Alcohol consumption in the new millennium – weighing up the risks and benefits for our health

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Summary

Alcohol is produced via the anaerobic fermentation of sugars by yeast and involves glucose molecules ($C_6H_{12}O_6$) being broken down to yield ethanol ($C_2H_5OH$), carbon dioxide ($CO_2$) and energy. The amount of alcohol contained in different alcoholic beverages varies considerably and is referred to as the ‘strength’ of the drink, which is expressed as the percentage of alcohol by volume (ABV). A standard unit of alcohol in the UK equates to 8 g or 10 ml of pure alcohol. Guidelines for sensible drinking set by the UK government are 3–4 units (24–32 g) a day or less for men, and 2–3 units (16–24 g) a day or less for women. Specific recommendations have been set for those planning pregnancy and pregnant women, who should drink no more than 1–2 units of alcohol once or twice a week and should avoid heavy drinking sessions. Guidelines for sensible drinking are similar in other countries. When taking into account the standard drink unit used to define guidelines, the most commonly recommended limit is 24 g/day for men and 20 g/day for women.

Older people are more likely to drink in line with the sensible drinking guidelines, as they tend to consume alcohol over a period of time, as opposed to consuming large quantities on one or two days of the week, a practice that is more popular among younger people. Consuming double the sensible drinking guidelines in one day is classified as binge drinking (an alternative definition is drinking until intoxicated). It has become recognised that binge drinking is a common phenomenon in the UK, particularly among younger people. Other factors, such as income, ethnicity and region of residence, are also recognised to be predictive of the quantity of alcohol consumed; for example, higher-income households are more likely to consume large quantities of alcohol and consume alcohol more frequently.

The quantity of alcohol consumed is an important factor in determining how it affects health and well-being. In terms of nutritional health, chronic excessive alcohol intake is well recognised to affect an individual’s nutrient status, because it reduces food intake and/or may interfere with the digestion, absorption, metabolism and utilisation of some nutrients. However, when consumed in moderation, alcohol is unlikely to interfere with the metabolism of nutrients or be associated with impaired vitamin function or depletion to an extent that may harm health,
provided that dietary intakes are adequate. Alcoholic beverages contain alcohol, carbohydrate and only small amounts of some vitamins, trace elements and minerals. They are therefore unlikely to contribute significantly to micronutrient intake. Any association that exists between moderate alcohol consumption and the absorption and metabolism of nutrients from the diet is dependant on a number of factors, including: the nutrient in question; habitual intake of the nutrient and the nutritional status of the individual; and the quantity of alcohol consumed over a period of time. Overall, little information exists about the implications of binge drinking on nutrient status; but it may alter food intake, and excessive alcohol consumption can cause disturbances to the digestive system. While infrequent binge drinking is unlikely to have any long-term nutritional implications (although it may affect nutrient status in the short-term), regular binge drinking may have a more negative impact.

Although alcoholic beverages tend to have few nutrients, they can be a significant source of energy, as alcohol provides 7 kcal per gram. Studies indicate that alcoholic beverages are usually additive to an individual’s normal energy intake and individuals are unlikely to compensate for energy from them during the day. The aperitif effect of alcohol may also encourage an increase in energy intake. It is therefore not surprising that alcohol intake has been associated with weight gain and an increase in body mass index (BMI) (with the exception of chronic excessive drinkers). However, there are a number of methodological problems that need to be considered when comparing the findings of studies investigating the association between alcohol intake and disease risk, including: potential misreporting of total intake; the use of an appropriate control group; and the need for comparable measurement units.

Consumption of alcoholic beverages has also been associated with increasing the risk of ill-health associated with pre-existing conditions. For example, it may contribute to ulcer development or aggravate symptoms of existing ulcers. There is evidence that moderate alcohol consumption during pregnancy may affect the development of the fetus’s central nervous system, and may result in low birthweight. However, there is currently no consensus as to whether alcohol should be completely avoided during pregnancy. However, it is well recognised that excess alcohol consumption during pregnancy can put the fetus at risk of fetal alcohol syndrome.

Quantity of alcohol consumed is particularly important when considering the association between alcohol and chronic diseases; for example, there is now strong evidence that light to moderate (1–3 drinks per day) consumption decreases the risk of a coronary heart disease (CHD) episode compared with abstainers (with particular benefits in men aged over 55 years and post-menopausal women). However, heavy drinking is associated with an increased risk of CHD. Further information is required to determine the association between drinking patterns and heart health. However, there is a consensus of opinion that it is the alcohol per se that influences heart health, rather than a particular type of alcoholic beverage; and other factors that affect an individual’s choice of drink may be important in distorting the findings from studies that show otherwise. As with risk of CHD, light to moderate alcohol consumption is associated with a decreased risk (of 30–40%) of type 2 diabetes, when compared with teetotallers. Heavy drinking may be associated with an increased risk, resulting
in a ‘J’ or ‘U’ shaped relationship between alcohol consumption and the incidence of type 2 diabetes. A ‘J’ shaped relationship has also been reported between alcohol consumption and blood pressure. It is widely accepted that heavy alcohol intake is a risk factor for high blood pressure, and evidence indicates that moderate intakes may exert a short-term lowering effect, while abstainers/teetotallers are reported to have a higher blood pressure. The type of stroke is important when considering the association between stroke and alcohol, as any alcohol consumption is associated with an increased risk of haemorrhagic stroke, but low intakes (i.e. one drink per day) may have a protective effect on ischaemic stroke. Heavy drinking is associated with an increased risk of both types of stroke.

Alcohol intake has also been associated with an increased risk of cancers at a number of sites. However, a consensus for a likely causal association has only been identified between alcohol and cancers of the upper-aero digestive tract and liver. The mechanisms to explain the positive associations between alcohol and breast cancer, and alcohol and colorectal cancer, have yet to be identified. It is unclear whether some types of alcoholic beverage are more strongly associated with increased risk of cancer than others. Mechanisms identified to date suggest that it is the alcohol per se that exerts damage and, therefore, quantity of alcohol consumed is the most important variable.

Studies looking at the relationship between alcohol and mortality suggest that 10–80 g (approximately 1–8 drinks) of alcohol each week is optimal to reap the health benefits, but increased risk is noted at higher intakes (thus indicating a ‘U’ shaped relationship between alcohol intake and mortality). Gender and age are important factors when considering the association between alcohol and mortality. For example, for younger people, the benefits of alcohol may be outweighed by the increased risk of other diseases (e.g. alcohol-related cancers, liver cirrhosis) and increased risk of violence and accidents. Aggressive behaviour, increased risk taking and decreased responsiveness to social expectations, which may lead to personal ‘harm’, including accidents and violence, are usually the result of alcohol intoxication (i.e. through binge drinking).

Excess alcohol consumption may also result in a ‘hangover’, which disrupts normal life through symptoms of fatigue and increased anxiety, and has economic consequences in the workplace through poor performance or absenteeism. The extent to which a hangover can affect an individual’s performance, including psychomotor, recognition and managerial skills, is still being debated. Binge drinking is also associated with an increased risk of sexually transmitted diseases and unplanned pregnancies. Although the cost of binge drinking in terms of long-term health has yet to be established, the significant economic cost is well recognised. A number of population-based initiatives are underway to try and change the ‘drinking culture’, and thus reduce the incidence of binge drinking. Initiatives include: advertising campaigns; changes to alcohol advertising regulations; and changes to pub licensing regulations.

**Keywords:** alcohol, binge drinking, guidelines for sensible drinking, moderate alcohol intake
1. Introduction

This paper provides an overview of guidelines for sensible drinking and current drinking habits both in the UK and around the world. Focusing on moderate alcohol consumption, the current evidence regarding the nutritional implications of alcohol in the diet, and its effects on disease risk are also considered. Finally, the social implications of the topical binge drinking phenomenon are discussed, thereby providing a timely and useful overview of the potential risks and benefits of alcohol consumption to our health.

More than 90% of adults in Britain drink alcohol (Strategy Unit 2004). For the majority of people who drink alcohol, it is associated with pleasure, socialising and relaxing (MORI 2000). When consumed in moderate amounts, it is an accepted element of a healthy, balanced diet and lifestyle, with few adverse effects to nutritional health or risk of disease, and possibly some benefits. However, when alcohol is consumed in excess, for example because of chronic abuse or binge drinking, it can adversely affect the health and well-being of the individual, and cause detriment to society. In particular, binge drinking is associated with an increased risk of injury or assault of the drinker and individuals in the vicinity. Chronic excessive consumption is associated with an increased risk of a number of adverse health conditions, including myopathy, liver cirrhosis, liver cancer, pancreatitis, kidney problems and problems with the central, peripheral and autonomic nervous system, such as cerebral dementia. It is also likely that chronic excessive drinkers will suffer from some nutritional deficiencies (Peters & Preedy 1999; Rajendram & Preedy 2005). Fortunately, it is only the minority of the population who are classified as chronic excessive drinkers, and for this reason, this briefing paper will focus primarily on the risks and benefits of light or moderate alcohol intake. Preedy and Watson (2004) provide a comprehensive overview of alcohol-related harms, including those related to chronic excessive consumption of alcohol.

2. An introduction to alcohol and alcoholic beverages

Alcoholic beverages contain ethanol, which is chemically an aromatic compound containing a hydroxyl group. Ethanol in beverages is commonly referred to as alcohol (International Food Information Service 2005). By law, ethanol for human consumption is only allowed to be derived from the fermentation of agricultural crops (e.g. cereals, grapes). Pure ethanol is also known as absolute alcohol, the production of which is subject to strict regulations, as is its use (e.g. in research establishments and the chemical industry). Because drinks containing ethanol/alcohol are subject to taxation in many countries, when sold for other purposes it is rendered unfit for consumption. For example, ethanol produced for domestic use contains a purple dye and pyridine (which has an unpleasant smell) and is known as methylated spirit (‘methyls’) and ethanol for industrial use contains 5% methanol (CH₃OH) (which is poisonous), yielding industrial rectified spirit. As the term ‘alcohol’ has been widely adopted as a colloquial equivalent of ethanol, in this briefing paper, alcohol is used synonymously with ethanol.

2.1 How is alcohol produced?

Alcohol is produced via the anaerobic fermentation of sugars by yeast. In essence, the overall process involves glucose molecules (C₆H₁₂O₆) being broken down in the absence of oxygen to yield ethanol (C₂H₅OH), carbon dioxide (CO₂) and energy. In wine or beer making, the carbon dioxide bubbles off and the amount of alcohol produced is controlled by the amount of yeast and the duration of fermentation (i.e. a larger quantity of yeast and longer fermentation produces more alcohol). The net overall reaction can be summarised as:

\[ C₆H₁₂O₆ \rightarrow 2C₂H₅OH + 2CO₂ + \text{energy} \]

2.2 The production of alcoholic beverages

Sugar, preferably glucose, is necessary to make alcohol. The sugars in fruit can be fermented by yeast to produce wine and cider. Alcoholic beverages can also be made from grains, such as barley and rye (traditionally in Europe), corn (traditionally in the Americas) and rice (traditionally in the Far East). As these grains do not contain fermentable sugars, the starch in the grain has to be hydrolysed (broken down, for example to maltose and glucose), before fermentation can occur. Alcoholic beverages produced in this way include ales, beers, lagers and rice wines. Grains also form the basis of spirits such as whisky and vodka, whilst liqueurs are made from distilled spirits (produced through the evaporation and condensation of alcohol, which produces liquid with a higher alcohol content), which are sweetened and flavoured. Fortified wines, such as port and sherry, are made by adding spirit to wine. About 10–15 years ago, pre-mixed drinks (referred to as ‘alco-pops’) were introduced to the market, further adding to the selection of alcoholic beverages available.
Methods of production of alcoholic beverages have developed through various traditions and practices and, more recently, large-scale production processes. Although alcoholic fermentation is a rapid process, the production of alcoholic beverages can take many years because of the different processes involved and the desired characteristics of the final products. For example, many wines are best left to ‘age’, rather than drunk ‘young’, and some types of whiskey may be left to mature for decades. The distinctive flavours of different types of alcoholic beverage also arise from the addition of other specific ingredients, such as hops in beer and juniper berries in gin.

2.3 Calculation of alcohol content in beverages

The ‘amount’ of alcohol contained in alcoholic beverages varies considerably and is referred to as the ‘strength’ of the drink, which, by law in the European Union (EU), should be expressed as the percentage of alcohol by volume (ABV). This information is mandatory for beverages containing more than 1.2% ABV. It should be noted that percentage ABV is not the same as the percentage of alcohol by weight (percentage w/v), because alcohol is less dense than water (relative densities 0.79 and 1.00, respectively). For example, 5% ABV is equivalent to 4 g of alcohol per 100 ml (4% by weight). The alcohol content of a range of strengths ‘by volume’ is shown in Table 1, and the typical percentage ABV of different types of alcoholic beverages is shown in Table 2.

2.4 Other measures of alcohol

Drinks containing alcohol are also ascribed a value of ‘units’, which relate to the amount of alcohol they contain. ‘Units’ were a concept developed for clinical practice in 1987 and have since been used as the basis of sensible drinking guidelines (discussed in section 3). One unit equates to 8 g (or 10 ml) of pure alcohol.

The list below shows the number of units of alcohol in common drinks:

- a pint of ordinary strength lager – 2 units;
- a pint of strong lager – 3 units;
- a pint of bitter – 2 units;
- a pint of ordinary strength cider – 2 units;
- a 175-ml glass of red or white wine – 2 units;
- a pub measure of spirits – 1 unit;
- an alco-pop – around 1.5 units.

Key points

- Ethanol in beverages is commonly referred to as alcohol.
- Alcohol is produced via the anaerobic fermentation of sugars by yeast, and involves glucose molecules \((C_6H_{12}O_6)\) being broken down to yield ethanol \((C_2H_5OH)\), carbon dioxide \((CO_2)\) and energy.
- The amount of alcohol contained in different alcoholic beverages varies considerably and is referred to as the strength of the drink, which is expressed as the percentage of alcohol by volume (ABV).
- A unit of alcohol equates to 8 g or 10 ml of pure alcohol.

3. Guidelines for sensible drinking

3.1 UK guidelines

The Department of Health currently advises that there are no significant health risks associated with consuming up to 3 to 4 units (24–32 g) of alcohol a day for men, and up to 2 to 3 units (16–24 g) a day for women. However, consistently drinking more than these levels per day is linked with a progressive increase in risks to

Table 2: Typical percentage alcohol by volume (ABV) of alcoholic beverages (source: Chan et al. 1994)

<table>
<thead>
<tr>
<th>Alcoholic beverage</th>
<th>% ABV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low alcohol beer</td>
<td>0–2</td>
</tr>
<tr>
<td>Alco-pops</td>
<td>4–5.5</td>
</tr>
<tr>
<td>Beer, cider</td>
<td>4–6</td>
</tr>
<tr>
<td>Wines</td>
<td>9–13*</td>
</tr>
<tr>
<td>Spirits</td>
<td>37–45</td>
</tr>
<tr>
<td>Liqueurs</td>
<td>20–40</td>
</tr>
<tr>
<td>Fortified wines</td>
<td>18–25</td>
</tr>
</tbody>
</table>

*Since these data were published, wines with a higher percentage ABV have become widely available; it is not unusual to find wines with 14.5% ABV (average strength is about 12.5% ABV).
health, many of which are discussed in detail in section 8. It should be noted that these figures are guidelines, not recommendations or targets. In 1992, the recommended maximum levels of alcohol consumption were set at no more than 21 units (168 g) per week for men and 14 units (112 g) for women (Department of Health 1992). However, following growing evidence that the health implications of occasional sessions of heavy drinking differed from the health implications of consuming the same amount of alcohol daily over the course of a week, daily upper limits on alcohol intake were formulated, in a bid to discourage heavy drinking sessions (Department of Health 1995). A history of sensible drinking messages can be found in the 1995 Department of Health report, entitled: Sensible Drinking. The report of an inter-departmental working group.

The Prime Minister’s Strategy Unit (2004) has recognised that since the revised guidelines came into place in 1995, variations in the size and strength of alcoholic drinks have been introduced, making the units unstandardised. For example, the average strength of wine is now approximately 12.5%, whereas unit measures are based on 9% strength. Typical pub measures have also changed for some drinks; for example, a glass of wine is now frequently served as a 175-ml measure. Therefore, whereas a small 125-ml glass of 9% strength wine would equate to one unit, wine served in today’s measures provides around two units. This report also investigated drinkers’ awareness of the alcohol guidelines. Eighty per cent of drinkers surveyed had heard of the term ‘alcohol units’, but only 25% understood the meaning of ‘a unit’. Because of this poor understanding, and the inconsistency between the strength and size of alcoholic beverages, the government plans to reconsider the presentation of sensible drinking guidelines to develop a much clearer format, focusing on the types and amounts of alcoholic drinks currently available (Prime Minister’s Strategy Unit 2004).

Specific guidelines for population groups

For some population groups, such as pregnant women and people with diabetes, more stringent guidelines about sensible drinking exist, some of which are outlined below.

Guidelines for intake of alcohol during pre-pregnancy

Excessive intakes of alcohol have been shown to affect reproduction in women, influencing the ability to conceive and the viability of conception (Goldberg 2002). The Department of Health recommends that women who are pregnant, or trying to become pregnant, should drink no more than 1–2 units of alcohol once or twice a week and should avoid heavy drinking sessions (Department of Health 1995). It should also be noted that chronic excessive consumption of alcohol has a detrimental effect on the absorption and utilisation of folate (see section 6 for more information). Adequate folate intakes are important before and during the first 12 weeks of pregnancy to protect the fetus against neural tube defects. For more information on nutrition before and during pregnancy, see Williamson (2006).

Alcohol intake and breastfeeding

Alcohol can pass to the baby in small amounts through breastmilk. It is thought that less than 2% of maternal alcohol intake is passed into the breastmilk, but it can change the taste of the milk, which may influence the baby’s acceptance and therefore short-term uptake of breastmilk (Mennella 2004; Department of Health 2005). Current advice from the Department of Health (2005) is that breastfeeding women should keep below the daily limit of 2 to 3 units, and avoid drinking alcohol just before a feed.

People with diabetes

Precautions regarding alcohol intake that apply to the general public are particularly important for people with type 1 diabetes, in order to help manage blood glucose levels. Alcohol markedly suppresses gluconeogenesis and has a hypoglycaemic effect (Lieber 1994). For those on insulin or sulphonylurea drugs, alcohol can cause severe hypoglycaemia that can result in brain damage or death. In order to manage blood glucose levels, people with type 1 diabetes are recommended to:

- avoid over-consumption of alcohol (i.e. not exceed safe drinking limits);
- choose ordinary types of lager rather than the higher alcohol types;
- never drink on an empty stomach, and top up with a snack if necessary;
- have a snack (such as cereal or toast) before going to bed and check blood glucose concentrations to reduce the risk of a hypoglycaemic event later on (Thomas 2001).

3.2 Guidelines for sensible drinking around the world

Guidelines for sensible drinking limits vary between countries, and these have been outlined in table 3. Some countries use more than one set of guidelines; for example, European wine-producing countries, such as France
<table>
<thead>
<tr>
<th>Country</th>
<th>Source</th>
<th>Guidelines (grams of alcohol)</th>
<th>Alcohol content of standard drink</th>
<th>Additional comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>National Health &amp; Medical Research Council</td>
<td>Not to exceed 4 units/day (40 g/day) or 28 units/week (280 g/week)</td>
<td>10 g</td>
<td>Guidelines recommend not drinking more than 1–2 drinks/h and having 1–2 alcohol-free days/week. Lists special populations that should not drink, including pregnant women and youths.</td>
</tr>
<tr>
<td>Austria</td>
<td>Federal Ministry for Labour, Health &amp; Social Affairs</td>
<td>24 g/day</td>
<td></td>
<td>Hazardous limit (unsatisfactory risk for health consequences) defined as 60 and 40 g of alcohol per day (men and women, respectively). Based on British 'Sensible Drinking Guidelines'.</td>
</tr>
<tr>
<td>Canada</td>
<td>Centre for Addiction &amp; Mental Health and Addictions Research Foundation</td>
<td>Not to exceed 2 units/day (27.2 g/day) or 14 units/week (190 g/week)</td>
<td>13.6 g</td>
<td>Moderate drinking means no more than 1 drink a day and no more than 7 drinks a week. More than 4 drinks on one occasion, or more than 14 drinks a week is a risk to health and safety. If you are pregnant or breastfeeding, avoid alcohol.</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>National Institute of Public Health</td>
<td>24 g/day</td>
<td></td>
<td>Recommend that children under the age of 15 should not drink.</td>
</tr>
<tr>
<td>Denmark</td>
<td>National Board of Health</td>
<td>Not to exceed 21 units/week (252 g/week)</td>
<td>12 g</td>
<td>Recommend that people should not drink and drive.</td>
</tr>
<tr>
<td>Finland</td>
<td>Oy Alko AB</td>
<td>Not to exceed 15 units/week (165 g/week)</td>
<td>11 g</td>
<td></td>
</tr>
<tr>
<td>France</td>
<td>Ministry of Health, Family &amp; Persons with Disability</td>
<td>Not to exceed 20 g/day (60 g/day)</td>
<td>12 g/beer, 8 g/wine</td>
<td>Recommend that pregnant women should not drink and that people should not drink and drive.</td>
</tr>
<tr>
<td>Ireland</td>
<td>Department of Health</td>
<td>21 units/week (2.10 g/week)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Italy</td>
<td>Ministry for Agriculture &amp; Forestry and National Institute for Food &amp; Nutrition</td>
<td>Not to exceed 2–3 units/day (24–36 g/day)</td>
<td>12 g</td>
<td>Recommend that pregnant women do not drink, students should not drink more than one unit at a time and avoidance of alcohol if taking medication.</td>
</tr>
<tr>
<td>Israel</td>
<td>Ministry of Education, Psychological &amp; Counseling Services</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Country</td>
<td>Source</td>
<td>Guidelines (grams of alcohol)</td>
<td>Alcohol content of standard drink</td>
<td>Additional comments</td>
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<td>------------------------</td>
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</tr>
<tr>
<td>Japan</td>
<td>Ministry of Health, Labour &amp; Welfare</td>
<td>1–2 units/day (19.75–39.5 g/day)</td>
<td>19.75 g</td>
<td>Advised not to drink on at least 2 days within a week and avoidance of alcohol when pregnant, driving or operating machinery and if an adolescent. Women with a low bodyweight are advised to drink less than the recommended daily limit.</td>
</tr>
<tr>
<td>The Netherlands</td>
<td>Stichting Verantwoorde Alcoholvergunning</td>
<td>Not to exceed 3 units/day (29.7 g/day)</td>
<td>9.9 g</td>
<td>Should not exceed 6 units/day (60 g/day) for men, 4 units/day (40 g/day) for women on one single occasion.</td>
</tr>
<tr>
<td>New Zealand</td>
<td>Alcohol Liquor Advisory Council</td>
<td>Not to exceed 3 units/day (30 g/day) or 21 units/week (210 g/week)</td>
<td>10 g</td>
<td>Should not exceed 2 units/day (20 g/day) or 14 units/week (140 g/week).</td>
</tr>
<tr>
<td>Norway</td>
<td>Directorate for Health &amp; Social Welfare</td>
<td></td>
<td></td>
<td>Recommend situational abstinence, such as when driving, during pregnancy, at work or in the company of children and young people.</td>
</tr>
<tr>
<td></td>
<td>Alcolutt</td>
<td></td>
<td></td>
<td>Alcolutt suggestions include: never drink on an empty stomach; show respect to people who do not drink alcohol; women cannot tolerate as much alcohol as men, adolescents should not drink.</td>
</tr>
<tr>
<td>Poland</td>
<td>State Agency for Prevention of Alcohol Related Problems</td>
<td>2 units/day (20 g/day) up to 5 times/week (not to exceed 100 g/week)</td>
<td></td>
<td>Suggest two alcohol-free days/week (based on WHO recommendations).</td>
</tr>
<tr>
<td>Portugal</td>
<td>National Council on Food and Nutrition</td>
<td>2–3 units/day (28–42 g/day)</td>
<td>14 g (unofficial)</td>
<td>Based only on wine consumption.</td>
</tr>
<tr>
<td>Romania</td>
<td>Ministry of Health</td>
<td>Not to exceed 32.5 g beer/day or 20.7 g wine/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spain</td>
<td>Ministry of Health and Spanish Institute for the Investigation of Beverage Alcohol</td>
<td>Not to exceed 3 units/day (30 g/day)</td>
<td>10 g</td>
<td>Wine officially considered as an integral part of a Mediterranean diet.</td>
</tr>
<tr>
<td></td>
<td>Basque Country: Department of Health &amp; Social Security</td>
<td>Not to exceed 70 g/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Catalonia: Central Authority</td>
<td>Not to exceed 4–5 units/day (32–50 g/day)</td>
<td>8–10 g</td>
<td></td>
</tr>
<tr>
<td>Sweden</td>
<td>Swedish Research Council</td>
<td>Not to exceed 20 g/day</td>
<td>Not to exceed 20 g/day</td>
<td>Recognise that a moderate alcohol intake may have certain positive medical effects.</td>
</tr>
<tr>
<td>Country</td>
<td>Source</td>
<td>Guidelines (grams of alcohol)</td>
<td>Additional Comments</td>
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</tr>
<tr>
<td>Switzerland</td>
<td>Swiss Federal Commission for Alcohol Problems and Swiss Institute for</td>
<td>Not to exceed 2 units/day (24 g/day)</td>
<td>List exceptional drinking guidelines: not to exceed 4 units/event, not to exceed 1 unit/h. No alcohol for youngsters; no alcohol during sports; no alcohol while operating machinery or before driving. Women have to be particularly cautious.</td>
<td></td>
</tr>
<tr>
<td>United</td>
<td>Department of Health</td>
<td>3–4 units/day (24–32 g/day),</td>
<td>Women who are pregnant or who are trying to get pregnant should drink no more than 1–2 units of alcohol per week. Recognises that moderate drinking for men over 40 and post-menopausal women confers health benefits, including lower risk of coronary heart disease, ischaemic stroke and gallstones.</td>
<td></td>
</tr>
<tr>
<td>Kingdom</td>
<td>not to exceed 21 units/week (168 g/week)</td>
<td>2–3 units/day (16–24 g/day),</td>
<td>Uses ‘Sensible Drinking Guidelines* as part of national alcohol strategy.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>not to exceed 14 units/week</td>
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<td></td>
<td></td>
<td>(112 g/week)</td>
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<tr>
<td></td>
<td>Scottish Executive</td>
<td>3–4 units/day (not to exceed 32 g/day)</td>
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<tr>
<td></td>
<td>United States</td>
<td>1–2 units/day (14–28 g/day),</td>
<td>Recognise that moderate drinking may lower the risk of coronary heart disease among men over 45 and women over 55. Exceeding moderate consumption can raise the risk for accidents, high blood pressure, stroke, violence, suicide, birth defects and certain cancers. A safe level of alcohol intake has not been established for women at any time during pregnancy. Advise to avoid drinking before or when driving, and to consume alcohol with food to slow absorption.</td>
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<tr>
<td></td>
<td>not to exceed 14 units/week (196 g/week)</td>
<td>1 unit/day (14 g/day), not to exceed 7 units/week (98 g/week)</td>
<td></td>
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<tr>
<td></td>
<td>National Institute of Alcohol and Alcoholism</td>
<td>Not to exceed 4 units/day (56 g/day) or 14 units/week (196 g/week)</td>
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<tr>
<td></td>
<td>American Heart Association</td>
<td>Not to exceed 2 units/day (28 g/day)</td>
<td></td>
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</tr>
</tbody>
</table>

Source: based on information from the ICAP (2004).

*See section 2 for information on sensible drinking guidelines.
and Spain, subcategorise the guidelines into different drink types, i.e. beer and wine. Overall, most countries have relatively similar recommendations when taking into account the standard drink unit used, the most common recommendation being a limit of 24 g/day of alcohol for men and 20 g/day for women. The highest recommendation originates from the Basque region of Spain, where it is recommended not to exceed more than 70 g/day for men and women. The alcohol content of the Japanese 'standard drink' (also referred to as a unit) is the highest recorded, being over double the amount used for a unit in the UK (19.75 g and 8 g, respectively). However, when comparing average consumption of alcohol (litres of alcohol per adult per annum), it is evident that Japan has a lower alcohol consumption than the UK (the equivalent of 6 L of absolute alcohol consumption compared with an average of 9 L in the UK) (World Health Organization 1999). One reason why the Japanese have a lower alcohol intake could be the higher prevalence of alcohol flushing responses, known to be particularly prevalent in those of East Asian descent, due to a genetic polymorphism. Symptoms, which include facial flushing, palpitations and drowsiness, are believed to discourage alcohol intake among those who suffer from the condition (Vines 1999; Yokoyama et al. 2003).

Key points

- The standard drink unit in the UK equates to 8 g of alcohol. Guidelines for sensible drinking set by the UK government are 3–4 units (24–32 g) a day or less for men, and 2–3 units (16–24 g) a day or less for women.
- Specific recommendations have been set for those planning pregnancy and pregnant women, who should drink no more than 1–2 units of alcohol once or twice a week and should avoid heavy drinking sessions.
- Guidelines for sensible drinking are fairly similar between other countries. When taking into account the standard drink unit used to define guidelines, the most commonly recommended limit is 24 g/day for men and 20 g/day for women.

4. Alcohol consumption

4.1 Current intakes in the UK

In the UK, average consumption of alcohol is assessed (via self-reporting) from national surveys, including the National Diet and Nutrition Survey (NDNS), the Health Survey for England (HSE) and the General Household Survey (GHS). The most recent NDNS revealed that, over 7 days, 60% of men and 44% of women consumed more than the recommended amount of alcohol on at least one day of the week: i.e. men consumed more than 4 units (32 g) and women more than 3 units (24 g), per day] (Hoare et al. 2004). On average, men obtained a significantly higher proportion of total energy from alcohol than women (6.5% and 3.9%, respectively); however, the median values were much lower, suggesting that a small proportion of respondents were deriving a much larger percentage of energy from alcohol (Henderson et al. 2002). When interpreting the findings of surveys used to estimate alcohol intake, it is important to recognise that under-reporting is a particular problem. Other difficulties associated with measuring alcohol intake are discussed in more detail in section 8 of the briefing paper.

Official UK dietary surveys often subcategorise respondents' intake of alcohol into the following categories:

- low: drinking less than 7 units (approximately 56 g) for women and 10 units (80 g) for men in 1 week;
- moderate: drinking 8–14 units (64–112 g) for women and 11–21 units (88–168 g) for men in 1 week;
- fairly high: drinking 15–25 units (120–200 g) for women and 22–35 units (176–280 g) for men in 1 week;
- high: drinking 26–35 units (208–280 g) for women and 36–50 units (288–400 g) for men in 1 week;
- very high: drinking more than 36 units (288 g) for women and more than 51 units (408 g) for men in 1 week;
- binge drinking: exceeding twice the advised daily limits on one single occasion.

It is these definitions that are used throughout this section.

Data from the GHS (ONS 2001) estimate that:

- half the adult UK population (26.3 million) are low to moderate drinkers;
- 6.4 million adults are fairly high to high drinkers;
- 1.8 million adults are very high drinkers.

Overlapping these categories are binge drinkers, who drink more than twice the daily guidelines on a single occasion (Strategy Unit 2003). According to the most recent GHS, 22% of men and 9% of women drink more than double the recommended alcohol intake on at least one day of the week (ONS 2006a). These figures are substantially lower than those from other surveys, thus emphasising the difficulty in collecting accurate information in this area. For example, reports of binge drinking from the NDNS identified that 39% of men and 22% of women exceed the daily guidelines by more than double on at least one day of the week (Hoare et al. 2004). The 2004 HSE found that a larger percentage of younger
men had a binge drinking occasion in the week before the survey; for example, 39% of men aged 16–24 years, compared with 13% of those aged 35–44 years, could be classified as binge drinkers (Department of Health 2006a). The same trend was evident among women, with 27% of 16–24-year-olds compared with 15% of those aged 35–44 years having had a binge drinking occasion during the week before the survey (Department of Health 2006a). The survey found that respondents in the older age groups tended to binge drink less but, in contrast, drank on a more frequent basis; for example, 52% of men and 32% of women aged 55–64 years consumed alcoholic beverages on three or more days a week compared with 29% of men and 14% of women in the age group of 16–24 years (Department of Health 2003).

Regional variations

Few regional variations in drinking habits have been identified from the dietary surveys, although the NDNS found that people living in northern regions were more likely than those in the south-east and London to consume alcohol (83% and 71%, respectively) (Henderson et al. 2002). Similar trends were found in the GHS; Scotland is the most likely area in which heavy drinking occurs on at least one day of the week, compared with England or Wales (Walker et al. 2002). Evans et al. (2000) propose that the effects of heavy drinking at weekends have been reflected in trends in coronary heart disease (CHD)-related mortality, with deaths being more common on Mondays in Scotland. The associations between alcohol intake and risk of disease are considered in more detail in section 8.

Economic variations

Dietary surveys have generally found that higher-income households are likely to consume more alcohol and consume alcohol more frequently. For example:

- the GHS estimated that respondents with a gross weekly income of more than £1000 were almost twice as likely to exceed the daily limits on at least one day per week than those with a gross weekly income of £200 or less (Walker et al. 2002);
- the latest NDNS found that men and women in receipt of benefits were significantly less likely than those in non-benefit households to have recorded consuming alcohol during the survey week (59% of men and 55% of women compared with 84% of men and 71% of women, respectively) (Henderson et al. 2002).

Ethnicity

The HSE (Department of Health 2006c) found that all minority ethnic groups (black Caribbean, Indian, Pakistani, Bangladeshi and Chinese) consumed less alcohol than the general population, with the exception of the Irish, who drank more frequently and were more likely to drink more than the guidelines. Among ethnic minority groups, Pakistani and Bangladeshi groups most commonly reported not consuming alcohol in the last 12 months, or being a non-drinker, and this was more commonly reported in women than men.

4.2 Recent trends in alcohol intake in the UK

In the past 10 years, surveys have continued to show an increase in alcohol consumption, particularly among certain subgroups of the population. For example, in young women (aged 16–24 years), the prevalence of fairly high alcohol intakes has increased significantly, from 9% of women drinking more than 21 units per week in 1993 to 21% in 2002 (as figure 1 indicates), with fewer women drinking lower quantities (Department of Health 2003).

Among all women, the average number of units consumed per week has increased by just over 1 unit since 1993, and in 2002, the majority of women were con-
suning 1–7 units per week. Among men, the intake of alcohol has also remained steady in the past 10 years, with the average alcohol intake increasing by less than 1 unit. There has been a slight increase in the percentage of men consuming more than the weekly guideline maximum alcohol intake, in all age groups (as figure 2 indicates). However, it should be noted that the largest increase has been among the youngest age group (16–24 years). It is not possible to confirm that the trends among young women and men are due to binge drinking alone, owing to the way the data are collected over a period of time and averaged out, rather than being day-specific. However, high alcohol consumption over the week, or on one day, is associated with adverse health consequences as outlined in section 8, and therefore this is a worrying trend.

The proportion of people aged 16 years or below consuming alcohol is also a concern; the Office for National Statistic (ONS 2006a) reported that 23% of boys and girls aged 11–15 years drank alcohol during any week. The survey also reports that the quantity of alcohol being consumed is increasing; for example, in 1990 young drinkers (aged 11–15) were consuming on average of 5.3 units per week, but in 2004 this had increased to 10.4 units per week (ONS 2006a). In each year, among those who drank, boys consumed more than girls, although recently the greatest increase has been among girls aged 14 years. The types of alcohol consumed by underage drinkers varies depending on a number of factors, including experience of drinking. Research by McKeganey et al. (1997) and by Alcohol Concern (2001) suggests that alco-pops may ‘introduce’ alcohol to under 18 year olds who then go on to consume stronger alcoholic beverages (McKeganey 1998).

Despite these worrying trends, studies suggest that drinking at a young age does not necessarily reflect drinking patterns in adult life (Strategy Unit 2003). However, the effects of alcohol consumption are wide ranging, impacting on society, as well as on individuals’ health as outlined in section 8, and the potential implications of drinking at such a young age should not be overlooked.

4.3 Trends around the world

Overall, Europe has the highest alcohol consumption worldwide, with rates increasing year on year, which is mainly associated with the increased alcohol consumption among the younger European population (Health Promotion Agency 2001). When comparing per capita alcohol intake in the UK with other European countries, it is evident that the UK has an annual per capita intake close to the European average (fig. 3). However, if consumption continues to rise among the younger population at its current rate, the UK population could be the highest consumers on a per capita basis within the next 10 years (Strategy Unit 2003).

In the past, wine-producing countries have tended to have the highest per capita alcohol intake, although this is now stabilising compared with non-wine-producing countries, presumably due to increased accessibility and lower costs. People from countries in the Mediterranean region tend to drink more wine-based beverages, in comparison with those from countries in the north of Europe, that tend to drink more beer-based alcoholic drinks (Health Promotion Agency 2001; Sieri et al. 2002; Strategy Unit 2003). It has been suggested that this may be one of the reasons that CHD is less common in these areas, a phenomenon referred to as the ‘French paradox’, which is discussed in more detail in section 8. As figure 3 indicates, Turkey and Israel have considerably lower intakes than any other European country, which could be due to religious and cultural practices that prohibit alcohol (Neumark et al. 2001). The highest per capita consumption is recorded for Luxembourg, but it is accepted that a significant proportion of alcohol bought in Luxembourg is for consumption by visitors from outside the country (Rehm et al. 2001). The meth-
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Methodology used to collect the data and variations between countries in the way the data are presented are also likely to account for at least some of the apparent differences in alcohol consumption.

**Key points**

- On average, men obtain a significantly higher proportion of total energy from alcohol than women.
- Half of the adult UK population are *low to moderate* drinkers. Despite *high* and *very high* drinkers being a minority, evidence from the GHS suggests that this figure is growing.
- In the UK, some variations in drinking habits between subgroups of the population have been identified by the NDNS, for example:
  - people living in northern regions of England are more likely than those in the south-east and London to consume alcohol;
  - higher-income households are more likely to consume large quantities of alcohol and consume alcohol more frequently;
  - compared to other ethnicities, the Irish drink the most alcohol; all other minority ethnic groups consume less alcohol than the average for the general population.
- It has become recognised that binge drinking is a common phenomenon in the UK, particularly among younger people. The NDNS found that 39% of men and 22% of women exceed the daily guidelines by more than double on at least one day of the week.
- Data from the HSE indicate that the percentage of men and women who binge drink decreases with increasing age, but in contrast, older people drink on a more frequent basis.
- The proportion of people aged 16 years or below consuming alcohol is also rising.
- The UK has an average per capita alcohol intake close to that of the European average.
- Overall, Europe has the highest alcohol consumption worldwide, with rates increasing year on year.

5. Absorption and metabolism of alcohol

5.1 Absorption of alcohol

Alcohol is a water-soluble molecule. It is absorbed throughout the entire gastrointestinal tract, and this is
mainly controlled by gastric emptying. Once absorbed, it circulates in the blood and is distributed through the water in the body by the process of diffusion. Alcohol will diffuse more rapidly into organs with a rich blood supply, such as the lungs, but little alcohol enters body fat, owing to its poor solubility in fat. Exposure to alcohol is greatest in the liver, as blood is received directly from the stomach and small bowel via the hepatic portal vein (Paton 2005).

Factors affecting absorption of alcohol and blood alcohol concentrations

Absorption of alcohol is quickest when drinks containing alcohol are drunk on an empty stomach. The type of alcoholic beverage will also affect the rate of absorption; drinks with a concentration of alcohol of 20–30% ABV are most rapidly absorbed (e.g. fortified wines). Aerated drinks are also recognised to get into the system more quickly, owing to easy absorption (Paton 2005). The amount of alcohol that has been absorbed can be measured by the blood alcohol concentration. The rate of absorption will vary between individuals and, therefore, after two individuals have drunk the same amount and type of any one drink, they may have different blood alcohol concentrations. This will be due to a number of factors, such as the speed at which the drink was consumed and whether food was eaten while drinking, as this may interfere with the absorption from the stomach and affect the rate of gastric emptying. Some drugs are also known to inhibit or enhance the rate of gastric emptying, thus slowing down or increasing the rate of absorption, and subsequently affect the concentration of alcohol in the blood. Gender and body size are also recognised to be important determinants of the rate at which alcohol is absorbed. For example, an average woman drinking the same amount as an average man of the same size will become intoxicated more quickly, because she has a higher percentage of body fat. As alcohol is poorly soluble in fat and women have a lower percentage of lean tissue, there is consequently a higher concentration of alcohol found in the lean tissue. Moreover, as women tend to be of smaller stature, they will also have a smaller volume of blood, resulting in a higher concentration of alcohol in the blood (Paton 2005).

5.2 Alcohol metabolism

Once absorbed, alcohol must be metabolised immediately, for two reasons. First, the body has no capacity to store alcohol, in contrast to the other energy-yielding macronutrients (protein, fat and carbohydrate), and second, alcohol has toxic properties. The liver is the principal site of alcohol metabolism, but some alcohol is also immediately metabolised in the stomach by alcohol dehydrogenase (the enzyme responsible for breaking down alcohol), found in the gastric mucosa (James & Ralph 2000). Some alcohol (2–5%) is excreted unchanged in the urine, sweat or breath.

In a healthy person, the majority of alcohol consumed is metabolised, and therefore removed from the blood at a constant rate; on average, about 6 g per hour (James & Ralph 2000). However, the rate at which alcohol is metabolised varies considerably between individuals and is affected by a number of factors, including drug intake, frequency (and usual quantity) of alcohol consumption, age, bodyweight and liver size, and some differences may be genetic (Truswell 1998). Gender has also been found to be associated with the speed at which alcohol is metabolised. Women have less alcohol dehydrogenase activity in the gastric mucosa, and so they metabolise alcohol more slowly and have a steeper rise in blood alcohol concentration when drinking. It has also been suggested that the microsomal ethanol-oxidising system (MEOS) pathway (another mechanism of breaking alcohol down, see below) operates more slowly in women (Frezza et al. 1990).

Metabolic pathways of ethanol utilisation

Most of the absorbed alcohol (C\textsubscript{2}H\textsubscript{5}OH) is oxidised to acetaldehyde (CH\textsubscript{3}CHO). Three separate enzymes or enzyme systems have been identified to catalyse this reaction, as outlined in figure 4 (and explained in more detail below). Although quantitative data on their respective roles are difficult to obtain, it is thought that the different systems are involved in different ways in the metabolism of alcohol.

The principal route of alcohol metabolism is via the Nicotinamide adenine dinucleotide (NAD)-linked enzyme alcohol dehydrogenase (ADH), which is in the cytoplasm of liver cells and in the gastric mucosa and which catalyses the reaction:

\[
\text{C}_2\text{H}_5\text{OH} + \text{NAD}^+ \rightarrow \text{CH}_3\text{CHO} + \text{NADH} + \text{H}^+ 
\]

Low levels of alcohol are metabolised to acetaldehyde by ADH. The hepatic MEOS is induced in the liver and other tissues by repeated ingestion of alcohol and, as a result, alcoholics are thought to have an induced MEOS, whereas for the general population, the role of this system in ethanol metabolism is small. This system alters the energy coupling of alcohol oxidation to oxidative phosphorylation and heat is generated. The MEOS requires the co-enzyme NADPH and oxygen to catalyse the reaction as shown:
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C₂H₅OH + NADPH + H⁺ + 2O₂ → CH₃CHO + 2H₂O₂ + NADP⁺

This reaction also involves a cytochrome similar or identical to cytochrome P450, which is also involved in other inactivation or detoxification reactions, including those linked with some drugs. Ethanol can, therefore, compete with drugs for this enzyme, which may be why alcohol ingestion can increase the circulating concentration of many drugs and why for many drugs alcohol is contra-indicated.

The third pathway that is thought to exist is an indirect one. This pathway is not thought to play a major role in alcohol metabolism, but it may be used under certain physiological conditions. Catalase, in addition to catalysing the breakdown of hydrogen peroxide (H₂O₂), can also catalyse the oxidation of alcohols, including ethanol, as the chemical formula shows:

C₂H₅OH + H₂O₂ → CH₃CHO + 2H₂O

Oxidation of acetaldehyde to acetate (CH₃COO⁻)

Acetaldehyde is the product of all three systems outlined above; it is toxic (more so than alcohol) and has to be metabolised further to acetate. This reaction is catalysed by the enzyme acetaldehyde dehydrogenase (ALDH), and can be represented as:

CH₃CHO + NAD⁺ + H₂O → CH₃COO⁻ + NADH + 2H⁺

A large proportion of acetate is rapidly converted in other tissues to non-toxic acetyl co-enzyme A (acetyl CoA) by the enzyme acetyl-CoA synthetase, then further oxidised via the tricarboxylic acid cycle (TCA cycle) (Krebs cycle). This process provides most of the adenosine triphosphate (ATP) that results from ethanol oxidation.

Generation of energy

Finally, there are biochemical reactions in the liver which are involved in the regeneration of co-enzymes required for efficient ethanol metabolism. Most of the NADH is reoxidised to NAD⁺ by the mitochondrial electron transfer chain, which ultimately results in the production of more energy. NADP⁺ is reduced by the pentose phosphate pathway, thus alcohol-derived energy is merely a by-product of alcohol detoxification. Whether these detoxification mechanisms simply dissipate the energy provided by alcohol, or whether the energy in alcohol is incorporated into the energy budget in the same way as protein, fat and carbohydrate, has been the subject of some considerable debate. One theory has suggested that the energy produced as a result of alcohol detoxification is wasted as heat, rather than used for biochemical work, a so-called ‘futile cycle’ (Lieber 1991a). However, as discussed in section 8, evidence indicates no significant wastage of energy when alcohol is metabolised via the principal route.

Genetic differences in alcohol and acetaldehyde metabolism, alcohol sensitivity and alcoholism

In the past few years, several studies have been published about the polymorphisms of the genes that code for enzymes responsible for the breakdown of acetalde-
hyde – the alcohol dehydrogenase family (see Couzigou 1999). In humans, there are at least four or five isoenzyme classes of ALDH, of which ALDH2 is one. Individuals homozygous for the ALDH2*2 allele have no ALDH2 activity. This results in an elevated level of acetaldehyde after alcohol consumption, which causes unpleasant side effects, such as facial flushing and headaches. A high percentage of individuals of Asian origin (e.g. up to 50% of Japanese and Chinese people) are recognised to have a mutation of the acetaldehyde dehydrogenase ALDH2 gene, and alcoholism has been found to be lower among these individuals, perhaps because they avoid alcohol consumption (Truswell 1998). It has been hypothesised that this is also the reason for lower intake of alcohol among this subgroup of the population, which was illustrated in section 4.

**Key points**

- The rate at which alcohol is absorbed from the digestive tract varies between individuals, as it is dependent on many factors, such as gender, body size, the speed at which the drink was consumed or whether food was eaten while drinking.
- Once absorbed, alcohol must be detoxified and metabolised immediately, as it has toxic properties. The liver is the principal site of alcohol metabolism.
- The rate at which alcohol is metabolised also varies considerably between individuals. On average, it is metabolised at a rate of about 6 g per hour (a little less than one unit/hour), but this depends on a number of factors, e.g. gender, frequency of alcohol consumption, age and stature.
- Alcohol is metabolised in three stages:
  - there are three potential pathways in the first stage of alcohol metabolism, in which alcohol is oxidised to acetaldehyde. The most commonly used route is via the NAD-linked enzyme ADH;
  - acetaldehyde is more toxic than alcohol, and therefore is oxidised quickly to acetate via the enzyme ALDH;
  - acetyl CoA synthetase converts a large proportion of acetate to acetyl CoA, where it is further oxidised to provide energy, via the Krebs cycle.
- Due to a genetic polymorphism, some individuals are unable to oxidise acetaldehyde efficiently. Therefore, when they consume alcohol, they are exposed to high levels of acetaldehyde, which causes unpleasant side effects, such as headaches and facial flushing. This is a particular problem among the Asian population.

**6. Alcohol and nutrition**

**6.1 Nutrient composition of alcoholic beverages**

Alcohol is the second highest source of energy, on a per gram basis, of all the macronutrients, providing 29 kJ/g (7 kcal/g). As table 4 indicates, most alcoholic beverages contain only small amounts of nutrients – primarily carbohydrate and very small amounts of protein. Beers also contain small amounts of some micronutrients, primarily B vitamins and some minerals, and wines contain several trace elements, including iron, potassium, copper and sodium. As with beer, the quantity of vitamins, minerals and trace elements in wine varies considerably and depends on a number of factors, including the raw ingredients and production method. For example, in wine the micronutrient content will be affected by the type of

<table>
<thead>
<tr>
<th>Table 4 Nutrient content of specific alcoholic beverages, per 100 ml</th>
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<tbody>
<tr>
<td><strong>Alcohol (g)</strong></td>
</tr>
<tr>
<td>Bitter</td>
</tr>
<tr>
<td>Lager</td>
</tr>
<tr>
<td>Guinness</td>
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<tr>
<td>Cider</td>
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<tr>
<td>Red wine</td>
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<tr>
<td>White wine</td>
</tr>
<tr>
<td>Port</td>
</tr>
<tr>
<td>Spirits (37.5% ABV)</td>
</tr>
<tr>
<td>Alco-pops</td>
</tr>
<tr>
<td>Cream liqueurs</td>
</tr>
</tbody>
</table>

Source: Chan et al. (1994); personal correspondence.

ABV, alcohol by volume; Tr, trace.
grape and its harvest time, production method, additives to the wine (e.g. vitamin C is added to some wine to stabilise it), storage, and yeast variety used in the fermentation process (yeast contains high levels of some B vitamins). As shown in table 4, liqueurs may also contain protein and fat owing to the addition of egg or cream.

Values for alcoholic beverages given in food tables are expressed per 100 ml, rather than the usual method of per 100 g. This is because alcoholic beverages are normally measured by volume (see section 2). To determine nutrient composition per 100 g, values should be divided by specific gravity. The precise specific gravity varies according to the composition of the drink, and generally increases with the amount of solids (mainly sugars) and decreases with the amount of alcohol. For example, the specific gravity of 40% volume spirits is 0.95, that of medium white wine is about 1.005, and that of draught bitter is about 1.004. For comparison, the specific gravities of ethanol and water are 0.79 and 1.00, respectively. For more information, see Chan et al. (1994).

6.2 The effect of alcohol on energy intake
As well as being a source of energy, it has been suggested that consuming alcohol may affect an individual’s food intake, thereby having an indirect, as well as direct, effect on overall energy consumption. The effects of alcohol on food intake have been investigated in two ways. First, laboratory-based studies have been used to measure physiological indicators associated with food intake, such as appetite, hunger or satiation, following various pre-loads and test meals. Second, observational studies of free-living people have considered trends in energy intake according to levels of alcohol consumption. Overall, these studies have demonstrated that energy derived from alcohol is additive to the individual’s normal diet, i.e. individuals are unlikely to reduce their food intake to compensate for the energy from alcohol during the day (Westerterp et al. 1999). Interestingly, reports of small laboratory studies (Foltin et al. 1993; Westerterp-Plantenga & Verwegen 1999) have indicated that alcohol can have an aperitif effect (i.e. act as an appetite stimulant), resulting in a greater overall intake of energy from a test meal after consumption of alcohol. The duration of this effect has been found to vary, along with the extent to which it affects subsequent food intake (i.e. amount of food consumed) (Yeoman 2004). The mechanism by which alcohol stimulates appetite is not yet known; however, as alcohol intake affects many neurotransmitters, hormonal and other afferent signals (such as leptin, a hormone involved in appetite regulation), changes to any one or all of these could affect food intake (Yeoman et al. 2003). Yeoman (2004) also reported that the disinhibition of restrained eating or decreased satiation from foods may be another possible mechanism for the short-term stimulation of appetite following an alcoholic beverage.

6.3 Nutritional implications of chronic excessive alcohol consumption
Chronic excessive consumption of alcohol is well recognised to affect an individual’s nutritional status and is well documented to be associated with nutrient deficiencies and malnutrition. It is believed that this is for two reasons: first, alcoholics are recognised to have a poor diet, perhaps due to a disturbance in normal eating behaviour resulting from a disruption to ‘normal life’, which may result in primary malnutrition (Santolaria & González-Reimers 2004); and second, when consumed in large quantities, alcohol interferes with the digestion, absorption, metabolism and utilisation of some nutrients (Lieber 2000; Santolaria & González-Reimers 2004). This malabsorption of nutrients from the diet may result in secondary malnutrition, as figure 5 illustrates.

Poor utilisation of nutrients because of inefficient metabolism or retention may also be a consequence of alcohol-related diseases, such as chronic pancreatitis and liver disease. Although it was once thought that diseases of alcoholics, such as liver cirrhosis, were the result of poor nutrition alone, it is now recognised that poor nutrition is usually an effect, rather than the cause (James & Ralph 2000; Santolaria & González-Reimers 2004).

Deficiencies of particular nutrients, such as thiamin and folate, are well recognised among chronic excessive consumers of alcohol. For example, thiamin deficiency due to excessive alcohol intake is referred to as Wernicke–Korsakoff syndrome and is characterised by
mental confusion and short-term memory loss (James & Ralph 2000). A more severe form, Wernicke’s encephalopathy, starts suddenly and features areas of bleeding in the brain, resulting in states of confusion, loss of bodily co-ordination and abnormal eye movements (Youngson 2001). A large amount of information outlining the implications of chronic excessive alcohol intake on nutritional status exists. However, a detailed overview of this is beyond the scope of this report, which focuses primarily on the implications of moderate alcohol intake.

**The nutritional implications of moderate alcohol consumption**

Few studies have been undertaken to assess the impact of moderate alcohol intake on nutritional status, and there is a lack of consistency in the results of those studies that have taken place (Jacques et al. 1989; Guillard et al. 1994; Lecomte et al. 1994; Walmsley et al. 1998).

Three possible scenarios, or a combination of all of them, should be considered when exploring the nutritional implications of low/moderate alcohol intake:

- Moderate alcohol consumption may increase intakes of some nutrients, either because of the contribution of nutrients found in alcohol (such as B vitamins in beer), or because of interactions between alcohol and nutrients. For example, Walmsley et al. (1998) report higher intakes of B vitamins with increasing alcohol consumption, which was partially explained by the high content of certain B vitamins in beer;

- Even at low intakes, alcohol may adversely affect the digestion, absorption, metabolism or utilisation of nutrients, which could have adverse consequences on nutritional status when alcohol is consumed on a regular basis. It is well recognised that chronic excessive alcohol exposure impairs folate absorption, for example by inhibiting expression of the reduced folate carrier and decreasing the hepatic uptake and renal conservation of circulating folate (and subsequent excretion in urine), although it is less clear to what extent moderate alcohol intake affects folate absorption (Halsted et al. 2002; Mason & Choi 2005). A study by Laufer et al. (2004) demonstrated that moderate alcohol intake may have adverse effects on vitamin B12 status, which has been attributed to intestinal malabsorption and altered binding of intrinsic factor (Shaw et al. 1990). Previous studies in this area have found inconsistent results, which Laufer et al. (2004) attributed to methodological weaknesses (Jacques et al. 1989; Van der Gaag et al. 2000);

- Substituting food with drink, which in turn reduces nutrient intake, is a common problem among alcoholics. However, conclusions from a number of studies to date have found that alcohol does not habitually substitute food intake among light to moderate alcohol consumers, and therefore nutrient loss should not be expected from reduced food intakes (Colditz et al. 1991; Veenstra et al. 1993; Männistö et al. 1996; Walmsley et al. 1998).

The variation in results of studies that have looked at nutrient status and moderate alcohol intake may be because the likelihood of any of these scenarios occurring will vary depending on a number of factors, including: the nutrient in question; an individual’s habitual intake of the nutrient; the quantity of alcohol consumed and over what period of time; and the nutrient status of the individual (van den Berg et al. 2002). So many confounding factors make it particularly difficult to disentangle relationships between moderate alcohol intake and nutritional status for any one nutrient.

**The nutritional implications of binge drinking**

There is little published information available about the implications of binge drinking on nutritional status. Due to the potential complications of carrying out studies in this area, it is difficult to know the true effects of binge drinking on our diet and nutrient status. Taking account of common drinking behaviours, there are perhaps a number of factors that will affect the nutrient intake of binge drinkers, and for regular binge drinkers, this may lead to a compromised nutrient status. For example, it is common among individuals who go out to drink in order to get drunk (i.e. binge drink) to avoid eating before going out (in order to get drunk more quickly) and forget to eat after the evening out. Excessive alcohol intake over a short period of time may also lead to adverse gastrointestinal conditions, such as vomiting or diarrhoea, following the drinking episode, which may reduce the absorption of nutrients. This may be the result of fluid overload, as alcohol passes rapidly into the colon, or the effect of microorganisms inducing an acetaldehyde-stimulated colonic response. The metabolism of nutrients may also be affected by the large quantities of alcohol consumed. Bearing in mind these possible implications of binge drinking, infrequent binge drinking is unlikely to have any long-term consequences on nutrient status. However, regular binge drinking may have a more negative impact.

### 6.4 Other components of alcoholic beverages

**Phenolic and polyphenolic compounds**

Some beverages contain plant-derived bioactive phenolic compounds such as flavonoids. Plants produce
thousands of phenolic and polyphenolic compounds as secondary metabolites. These bioactive compounds are essential to the plant’s physiology, being involved in diverse functions, such as structure, pigmentation, pollination, pathogen and predator resistance, and growth and development. These phenolic and polyphenolic compounds have a diverse range of effects in vitro that are suggestive of a putative role in the prevention of chronic diseases, such as cardiovascular disease (CVD) and cancer. However, their precise role remains largely unclear, and research has been made more difficult by the fact that absorption of the parent compounds is generally low and it has been necessary to study the fate following absorption of a diverse range of metabolites. Many of these polyphenols can be found in alcoholic beverages. For example, red wines, and to a much lesser extent, white wines, are a rich source of a variety of phenolic and polyphenolic compounds. In the making of red wine, with prolonged extraction, the fermented ‘must’ may contain up to 40% of the phenolics originally present in the grapes. The production of white wine results in either low levels or an absence of skin- and seed-derived phenolics, so the overall level of phenolics is much lower than that found in many red wines. The range of concentrations also differs according to the geographical origin of the grapes.

Whiskies also contain complex mixtures of phenolic compounds. These include quercetin, vanillin, and ellagic, gallic and syringic acids, which are extracted from the wooden casks during maturation (Duthie & Crozier 2003). The phenolic profile depends on how long the spirit has been matured, the species of oak from which the casks are made, the pre-treatment of the cask, e.g. charring the wood, prior use of the cask for bourbon or sherry storage, and the number of times the cask has previously been used for maturation. Phenolics may also arise from derivatives of malt and from peat smoke, and from the breakdown of wood lignin during cask charring and whisky maturation. In in vitro studies, the presence of phenolics conveys considerable anti-oxidant activity to whiskies. However, there appear to be few epidemiological studies that have attempted to relate whisky consumption per se to the incidence of heart disease and cancer, and as noted in section 8, the type of drink consumed may be predictive of lifestyle factors, which can cause problems with confounding in epidemiological studies.

Beer contains a range of phenolic and polyphenolic compounds which come from the barley and hops used in beer production. Total phenolic concentrations are comparable to those found in white wine and may approach levels found in phenolic-rich red wines (Duthie & Crozier 2003). Consequently, beer exhibits considerable antioxidant activity in model systems, and the phenolics and/or their metabolites appear to be readily absorbed, resulting in increases in antioxidant capacity of plasma after consumption (Fantozzi et al. 1998; Bourne et al. 2000; Ghiselli et al. 2000). However, relevance of this to health still remains unclear (Duthie & Crozier 2003).

Congeners

Alcoholic fermentation (i.e. the fermentation of fruits and grains to produce alcohol) produces about 95% alcohol and 5% fusel oil, a mixture of organic acids, higher alcohols (propyl, butyl and amyl), aldehydes and esters. These bioactive compounds are known collectively as congeners and distil over with the alcohol. On maturation of the liquor, fusel oil changes and imparts the special flavour to the spirit, and therefore it is understood that congeners contribute to the taste, smell and appearance of alcoholic beverages (Swift & Davidson 1998). Many of the symptoms of a hangover (see section 10) are attributed to congeners in alcoholic beverages, and therefore they may have some short-term effects on health (Bender & Bender 2000; Wiese et al. 2000).

Key points

- Once absorbed and metabolised, alcohol contributes to an individual’s energy balance, providing 7 kcal per gram.
- Most alcoholic beverages contain alcohol, carbohydrate and only small amounts of protein, some vitamins, trace elements or minerals.
- Overall, studies have demonstrated that energy derived from alcohol is additive to the individual’s normal diet, and individuals are unlikely to compensate for energy from alcohol during the day. The aperitif effect of alcohol may also encourage an increase in energy intake.
- Chronic excessive alcohol intake is well recognised to affect an individual’s nutrient status; because it reduces food intake, and/or as a result of alcohol interfering with the digestion, absorption, metabolism and utilisation of some nutrients. Deficiencies of some nutrients, such as thiamin and folate, are more common among alcoholics, because these nutrients are particularly sensitive to the effects of alcohol.
- Any association that exists between moderate alcohol consumption and nutrient absorption and metabolism, is likely to be dependant on a number of factors, including: the nutrient in question; habitual intake of the...
nutrient and the nutrient status of the individual; and the quantity of alcohol consumed over a period of time.

- Overall, when consumed in moderation, alcohol is unlikely to interfere with the metabolism of nutrients, or be associated with impaired vitamin function or depletion, to an extent that may harm health, provided that dietary intakes are adequate.
- Little information exists about the implications of binge drinking on nutrient status; but it may alter food intake, and excessive alcohol consumption can cause disturbances to the digestive system. Although infrequent binge drinking is unlikely to have any long-term nutritional implications, regular binge drinking may have a more negative impact.
- Wine, beer and whiskies all contain a range of phenolic and polyphenolic compounds in varying quantities. These bioactive compounds have potential health-promoting attributes, but their precise roles remain largely unclear.

7. The burden of disease and mortality related to alcohol

The number of alcohol-related deaths in England and Wales has been rising since the 1980s, with a more substantial increase in recent years, as figure 6 indicates.

Alcohol-related deaths are more common among men than among women; for example, in 2004 the male death rate was 17.7 per 100 000 population, compared with the rate for women of 8.5 deaths per 100 000 population (Office for National Statistics 2006b). The proportion of deaths related to alcohol and the most common causes of alcohol-related death vary by age. For example, among the young, road traffic accidents, suicide and alcoholic liver disease are more common causes of alcohol-related death (Britton & McPherson 2001). A number of published papers have tried to establish the relationship between alcohol consumption and risk of death. For example, using data from the Whitehall cohort following more than 10 000 civil servants for 11 years, a ‘U’ shaped relationship between alcohol intake and mortality was reported. Non-drinkers and those drinking more than 248 g per week had approximately a twofold increased risk of mortality, compared with moderate drinkers, consuming 10–80 g of alcohol each week (Britton & Marmot 2004). The optimal frequency of drinking was between once/twice a week and daily, after adjustment for average volume consumed per week (Britton & Marmot 2004).

The Copenhagen Heart Study, involving more than 12 000 individuals (aged 30–79 years) followed for 12 years, reported an increase in risk of mortality with increasing alcohol consumption, with a significant relative risk of 1.44, for 42–69 drinks (approximately 400–700 g) per week, which increased to a relative risk of 2.29 for 70+ drinks per week. However, individuals drinking 1–6 drinks (10–60 g) per week had a 35% lower death rate compared with those who never drank (Gronboek et al. 1994). Data from more than 490 000 participants of the US Cancer Prevention Study II have been analysed to assess alcohol consumption and mortality among middle-aged and elderly adults (Thun et al. 1997). The study concluded that moderate alcohol consumption slightly reduced overall mortality, with overall death rates of those reporting about one drink/day (approximately 10 g) being 20% below those of non-drinkers; but above one drink per day, the overall death rate among drinkers increased (Thun et al. 1997). All these studies included participants aged 30 years and over only, and as noted, age is particularly important when considering the balance of adverse and beneficial effects of alcohol intake.

Analysis by Britton and McPherson (2001) concluded that there is only likely to be a net favourable mortality balance associated with light/moderate alcohol consumption among men aged 55 years and over and women aged 65 years and over, who have a much higher risk of CVD. There is also a pronounced gender effect on CHD-related mortality, with women reaping the benefits, and harms, of alcohol at a much lower level of intake, as discussed in section 8. The relationship between mortality and alcohol consumption is also affected by choice of the control population (i.e. light drinkers or abstainers) which is discussed further in section 8.1.

![Figure 6](image_url) Alcohol-related death rates across England and Wales, 1991–2004. Source: ONS (2006b).
It should be noted that none of the studies cited above have considered the effect of the pattern of alcohol consumption, which is particularly important to some causes of alcohol-related death, such as accidents and violence, but also possibly stroke and some cases of CHD (as discussed in section 8), and this may account for some of the variation in alcohol-attributed deaths between European countries reported by Britton et al. (2003). The Whitehall II Cohort Study also reported that drinking only once a month, or on special occasions, had a 50% increased risk of mortality (Britton & Marmot 2004).

For the younger age groups, the benefits may be outweighed by the increased risk of other diseases (e.g. alcohol-related cancers, liver cirrhosis) and increased risk of violence and accidents. White et al. (2002) have considered the younger population in their analysis, based on population data and relative risk of mortality from published systematic reviews, and made some recommendations for sensible drinking limits depending on age, assuming that no more than a 5% increase in risk of mortality is an acceptable compromise. A limit of 1 unit per day for women up to the age of 44 years, 2 units per day for women aged 45–74 years and 3 units per day for those aged over 75 years are recommended. More tiers have been recommended for men: those up to 34 years – 1 unit per day; 35–44 years – 2 units per day; 45–54 years – 3 units per day; 55–84 years – 4 units per day, and 85+ years – 5 units per day (White et al. 2002).

Mortality data have commonly been used as a source of information to identify health problems for a population. However, as medical techniques have become more advanced, death is more easily averted, and therefore the mortality statistics are unable to provide a comprehensive picture of the health problems in a country. Other measures to summarise population health have been introduced, such as disability-adjusted life years (DALYs). DALY is a measure of the years ‘lost’ to premature death and the years of healthy life ‘lost’ to living in a poor state of health. This means that, if a condition is associated with morbidity, rather than mortality (such as chronic excessive alcohol intake), the DALY measure will be larger. For example, Connor et al. (2005) calculated that 17 200 years of life were lost from premature deaths due to alcohol in New Zealand in 2002, while 26 000 DALYs were lost. DALYs due to alcohol are in some cases greater compared with other common diseases; for example, alcohol accounted for 4% of DALYs for the USA in 1996, compared with only 0.4% of deaths; whereas ischaemic heart disease accounts for 11% of DALYs, and a substantial 24.7% of deaths (McKenna et al. 2005). This emphasises the potential harm that alcohol can have on poor health and subsequent quality of life.

**Key points**

- Studies looking at the relationship between alcohol intake and mortality suggest that 10–80 g (approximately 1–8 drinks) of alcohol each week is optimal to reap the health benefits; thus indicating a ‘U’ shaped relationship between alcohol intake and mortality.
- Gender and age are important factors when considering the association between alcohol intake and CHD-related mortality.
- For younger people, the benefits of alcohol intake may be outweighed by the increased risk of other diseases (e.g. alcohol-related cancers, liver cirrhosis) and increased risk of violence and accidents.
- Alcohol is commonly associated with morbidity, rather than mortality.
- DALYs due to alcohol are in some cases greater compared with other common diseases.

### 8. Alcohol and disease risk

There is now a large body of scientific literature that supports the hypothesis that consuming alcohol modifies the risk of several diseases. The extent to which it affects the risk of disease will vary, depending on a number of factors relating to the alcohol consumed, such as the quantity and type of drink, and individual characteristics such as genetic susceptibility. Chronic excessive alcohol drinkers are also susceptible to a number of conditions not commonly seen among the general population, such as alcoholic neuropathy. Fortunately, it is only a minority of the population who can be classified as chronic excessive drinkers, and therefore, this section will focus on the relationship between light/moderate alcohol consumption and risk of common non-communicable diseases, including obesity, heart disease, stroke, high blood pressure (hypertension) and cancer.

#### 8.1 Methodological problems of studies assessing associations between alcohol consumption and disease risk

There are many methodological problems facing researchers attempting to investigate the association between alcohol consumption and chronic disease risk. In particular, it is very difficult to assess alcohol consumption accurately. Many studies rely on self-reported intakes, and misclassification is common as respondents may misreport their intakes either intentionally (e.g. for fear of being stigmatised) or unintentionally (e.g. from...
over- or underestimation) (ILSI 1999). Inappropriate classification of drinkers could lead to erroneous associations between measured intake and risk of disease. The problems in reliably assessing individuals’ alcohol intake also make it difficult for studies to ensure a heterogenous sample with representation from all drinking ‘categories’.

‘Non-drinkers’ or ‘abstainers’ are usually the selected reference population. The appropriateness of this has been debated, as individuals may choose not to drink because of ill-health (these people are sometimes referred to as ‘sick quitters’), or may follow a ‘healthier’ diet and lifestyle, as illustrated among those taking part in the European Prospective Investigation into Cane and Nutrition (EPIC) Heidelberg study (Ruf et al. 2005). Either of these factors may put the reference sample at an increased or decreased risk of certain diseases (ILSI 1999).

Drinking habits may also change substantially over time, which creates a problem for prospective studies that measure intake on one occasion and follow up subjects for prolonged periods. It is possible that drinking patterns (i.e. daily, drinking with meals, binging drinking, etc.) influence associations between alcohol consumption and disease risk. It was once thought that the type of drink consumed might also be relevant in considering disease risk, although there is now a consensus that it is the alcohol content per se that is important. However, what is becoming increasingly apparent is that the type of drink is a predictor for specific lifestyle factors that need to be considered. For example, studies have found wine drinkers to have a healthier diet than people who drink beer or spirits (McCann et al. 2003; Johansen et al. 2006). Therefore, investigations into this area need to adjust sufficiently for lifestyle factors such as diet, smoking and physical activity, because variations in lifestyle may account for at least some of the differences in disease risk (Grønboek 1999; Tjønneland et al. 1999).

Comparing findings from different studies can also be difficult, as studies use different measurement units. Respondents may recall the number of drinks they drank, but information on the size of the drink and its alcoholic strength may not be available. Some studies may translate the information into grams of alcohol, while others refer to ‘standard units’ as discussed in section 3. The latter is a particular problem when comparing international studies, as standard units of alcohol vary across the world. A further complication with regards to measurement units is the inconsistent use of ‘categories’ of drinker (i.e. heavy, moderate and light) between studies. The categories used to describe weekly drinking habits (e.g. fairly high, high, etc.) in the UK dietary surveys were outlined in section 4; however, these terms are not consistent with the terminology used in the majority of studies discussed below.

8.2 Alcohol and bodyweight

Most prospective data support the conclusion that heavy alcohol intake (classified as >30 g or >3 drinks per day) is associated with an increased risk of weight gain and obesity, while light to moderate drinking is not (Wannamethee & Shaper 2004). The findings of cross-sectional studies have not been as consistent, except that very heavy consumers (consumption of >49 g/day) have a low body mass index (BMI) (reasons for which were discussed in section 4) (Suter 2005). These inconsistent findings may be due to the various methodological problems linked to studies measuring alcohol, as outlined at the beginning of this section, particularly the misreporting of intake, which is recognised to be a particular issue in overweight/obese subjects. Other factors that cannot be controlled for in epidemiological studies may also influence the relationship between alcohol consumption and bodyweight. For example, it has been suggested that much of the inconsistency between these studies may have arisen from the confounding effects of cigarette smoking (i.e. stimulation of energy expenditure, effect on decreasing appetite and subsequent food intake). This has been demonstrated by findings from the British Regional Heart Study, which found current smokers to have a lower mean BMI than non- (and ex-) smokers at almost every level of alcohol intake, and the strongest influence of alcohol on bodyweight was seen in non-smokers (Wannamethee & Sharper 1992; 2003). According to Suter (2005), other factors may influence the relationship between alcohol intake and bodyweight, including: the frequency of drinking (affecting the amount of alcohol consumed and the metabolism of alcohol and other macronutrients); gender; genetic background; and the composition of the diet.

A number of studies have reported associations between alcohol intake and distribution of bodyweight, particularly abdominal obesity; however, the exact relationship is unclear and is complicated by a variety of factors. For example, analysis of data from the large US prospective male Health Professionals’ Follow-up Study reported that changes in alcohol consumption over time were not significantly related to waist gain (Koh-Banerjee et al. 2003). However, the reported alcohol intake among this cohort was only approximately 11.5–14.9 g/day, and small-scale prospective studies suggest that quantity and frequency of alcohol intake may be an
important factor. For example, a case–control study by Dorn et al. (2003), involving approximately 2000 participants (with a mean age of 52 years), reported alcohol consumption to be associated with lower levels of centrally located fat, compared with abstinence, and in particular, small amounts of alcohol on a regular basis were associated with the smallest abdominal measurements. Participants with the most intense drinking (3–4 drinks/day) on a sporadic basis had some of the largest abdominal measurements.

Greenfield et al. (2003) also reported that a moderate alcohol intake (equated to 1–2 drinks per day) was significantly associated with less abdominal fat, compared with abstainers. This case–control study investigated the association between alcohol intake and abdominal fat among approximately 400 monozygotic twins to minimise genetic and environmental effects. Finally, some research has suggested that the relationship between alcohol and fat distribution may alter according to the type of alcoholic beverage, but the data are inconsistent (Sakurai et al. 1997; Dallongeville et al. 1998; Dorn et al. 2003).

As alcohol often seems to supplement, rather than displace, food-derived energy intake, as discussed in section 6, the lack of a clear relationship between alcohol consumption and bodyweight has led to the suggestion that energy from alcohol may have an effect on normal energy regulation. Possible explanations for this have considered the low biological efficiency of alcohol as an energy source, the thermogenic effects of alcohol and the interaction of alcohol with lipid metabolism. These possible explanations are considered in more detail below.

Thermogenic effect of alcohol

A review of studies considering the thermogenic effect of alcohol has shown that in non-alcoholic subjects, the extra energy expenditure associated with alcohol ingestion ranges from 10% to 15% of the energy content of the alcohol ingested (Westerterp et al. 1999). This is entirely in keeping with the thermogenesis associated with the other macronutrients. However, a recent review by Suter (2005) states that the thermic effect of alcohol among moderate consumers is 15–25% of the energy content of the alcohol intake, which would result in alcohol contributing less energy to the overall energy balance. It should also be noted that alcohol has a different thermogenic effect in heavy alcohol consumers, as the mechanism by which the alcohol is metabolised leads to increased thermogenesis (see below for more discussion) (Suter 2005). The alcohol-related ill-health, common to heavy drinkers, such as liver cirrhosis, may also have an impact on the thermogenesis of the alcohol consumed (Suter 2005).

Biological efficiency of alcohol

As explained in section 5, among heavy alcohol consumers (and habitual daily moderate alcohol consumers), a larger fraction of the alcohol energy will be metabolised via the MEOS. This system alters the coupling of alcohol oxidation to oxidative phosphorylation, by using NADPH, thus generating heat and reducing the amount of energy available (James & Ralph 2000; Suter 2005). It is suggested that this is a contributing factor for the lower BMIs of very heavy alcohol consumers compared with moderate drinkers or abstainers, and this has been supported by experimental studies with alcoholics, which found that when substituting 50% of dietary energy from carbohydrate with energy from alcohol, subjects lost weight (Lieber 1991a). These findings support the idea of alcohol inducing energy wastage among very heavy alcohol consumers, as the thermogenic effect of alcohol is higher than its biological efficiency. The dose of alcohol required for induction of the MEOS varies from one individual to another, which may provide a further explanation for the variability in results of studies of alcohol and bodyweight (Suter 2005). No significant wastage of energy is reported when alcohol is metabolised through the principal route, i.e. via the NAD-linked enzyme ADH (Lieber 1991a) (see section 4).

Alcohol and lipid oxidation

There are three possible mechanisms by which alcohol may interfere with lipid metabolism to encourage weight gain. First, it is well recognised that ingestion of alcohol leads to the suppression of fat oxidation by approximately 30%, while the alcohol is metabolised; this in turn inhibits fat mobilisation (Feinman & Lieber 1999; Jéquier 1999; Santolaria & González-Reimers 2004). A study by Siler et al. (1999) also indicated that moderate alcohol consumption led to an increase in hepatic de novo lipogenesis, thus increasing the fat mass directly. Therefore, the interaction of ethanol and lipid metabolism is relevant to the effect of alcohol consumption on bodyweight and body composition, because it favours lipid storage (Feinman & Lieber 1999).

Summary

As discussed in section 5, studies indicate that alcohol-derived energy is usually additive to the normal diet and
therefore promotes an increase in energy intake; therefore, one would expect alcohol intake to be associated with weight gain and a higher BMI (with the exception of chronic heavy drinkers whose diet and metabolism may be altered). Recent prospective studies considering weight gain and alcohol consumption have demonstrated that people who drink more than 3 drinks a day tend to be heavier, compared with low alcohol drinkers or abstainers. No association has been demonstrated at lower intakes, but other confounding factors may be obscuring the relationship.

8.3 Alcohol and coronary heart disease

In the past decade, there have been many epidemiological studies providing evidence that low to moderate intakes of alcohol may reduce the risk of heart disease, with one of the largest prospective cohort studies reporting an adjusted relative risk of 0.69 for angina and 0.65 for myocardial infarction among middle-aged men consuming one drink per day, compared with non-drinkers and occasional drinkers (<1 drink per week) (Camargo et al. 1997). It is evident from these studies that the amount of alcohol consumed is important when considering this relationship, along with gender. A comprehensive meta-analysis looking at the average volume of alcohol consumed in relation to CHD mortality risk found consumption of 0–20 g/day to be associated with a decrease in risk; this inverse association was noted for those consuming up to 72 g/day in both genders. Consuming 89 g/day or more was associated with an increased risk of CHD (Corrao et al. 2000). However, the major protective effect occurred at 10 g/day for women compared with 25 g/day for men, thus emphasising that gender is an important determinant. The meta-analysis noted that the estimates of degree of protection, based on the findings of 28 good quality cohort studies, were smaller than estimates based on all studies combined.

Other factors that may influence the association between alcohol intake and risk of CHD

More recently, it has been recognised that the pattern of alcohol consumption (amount of alcohol consumed per session or number of drinking occasions per week) may influence CHD risk (Agarwal 2002; Rehm et al. 2003b; Warner Kershner et al. 2004). To date, ‘drinking patterns’ have not been considered in many epidemiological studies, and therefore the influence of this factor may have been underestimated (Rehm et al. 2003a). A review of studies by Puddey et al. (1999) indicates an increased risk of major coronary events among drinkers with episodic heavy drinking patterns, thus adding to the current public health concern around people binge drinking.

This relationship still exists when comparing individuals who undertake heavy drinking sessions with those who consume similar amounts of alcohol spread over 1 week. For example, findings from the Health Professionals’ Study found that, for a given level of weekly alcohol consumption, spreading alcohol consumption across the week was potentially more beneficial to cardiovascular health than restricting it to a smaller number of days (Mukamal et al. 2003). These findings are generally in line with those from other large prospective cohort studies, although Tolstrup et al. (2006) reported that drinking patterns may be important to CHD risk among men, independent of total alcohol intake (i.e. the lowest hazard ratio was among men consuming 21 units or more per week over 7 days). Whereas among postmenopausal women, total alcohol intake may be more important, with the lowest risk reported among those consuming 14 drinks or more per week over 2–4 days.

Ecological studies, paradoxically indicating a low CHD mortality rate in populations with high smoking rates and consuming a diet high in saturated fat (e.g. the French), have led to the suggestion that some alcoholic beverages, namely red wine, are more protective than others. This concept has been supported by the presence of bioactive compounds, namely polyphenols, found in red wine (see section 6 for more discussion). A number of observational case–control and cohort studies have considered whether consuming one type of alcoholic beverage compared with another impacts on CHD risk. Current consensus is that it is the alcohol per se that is responsible for the positive heart health effects (Rimm et al. 1996; Burns et al. 2001; Wollin & Jones 2001; Rehm et al. 2003b; Warner Kershner et al. 2004); and this has been demonstrated by data from the Health Professionals’ Follow-up Study (Mukamal et al. 2003).

Studying some of the mechanisms recognised to be involved in the protective effect of alcohol, including changes in concentrations of high density lipoprotein (HDL) cholesterol, fibrinogen and triglycerides, Rimm et al. (1999) calculated that 30 g of alcohol a day should reduce risk of coronary disease by 24.7%. These mechanisms and others are discussed in more detail later in this section.

8.4 Alcohol and blood pressure

An association between blood pressure and alcohol intake is widely accepted, with heavy drinking patterns (in particular, binge drinking) being a risk factor for high
blood pressure, or hypertension (Dyer et al. 1981; Witteman et al. 1990; Marmot et al. 1994). Evidence indicates that a reduction in alcohol intake among heavy drinkers can reduce systolic and diastolic blood pressure among hypertensive or non-hypertensive people (Xin et al. 2001). For example, findings from a meta-analysis of randomised controlled trials indicated alcohol intake reduction in heavy drinkers (those consuming 3–6 drinks per day) results in a 3.31-mmHg reduction in systolic blood pressure and 2.04-mmHg reduction in diastolic blood pressure. Therefore, those diagnosed with hypertension should reduce their intake of alcohol in order to improve their condition (Dickinson et al. 2006).

However, there is much debate about the overall relationship between alcohol and blood pressure. For example, evidence indicates that moderate intakes of alcohol may lead to short-term lowering of blood pressure (Witteman et al. 1990; Gillman et al. 1995), whereas abstainers/teetotallers have a higher blood pressure, suggesting a ‘J’ shaped relationship (Papadakis et al. 2000; Agarwal 2002). Reviews have also described a threshold phenomenon, whereby the risk of high blood pressure increases if intake exceeds 3 standard drinks per day (Belin et al. 1996; Keil et al. 1998; Grobbee et al. 1999; Klatsky 2003). A consensus view on this issue has yet to be agreed. The variation in findings may be due to methodological differences. For example, a systematic review of nine studies by McFadden et al. (2005) suggested a potential reduction in blood pressure in the hours after exposure to alcohol, followed by a rise in blood pressure, thus indicating the importance of the timing of the blood pressure measurement. A review by Lip and Beevers (2003) summarises other factors that have been found to influence the possible relationship between alcohol and blood pressure, including type of beverage and pattern of drinking. For example, heavy ‘weekend drinkers’ have been found to have a higher blood pressure when compared than those who drink the same quantity over a week (Wannamethee & Shaper 1991).

8.5 Alcohol and stroke

A number of epidemiological studies have investigated the relationship between alcohol intake and stroke. These have shown alcohol to both reduce and increase the risk of stroke, depending on the quantity of alcohol consumed, drinking habits (e.g. binge vs. regular drinking), and the type of stroke (i.e. ischaemic stroke or haemorrhagic stroke). The variation in results is not surprising, as different types of stroke have different causes: ischaemic stroke is caused by a blockage in a blood vessel in the brain, commonly arising from a blood clot formed somewhere else, and therefore has a similar aetiology to that of CHD; haemorrhagic stroke is caused by a rupture of a blood vessel supplying the brain, thus releasing blood into the brain (see Stanner 2005 for more information). Therefore, it is unlikely that the effect of alcohol would be the same for both types of stroke. It is now believed that findings from earlier studies, which did not differentiate between the different type of stroke and the quantity of alcohol consumed, obscured the relationship between alcohol intake and stroke (Grobbee et al. 1999; Mazzaglia et al. 2001).

Although a recent comprehensive review reported a lack of association between light to moderate drinking and haemorrhagic stroke (Warner Kershner et al. 2004), meta-analyses that have explored this relationship have reported a positive, linear relationship between alcohol intake and haemorrhagic stroke (Corrao et al. 1999; Reynolds et al. 2003). In line with these findings, the most recent systematic review undertaken by Mazzaglia et al. (2001) concluded that there is a causal association between haemorrhagic stroke and heavy drinking (classified as more than 40 g/day).

Turning to ischaemic stroke, although there is a variation in the findings of studies, the consensus of opinion is that there is a positive association between ischaemic stroke and heavy alcohol intake. However, as illustrated by the meta-analyses and reviews, results from studies investigating the association between moderate alcohol consumption and ischaemic stroke are inconsistent (Puddey et al. 1999; Britton & McPherson 2001; Mazzaglia et al. 2001; Reynolds et al. 2003). In the most recent meta-analysis considering this association, those consuming 12 g or less of alcohol each day had a significantly lower risk of ischaemic stroke (Reynolds et al. 2003). However, more prospective studies are needed to clarify this association, as findings from the Health Professionals’ Follow-up Study indicated no association between 0.1–9.9 g of alcohol a day and risk of ischaemic stroke (Mukamal et al. 2005a). As ischaemic stroke is the most common type in Western countries, accounting for 75–85% of all strokes, any reduction in its risk could have a significant impact on public health.

Other factors that may influence the association between alcohol intake and risk of stroke

A small number of studies have considered whether the type of alcohol has any effect on the risk of stroke. To date, evidence indicates that wine (in moderation) may have protective effects, while other beverages do not (Constant 1997; Truelsen et al. 1998; Malarcher et al. 2001; Mukamal et al. 2005a). For example, the large
prospective Copenhagen City Heart Study found no association between risk of stroke and intake of spirits and beer, yet daily, weekly and monthly intakes of wine (mode = 1–7 units/week), compared with no wine, were associated with a lower risk of stroke (Truelsen et al. 1998). Assuming that studies have used appropriate methods to control for confounding lifestyle factors, this relationship may not be due to the difference in wine and beer composition, but could be due to wine being more commonly consumed with a meal, thus slowing down its metabolism. Although not always taken into consideration, patterns of drinking may be an important factor in understanding the relationship between alcohol consumption and stroke. For example, consumption of a large quantity of alcohol in a few hours is associated with an increased risk of both haemorrhagic and ischaemic stroke (Grobbee et al. 1999; Mazzaglia et al. 2001). Drinking pattern could therefore account for some of the variation in the results of different studies, and hence recent reviews recommend that future research should ensure this factor is considered.

Hypertension (as discussed above) is one of the most important risk factors for stroke and, therefore, alcohol-induced fluxes in blood pressure might be in part responsible for the association between alcohol consumption and stroke (Reynolds et al. 2003; Frayn 2005). Some studies have controlled for hypertension when assessing the relationship between alcohol intake and stroke, which Puddey et al. (1999) suggest may attenuate any association; however, a consensus on this issue has yet to be met. Other possible explanations for the variation in findings include possible racial and gender inter-relationships (Puddey et al. 1999). Alternatively, it is feasible that raw data (e.g. death certificates) used by the researchers may have been inaccurate, as there are few reliable methods of distinguishing between different forms of stroke (Mazzaglia et al. 2001).

Sudden death

Although findings have varied, some studies have reported an association between cardiovascular death and heavy episodic drinking (Britton & McKee 2000; Malyutina et al. 2002; Mukamal et al. 2003b). Those studies that did not account for drinking patterns found no significant results (Wannamethee & Shaper 2002; Marques-Vidal et al. 2004). A systematic review by McKee and Britton (1998) suggests that heavy drinking occasions are mainly associated with physiological mechanisms, increasing the risk of sudden cardiac death and other related conditions, such as increased blood clotting and reduced threshold for ventricular fibrillation. This association between heavy drinking occasions and cardiac deaths, particularly sudden cardiac deaths, came to light from experiences in Eastern Europe, where heavy episodes of binge drinking are common. In particular, an observational study by Chenet et al. (1998) reported a significant increase in sudden cardiac death at weekends, which is markedly similar to the pattern of deaths that are seen from alcohol poisoning and from accidents and violence, commonly associated with binge drinking.

Proposed mechanisms for how light/moderate alcohol intake may increase the risk of cardiovascular conditions

Two mechanisms are considered here to explain how light to moderate alcohol intake may increase the risk of hypertension. It is well recognised that hypertension can increase the risk of stroke through various mechanisms, as reviewed by Frayn (2005). However, the different dose-dependant effects and divergence between acute and chronic intake makes the mechanism of the interaction between alcohol and hypertension complex. Grobbee et al. (1999) suggest that the pressor effect caused by a high alcohol intake could be due to a neurogenic mechanism, a humoral mechanism or caused directly through the actions on the vessels that maintain peripheral resistance, resulting in hypertension induced by alcohol.

Alcohol has been found to have anti-coagulant effects, which may play an important role in increasing the risk of haemorrhagic stroke. However, overall, the mechanisms associating moderate alcohol consumption and increased risk of haemorrhagic stroke, and heavy alcohol intake and both haemorrhagic and ischaemic stroke, are unclear.

Recognised mechanisms for light/moderate alcohol intake reducing the risk of cardiovascular conditions

A number of mechanisms have emerged from both experimental and observational epidemiological studies that may explain the reported associations between alcohol consumption and CVD, and these are outlined below.

Blood lipids Reviews of the relationship between alcohol intake and blood lipids indicate that moderate alcohol intake has been shown to lead to an increase in high density lipoprotein (HDL) cholesterol (a lipoprotein that binds with cholesterol and brings it back to the liver for elimination or re-processing), and a decrease in low density lipoprotein (LDL) oxidation (which is involved in the development of atherosclerotic plaques) (Grobbee
et al. 1999; Puddey et al. 1999; Papadakis et al. 2000; Mazzaglia et al. 2001; Agarwal 2002; Warner Kershner et al. 2004). There has been some debate as to whether the pattern of drinking may be important in determining the effects of alcohol on blood lipids. Evidence indicates that this is not the case (Peasey et al. 2003; Puddey et al. 1999).

Antioxidant properties As alcoholic beverages are originally derived from plants, they contain some bioactive substances found in plants and plant-derived foods (Duthie & Crozier 2003). Of all the alcoholic beverages, wine has the highest concentration of bioactive substances, and in particular, polyphenols. Polyphenols are thought to have anti-oxidant activity which may be beneficial in protecting against the oxidation of LDL-cholesterol. Although studies have confirmed that polyphenolic compounds, from red wine in particular have antioxidant activity in vitro, it has been less clear whether such antioxidant effects occur in vivo. Furthermore, the bioavailability of polyphenols is an area of particular uncertainty (see section 6). There is also large individual variation in the absorption of bioactive substances between subjects.

Haemostatic factors The process of blood clotting (known as haemostasis) functions as a careful balance between blood flowing and stopping, and between clotting and clots dissolving. If blood is prone to clot too little, then there is a risk of haemorrhage; too much, and there is a risk of blood clots forming where they are not wanted, such as in an artery supplying blood to the brain, causing a stroke, or to the heart, causing a heart attack. Moderate alcohol consumption is recognised to have anti-coagulant effects as it causes:

- a decrease in levels of plasma fibrinogen (the precursor of the insoluble blood-clotting protein, fibrin), which reduces the risk of blood clots;
- a decrease in platelet adhesiveness, and a subsequent decrease in platelet aggregation;
- changes in the concentration of fibrinolytic factors, including clotting factor VII and tissue plasminogen activator, thus reducing platelet aggregation.

Homocysteine High levels of homocysteine in the blood (above those considered normal) are associated with an increased risk of CHD and stroke. Homocysteine is an amino acid produced as the body metabolises protein. Although alcoholics have been shown to have raised homocysteine levels, levels have been found to be lower in individuals who are moderate alcohol drinkers (Bree et al. 2002).

Several of the B vitamins are involved in homocysteine metabolism, and studies have found that supplementation, particularly with folic acid, can lower homocysteine levels (see Clarke 2005). Folate intake is also recognised to be of particular importance when assessing the association between alcohol intake and risk of chronic disease. For example, findings from the large prospective cohort Nurses’ Health Study indicate that higher dietary folate intakes (above 400 µg/day) can diminish the risk of CVD associated with high intakes (30 g/day) of alcohol, but the expected benefit of moderate alcohol intake was not observed when folate intake was low (Jiang et al. 2003). This finding could be due to the relationship between folate and serum homocysteine. However, there appears to be a variation in the association between folate, alcohol and risk of CVD with age, as the positive association between heavy alcohol intake, low folate and risk of disease seems to be most apparent in younger women below the age of 60 years (Jiang et al. 2003).

Endothelial function The endothelium forms the interface between the artery wall and constituents of the blood. It plays an important role in the regulation of vascular tone, for example by inhibiting platelet aggregation and adhesion. Endothelial dysfunction is thought to presage atherosclerosis; therefore, healthy functioning of the endothelium is important. There are some human studies linking moderate alcohol intake, particularly red wine, with better endothelial function (Teragawa et al. 2002).

Other emerging risk factors for CVD Moderate alcohol consumption (up to 40 g/day), compared with non-drinking and heavy drinking, has been found to have anti-inflammatory and immunomodulatory effects, which may influence the inflammatory processes that characterise the development of atheroma (Imhof et al. 2004). See Stanner (2005) for more information.

Associations between alcohol intake and CVD-summary

It is evident that there are disparities in the relationships between alcohol consumption and various cardiovascular conditions, with complex inter-relationships between conditions. For example, alcohol is associated with an increased risk of haemorrhagic stroke, but may have a protective effect on ischaemic stroke. Heavy drinking (more than 40 g/day), however, is associated with an increased risk of both types of stroke. As the majority of cases of stroke are ischaemic in nature, it is important to
identify the true characteristics of the relationship between stroke and alcohol intake. Drinking patterns also may be important in relation to the risk of stroke, and future studies in this area should consider this factor.

The impact of drinking patterns on heart health is also unknown, although there is now strong evidence that moderate consumption (10 g/day for women, and 25 g/day for men) decreases the risk of a CHD episode, with particular benefits to men aged over 55 years and post-menopausal women; however, heavy drinking (classified as more than 89 g/day) is again associated with an increased risk. In particular, a number of studies indicate a positive association between heavy episodic drinking and CVD-related death. The consensus of opinion among scientists is that it is the alcohol per se that influences heart health, rather than the type of alcoholic beverage, and other factors that affect an individual's choice of drink may be important in distorting the findings from studies that show otherwise.

A number of potential mechanisms have been identified to account for the risk reduction associated with moderate alcohol intake and CHD, and the possible benefits associated with ischaemic stroke. However, there are fewer explanations to account for the increased risk of CVD conditions associated with heavy consumption of alcohol, or the positive linear relationship between alcohol and haemorrhagic stroke.

8.6 Alcohol and type 2 diabetes

Evidence indicates that moderate alcohol consumption (1–3 drinks/day) is associated with a decreased risk (of 30–40%) of type 2 diabetes, when compared with teetotallers or the lowest consumers (Conigrave & Rimm 2003; Zilkens & Puddey 2003; Howard et al. 2004; Van de Wiel 2004; Carlsson et al. 2005; Koppes et al. 2005). However, heavy alcohol consumption appears to be associated with an increased risk; therefore, the association between alcohol and type 2 diabetes is thought to be 'J' or 'U' shaped, with risk beginning to rise at around 4 drinks per day (Conigrave & Rimm 2003; Howard et al. 2004; Carlsson et al. 2005; Koppes et al. 2005). It should be noted that a number of confounding factors may complicate the relationship between alcohol intake and risk of diabetes; gender, BMI, frequency of drinking and type of alcoholic beverage have all been found to be important.

**Possible mechanism**

Although the acute effects of alcohol may induce a state of insulin resistance, studies indicate that a moderate amount of alcohol intake results in enhanced insulin sensitivity in the long-term, and therefore this may account for the possible beneficial effect of moderate alcohol consumption on risk of type 2 diabetes, as reported in a number of review papers (Zilkens & Puddey 2003; Van de Wiel 2004; Koppes et al. 2005). This effect on insulin sensitivity may also be one of the mechanisms involved in the protective effect of alcohol with regard to atherosclerotic disorders. The cardio-protective effects of alcohol are particularly relevant to people with type 2 diabetes, for whom coronary risk factors are highly prevalent. Similarly, heavy alcohol consumption should be avoided by those with diabetes because of its detrimental effects on lipid metabolism (see section 8.2), blood pressure (see section 8.4) and other cardiovascular risk factors (see section 8.5).

8.7 Alcohol and cancer

There is strong and consistent convincing evidence from epidemiological and experimental studies that alcohol is an independent risk factor for oral, pharyngeal, oesophageal and liver cancers (WCRC 1997; Department of Health 1998; Key et al. 2004). There is also considerable evidence to show a small increase in risk of breast cancer with increasing alcohol consumption (Key et al. 2004). Reviews by Pöschl and Seitz (2004) and Morris-Brown (2005) have supported a weak association between increasing alcohol consumption and increased risk of colorectal cancer. Alcohol has also been considered to cause an increased risk of cancer of certain other organs, such as the stomach, lung and cervix (Bagnardi et al. 2001); however, this briefing paper will focus on cancers of the upper digestive tract, the liver, breast and colorectal cancer.

The increase in cancer risk appears to be primarily due to alcohol per se, rather than specific alcoholic beverages, although some studies have found that a stronger relationship exists between cancers of the upper digestive tract and distilled spirits (Longnecker 1995; Pöschl & Seitz 2004). Pattern of alcohol intake has been found to affect the association between alcohol and some chronic diseases, although very limited information is available on drinking pattern and cancer risk. A large prospective study showed that consumption of wine outside a meal was associated with a relative risk of cancer death of 1.7 for women and 3 for men, compared with those consuming wine with a meal. Also, one study indicated that subjects who drank outside of meals were at a higher risk of cancers of the upper aero-digestive tract, after allowance for drinking status and number of drinks per day. The association was particularly marked for...
cancers of the pharynx (odds ratio 1.8), oesophagus (odds ratio 1.7) and oral cavity (odds ratio 1.5) (Dal Maso et al. 2002). A detailed discussion of the mechanisms by which alcohol affects these organs/sites is beyond the scope of this briefing paper, but some of the most likely are briefly considered later in this section.

**Cancers of the upper aero-digestive tract**

Alcohol has a carcinogenic dose–response effect at sites of the upper aero-digestive tract, which is independent from smoking, and in some studies, smoking and alcohol have been found to interact in a multiplicative fashion (Department of Health 1998; Doll et al. 1999; Bagnardi et al. 2001). In Westernised countries, alcohol and tobacco are the main risk factors for 75% of oral and pharyngeal cancers (International Agency for Research on Cancer 1988). However, the incidence of these forms of cancer is relatively low in the UK, even among people who both smoke and drink alcohol; therefore, other mechanisms (e.g. genetics) are probably responsible, with alcohol and smoking playing a contributory role (Morita et al. 2002; Ogden 2005).

**Liver cancer**

Chronic excessive alcohol consumption can lead to liver cirrhosis, which can develop into hepatocellular carcinoma (Boffetta & Hashibe 2006). Excessive alcohol consumption is the main diet-related risk factor for liver cancer in Western countries. A meta-analysis by Corrao et al. (2004) reported a relative risk of 1.8 for hepatocellular carcinoma for the heaviest consumers (100 g/day). However, the direct correlation between alcohol consumption and the development of hepatocellular carcinoma is not straightforward, as the disease does not develop in all patients with cirrhosis. There is evidence that alcohol, the metabolism of alcohol, or both, affect cell signalling pathways that regulate normal and abnormal hepatocyte function, proliferation and apoptosis (McKillop & Schrum 2005). It is thought that the specific effect of ethanol on hepatocyte signalling integrity varies according to acute or chronic exposure to alcohol.

**Breast cancer**

A large number of epidemiological studies indicate that alcohol intake increases the risk of breast cancer, and the risk rises with increasing consumption of alcohol, even in small amounts (*i.e.* one drink per day) (WCRF 1997). Breast cancer is the most commonly diagnosed cancer among women across the UK. Although a number of other factors (e.g. family history, hormonal factors, age) contribute to the risk of breast cancer, alcohol is one of the few recognised dietary risk factors. The mechanism is unknown but may involve increased levels of circulating oestrogen. Various reviews and meta-analyses have been undertaken to assess the potential risk of breast cancer associated with alcohol intake (Longnecker 1994; Corrao et al. 1999; Bagnardi et al. 2001; Collaborative Group on Hormonal Factors in Breast Cancer 2002; Hamajima et al. 2002; Boffetta & Hashibe 2006). Longnecker (1994) reported a 10% increase in the risk of breast cancer with every 10 g of alcohol consumed each day (about one drink per day). Yet more recently, a collaborative reanalysis of data from 53 epidemiological studies found a smaller increase in the risk of breast cancer, of 7%, with every additional 10 g of alcohol consumed per day (Collaborative Group on Hormonal Factors in Breast Cancer 2002).

**Colorectal cancer**

Epidemiological studies have generally reported a positive association between alcohol consumption and risk of colorectal cancer. The most recent meta-analysis in this area reported a pooled multivariate risk of 1.16 (bordering on significance) with 30–44 g/day of alcohol, and a significant multivariate risk of 1.41 for those consuming 45 g/day of alcohol or more, when compared with non-drinkers (Cho et al. 2004). This meta-analysis indicated a dose–response, with alcohol intake of about 30 g/day (3 drinks) increasing the risk of colorectal cancer, while two other meta-analyses reported an increased risk from about 2–3 drinks per day (Longnecker et al. 1990; Bagnardi et al. 2001). However, findings related to specific alcoholic beverages or different anatomic sites in the large bowel have been inconsistent.

**Proposed interactions between alcohol and cancer-causing mechanisms**

Multiple (possibly site-specific) mechanisms may be involved in the associations between alcohol and cancer, although, perhaps most importantly, an individual’s risk of alcohol-related cancer is modulated by genetic factors (Boffetta & Hashibe 2006).

In animal models, alcohol does not initiate cancer, but can increase the effect of substances that are carcinogenic. However, when alcohol is metabolised to acetaldehyde, it becomes highly toxic, and is believed to be mutagenic and carcinogenic, and subsequently, it is thought that it is acetaldehyde that is responsible for the carcinogenic effect associated with alcohol consum-
tion. Binding to DNA and the formation of stable adducts are possible mechanisms by which acetaldehyde could trigger the occurrence of replication errors, or mutations in oncogene or tumour suppressor cells. See Pöschl & Seitz (2004) for further information.

The publication of the review on food, nutrition, physical activity and the prevention of cancer in 2007 by the World Cancer Research Fund, will provide a much needed, detailed overview of the evidence base in this area (for more information, see http://www.wcrf.org/research/second_wcrf_aicr_report.lasso).

Summary

Risk of cancer at a number of sites has been associated with alcohol intake. A consensus for a likely causal association has been identified between alcohol and cancers of the upper aero-digestive tract and the liver. However, mechanisms to explain the positive association between alcohol and breast cancer, and between alcohol and colorectal cancer, have yet to be identified. There is a linear dose–response relationship between alcohol intake and risk of cancer, although the magnitude of the risk varies depending on cancer site. However, it is likely that smoking will have a multiplicative effect at all sites.

Although the mechanisms identified to date suggest that it is the alcohol \textit{per se} that exerts damage and therefore the quantity of alcohol consumed is the most important variable, it remains unclear whether the type of alcoholic beverage consumed is an important factor in determining the association between alcohol intake and cancer.

Key points

- There are a number of methodological problems that need to be considered when exploring the associations between alcohol consumption and disease risk, including: misreporting of total intake; the use of an appropriate control group; and the need for comparable measurement units.
- Alcohol intake is associated with weight gain and an increase in BMI (with the exception of chronic excessive drinkers), and this is supported by recent prospective studies. However, a number of other factors may obscure the relationship, and may account for the variation in findings from studies.
- Alcohol is associated with an increased risk of haemorrhagic stroke, but low intakes (\textit{i.e.} one drink per day) may have a protective effect on ischaemic stroke. Heavy drinking is associated with an increased risk of both types of stroke, and this has been the predominant finding of studies. Drinking patterns also may be important in relation to the risk of stroke.
- There is now strong evidence that light to moderate (1–3 drinks per day) alcohol consumption decreases the risk of a CHD episode, compared with abstainers, with particular benefits in men aged over 55 years and post-menopausal women; however, heavy drinking is once again associated with an increased risk.
- Further information is required to determine the association between drinking patterns and heart health. However, there is a consensus of opinion that it is the alcohol \textit{per se} that influences our heart health, rather than a particular type of alcoholic beverage; and other factors that affect an individual's choice of drink may be important in distorting the findings from studies that show otherwise.
- Light to moderate alcohol consumption is associated with a decreased risk (of 30–40\%) of type 2 diabetes, when compared with teetotallers. Heavy drinking may be associated with an increased risk, resulting in a ‘J’ or ‘U’ shaped relationship between alcohol consumption and the incidence of type 2 diabetes (similar to that seen with CHD).
- Cancers at a number of sites have been associated with alcohol intake; however, a consensus for a likely causal association has only been identified between alcohol and cancers of the upper-aero digestive tract and the liver. The mechanisms to explain the positive associations between alcohol and breast cancer, and evidence for the positive association between alcohol and colorectal cancer, have yet to be identified.
- There is a linear dose–response relationship between alcohol intake and risk of cancer, although the magnitude of the risk varies depending on cancer site. However, it is likely that smoking will have a multiplicative effect at all sites. It is unclear whether some types of alcoholic beverage are more strongly associated with an increased risk of cancer than others. Mechanisms identified to date suggest that it is the alcohol \textit{per se} that exerts damage.

9. Alcohol and other conditions

Experience from clinical cases suggests that consuming even moderate amounts of alcohol may cause an adverse reaction in some individuals. Alcohol should also be avoided in certain circumstances, such as pregnancy.

9.1 Adverse reactions to alcohol for some susceptible individuals

A small number of studies have investigated why red wine may cause headaches and migraines among those
susceptible, when other alcoholic beverages do not. Although small scale, these studies have confirmed that alcohol per se is not the cause of these adverse reactions (Littlewood et al. 1988). The UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (2000) reported on the potential ‘allergic’ response to red wine, and suggested that the phenolic flavonoid content of the wine may be responsible. The report also noted that the headaches may be mediated by histamine, a vasoactive amine, which has been suspected of causing headaches. Jarisch and Wantke (1996) verified this association among those suffering histamine intolerance, but other explanations are needed to account for those individuals who suffer headaches and migraines from alcoholic beverages other than wine.

9.2 Alcohol and peptic ulcers

It was previously thought that an association existed between alcohol consumption and benign digestive disorders, such as peptic ulcer. However, it is now thought that diet does not have a significant role, as duodenal ulcers are primarily due to a Helicobacter Pylori infection, or intake of non-steroidal inflammatory drugs (Cummings 2000; Yamada et al. 2005). However, alcohol can be a ‘problem food’ for anyone with an ulcer, or at risk of an ulcer, and should be avoided or consumed sparingly. Alcoholic beverages typically increase the acidity of the stomach and therefore can contribute to ulcer development or aggravate symptoms of existing ulcers (NHS Direct 2006).

9.3 Alcohol during pregnancy

The potentially damaging consequences of heavy alcohol intake during pregnancy for the embryo have been discussed in the scientific literature since the early 1970s, with the most widely recognised consequence being fetal alcohol syndrome (FAS). FAS is characterised by reduced birthweight and length, including a small head size and an underdeveloped brain, a variety of congenital abnormalities, e.g. of the heart or joints, and a characteristic facial appearance. Affected children are at a high risk of mental retardation and generally have stunted physical growth. However, FAS is not the only outcome of pre-natal alcohol exposure, and it has been suggested that it can present as a spectrum of disorders. Fetal alcohol spectrum disorder (FASD) is an umbrella term to describe a range of effects that can occur due to the presence of alcohol during the pre-natal stage. FASD is characterised by the presence of some of the criteria for FAS and is associated with lesser degrees of harm from maternal alcohol consumption. It is thought that a number of factors affect the severity of the condition, including the amount of alcohol that the fetus is exposed to, the timing of exposure, patterns of drinking and genetic factors (although FASD is non-hereditary, genes may be an important determinant of the degree of the risk) (Riley & McGee 2005). There is little known about the long-term development of affected children; however, experts in this field suggest that appropriate early care and interventions for children with heavy pre-natal alcohol exposure may improve the mental development of children born with FAS (Spohr et al. 1993; Riley & McGee 2005).

Today, there is a growing body of evidence suggesting that moderate alcohol intake during pregnancy may have an adverse affect on the fetus, and subsequently the infant and child, and binge drinking in early pregnancy may be particularly harmful. For example, alcohol consumption during pregnancy is associated with an increased risk of miscarriage, although it is unclear whether this is related to the quantity consumed or drinking habits in relation to the stage of pregnancy (Royal College of Obstetricians and Gynaecologists 2006). Alcohol intake during pregnancy is also believed to affect fetal growth and development; for example, using prospectively collected data from more than 31,000 pregnancies, Mills et al. (1984) showed that those consuming under one drink each day had an odds ratio of producing a small-for-date newborn of 1.11, compared with an odds ratio of 1.62 for those drinking 1–2 units/day and 1.96 for those drinking 3–5 units/day. However, it is unclear how drinking habits in relation to the stage of pregnancy affect birthweight. One study showed that among women who continued to drink more than 2 units/day during the third trimester, 45% had an infant with a birthweight below the tenth percentile, whereas those who had reduced or discontinued their alcohol consumption during this period had no excess of low-birthweight babies (Rossett et al. 1980).

More recently, there have been a number of studies reporting a possible association between maternal alcohol intake during pregnancy and neurological development of the fetus. For example, Hepper et al. (2005) found that among pregnant mothers consuming an average of 4.2 units/week, spontaneous startles in fetuses were higher, compared with those not exposed to alcohol; thus suggesting that alcohol exerts a delaying effect on the natural decrease in the incidence of spontaneous startles, and as fetal startles have been linked to brainstem function and central nervous system (CNS) development, they may be an important indicator of delayed development of the CNS. Earlier studies have found
other effects of mothers’ alcohol intake during pregnancy on the fetus, such as altered breathing movements (Fox et al. 1978; McLeod et al. 1983), although the significance of these findings is uncertain (Royal College of Obstetricians and Gynaecologists 2006).

Long-term implications, such as aggressive behaviour among children whose mothers drank alcohol during pregnancy, have also been noted (Sood et al. 2001). A long-term prospective study on pre-natal alcohol intake and offspring development observed neurobehavioural effects from the first day of life through to 14 years of age. The stratified cohort sample of more than 1500 women drank on average one drink per day before pregnancy and less than one drink per day during mid-pregnancy. The study found that learning problems were more commonly observed among children aged from 7 to 14 years, but problems with attention and speed of processing information were observed across the 14-year period. The effects were dose-dependent and generally without a threshold (Streissguth et al. 1994). However, other studies considering child developmental outcomes have found no relationship at approximately one drink per day during pregnancy (Jacobson et al. 1998, 1994).

From the research undertaken to date, it is not clear whether the effects of alcohol intake during pregnancy fit a threshold or dose–response model, or may be related to the stage of development of the fetus during exposure. The stage of pregnancy at which the mother consumes alcohol may be particularly important as the brain passes through some developmental phases during which it is more vulnerable to teratogenic agents, such as alcohol (Coles 1994). The pattern of drinking is also thought to be important; for example, alcohol may be less harmful if consumed with a meal, but most harmful when consumed in excess in one session (Plant et al. 1999). Genetic factors affecting maternal or fetal metabolism, as well as interaction with other harmful behaviours, may also be important (Autti-Rämö 2002). Furthermore, differences in outcome may be accounted for by differences in study design and in particular outcome measures. It is therefore an area in which further research is needed.

**Recommendations for intake during pregnancy**

There is currently no consensus of opinion across the world on what is a safe level of alcohol consumption during pregnancy. In the USA, pregnant women have been advised not to drink alcoholic beverages and alcohol (US Surgeon General’s Office 2005). Currently, the Department of Health recommends that women should drink no more than 1–2 units of alcohol, once or twice a week at any stage of pregnancy, and should avoid binge drinking (Department of Health 1995). The Midwives’ Information and Resource Service (2003), supports this advice for pregnant women, and reassures women that light, infrequent drinking constitutes no risk to their baby. Most recently, clinical guidance on antenatal care published in the UK by the National Institute of Clinical Excellence (1997) stated that women should limit their alcohol consumption to no more than one standard unit per day, noting that alcohol has an adverse effect on the fetus.

**9.4 Alcohol, bone mineral density and fracture risk**

A number of studies have considered the effects of alcohol consumption on bone health in terms of bone mineral density (BMD) and risk of fracture. In relation to moderate alcohol intake and BMD, some studies indicate a beneficial effect, while others report no association (Ganry et al. 2000; Tudor-Locke & McColl 2000; Bainbridge et al. 2004) [see Jugdaohsingh et al. (2006) for a recent review]. Evidence for a beneficial effect of moderate alcohol intake on bone health has mainly been observed in studies among post-menopausal women; however, Jugdaohsingh et al. (2006) suggest that this may be primarily because such studies have focused on this subgroup of the population, rather than a specific protective effect in this group. Proposed mechanisms for this positive association include a reduction in bone remodelling via decreases in parathyroid hormone concentrations and an increase in calcitonin production (a hormone involved in the metabolism of bone); the production of endogenous hormones, including oestradiol; the high silicon (which may promote bone formation) content of beer; and the phenolic constituents of alcoholic beverages (Dickerson et al. 2003; Jugdaohsingh et al. 2004).

Although studies are indicating a beneficial effect of moderate alcohol intake on BMD, Jugdaohsingh et al. (2006) report that there are only limited data suggesting a decreased risk of fracture. Indeed, some studies report an increased risk (usually associated with an increase in alcohol intake) predominately; although the data suggest no overall risk (Iich & Kerstetter 2000; Dickerson et al. 2003; Holick & Dawson-Hughes 2004; Jugdaohsingh et al. 2006). The association between high alcohol intake and fracture risk has been put down to an increased risk of falls and other types of trauma (Holick & Dawson-Hughes 2004).

Chronic alcohol abuse has been recognised as a risk factor for low bone density, associated with osteoporo-
Alcohol consumption and our health

sis and an increased risk of fractures (Holick & Dawson-Hughes 2004). Once again, the mechanism for these associations is not clearly understood. However, some potential factors include: poor nutrition and malabsorption of critical nutrients; alterations in the metabolism of vitamin D and calcium; potential toxic effects of excessive alcohol intake on osteoblasts, thus impairing their function; and an increased risk of falls, due to reduced co-ordination (discussed in more details in section 6) (Iich & Kerstetter 2000).

9.5 Alcohol and renal dysfunction

Chronic excessive alcohol intake is recognised to adversely affect the kidneys. However, when considering moderate alcohol intake and renal function (measuring creatinine levels and glomerular filtration rates), the association is less clear. For example, data from the Nurses’ Health Study cohort have suggested no adverse effect of alcohol (with intakes up to 60 g/day) on renal function among women, when compared with non-drinkers (Knight et al. 2003). However, data from the large US male Physicians’ Health Study cohort found an inverse relationship between moderate alcohol consumption (7 drinks/week) and the risk of developing renal dysfunction (Schaeffner et al. 2005). Drinking less than 2 drinks per day also appeared to be protective of end-stage renal disease in a case–control study, while drinking more than 2 drinks per day was associated with an increased risk (Perneger et al. 1999).

Key points

- Some individuals are susceptible to adverse reactions to alcohol, although there is no clear cause; it is not thought that alcohol per se is responsible.
- Although it is well recognised that alcohol is not a cause of peptic ulcers, it may contribute to ulcer development or aggravate symptoms of existing ulcers.
- Excess alcohol consumption during pregnancy can put the fetus at risk of fetal alcohol syndrome, while lesser degrees of harm to the fetus may result in FASD.
- There is also evidence that moderate alcohol consumption during pregnancy may affect the development of the fetus’s CNS. Aggressive behaviour and delayed neurobehavioural development have also been reported among children of mothers who drank in moderation during pregnancy, along with low birthweight. However, there is no consensus regarding the impact of moderate alcohol intake during pregnancy, to the fetus or to the child in later life.

- The most recent advice from an authoritative source in the UK states that women should limit their alcohol consumption to no more than one standard unit per day, noting that alcohol has an adverse effect on the fetus.
- Some studies have reported that moderate alcohol intake is positively associated with BMD, while others have shown no effect. Moderate alcohol intake may have a greater effect on BMD in post-menopausal women.
- It is unclear whether there is any association between alcohol intake and risk of fracture, over and above that due to an increased risk of falls and other types of trauma.
- Evidence indicates no adverse effect of moderate alcohol intake on renal function, although it is less clear whether there is a beneficial effect.

10. Alcohol and social issues

Many of the adverse social consequences related to alcohol are linked to drinking alcohol in excess, through either binge or chronic excessive drinking. It has recently been recognised that binge drinking has become popular among teenagers and young adults, and in particular among men, although women’s drinking has been rising in the past 10 years, and therefore received more attention from the media (Strategy Unit 2004). Binge drinking has been defined as consuming double the daily sensible drinking guidelines (i.e. at least 6 and 8 units per day for women and men, respectively), or in some cases, drinking until inebriation. It is difficult to confirm the long-term health effects of binge drinking, primarily because it is difficult to identify this pattern of drinking among individuals taking part in epidemiological studies. However, data are available on the economic cost of binge drinking to society, and the personal costs are easy to identify. For example, alcohol intoxication induces a range of short-term psychological and psychomotor effects that increase an individual’s risk of injury, or assault, which subsequently involves the use of public services, such as police and ambulance. As outlined at the end of this section, the UK government is taking a population approach to discourage binge drinking, and this is set out in the Alcohol Harm Reduction Strategy (Strategy Unit 2004), along with ways in which to manage and help chronic excessive drinkers.

10.1 Short-term psychological and psychomotor effects of alcohol intake

Consumption of alcohol may result in changes in mood and emotion, and alcohol intoxication may induce a
range of short-term psychological and psychomotor effects. As table 5 indicates, these may result in increased aggressive behaviour, increased risk taking and decreased responsiveness to social expectations. Therefore, for some individuals, in certain situations the effects of alcohol consumption, particularly to intoxication, can lead to personal ‘harm’, including accidents, violence, assault and risky sexual behaviour.

**Acute ill health as a result of drinking excessive amounts of alcohol**

Excess alcohol intake can cause short-term adverse effects to health and well-being, colloquially termed as a ‘hangover’. Although this does not impinge on National Health Service (NHS) resources, work and normal life will be disrupted because of fatigue and increased anxiety (Finnigan et al. 1998, 2005; Wiese et al. 2000; McKinney & Coyle 2004, 2006). The extent to which excess alcohol will affect psychomotor and cognitive function is debatable, following inconsistent results from a number of studies that have explored this topic.

In particular, aviation research has examined the performance of pilots in flight simulators, and found increased errors on some tasks after consumption of alcohol the night before (Morrow et al. 1990; Taylor et al. 1994). However, other studies that have measured vigilance, dual tasks, including reaction times and probed memory tasks, found no significant differences between those who had consumed excess alcohol the night before and those who had not (Lemon et al. 1993; Finnigan et al. 1998, 2005). Studies considering executive- or managerial-type cognitive functions after drinking the night before, have also been inconsistent (Streufert et al. 1995; Verster et al. 2003; Weissenborn & Duka 2003). This may be a result of differences between studies, in terms of the alcohol dosage, test method and specific tests of performance. However, studies have consistently found that participants feel tired and anxious after excess alcohol consumption the night before, and this could account for poor performance and absenteeism in the workplace, costing in the region of £6.4 billion per year in the UK (including absenteeism from alcohol-related injuries) (Strategy Unit 2003).

Although some research has investigated the implications of excess alcohol consumption on the body, little is known about the physiology underlying the conditions (Swift & Davidson 1998; Slutske et al. 2003). To date, hangover symptoms have been attributed to several factors, including the direct effects of alcohol on the body (see table 6), the effects of alcohol metabolites and other ingredients of alcoholic drinks (such as congeners, see section 6), and the effects of alcohol withdrawal, which result from compensatory changes in the CNS that take place in response to heavy consumption of alcohol (Swift & Davidson 1998).

There is no consensus definition of a hangover and few published estimates of the incidence of hangovers. A

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**Table 5 Effects of specific quantities of alcohol on blood ethanol concentrations and mental function**

<table>
<thead>
<tr>
<th>Number of drinks</th>
<th>Blood alcohol level</th>
<th>Possible effects (mg/100 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 pint of beer</td>
<td>30</td>
<td>Increased likelihood of having an accident</td>
</tr>
<tr>
<td>2 glasses (125 ml) of wine</td>
<td>50</td>
<td>Increased cheerfulness, impaired judgement and loosening of inhibitions</td>
</tr>
<tr>
<td>1 double measure whisky</td>
<td>80</td>
<td>Loss of driving licence if caught</td>
</tr>
<tr>
<td>1.5 pints of beer</td>
<td>100</td>
<td>Loss of self-control, exuberance, quarrelsomeness, slurred speech</td>
</tr>
<tr>
<td>3 whiskies</td>
<td>200</td>
<td>Stagger, double vision, loss of memory</td>
</tr>
<tr>
<td>Half a (75 cL) bottle of wine</td>
<td>400</td>
<td>Sleepiness, oblivion, coma</td>
</tr>
<tr>
<td>2 pints of beer</td>
<td>500+</td>
<td>Death</td>
</tr>
</tbody>
</table>

Adapted from: Royal College of Physicians (1987).
review undertaken by Wiese et al. (2000) identified common symptoms of hangover as: headache; poor sense of overall well-being; diarrhoea; anorexia; tremulousness; fatigue; and nausea; and defined a hangover as at least two of these symptoms, occurring after the consumption and full metabolism of alcohol. Increased sympathetic nervous system activity, including increased systolic blood pressure, rapid heartbeat, tremor and sweating, may also accompany a hangover (Finnigan et al. 2005).

Swift and Davidson (1998) report that hangover symptoms may be influenced by the type of alcoholic beverage consumed and the amount drunk, and symptoms usually peak when the blood alcohol concentration returns to zero and may continue for up to 24 hours after that. Overall, the greater the amount and duration of alcohol consumption, the more likely it is that several symptoms will occur. However, results from studies considering this relationship vary and, therefore, it is difficult to reach a consensus.

Injury as a result of drinking excessive amounts of alcohol

According to research commissioned by the Strategy Unit (2003), on any one night 41% of attendees at accident and emergency (A&E) departments tested positive for alcohol consumption, and 14% were intoxicated. Furthermore, between midnight and 5 am, 70% of attendances were alcohol related. Thus, the consumption of alcohol places a significant burden on A&E services. Violent assaults, road traffic accidents, psychiatric emergencies or deliberate self-harm episodes are all commonly identified reasons for attendance among those who have consumed alcohol (Strategy Unit 2003). The interim analytical report from the Strategy Unit (2003) suggests that about £0.5 billion of A&E costs are alcohol related; however, this does not take account of follow-up treatment that individuals may require or hospital admissions, and therefore is only a small proportion of true costs. A number of these cases are a result of anti-social behaviour, public disorder and violence, thus creating work for the police, as well as the NHS staff.

10.2 Crime and public disorder as a result of drinking alcohol

Richardson and Budd (2003) reported that more than half of alcohol-related violence between strangers and acquaintances occurs in or around pubs, clubs or discos. It is not possible to identify/record the causes of such events, but it is recognised that impaired cognitive skills may lead to intoxicated individuals misreading social cues and responding inappropriately to social situations with ill-treatment of others. The situation may be made worse as consumption of alcohol results in an increased probability of aggressive behaviour. Room et al. (2005) report that this is due to altered messaging through brain receptors and neurotransmitters, which may also result in impaired motor skills, causing a reduction in co-ordination, thus resulting in individuals knocking into other people or causing victims to be less able to defend themselves (Sumner & Parker 1995; Strategy Unit 2003).
In particular, excessive drinking is associated with an increased risk of partner violence; approximately a third of incidents of partner violence were committed when the perpetrator had been drinking alcohol (Strategy Unit 2003). Partner violence is frequently linked to chronic alcohol misuse; for example, rates of alcohol abuse and alcohol dependence among perpetrators may be 2–7 times higher than in the general population (Strategy Unit 2003).

Surveys suggest that alcohol is a factor in more than 30% of city centre arrests. Detainees under the influence of alcohol also cause problems for custody officers in terms of noise, hygiene and disruptive behaviour, and therefore need close supervision. This is reflected in the increase in costs of £59 to process an alcohol-related arrestee (as at 2003), compared with an arrestee who has not been drinking alcohol (Maguire & Nettleton 2003; Strategy Unit 2003), thus emphasising the economic burden of binge drinking on public services. The overall costs of alcohol-related crime are significant; Leontaridi (2003) has estimated that £1.7 billion is spent on alcohol-related offences and a further £30 million is spent on alcohol-specific offences, taking account only of costs incurred by the police, the Crown Prosecution Service, Magistrates and Crown courts, legal costs and costs to the prison and probation services.

Particular crimes commonly related to drinking in excess include drink driving and sexual assaults. Although drink driving has been declining, along with the number of casualties from road accidents involving alcohol, 5% of all road accidents in 2000 and 17% of deaths were associated with drivers over the legal alcohol limit. An estimated 85 000 cases of drink driving each year cost crime-related public services £0.5 billion a year (including legal costs) (Strategy Unit 2003). Fewer cases of alcohol-related sexual assaults are brought to the attention of the police, than actually take place (19 000 cases in 2003). Intoxicated victims are less likely to report their victimisation, and a large number of victims of sexual attacks are unreported, making the true burden unclear. Experience suggests that offender drinking increases the likelihood and severity of victim injury, and reported alcohol-related sexual assaults are more likely to occur between people who do not know each other well, when compared with other sexual assaults.

10.3 Alcohol-related harms and other public health issues

Excessive alcohol consumption is also linked to other public health issues, e.g. sexually transmitted diseases and unplanned pregnancies, as consumption of alcohol is recognised to increase the likelihood of unsafe sex (Cooper 2002; Rassool & Winnington 2003). This could be because consuming too much alcohol makes it more difficult to make judgements and assess risks; it lowers inhibitions and reduces the sense of self-control. Furthermore, alcohol has also been found to increase perceived attractiveness of the opposite gender and is also associated with contexts in which people are more likely to meet potential sexual partners, such as parties, pubs and clubs, thus increasing the chance of a sexual encounter (Alcohol Concern & Drugscope 2002; Jones et al. 2003; Strategy Unit 2003; Paton 2005).

Alcohol consumption can also be linked to another prominent public health concern, namely obesity. Obesity is the result of energy intake being higher than energy expenditure for an extended period of time. As noted in section 6, alcohol is the second most energy-dense nutrient and, in most cases, the energy derived from alcohol is consumed in addition to energy from food. Therefore, it could be considered that moderate to heavy alcohol consumption may increase the risk of obesity. This is discussed in more detail in section 8.

10.4 The economics of alcohol consumption

The total cost of alcohol-related harm was calculated by the Strategy Unit (2003) at £20 billion per annum. However, these costs (and all of the costs considered in this section) are related to the consumption of alcohol in excess (i.e. through binge drinking and chronic excessive drinking). The value of the alcoholic drinks market is more than £30 billion per annum (Strategy Unit 2003), and it is estimated that in the UK, one million jobs are linked to the production and sale of alcohol (ONS 2001). Money is also generated from taxes on alcohol purchases; however, with £20 billion per annum being spent on dealing with alcohol-related harm, and more money being spent on prevention efforts, the net profit for the economy is debatable.

10.5 Population-based initiatives underway to reduce the incidence of binge drinking

As outlined in section 4, the phenomenon of binge drinking is not just affecting the UK; all over Europe, the younger population are drinking more. Like the UK, other EU governments have undertaken education campaigns to warn people of the risks of drinking in excess to their personal safety. For example (see figure 7), in The Netherlands, a confrontational image of an unconscious, partially dressed young girl has been used to
remind girls of the potential consequences of drinking too much (Sheldon 2006).

A similar type of advertising campaign has been used in the UK: a cinema advertisement highlighting the ‘Jekyll and Hyde’ effect that binge drinking can have on women who drink to get drunk was shown in cinemas across the UK in 2004 (Portman Group 2006). The public health White Paper (Department of Health 2004b) made a commitment to work with the Portman Group (http://www.portmangroup.org.uk) to develop a new information campaign with the aim of cutting down binge drinking. It is difficult to evaluate the effectiveness of these types of campaigns, but it is suggested that they have generally been found to be ineffective (World Health Organization 2001; Room et al. 2005). The White Paper also committed to support action by Office of Communications (Ofcom) to strengthen the rules of broadcasting, particularly to protect under-18s, based on the idea that advertising has some influence on young viewers’ attitudes to drinking (DCMS 2006). Since January 2005, new rules forbid alcohol commercials that have a strong appeal to under-18s, and prevent adverts for alcohol that include even subtle links between sex and drinking (or a brand of alcohol), or drinking and daring, aggressive, irresponsible or anti-social behaviour (DCMS 2006).

Other government-led initiatives to reduce the adverse impact that alcohol can have on society include tougher enforcement measures by the Police and Trading Standards against irresponsible drinkers and vendors who are fuelling anti-social behaviour, including issuing £40 fixed penalty notices for being drunk and disorderly, and naming and shaming off-licences, bars and clubs after conviction of selling to underage drinkers. Such measures were tested for 8 weeks over the 2005 festive season as part of a wider Alcohol Harm Reduction Strategy for England. It was thought that such measures would kick-start a culture change where it will be less acceptable to get drunk and behave in an anti-social or violent manner (Department of Health 2006a). It is hoped that the option to sell alcohol 24-hours-a-day will also make changes to the drinking culture in England and Wales. New licensing laws came into force in November 2005, superseding those from 1964, with the aim of bringing Britain more in line with other European countries, to allow for more responsible drinking and to avoid the rush to make last orders at the bar. It is hoped that the variation in closing times of establishments in the same area will also avoid confrontation flashpoints, and therefore prevent crime and disorder and improve public safety (DCMS 2006).

As outlined by the World Health Organization (2004), the prevention of alcohol-related problems requires a comprehensive approach, combining preventative policies at a national and local level. Other activities and initiatives to reduce the incidence of harm caused by excess consumption of alcohol are outlined in the Alcohol Harm Reduction Strategy for England (Strategy Unit 2004), including local initiatives, education to children, and liaison and co-operation with the drinks industry.
Key points

- Alcohol intoxication, such as through binge drinking, may induce a range of short-term psychological and psychomotor effects that may cause increased aggressive behaviour, increased risk taking and decreased responsiveness to social expectations, and may lead to personal ‘harm’, including accidents, violence, assault and risky sexual behaviour. Consequently, binge drinking places a significant burden on A&E services and the police.
- Excess alcohol consumption may also result in a ‘hangover’ that disrupts normal life, due to symptoms of fatigue and increased anxiety, and has economic consequences in the workplace through poor performance or absenteeism. The true extent to which a hangover can affect an individual’s performance, including psychomotor, recognition and managerial skills, is still being debated.
- Common symptoms of a hangover are: headache; poor sense of overall well-being; diarrhoea; anorexia; tremors; fatigue; and nausea. Hangover symptoms have been attributed to several factors, including: dehydration and electrolyte imbalance; gastrointestinal disturbances; low blood glucose; and disruption to normal biological rhythms.
- Binge drinking is also associated with an increased risk of public health issues, including sexually transmitted diseases and unplanned pregnancies.
- Although the cost of binge drinking in terms of long-term health has yet to be established, the significant economic cost is well recognised.
- A number of population-based initiatives are underway to try and reduce the incidence of binge drinking. These include: advertising campaigns; changes to alcohol advertising regulations; and changes to pub licensing regulations.

11. Conclusions

Alcohol has been produced for thousands of years, via the anaerobic fermentation of sugars by yeast, and involves glucose molecules ($C_6H_{12}O_6$) being broken down to yield ethanol ($C_2H_5OH$), carbon dioxide ($CO_2$) and energy. Alcoholic beverages primarily consist of alcohol and carbohydrate, and offer few other nutrients; yet per gram, alcohol is the second highest source of energy of all the macronutrients, therefore alcoholic drinks make a notable contribution to energy intakes. Fortunately though, when consumed in moderation, alcohol does not seem to interfere with the absorption and metabolism of nutrients and, therefore, in moderation can be part of a healthy, balanced diet. This is particularly important, as more than 90% of adults in Britain drink alcohol (Strategy Unit 2004).

For the majority of people who drink alcohol, it is associated with pleasure, socialising and relaxing (MORI 2000). When consumed in moderate amounts, it is an accepted element of a healthy diet and lifestyle, with few adverse effects to nutritional health or risk of disease, and possibly some benefits. Guidelines for sensible drinking have been set by the UK government, and take account of the risks to health and society. The guidelines are 3–4 units (24–32 g) a day or less for men, and 2–3 units (16–24 g) a day or less for women, which are fairly similar to those of other countries.

The UK has an overall alcohol intake close to that of the European average. Half of the adult UK population are classified as low to moderate drinkers. Despite high and very high drinkers being a minority, evidence from the GHS suggests that this figure is growing. This is of concern to public health professionals, as alcohol poses a considerable threat to health if consumed in excess. Unfortunately, it is all too easy to consume in excess; humans lack biological curbs on alcohol intake, and the biological effects of alcohol consumption increasingly impair the capacity for judgement about how much we should consume. Furthermore, the effects of excess consumption (i.e. the hangover) are too far removed in time from the triggering behaviour, to act as conditioning. Taking these factors into account, it is not surprising that many people drink until they ‘get drunk’, or binge drink. Binge drinking is a common phenomenon in the UK, particularly among younger people, whereby violence and accidents pose a greater threat to health and well-being, and are the main cause of alcohol-related mortality. The ‘older’ binge drinking population are at a higher risk of cardiovascular death, such as a stroke or sudden cardiac death.

Individuals who do not necessarily binge drink but drink more than the government-recommended limits on a daily basis (i.e. more than 3 drinks a day for men, and 2 drinks a day for women) are also putting themselves at an increased risk of chronic conditions, such as CVD, high blood pressure, diabetes and weight gain. Alternatively, light to moderate alcohol consumption (i.e. consuming alcohol in line with the recommendations) can reduce the risk of these diseases, with the most benefits being seen in men aged over 55 years and post-menopausal women. However, there is an inverse association between alcohol intake and risk of cancer, and for some cancers, such as breast cancer, just one drink a day can increase an individual’s risk by about 7%. For younger people, the benefits of alcohol may be outweighed by the increased risk of other diseases (e.g. © 2006 British Nutrition Foundation Nutrition Bulletin, 31, 286–331
alcohol-related cancers, liver cirrhosis) and increased risk of violence and accidents.

Looking at alcohol consumption more holistically, it provides a focus for social gatherings and relaxation, particularly for young adults, and therefore assists in integration and social development. However, when consumed in excess, it can accentuate emotions and may result in aggressive behaviour, which poses another risk. Given the current levels of intake and, in particular, the rise in binge drinking, action is required to encourage a shift in the drinking culture, to discourage drinking in excess. Targeted publicity of the potential harms of excess drinking is already underway, although far-reaching dissemination is essential to ensure that this is effective.

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References


