraser *et al*. 1983), it is possible that the postprandial reduction in the plasma HDL concentration could be counterbalanced by the ingestion of alcohol with a meal and/or as an aperitif. The 'Aperitif Study' investigated postprandial metabolism in fourteen young men (aged 27·2 (SE 1·2) years, BMI 23·7 (SE 0·6) kg/m^2) after ingestion of a meal (1 g fat/kg body weight) with or without alcohol (0·5 g/kg body weight) with the meal and with (0·2 g alcohol/kg body weight) or without (water) an alcoholic aperitif 90 min before the test meal. The amount of alcohol given as an aperitif was 15·3 (SE 0·4) g and the amount given with the meal was 38·2 (SE 0·9) g. Each subject received each of the following combinations: alcoholic aperitif + water with the meal; alcoholic aperitif + alcohol with the meal; water before the meal + alcohol with the meal. During the 90 min aperitif phase of the study a blood sample was taken every 30 min. At 90 min after the meal a blood sample was taken every 60 min and analysed for glucose, insulin, triacylglycerols, total cholesterol, HDL-cholesterol, blood alcohol concentration and other variables. The peak triacylglycerol concentration, as well as the triacylglycerol area under the curve (for the whole 8 h period), was highest after the alcoholic aperitif + alcohol with the meal intervention ($P < 0{\cdot}05$).

The suppression of NEFA by alcohol or ingestion of food is well known (Suter *et al*. 1992). During the last few years evidence has accumulated that NEFA and/or signals induced by NEFA have a key role in the induction as well as the progression of atherosclerosis, and also in the development of chronic diseases including insulin

resistance (Zilversmit, 1995; Castro-Cabezas *et al*. 2001; Boord *et al*. 2002; Yli-Jama *et al*. 2002; Esenabhalu *et al*. 2003). Endothelial dysfunction probably represents one of the early steps in atherogenesis, and it seems that NEFA overload contributes to vascular dysfunction (Esenabhalu *et al*. 2003). Increased NEFA and lipotoxicity may represent a central phenomenon in the development of the metabolic syndrome (Listenberger *et al*. 2003; Unger, 2003). Thus, any strategy that has a favourable affect on NEFA metabolism (e.g. NEFA plasma concentration) would represent protection from these diseases. Data from the Italian National Research Council Study suggests that alcohol consumed with a meal could be more cardio-protective than alcohol consumed alone (Trevisan *et al*. 1987). This effect could be a result of the potential effects of alcohol on lipid particle concentration as well as the effects of the lower concentration of NEFA. Furthermore, the ingestion of alcohol together with a meal leads to lower peak blood alcohol concentrations. In the aperitif study the postprandial suppression of the NEFA concentration was greater after the alcoholic aperitif + alcohol with the meal and the water before the meal + alcohol with the meal interventions than after the alcoholic aperitif + water with the meal intervention. The suppression of the NEFA after the water before the meal + alcohol with the meal and the alcoholic aperitif + alcohol with the meal interventions was identical. The area under the curve for the NEFA was significantly higher with the alcoholic aperitif + water with the meal than with the other interventions ($P < 0{\cdot}05$). Elevated fatty acids impair the effect of insulin on glucose uptake in skeletal muscle and induce endothelial dysfunction, and thus enhance atherogenesis (Steinberg & Baron, 2002). Inflammation and inflammatory mediators are major participants in atherogenesis as well as in the pathogenesis of the insulin resistance syndrome, and complement factors are involved in cellular NEFA uptake (Meijssen *et al*. 2002). During the postprandial phase elevated NEFA may even have a higher pathophysiological potential.

Alcohol consumption may lead to an increase in fasting HDL-cholesterol levels but alcohol does not appear to counterbalance the postprandial fall in HDL-cholesterol. Even a preload of alcohol in the form of an aperitif did not counterbalance the HDL-cholesterol-lowering effect of the meal. Also, when investigated with the combination of alcohol and pre-meal exercise HDL-cholesterol declined independently of alcohol intake and/or exercise training (Suter *et al*. 2001). This outcome may be a result of an altered lipid exchange during the postprandial phase associated with the alcohol-induced increase in the triacylglycerols (Gotto, 2002). Regular exercise represents the other central strategy for increasing fasting HDL-cholesterol (Durstine *et al*. 2002). Recently, the effect of a preprandial walking session on lipidaemia 4 h after a meal was investigated (P Suter and S Pollakis, unpublished results). A preprandial walking session of 1 h duration (distance 6·6 km) led to a significant reduction in the postprandial decline in the HDL-cholesterol level 4 h after the ingestion of a standard meal (1 g fat/kg body weight) without alcohol. In this study no alcohol was ingested. Plasma HDL-cholesterol concentration declined by 0·07 (SE 0·01) mmol/l on the control day and by 0·01 (SE 0·01) mmol/l on the intervention day ($P = 0{\cdot}04$). In addition, the postprandial increase in the insulin concentration at 4 h was 38 % lower on the intervention day as compared with the control day. In this small group of ten randomly-selected healthy elderly subjects (aged ≥65 years) the plasma triacylglycerol concentration surprisingly was not affected by the walking intervention (this finding may be because of two prediabetic subjects who had an exaggerated postprandial lipid response). It can be argued that, considering the alcohol-induced increase in fasting HDL-cholesterol, the observed postprandial reduction in HDL-cholesterol is of only minor pathophysiological importance. Currently, the pathophysiological importance of the postprandial reduction in HDL-cholesterol is not known. However, it is possible that because of the large impact of a small change in HDL-cholesterol on the risk of CAD and the persistence of the postprandial reduction for several hours, an increased risk may result from this reduction in the long term. There is an inverse relationship between the fasting HDL-cholesterol concentration and the fasting triacylglycerol concentration and postprandial lipidaemia (triacylglycerol area under the curve and peak triacylglycerol concentration; Gotto, 2002). Alcohol suppresses NEFA, which may confer considerable toxicity and may thus have protective effects for certain chronic diseases (CAD and diabetes). In view of the potential proatherogenic effects of the postprandial lipid changes, a preprandial exercise session (especially before a fat-rich meal) can be recommended. If alcohol is consumed it should be ingested together with a meal. To minimise the risk of overweight, energy intake should be according to energy requirements (Suter *et al*. 1997).

Conclusion

Light to moderate amounts of alcohol may have cardio-protective effects through different mechanisms. There is strong evidence to suggest that alcohol favourably modulates the pathophysiological effects of existing cardiovascular risk factors. There is increasing evidence that a major part of the pathogenesis of atherosclerosis occurs during the postprandial phase, and epidemiological evidence suggests that alcohol ingested with a meal does elicit greater protection than alcohol without a meal. Despite the favourable effect of alcohol on HDL-cholesterol, alcohol cannot prevent the temporary postprandial fall in HDL-cholesterol. Nevertheless, it appears that moderate amounts of alcohol modulate postprandial metabolism (e.g. suppression of the potentially-toxic NEFA). Thus, the cardio-protective effects of moderate amounts of alcohol can be increased by the ingestion of alcohol with a meal.

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