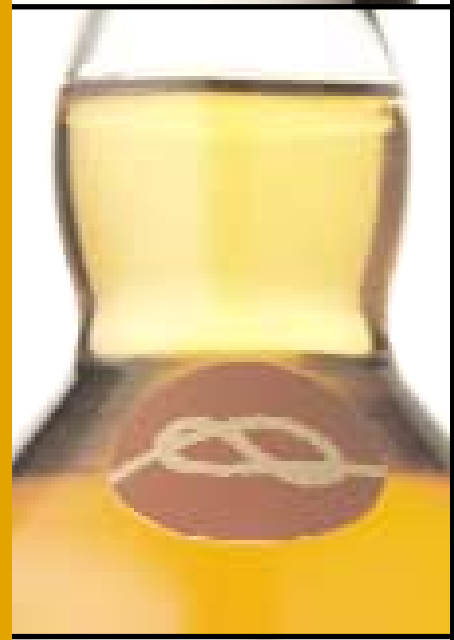
AUSTRALIAN ALCOHOL GUIDELINES

Health Risks and Benefits



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National Health and
Medical Research Council

NHMRC
National Health &
Medical Research Council

NUMBER OF STANDARD DRINKS

1.5

375ml
Full Strength Beer
4.9% Alc./Vol

1

375ml
Mid Strength Beer
3.5% Alc./Vol

0.8

375ml
Light Beer
2.7% Alc./Vol

1.5

375ml
Full Strength Beer
4.9% Alc./Vol

1

375ml
Mid Strength Beer
3.5% Alc./Vol

0.8

375ml
Light Beer
2.7% Alc./Vol

1

285ml Middy*
Full Strength Beer
4.9% Alc./Vol

0.7

285ml Middy*
Mid Strength Beer
3.5% Alc./Vol

0.5

285ml Middy*
Light Beer
2.7% Alc./Vol

1.5

375ml
Pre-mix Spirits
5% Alc./Vol

1.5

340ml
Alcoholic Soda
5.5% Alc./Vol

1

30ml
Spirit Nip
40% Alc./Vol

24

750ml
Bottle of Spirits
40% Alc./Vol

1

100ml
Standard Serve
of Wine
12% Alc./Vol

1.8

180ml
Average Restaurant
Serve of Wine
12% Alc./Vol

7

750ml
Bottle of Wine
12% Alc./Vol

36

4 Litres
Cask Wine
12% Alc./Vol

* NSW, WA, ACT = Middy; VIC, QLD, TAS = Pot; NT = Handle; SA = Schooner

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- fostering and supporting a high quality and internationally recognised research base;
- providing evidence based advice;
- applying research evidence to health issues thus translating research into better health practice and outcomes; and
- promoting informed debate on health and medical research, health ethics and related issues.

NHMRC web address: http://www.nhmrc.gov.au

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PART ONE:

DRINKING GUIDELINES

INTRODUCTION

Alcohol is widely used and enjoyed throughout our society, and for many people it forms part of an enjoyable and generally healthy lifestyle that includes good diet and exercise. Recent evidence has confirmed that, at low levels, alcohol has health benefits for some people, particularly in contributing to reducing the risk of heart disease from middle age onwards. It also, however, has the potential to cause much harm, and is second only to tobacco as a cause of drug-related deaths and hospitalisations, causing almost 3,300 deaths and 50,000 hospital episodes in 1997. People who drink regularly at higher levels place themselves at increased risk of chronic ill health and premature death, while an episode of heavy drinking places the drinker and others at risk of injury and death. These patterns of drinking also have substantial social and economic implications, not only for individuals, but also for families, workplaces, and society as a whole.

This guidelines document has been written to provide Australians with knowledge and understanding that will enable them to enjoy alcohol, if they choose to drink, while avoiding or minimising harmful consequences. In its current form, the document is intended primarily for professional groups, educators, and industry and policy makers. While this document may be of interest to people wanting to make decisions about their own drinking, it is not specifically aimed at the general public.

The guidelines themselves are set out in Part One of this document, and are divided into two sections:

- Guidelines for the whole population
- Guidelines for particular groups

Accompanying each guideline is a **Rationale**, providing a brief statement of the scientific basis for the guideline, and a **Comment** that adds to, qualifies and/or provides context for the guideline. Part Two of the document sets out in more detail the context and evidence base for the guidelines, and provides information that will help in applying the guidelines.

The guidelines are based on an **Australian Standard Drink**, which contains 10 grams (equivalent to 12.5 millilitres) of alcohol. All alcoholic beverages, by law, state on the label the number of standard drinks in the container. Figure 1 illustrates the number of standard drinks found in typical serving containers (although glasses, particularly for wine, are often much larger than those shown):

Figure 1: Typical standard drinks—a guide.
This figure appears on the inside back cover.

The guidelines set levels for *low risk* drinking. The following sections of Table 1 indicate low risk drinking levels for the general population (as set out in Guideline 1), and also indicate levels that are considered to be *risky*, and *high risk*, for men and

women of average or larger body size (ie over 160 cm in height and 50 kg in weight). The levels set in the tables may be too high for people of below-average body size.

Low risk levels define a level of drinking at which there is only a minimal risk of harm and, for some, the likelihood of health benefits. *Risky* levels are those at which risk of harm is significantly increased beyond any possible benefits. *High risk* drinking levels are those at which there is substantial risk of serious harm, and above which risk continues to increase rapidly.

Furthermore, the guidelines and Table 1 distinguish:

- *long-term risk*—the level of long-term risk associated with regular daily patterns of drinking, defined by the total amount of alcohol typically consumed per week; and
- *short-term risk*—the risk of harm (particularly injury or death) in the short-term that is associated with given levels of drinking on a single day. These levels assume that overall drinking patterns remain within the levels set for long-term risk, and that these heavier drinking days occur infrequently and never more than three times per week. Outside these limits, risk is further increased.

Table 1: Summary of guidelines for low risk drinking

1. Alcohol consumption at levels shown below is **not** recommended for people who:
   - have a condition made worse by drinking (see p.9)
   - are on medication (see p.12)
   - are under 18 years of age (see p.15)
   - are pregnant (see p.16)
   - are about to engage in activities involving risk or a degree of skill (eg driving, flying, water sports, ski-ing, operating machinery).

2. Otherwise risk levels for the following patterns of drinking are as follows*:

For risk of harm in the short-term:

	Low risk (standard drinks)	Risky (standard drinks)	High risk (standard drinks)
MALES On any one day	up to 6 *on any one day, no more than 3 days per week*	7 to 10 *on any one day*	11 or more *on any one day*
FEMALES On any one day	up to 4 *on any one day, no more than 3 days per week*	5 to 6 *on any one day*	7 or more *on any one day*

For risk of harm in the long-term:

	Low risk (standard drinks)	Risky (standard drinks)	High risk (standard drinks)
MALES			
On an average day	up to 4 *per day*	5 to 6 *per day*	7 or more *per day*
Overall weekly level	up to 28 *per week*	29 to 42 *per week*	43 or more *per week*
FEMALES			
On an average day	up to 2 *per day*	3 to 4 *per day*	5 or more *per day*
Overall weekly level	up to 14 *per week*	15 to 28 *per week*	29 or more *per week*

* **Note**:

1. It is assumed that the drinks are consumed at a moderate rate to minimise intoxication, eg for men no more than 2 drinks in the first hour and 1 per hour thereafter, and for women, no more than 1 drink per hour.
2. These guidelines apply to **persons of average or larger size**, ie above about 60 kg for men and 50kg for women. Persons of smaller than average body size should drink within lower levels.

(Table based on *International Guide for Monitoring Alcohol Consumption and Related Harm*, WHO, Geneva, 2000)

Guidelines for the whole population

GUIDELINE 1

To minimise risks in the short and longer term, and gain any longer-term benefits

For men

1.1 an *average* of no more than 4 standard drinks a day, and no more than 28 standard drinks over a week;

1.2 not more than 6 standard drinks in any one day;

1.3 one or two alcohol-free days per week.

For women

1.4 an *average* of no more than 2 standard drinks a day, and no more than 14 standard drinks over a week;

1.5 not more than 4 standard drinks in any one day;

1.6 one or two alcohol-free days per week.

Note:

These drinks should be spread over several hours. It is assumed that the drinks are consumed at a moderate rate to minimise intoxication, eg for men no more than 2 drinks in the first hour and 1 per hour thereafter, and for women, no more than 1 drink per hour.

This guideline assumes that the drinker is not on medication, does not have a family history of alcohol-related problems or a condition that is made worse by drinking, is not pregnant and, is not about to undertake any activity involving risk or a degree of skill, including driving, flying, water sports, ski-ing, or using complex or heavy machinery or farm machinery, etc.

Rationale:

Drinking within the average drinking levels set in guidelines 1.1 for men and 1.4 for women minimises the longer-term risks of ill health and death related to alcohol, and maximises the potential longer-term health benefits. The levels are based on strong evidence, which continues to accumulate from many studies, on overall death rates in relation to different levels of drinking. The evidence takes into account the health risks and benefits of alcohol. It relates to both average levels of drinking over time, and the harm caused by occasional heavy drinking days (above 6 standard drinks for men or 4 for women).

Studies from several countries, including Australia, show that drinking above the daily limits set in guidelines 1.2 for men and 1.5 for women significantly increases the short-term risk of health and social problems, including risk of injury or death from accident, assault and self-harm.

A given amount of alcohol usually affects women more than men because of differences in the way their bodies process alcohol.

The evidence base for Guideline 1 is discussed on page 34. See also pages 23, 39, 41, 45 and 65.

Comment:

The levels set in this guideline may be too high for people of below-average body size (ie below about 60kg for men and 50kg for women).

The weekly levels of drinking set in guidelines 1.1 and 1.4 allow for some variation from day to day. Guidelines 1.2 and 1.5 set limits to this variation. While they specifically target the short-term risk from intoxication, they also contribute to reducing the long-term risk of harm.

The safety of drinking depends not only on how much a person drinks, but also on the rate of drinking, the environment, and what the person is doing during and after drinking. Eating while drinking helps to reduce intoxication and, therefore, the risk. The risk of injury or violence, depression and suicide attempt are all influenced by the setting in which people are drinking (see Guideline 3).

ALCOHOL-FREE DAYS—A Prudent Approach

Comment: While the evidence for alcohol-free days as a means of reducing health risk is limited, regular alcohol-free days may help drinkers to remain in control of their drinking and reduce its habit-forming potential. This is especially important for people drinking above the guideline limits. Research shows that to become dependent on something, a person must use it regularly. There is evidence that everyone who drinks over time, even at low levels, acquires some physiological tolerance to alcohol, but this occurs less in people who drink only intermittently, and they are also less likely to experience withdrawal symptoms. A mild degree of dependence is common in the Australian population. One early sign of this, for example, is finding it difficult to stop drinking after two or three glasses.

Dependence is further discussed on page 75

GUIDELINE 2

When undertaking activities that involve risk or a degree of skill

2.1 to avoid the risk of harm to the drinker and others, do not drink alcohol before or during such activities.

Rationale:

Very low levels of alcohol can affect judgement and performance, and even a very small effect may be relevant where a high degree of skill is needed, where the risk is already high, or where the safety of others is involved.

The evidence base for Guideline 2 is discussed on pages 37, 39, 46–49 and 65–67.

Comment:

Situations where this guideline is relevant include recreational and occupational activities such as flying, water sports, ski-ing, using complex or heavy machinery or farm machinery, and driving.

In occupational settings such as flying commercial aircraft, driving public or heavy vehicles, or operating commercial vessels, machinery, mobile plant or farm equipment, blood alcohol levels are stipulated by legislation and/or company policy (eg no alcohol within 24 hours of flying).

While State and Territory laws allow a blood alcohol concentration (BAC) of up to 0.05 while driving, for full licence holders, there is evidence of impairment at lower blood alcohol levels, and research shows that crash risk is increased 5-fold at 0.05. To meet legal BAC limits, learner drivers and provisional licence holders need to avoid drinking for several hours before driving.

The more alcohol that is consumed, the longer it takes for BAC to return to zero. It generally takes about one hour for one standard drink to pass through the bloodstream, but after a heavy drinking session, BAC may still be over 0.05 the next morning. Furthermore, even after BAC returns to zero, evidence shows that judgement and performance may continue to be impaired for a time, from the continuing effects of hangover.

While it varies significantly from person to person, BAC will, in general, remain below 0.05 if a man of average size drinks no more than two drinks in the first hour and one per hour thereafter, and if a woman of average size drinks no more than one standard drink per hour.

GUIDELINE 3

When responsible for private and public drinking environments

3.1 actively promote responsible drinking;

3.2 strive to make sure that those being served alcohol do not become intoxicated, and suggest alternatives to alcohol;

3.3 refuse to serve alcohol to people who are intoxicated;

3.4 minimise the potential for harm in the setting;

3.5 closely supervise or monitor young people.

Rationale:

There is good evidence that both licensed and private drinking settings can influence the risk of various forms of short-term harm associated with drinking.

The evidence base for Guideline 3 is discussed on page 43. See also pages 23, 46–49, 61 and 65.

Comment:

This guideline applies to any environment where alcohol is served, including private homes and parties, workplaces (for social functions), and any public setting (hotels and bars, clubs, public events, planes etc).

Effective strategies to reduce harm include providing non-alcoholic drinks, making food available, providing safe transport, and staff training. Some of these are legal requirements in licensed premises under State and Territory laws.

Guidelines for particular groups

GUIDELINE 4

People with a health or social problem that is related to alcohol, or made worse by alcohol (including alcohol dependence)

4.1 should consider not drinking at all;

4.2 are strongly advised to stop drinking for at least several weeks or months;

4.3 might then try drinking at low levels (substantially below Guideline 1)* under professional supervision;

4.4 should not drink if they have developed severe alcohol dependence;

4.5 should never drink if they have a severe health problem made worse by alcohol (eg cirrhosis, pancreatitis);

4.6 if they have hepatitis C or other forms of chronic viral hepatitis, should consider drinking only infrequently and well below the levels recommended in Guideline 1.

* The appropriate level will vary from person to person.

Rationale:

A goal of 'controlled drinking' within the guideline levels is feasible for some people who have a problem with drinking, especially if they have social supports and if they tackle the problem early, before they become severely dependent.

People who have significant alcohol dependence or major organ damage (eg damage to liver or pancreas) will incur further harm if they continue to drink.

People with chronic hepatitis C (or other forms of chronic hepatitis infection) who drink heavily have poorer health outcomes than those who drink less. There are few data, however, to suggest a level of consumption associated with acceptably low risk. Guideline 4.6 reflects advice currently given to these people in Australia.

The evidence base for Guideline 4 is discussed on pages 75 and 76. See also page 41.

Comment:

A range of health and social problems may be associated with alcohol. They include domestic violence, unsafe sex, financial and relationship problems, and physical conditions such as pancreatitis, high blood pressure, and gastrointestinal problems. These problems may or may not involve dependence.

Dependence on alcohol exists in varying degrees. It may be associated with withdrawal symptoms, both physical and psychological (eg anxiety, tremors), when drinking stops. The person may have difficulty in limiting drinking on any one occasion. For some, especially those with severe dependence, total abstinence will be the only realistic goal.

A number of professional, medical and voluntary agencies can help people who have problems with their drinking. Most States and Territories have an alcohol and drug telephone helpline, which can provide information on specialist services available (see Appendix 6).

It is crucial that the impact of such problems on families and partners be recognised, and their needs supported. At the same time, families offer a key resource in helping people with a drinking-related problem, and are often the first to acknowledge such problems.

GUIDELINE 5

People with a relative* who has, or has had, a problem with alcohol

5.1 are advised to be careful about how much they drink;

5.2 should take particular care to have regular alcohol-free days (one or two days per week);

5.3 might consider not drinking at all.

* first-degree relatives (parents, siblings) or second-degree relatives (grandparents, uncles, aunts, cousins).

Rationale:

People with a family history of alcohol-related problems, including alcohol dependence, are more at risk than the general population of being unable to control their level of drinking.

Alcohol-free days are particularly relevant for this group, as the nature of dependence suggests strongly that alcohol-free days help people to avoid alcohol-related problems by breaking the drinking pattern. (See Guideline 1)

The evidence base for Guideline 5 is discussed on pages 41 and 76.

Comment:

The degree of risk is related to both the closeness of the relatives who have an alcohol-related problem (first degree or second degree relatives) and the number of relatives involved.

GUIDELINE 6

People with a mental health problem (including anxiety or depression) and/or sleep disturbance

6.1 if they do drink, should take particular care to stay within the levels set in Guideline 1, and should consult with their doctor or pharmacist about possible side-effects;

6.2 may need to consider not drinking at all, if they find it difficult to keep their drinking within these guideline levels;

6.3 may need to stop drinking entirely if symptoms persist.

Rationale:

There is clear evidence that drinking above the levels set in Guideline 1, and particularly at high risk levels (see page 5), leads to poorer outcomes for people who have a mental health problem. In particular, people who are depressed and sometimes drink excessively are at much greater risk of self-harm and suicide, especially if also they drink regularly above guideline levels. There is also some evidence that alcohol use is associated with poorer outcomes for people suffering from schizophrenia.

Heavy drinking can also aggravate symptoms in people with milder degrees of anxiety and depression. While alcohol consumption may bring some relief from anxiety or stress in the short-term, it can worsen mood in the longer term, especially at higher levels of consumption.

It is not possible, based on current evidence, to define precise drinking levels above which these effects occur.

With some mental health problems, the risk of alcohol dependence is significantly increased, particularly where the person drinks to relieve anxiety or change their mood.

Most of the medications used for mental health problems interact with alcohol.

Alcohol can also disrupt sleep. Even one or two drinks may result in reduced sleep quality and early morning waking.

The evidence base for Guideline 6 is discussed on pages 38 and 50.

Comment:

Anyone with a mental health problem should discuss their alcohol intake with their doctor.

Carers can encourage people with a mental health problem to stay within guideline levels, or to abstain if necessary.

GUIDELINE 7

People taking medications or other drugs

7.1 should carefully read the labels and pamphlets with their medications (including herbal preparations), to check for harmful interactions with alcohol. Some people may need to reduce their drinking or stop drinking alcohol altogether;

7.2 are advised to be very cautious if drinking alcohol while using benzodiazepines, heroin, methadone or other central nervous system depressants;

7.3 if they are taking a number of medications, are at greater risk of increasing the effects of alcohol and/or decreasing the effectiveness of their medication. These people may need to reduce or stop drinking alcohol;

7.4 should consult their doctor or a pharmacist to discuss any aspect of their medication, including possible interactions with alcohol.

Rationale:

Alcohol can interact with many prescribed and over-the-counter medications, and this can alter the effect of alcohol and/or the medication. Even at low levels, drinking can cause problems, depending on the medications. Response also varies from person to person. Some interactions have serious implications for driving or operating machinery.

Alcohol dampens activity in the brain, and it can be particularly dangerous, or even lethal, when used with medications or other drugs, legal or illegal, that have similar effects (ie that depress the central nervous system).

The evidence base for Guideline 7 is discussed on pages 55–56.

Comment:

Health care providers should be aware of the possibility of interactions with alcohol, both with the medications they prescribe and with other medications people may take. Particular care is needed with medications for mental health problems and for blood pressure.

Some herbal preparations also interact with alcohol.

Many people take benzodiazepines safely under medical supervision. When combined with alcohol, however, benzodiazepines pose similar risks to other drugs, legal and illegal, that depress brain activity (notably heroin).

For up-to-date information on medications that may interact with alcohol, see the MIMS website, <http://www.mims.hcn.net.au>

GUIDELINE 8

Older people

8.1 are advised, if they drink, to consider drinking less than the levels set in Guideline 1.

Rationale:

A given amount of alcohol tends to produce a higher blood alcohol level in an older person because, with age, the body's total water content decreases.

At the same time, the risk of falling increases with older age. Driving may also be affected. Alcohol can increase the risks in both these areas.

In addition, many older people take medications that may interact with alcohol.

The evidence base for Guideline 8 is discussed on page 49. See also pages 45, 65 and 69.

Comment:

People age at different rates, and older people who drink need to reassess their drinking regularly. While alcohol has psychological, social and health benefits for many older people, the body's tolerance for alcohol decreases with age.

Some older people may need to reduce their drinking or stop it altogether to avoid harmful interactions with medications.

The potential benefits of alcohol in preventing heart disease can be achieved with as little as one to two standard drinks per day for men, and less than one per day for women. Similar benefits can be gained from strategies such as regular exercise, giving up smoking, and a healthy diet.

Guideline 9

Young adults (aged about 18–25 years)*

9.1 are especially urged not to drink beyond the levels set in Guideline 1;

9.2 should not drink at all for at least several hours before undertaking potentially risky activities (eg driving, swimming, boating);

9.3 should not mix alcohol with other mood altering drugs.

* While this guideline applies to people aged about 18–25 years, the issues and concerns overlap with those covered under Guideline 10, 'Young People'.

Rationale:

While many young adults drink at low risk levels, statistically this is the group that is most likely to be harmed by alcohol. Young adults have the highest alcohol consumption in Australia and are the group at highest risk in relation to alcohol-related injury, including road trauma, violence, sexual coercion, falls, accidental death (including drowning), and suicide. Younger, less experienced drinkers are at even higher risk due to their lower alcohol tolerance.

The evidence base for Guideline 9 is discussed on page 46. See also pages 37–38 and 61.

Comment:

To meet legal requirements in all States and Territories, both learner drivers and provisional licence holders must avoid alcohol in the hours before driving.

The more alcohol that is consumed, the longer it takes for blood alcohol level to return to zero. After a heavy drinking session, blood alcohol concentration (BAC) may still be over 0.05 the next morning. Furthermore, even after BAC returns to zero, evidence shows that judgement and performance may continue to be impaired for a time, from the continuing effects of hangover.

Any potential benefits of alcohol need to be weighed against increased risk-taking, loss of inhibitions, reduced decision-making skills, overdose related to low tolerance, and the increased risk of accidents, violence and unwanted sexual activity. The effect of alcohol in protecting against heart disease has been shown to be relevant only for people over about 40 years. There is, as yet, no evidence to suggest that it is relevant for younger age groups.

Strategies to minimise harm from drinking include alternating alcoholic and non-alcoholic drinks, eating while drinking, staying with friends rather than driving, and not accepting lifts with a drunk driver.

Guideline 10

Young people (up to about 18 years)*

10.1 should follow the recommendations under Guideline 9;

AND

10.2 if they choose not to drink, should be supported in this decision;

10.3 in settings where alcohol is available to them, should be supervised by adults at all times;

10.4 should keep any drinking to a minimum;

10.5 most importantly, should not drink to become intoxicated;

10.6 to become responsible adult drinkers, a gradual, supervised introduction to alcohol is recommended.

* While this guideline applies young people up to about 18 years of age, the issues and concerns overlap with those covered under Guideline 9, 'Young Adults'.

Rationale:

Younger people's ability to cope with alcohol is influenced by their physical size and stage of development. Young people generally have smaller body size than adults, and can therefore tolerate less alcohol. They also lack experience of drinking and its effects, and so have no yardstick by which to measure their drinking behaviour. This lack of exposure also means that they have built no physiological tolerance to alcohol.

There is evidence from the United States that the earlier the age that a young person starts to drink, the greater the risk of alcohol-related problems in later life.

The evidence base for Guideline 10 is discussed on page 46. See also pages 23, 37–38 and 61.

Comment:

Drinking by young people is very common in Australia, although there are also many young people who do not drink. There is a range of views about the optimal age to start drinking. A number of cultural groups in Australia introduce children to very dilute alcohol at a young age.

Loss of inhibitions and decision-making skills place young people at particular risk of violence, accidents and sexual coercion.

Helping young people to learn about drinking and the effects of alcohol within a safe and supportive environment can help them to manage their drinking in ways that minimise risk to themselves and others, both when they are young and throughout their adult years.

Guideline 10 should be interpreted within the context of the relevant State/Territory legislation in relation to alcohol and young people.

Guideline 11

Women who are pregnant or might soon become pregnant

11.1 may consider not drinking at all;

11.2 most importantly, should never become intoxicated;

11.3 if they choose to drink, over a week, should have less than 7 standard drinks, AND, on any one day, no more than 2 standard drinks (spread over at least two hours);

11.4 should note that the risk is highest in the earlier stages of pregnancy, including the time from conception to the first missed period.

Rationale:

Alcohol in a woman's blood stream enters that of her unborn child, and this may affect the child from conception onwards. It is difficult to identify exactly the lower levels of drinking at which alcohol may cause harm to the child and, for this reason, a woman may consider not drinking at all.

Nevertheless, while more high quality research is needed, the limited available evidence indicates that averaging less than one drink per day has no measurable impact on children's physical and mental development.

The evidence indicates that episodes of drinking above the guideline levels considerably increase the risk to the unborn child, including the risk of miscarriage, low birth weight, cognitive defects and congenital abnormalities. Heavy bouts of drinking maximise that risk.

The evidence base is discussed on page 77. See also pages 23 and 46.

Comment:

The most important consideration for women is to avoid a high blood alcohol level at any time during the pregnancy. The first weeks after conception are probably the most critical in relation to alcohol, and the woman is usually unaware of the pregnancy at this stage. The guideline is therefore important not only for women who are pregnant, but for those who may soon become pregnant.

The literature review undertaken for these guidelines found no definite evidence that low-level drinking causes harm to the unborn child. Other authorities have, nevertheless, recommended no drinking during pregnancy.

Women may choose not to drink at all, out of caution, especially if relevant risk factors are present: for example, if the mother has health problems such as high blood pressure or poor nutrition. Good antenatal care and good diet, including folate and vitamin B supplements, and not smoking are also very important.

BREASTFEEDING—A Prudent Approach

Women who are breastfeeding are advised not to exceed the levels of drinking recommended during pregnancy, and may consider not drinking at all.

Comment: Alcohol in the blood stream passes into breast milk. There is little research evidence available about the effect that this has on the baby, although practitioners report that, even at relatively low levels of drinking, it may reduce the amount of milk available and cause irritability, poor feeding and sleep disturbance in the infant. Given these concerns, a prudent approach is advised.

GUIDELINE 12

People who choose not to drink alcohol

12.1 should not be urged to drink to gain any potential health benefit, and should be supported in their decision not to drink.

Rationale:

There are many good health, family and social reasons why people choose not to drink. These include personal, cultural and religious considerations. Non-drinkers can use other strategies, such as regular exercise, giving up smoking, and a healthy diet, to gain protection against heart disease.

See pages 23, 31 and 40.

Comment:

The potential protection that alcohol provides against heart disease is of importance only for people in age groups where heart disease is a risk; generally from about age 40 onwards.

PART TWO:

CONTEXT AND EVIDENCE BASE

1 INTRODUCTION

WHY GUIDELINES?

Alcohol is widely used and enjoyed throughout our society. Evidence over several decades has confirmed that, at relatively low levels, alcohol has some health benefits from middle age onwards, and can form part of an enjoyable and generally healthy lifestyle that includes good diet and exercise. Alcohol also, however, has the potential to cause considerable harm. People who drink regularly at higher levels place themselves at substantially increased risk of chronic ill health and premature death, while an episode of heavy drinking places the drinker and others at increased risk of injury and death. For some, particularly among younger age groups, such drinking can form part of a wider pattern of risk-taking behaviour.

Australian Alcohol Guidelines: Health Risks and Benefits has been written to assist those Australians who choose to drink, to avoid or minimise any harmful consequences and enjoy alcohol. The document aims to provide clear, helpful and evidence-based information and advice in an area where there are many views, often conflicting information, and a range of social, political and commercial interests.

This document and the guidelines it contains have been developed by an expert working party representing the fields of drug and alcohol studies, public health, mental health, and consumers (see Appendix One for details). The advice provided is grounded in the best scientific evidence available, gathered in a comprehensive, systematic and up-to-date review of the scientific and medical literature.

The document has a three-fold purpose:

- to enable Australians to make informed choices about their drinking and health;
- to enable health professionals to provide evidence-based advice on drinking and health; and
- to promote individual and population health, and minimise harm from alcohol.

THE APPROACH TAKEN

The central concern in formulating the guidelines has been to minimise harm from alcohol and manage the risk associated with certain *patterns* of drinking, while acknowledging and maximising the social benefits, and the health benefits from middle age onwards. This emphasis on drinking patterns represents a change of direction from previous guidelines, where the focus has been largely on levels of alcohol consumption per se.

The issues involved are complex. The effects of alcohol vary over time and from person to person. They vary with age, gender, lifestyle, physical and mental health,

previous drinking history, medications and family history. Risk also depends on environmental factors such as the design of licensed premises and staff training, and many of these are amenable to change.

The guidelines are not intended to be prescriptive, but to provide a framework for decision making and the clearest possible advice, based on the best scientific evidence available and presented in language accessible to the general reader. There remains much we do not know, and the document also identifies limitations in our understanding, and areas requiring further investigation. The scientific evidence will continue to develop and change, and the guidelines will need to be regularly reviewed and up-dated as significant new evidence comes to light.

WHO WILL USE THIS GUIDELINES DOCUMENT?

Australian Alcohol Guidelines: Health Risks and Benefits is intended to provide a resource for a wide range of groups and individuals, including health professionals, community groups, industry, professional organisations, schools and educational organisations. The document will inform policy makers, planners, decision makers, and those responsible for the provision of alcohol, who have a broader responsibility to the community and whose decisions may influence the health of communities.

While the document may be of interest to people wanting to make decisions about their own drinking, it is not specifically aimed at the general public.

Detailed recommendations in relation to specific health conditions are beyond the scope of the document. Specialist professional organisations and societies are encouraged to develop additional guidelines to meet such needs, within the overall framework of these guidelines.

STRUCTURE OF THE DOCUMENT

Part One of *Australian Alcohol Guidelines: Health Risks and Benefits* sets out the guidelines themselves, along with a brief rationale and comments.

This section, Part Two, sets out the context for the guidelines, summarises the evidence base, and provides information that will help in applying the guidelines. Following this introductory chapter:

- Chapter 2 provides a broad overview of the effects of alcohol. It presents a summary of the extent of alcohol-related harm in Australia, and the levels of drinking that can lead to such harm. This is followed by a brief account of what happens to alcohol within the body, its immediate and longer-term effects on health, and the social consequences of excessive drinking. The chapter then considers the influence of the drinking environment and the type of drink, the different susceptibilities of various population groups, and the interaction of alcohol with medications and recreational drugs.

- Chapter 3 summarises patterns of drinking in the Australia community, providing a historical perspective and data from a series of household surveys.
- Chapter 4 summarises the evidence from the literature regarding the risks and benefits of alcohol in relation to a number of specific conditions. It draws heavily on the literature review commissioned by the NHMRC to underpin the guidelines (see section on 'The Evidence Base' below).
- Chapter 5 sets out some of the areas where there is a particular need for research to provide a better understanding or more evidence.

WHAT HAS CHANGED SINCE THE LAST GUIDELINES?

Australian Alcohol Guidelines: Health Risks and Benefits reflects the considerable advances in understanding that have occurred since 1992, when the National Health and Medical Research Council (NHMRC) published the previous Australian guidelines. Much has changed, not only in our knowledge, but also in our appreciation of the complexity of the questions involved, and in the emphasis and direction of research.

Perhaps the most important change, as mentioned above, is a broadening in perspective from overall *levels* of consumption—the average amount drunk—to incorporate *patterns* of drinking. While average levels of drinking remain the main issue in relation to long-term harm, there is increased recognition of the critical importance of patterns of drinking in relation to intoxication and the short-term risk it carries, and a greater appreciation of alcohol as a contributor to acute health problems.

See, in particular, Guidelines 1, 3, 9, 10 and 11.

The concept of 'patterns of drinking' refers to aspects of drinking behaviour other than the level of drinking, including when and where drinking takes place, the number of heavy drinking occasions and their characteristics, activities associated with drinking, personal characteristics of the drinker and drinking companions, the types of drinks consumed, and the drinking norms and behaviours that comprise a 'drinking culture'.

Balancing the focus on harm is a greater appreciation of the potential beneficial effects of alcohol: not only the social benefits, but also the degree of protection that alcohol affords against heart disease in middle-aged and older people. There is a better understanding of the biochemical mechanisms involved in this protective effect, and of the complex trade-off between benefit and harm.

Differing vulnerabilities are also better recognised and understood: for example, the different responses to alcohol of men and women, and different age groups, and differing genetic and ethnic susceptibilities to harmful consequences. There is

growing recognition of the issue of men's health and of the contribution of particular patterns of drinking to men's poor health outcomes. At the same time, there is increasing concern about potentially harmful changes in the levels and patterns of drinking among young women. There is also growing recognition of health issues affecting Indigenous communities.

In assessing the effects of alcohol at the population level, the focus of attention has broadened from numbers of deaths to incorporate personal years of life lost and various measures of injury, disability, illness, quality of life, and economic costs.

THE EVIDENCE BASE

The guidelines are based on what the Working Party has determined to be the best evidence available. Evidence-based medicine has been defined as 'the conscientious, explicit and judicious use of current best evidence in making decisions about the care of the individual patient. It means integrating individual clinical expertise with the best available external clinical evidence from systematic research' (Sackett et al 1996). Similarly, another authority has described evidence-based medicine as 'an approach to health care that promotes the collection, interpretation, and integration of valid, important and applicable patient-reported, clinician-observed and research-derived evidence. The best available evidence, moderated by patient circumstances and preferences, is applied to improve the quality of clinical judgements' (McKibbon et al 1995).

To establish the evidence base, a systematic review of the literature was commissioned by the NHMRC, and that literature review formed the primary evidence base for these guidelines. The group that carried out the literature review was selected by a competitive tender process, and comprised international experts in the field who had undertaken other similar reviews. The review group was headed by Professor Eric Single, PhD (Canada), and included Mary Jane Ashley, MD (Canada), Susan Bondy, PhD (Canada), Maureen Dobbins, PhD (Canada), James Rankin, MB, FRACP, FAFPHM (Australia) and Jürgen Rehm, PhD (Germany). The literature review, entitled, *Evidence Regarding the Level of Alcohol Consumption Considered to be Low-Risk for Men and Women,* is available on the Internet at <http://www.nhmrc.gov.au>. Studies included in the review were required to meet rigorous assessment criteria regarding the quality of the study design, and causality was assessed according to the guidelines of the International Agency for Research on Cancer (IARC) (see Appendix Three). These criteria are usually used to rank the strength of evidence for clinical interventions. The current guidelines, however, are not clinical interventions and therefore warrant the application of slightly different criteria in determining what 'evidence' is relevant. As yet, there is no accepted criteria for determining evidence in relation to public health issues, hence wherever possible the above criteria were applied (but it is noted that there is a case to be made for developing a new set of criteria specifically for guidelines in areas such as nutrition or injury prevention).

This document, *Australian Alcohol Guidelines: Health Risks and Benefits*, summarises the evidence gathered in the literature review, citing some of the better known and more substantial work in the field. For more detail, readers should consult the literature review itself. The literature review was further supplemented by additional literature that was identified through the consultative process and expert input. This included the input of the Expert Working Party (see Appendix One). The expertise of the Working Party was of particular relevance in areas that extended beyond the literature review, and evidence included during this process was required to meet similarly rigorous assessment criteria regarding the quality of the study design. Some additional meta-analyses were also undertaken specifically for this project (Lawrence & Holman 1999). Wherever possible, this document draws on peer-reviewed literature, and such literature constitutes the large majority of the references cited. Where peer-reviewed evidence was not available, other publications have been cited.

In adopting an evidence-based approach, it is relevant to note the importance of doing this pragmatically and realistically. As Muir Gray (1999) states, 'The absence of excellent evidence does not make evidence-based decision making impossible; what is required is the best evidence available, not the best evidence possible'. The evidence base in this area, while not perfect, contains a substantial body of rigorous data, and the best and most rigorous research evidence available was used to determine the guidelines. There are many areas where additional research is required. For instance, data on patterns of drinking and health outcomes are limited, although there are some good studies of drinking environments and their effect on behaviour and risk. In areas where data are non-existent or very limited, recommendations can often be based on good scientific or theoretical knowledge, or on the principles of biological science, neuroscience or behavioural science. Where such reasoning is used in the present guidelines, it is identified clearly (as, for example, in the recommendation for alcohol-free days).

These guidelines have been based on the best evidence available at the time of writing. They may be subject to different interpretation as the body of research builds in various areas. Where the evidence base is less strong, this is noted in the relevant part of the document, and Chapter 5 identifies areas and specific topics needing further research.

While randomised controlled trials are considered to be the 'gold standard' of evidence, they are often neither feasible nor possible for many public health interventions, and there is currently no agreed grading for levels of evidence that are specific and appropriate to this area (NHMRC 1999). Similarly, both logistical and ethical barriers often prevent the use of randomised controlled trials to assess alcohol toxicity. Valid conclusions can nevertheless be drawn, regarding the consequences of alcohol consumption, from well designed and conducted observational studies. Much of the available evidence derives from epidemiological studies (often using a cohort or case-control design) in which alcohol consumption is usually measured by self-report of frequency of drinking and quantity per

occasion (see Appendix Four for discussion about the structure and potential scope of surveys). Studies on level of drinking often underestimate true consumption. They have, nevertheless, proved to be useful indicators of health outcomes for some decades, suggesting that any error is probably relatively consistent across studies.

In addition, the data that form the basis for the general population guidelines are limited in the extent to which they can make allowances for individual variation, and this needs to be considered in applying the guidelines. There is wide variability in the way individuals react to alcohol, determined by factors including gender, body size, age, experience of drinking, and individual metabolism. Where the evidence base is less clear cut the Committee took a cautious and conservative position.

TERMINOLOGY

Where this document refers to 'alcohol', it refers specifically to ***ethyl alcohol***, also known as ***ethanol***. This is the type of alcohol found in drinks intended for human consumption (see page 29).

Wherever possible, the document defines levels of drinking precisely, using the Australian 'standard drink' (10g alcohol, see page 28). Where descriptive terms such as 'heavy', 'moderate' or 'light' are used, some quantitative descriptors are included where possible. The term 'binge drinking' is avoided as far as possible, as its meaning is ill defined and unclear.

Alcohol use is discussed, where possible, in terms of level of risk, as set out in Table 1, on page 5. This is consistent with public health messages in a range of other areas. The intention is to take a neutral stance, to enable people to assess their level of risk as objectively as possible, based on the scientific evidence. The Working Party has categorised risk associated with drinking using three risk levels:

- *Low risk* levels define a level of drinking at which there is only a minimal risk of harm. At this level, there may be health benefits for some of the population.
- *Risky* levels are those at which risk of harm is significantly increased beyond any possible benefits.
- *High risk* drinking levels are those at which there is substantial risk of serious harm, and above which risk continues to increase rapidly.

The 'low risk' levels are consistent with the levels recommended for men and women in Guideline 1.

Risk is also assessed in terms of:

- *long-term risk*—the level of long-term risk associated with regular daily patterns of drinking, defined by the total amount of alcohol typically consumed per week. This is the way alcohol-related risk has traditionally been assessed; and

- *short-term risk*—the risk of harm (particularly injury or death) in the short-term, that is associated with given levels of drinking on a single day. These levels assume that overall drinking patterns remain within the levels set for long-term risk, and that these heavier drinking days occur a maximum of three times per week. Outside these limits, risk is further increased.

It is this latter category that marks the current guidelines as being distinctly different from previous ones.

The terms 'alcoholism' or 'alcoholics' are avoided as they are ambiguous and stigmatising. Instead, the term 'dependence' is used. Alcohol dependence is a complex phenomenon that is discussed on page 75. In brief, it refers to situations where drinking is given priority over other behaviours that the person had previously found much more important. Dependence ranges from mild to severe. People with severe dependence drink regularly above guideline levels, often find it hard to limit how much they drink, and generally have marked tolerance to the effects of alcohol. If they stop drinking for a few hours, they experience both physical and psychological withdrawal symptoms, such as tremulousness and anxiety.

'Intoxication' is a widely used term with no consistent or formally agreed definition. It is usually taken to refer to an elevated blood alcohol concentration such that a person cannot function within their normal range of physical and cognitive abilities. Levels above 0.05 or 0.08 are sometimes taken as a proxy measure of intoxication. In lay terms, it is a subjective feeling, the experience of a substantial effect of alcohol on mood, cognition, and psycho-motor function. There are marked variations in the amount of alcohol different people need to consume in order to experience intoxication.

Further definitions of terms used in this document can be found in the Glossary and Acronyms section (page 125).

'A STANDARD DRINK'

A clear understanding of what constitutes a 'standard drink' is essential for the accurate interpretation and application of these guidelines. The present guidelines follow the previous Australian drinking guidelines (NHMRC 1992), in defining a standard drink as containing 10 grams of alcohol (equivalent to 12.5 millilitres of alcohol). Where possible, in discussing the evidence, amounts of alcohol are defined in grams and Australian standard drinks. The notion of a standard drink is used widely, but the definition varies from country to country.

In Australia, all bottles, cans and casks containing beverage alcohol are required by law to state on the label the approximate number of standard drinks they contain. Table 2 provides a rough guide.

It can sometimes be difficult to translate standard drinks into real life situations. There are no common glass sizes used across all public drinking environments, let alone in private homes, and people commonly underestimate what they drink. The

problem is compounded where large containers (jugs, casks, flagons) are shared, where glasses are topped up by another person, or where the composition of mixed drinks is not known (eg cocktails or punch at a party). Hence, it may be challenging to calculate accurately the numbers of standard drinks consumed.

Table 2: Numbers of Australian standard drinks in common containers of various alcoholic beverages

Light beer (2.7%):		
1 can or stubbie	=	.08 a standard drink
Medium light beer (3.5% alcohol):		
1 can or stubbie	=	1 standard drink
Regular beer (4.9% alcohol):		
1 can or stubbie	=	$1^1/_2$ standard drinks
1 jug	=	4 standard drinks
1 slab (cans or stubbies)	=	about 36 standard drinks
Wine (9.5%–13% alcohol):		
750-ml bottle	=	about 7 to 8 standard drinks
4-litre cask	=	about 30 to 40 standard drinks
Spirits:		
1 nip (30 ml)	=	1 standard drink
Pre-mixed spirits (around 5% alcohol):		
1 can (375 ml)	=	$1^1/_2$ standard drinks

There is a body of opinion that the term 'standard drink' should be replaced by 'standard unit', because of the ease with which a standard drink is confused with a (usually larger) serving of drink. While the Working Party shares this concern, it considers that any change that has major implications for public and professional education needs to be carefully considered and debated. 'Standard unit' is, however, the measure used in the United Kingdom. This issue should be pursued and reconsidered next time these guidelines are reviewed, if not before.

2 ALCOHOL AND ITS EFFECTS: AN OVERVIEW

WHAT IS ALCOHOL?

The term 'alcohol' describes a series of organic chemical compounds, but only one type, *ethyl alcohol* or *ethanol,* is found in drinks intended for human consumption, and this is the type that is the subject of this chapter. It should, however, be noted that some other forms of alcohol are particularly toxic to humans.

CLASSIFYING THE EFFECTS OF ALCOHOL

Any method of classifying the effects of alcohol needs to take into account:

- potential harm in the areas of social, population and individual health, and under the law;
- the potential health benefits and social benefits of alcohol;
- the potential to produce dependence in a proportion of people who drink at high levels for sustained periods;
- the continuum of risk that exists for individuals, from net benefit (where the risk of harm is slight and outweighed by potential benefits) to high risk;
- effects of alcohol in the short term (resulting from a single drinking episode) and the long term (generally resulting from regular drinking over time);
- the variations in short-term and long-term effects between population groups (including males and females, and different age groups), and to some extent in response to cultural factors;
- variations in effects from person to person, depending on a wide range of factors that include body size, gender, inherited characteristics, and the amount the person habitually drinks;
- variations over time in the drinking patterns and levels of individuals and populations.

Traditionally, alcohol-related harm has been thought of as resulting from long-term chronic heavy alcohol use, and numerous studies, going back to the turn of the century, have examined the relationships between access to alcohol, per capita consumption and alcohol-related harm (eg Stockwell 1997, Holder & Edwards 1995, Edwards et al 1994, Makela et al 1981, Single et al 1981, Moore & Gerstein 1980, Bruun et al 1975).

Over recent years, as discussed in the previous chapter, alcohol policy has seen a major shift from the almost exclusive preoccupation with harm from chronic long-term drinking to include harm that is acute, often more transient (but not necessarily less serious), and frequently experienced by the young and/or occasionally immoderate drinker (Roche 1998a). At the same time, alcohol problems

are now described by most countries and by the World Health Organization (WHO) as an important public health concern.

The increased understanding of patterns of drinking encompasses not only variations in drinking over time (including heavy drinking occasions), but the settings, activities and circumstances associated with drinking, personal characteristics of drinkers and their drinking companions, and types of beverages consumed. This has wide implications for alcohol policy (Rehm et al 1996).

In general, problems associated with alcohol use have been characterised as those relating to:

- **dependence**:
  - withdrawal symptoms, loss of control, social disintegration etc;
- **regular use**:
  - cirrhosis of the liver, cognitive impairment, pancreas damage, heart and blood disorders, ulcers etc;
- **intoxication**:
  - alcohol-related violence, risky behaviours, road trauma, falls etc.

A related approach is shown in Figure 2, based on the model developed by Thorley (1980). This shows the overlap between three separate aspects of drinking behaviour: regular heavy drinking, alcohol dependence, and acute alcohol-related problems.

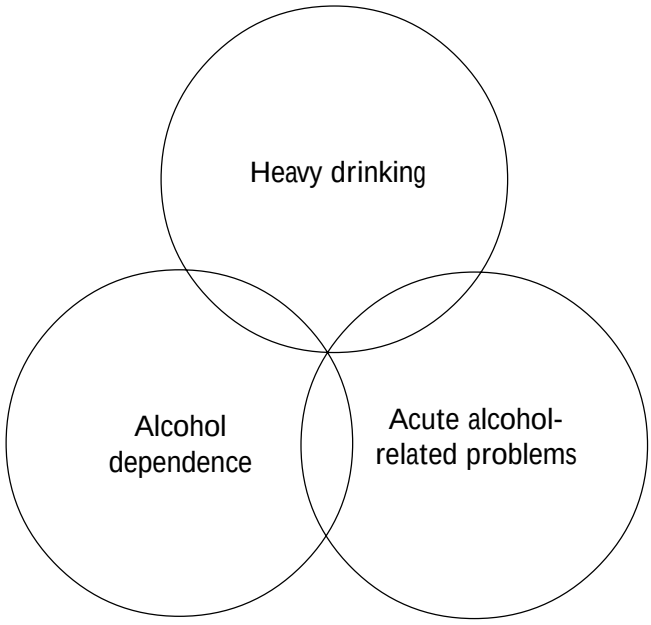

Figure 2. Relationship between aspects of drinking behaviour (based on Thorley 1980)

A more elaborate model developed by Rehm and Fischer (1997) is set out in Table 3. Their model distinguishes problems that are the result of a single drinking occasion from problems resulting from long-term drinking, and differentiates these categories on the basis of effects at various levels: physiological, psycho-physiological or mental, personal and social, and wider social and cultural.

Table 3: Conceptual schema of alcohol-related harm (adapted from Rehm and Fischer 1997)

	Potential consequences of single-occasion use	Potential consequences of long-term use
Physiological reactions	Overdose	Death (eg liver cirrhosis) Illness (eg gastritis, pancreatitis)
Psycho-physiological and mental reactions	Changed consciousness and control (eg hangover, suicide)	Dependence
	Injury to drinker	Depression
	Accidental death	Cognitive loss
Immediate personal and social/environmental reactions	Severe family and workplace disruption	Disruption of social and work relationships
	Injury to others, violence	
Wider social and cultural reactions	Criminal and informal sanctions	Stigmatisation, coercion to change, treatment, criminalisation of alcohol-related behaviour

QUANTIFYING THE TYPES OF HARM ASSOCIATED WITH ALCOHOL

Drinking alcohol has health, social and economic costs and benefits, for both individuals and populations. As the figures below and in the next section demonstrate, people who drink small quantities have better health outcomes than those who do not drink, although abstainers achieve very much better health outcomes than heavy drinkers. It does not follow, however, that abstainers, either at the individual or the population level, would achieve a better health outcome by drinking: there are many valid, and often compelling, health and social reasons why people choose not to drink.

See GUIDELINE 12

At high levels of consumption, harms far outweigh benefits, and the higher the consumption, the greater the costs to one's health and society. Determining the appropriate policies and controls to place around alcohol is a continual challenge

for society and one that is not easily resolved (Roche 1999). Increasingly, efforts have been directed at developing a rigorous evidence base to guide policy and clinical decision-making.

Drinking alcohol at risky and high risk levels for long-term harm was estimated to have caused 3,290 deaths in Australia in 1997, accounting for about 4 percent of all male deaths and 2 percent of all female deaths, and about 50,000 hospitalisations (Chikritzhs et al 2001). The short-term and long-term effects of excessive drinking make roughly equal contributions to these deaths.

Table 4 focuses on short-term effects, showing the proportions of various types of traumatic injury and deaths in Australia that are attributed to alcohol use.

Table 4: Proportions of types of traumatic injury and death attributed to alcohol in Australia (based on English et al 1995)

Harm	Proportion attributed to alcohol	
	Males (% of all male cases)	**Females** (% of all female cases)
Road injuries	37%	18%
Suicide	12%	8%
	Persons	
Fall injuries	34%	
Fire injuries	44%	
Drowning	34%	
Assault	47%	
Child abuse	16%	

Mathers et al (1999) estimated that the harm associated with alcohol consumption accounted for 4.9 percent of the total burden of disease and injury in Australia in 1996 (comprising 6.6 percent in males and 3.1 percent in females). However, the protective effect of low to moderate consumption is estimated to have averted 2.8 percent of the total burden (2.4 percent in males and 3.2 percent in females). In comparison, tobacco smoking accounted for 9.7 percent of the total burden, physical inactivity for 6.7 percent, hypertension for some 5.4 percent, obesity for 4.3 percent, inadequate fruit and vegetable consumption for 2.7 percent, and illicit drugs for 1.8 percent.

On a global basis, alcohol was estimated to account for 3.5 percent of the total burden of disease and injury in 1990. For men, alcohol was the leading cause of disability in developed countries, and the fourth leading cause of disability in developing countries, out of a list of 11 major risk factors. For women, however, alcohol was considered to be a much less significant contributor to disability (Murray & Lopez 1996).

Any consideration of alcohol-related problems needs to take into account the considerable variations that occur in drinking patterns over time. For example, some studies have suggested that over a year in a given population, about one third of existing problems will worsen, one third will remain the same, and one third will resolve and be replaced by a new population with problems (Vaillant 1998).

ECONOMIC IMPACT OF EXCESSIVE DRINKING

In addition to its health and social consequences, excessive drinking has a substantial economic impact. The costs accrue not only to the health care system, but, more significantly, to industry through absenteeism, premature retirement, and impaired or lost productivity. It has been estimated, for example, that alcohol cost the Australian community some $4.5 billion in 1992 (Collins & Lapsley 1996), the equivalent of 1.4 percent of the gross domestic product (WHO 2000). Therefore, compliance with these drinking guidelines has the potential to achieve considerable savings to the health care system, and to the economy as a whole. The realisation of these potential savings requires the implementation of a comprehensive range of policies to encourage low risk drinking, over and above the publication of these guidelines.

The 1998 National Drug Strategy Household Survey found that, of all those who reported drinking alcohol at least once during the previous 12 months, one in 20 (5 percent) reported missing school or work due to their drinking (Adhikari & Summerill 2000).

A recent Canadian cost study suggests that drinkers who would qualify for a diagnosis of alcohol dependence account for about 50 percent of the social costs related to alcohol (Rehm 1999). By implication, the remaining 50 percent of costs are incurred by drinkers who are not addicted. The majority of these are people who usually drink within guideline levels, but occasionally drink at high-risk levels. Understanding of the types of harm likely to result from these infrequent bouts of excessive drinking continues to grow (Stockwell et al 1996).

Table 5 shows the estimated economic cost of alcohol in a range of countries:

Table 5: Comparison of total tangible cost of substance abuse estimates for various countries

Study	Country	Year of data	Original total cost estimate[1] (millions, local currency)	Total cost relative to GDP[2] (%)
Adrian *et al* (1988)[3]	Canada	1984	11,840	2.7
Rice *et al* (1990)	U.S.	1985	70,340	1.7
Collins & Lapsley (1991)	Australia	1988	4,538	1.4
McDonnel & Maynard (1985)	U.K.	1983	1,614	0.5
Nakamura *et al* (1993)	Japan	1987	664,000	1.9
Single, Robson *et al* (1998)	Canada	1992	7,522	1.1
Harwood *et al* (1999)	U.S.	1992	148,000	2.4
Kopp *et al* (1999)	France	1997	F115,420	1.5

1 Total cost includes all indirect and direct costs, as specified by the author, unless otherwise indicated.

2 Original total cost figure is divided by the national GDP for the year of the study.

3 Estimates of external costs.

Source: WHO (2000)

LEVEL OF DRINKING AND LEVEL OF HARM: SETTING GUIDELINES FOR THE GENERAL POPULATION

Two main patterns of drinking create a risk of health problems for the drinker:

- excessive intake on a particular occasion; and
- consistent high-level intake over months and years.

See GUIDELINE 1

The former leads primarily to 'problems of intoxication' such as falls, accidents and violence at least partly precipitated by high blood alcohol levels. A pattern of regular drinking to intoxication over a number of years also predisposes the drinker to long-term physical health problems such as cardiovascular disease as well as these more immediate problems (Single et al 1999b). People who drink at a high level on a daily basis will usually develop significant tolerance to alcohol and may experience only a slight degree of intoxication, but are still putting themselves at increased risk of physical health problems in the longer term.

A significant new departure for these Drinking Guidelines is the establishment of upper daily limits to address the acute or short-term health risks, in addition to the limits set to minimise long-term or chronic risks. The previous NHMRC drinking

guidelines (NHMRC 1992) recommended that alcohol consumption should not exceed 4 standard drinks per day, or 28 per week, for a man, and 2 per day, or 14 per week, for a woman. These present guidelines confirm this level of drinking as an *average*, taken over a week; but they also take the view that, *within this weekly limit and under restricted circumstances,* drinking up to 6 standard drinks in one day for a man and 4 drinks for a woman will be low risk. *The following restrictions are crucial*: as well as remaining within the weekly limit, the drinker must not be about to drive or operate machinery, should not be on medication, should not have a condition which is exacerbated by drinking, and should not be pregnant or breast feeding.

There are two major reasons for this significant revision:

- The evidence from the relevant epidemiological studies for increased risk of long-term harm from alcohol is based on average consumption over a period of a month or more. As described below, analysis of information pooled from the best long-term studies shows that there is no increased risk of premature death for men who drink an *average* of up to 39g (4 standard drinks) per day and for women who drink an *average* of up to 19g (2 standard drinks) per day. Such average levels allow for day-to-day variation either side of these figures, and there is no evidence of significant additional harm up to the new upper limits for consumption on occasional days.
- A growing body of research shows that drinking above 60g (6 standard drinks) per day for men and 40g (4 standard drinks) for women is associated with significant additional risk of harm in the short term (WHO 2000, McLeod et al 1999). Risk increases rapidly with further drinking above these limits (McLeod et al 1999). This evidence stands behind the categorisation of risk set out in Table 1, page 5, which identifies 'risky' and 'high risk' drinking levels for single occasion drinking, as well as for average level of drinking over time. These levels are also consistent with those identified in recent international alcohol research guidelines (WHO 2000).

A large number of studies have looked at the mortality rates associated with different levels of drinking alcohol. A meta-analysis combining and summarising the results of many such studies was carried out in 1995 (English et al 1995) and updated specifically for these guidelines (Lawrence & Holman 1999). The meta-analysis results reflect the sum of both short-term and longer-term effects of alcohol. The results showed that:

- The lowest mortality rate (ie the lowest risk) was associated with an average of 1 standard drink per day for men and somewhat less for women. The mortality rate for people drinking at these levels was lower than that for non-drinkers.
- For women drinking an average of 2 to 3 standard drinks per day, the mortality rate was slightly (about 10 percent) higher than that for non-drinkers. At 4 to 5 standard drinks per day, the risk was 40 percent higher than for non-drinkers.

- For men drinking between 3 and 4 standard drinks per day, the mortality rate was the same as for non-drinkers. Risk only started to increase significantly for those drinking at least 4 standard drinks per day.

This evidence is the basis for the recommendation in Guideline 1 that, on average, men should not exceed 28 standard drinks per week, and women should not exceed 14 per week. The guideline is set at the point at which risk starts to rise significantly above the level of risk for non-drinkers (rather than the level associated with the lowest mortality rate overall).

PROCESSING ALCOHOL IN THE BODY

Alcohol, when it is swallowed, is rapidly absorbed into the blood stream, and its effects on many organs and systems of the body are evident within minutes. This section and the ones that follow give a brief overview of that process and trace the effects of alcohol in the short and the long term.

After being swallowed, a small amount of the alcohol is broken down (metabolised) by enzymes in the gut, but most of it is absorbed directly into the blood stream, some from the stomach and some from the first few centimetres of the small intestine. This occurs very rapidly, but the actual rate varies not only from person to person, but also from time to time for the one person. Food in the gut slows down the absorption, and it is influenced by the temperature of the drink and its alcohol concentration.

In the blood stream, alcohol is carried throughout the body and distributed in the body fluids (but not in body fat). Its effects on the brain are evident within about five minutes of being swallowed. A small amount, about 2 percent, is excreted unchanged in sweat, breath and urine, enabling 'blood alcohol concentration' to be calculated through breath testing. This measurement, which can also be done directly through a blood test, provides an index of the concentration of alcohol throughout the body. The risk of accident or injury increases rapidly with increasing blood alcohol levels, largely through the effects on the brain (see below). For a given amount of alcohol consumed, women have a higher blood alcohol concentration and hence a higher risk of accident or injury.

Blood alcohol levels fall slowly over time, after drinking has stopped, as the alcohol in the blood is gradually broken down by the liver. It generally takes about an hour to clear one standard drink, although this varies quite widely from person to person. Men generally have a larger liver than women and this, coupled with differences between men and women in the actual metabolic process (the enzymes involved), means that, on average, men clear alcohol from the blood stream more rapidly than women do.

When alcohol is broken down (both in the gut and the liver), acetaldehyde is formed, and this is further broken down into water and acetic acid, then to carbon dioxide. In 90 percent of people of Caucasian extraction, this breakdown occurs almost instantaneously, but in about 10 percent of Caucasians and about 90 percent of people of Oriental extraction, this step is interrupted and even after only a small

amount of alcohol, the build-up of acetaldehyde causes obvious flushing (the 'flushing response'), nausea, vomiting and a range of other unpleasant effects.

SHORT-TERM EFFECTS FOR THE DRINKER

Alcohol has a range of short-term effects. The following sections provide an overview of these effects, briefly discussing effects on the brain, stress, sleep and sexual function, the gut and pancreas, the heart and circulation, and risky behaviour, injury and death. More detailed evidence relating to specific health conditions is presented in Chapter 4, page 65. Specific differences relating to gender and age are discussed on pages 45, 46 and 49.

Effects on the brain

Alcohol affects almost all cells and systems in the body, but its most obvious and most immediate effects are on the brain. It inhibits many of the brain's functions, dampening the arousal centres. The initial effects of alcohol may reduce tension or inhibitions, making the person feel more relaxed or excited. With more alcohol, the dampening effects extend to motor and sensory centres in the brain, so the person reacts more slowly to stimuli. For example, when driving, it takes longer to apply the brakes if the car ahead stops suddenly.

See Guideline 2

With increasing alcohol concentrations in the body, movement and coordination rapidly become more difficult. Speech, thinking and the senses are affected, and without the cognitive or verbal capacity to resolve conflicts, physical violence becomes more likely (Marsh & Kibby 1992). Both alcohol and acetaldehyde in the bloodstream also affect the centre in the brain that controls vomiting.

If the blood alcohol concentration reaches a sufficient level, it leads to unconsciousness and eventually, because it slows down all systems of the body, it will inhibit breathing. This may be fatal, particularly as the person is also likely to be vomiting. The danger is much higher for people using other drugs that also depress the central nervous system (including illicit drugs and benzodiazepines), as these may also affect breathing. A high proportion of people who die from heroin overdose also have a high blood alcohol concentration. Zador et al (1996), for example, report that alcohol was detected in 45 per cent of heroin-related deaths.

Alcohol also affects the pituitary gland at the base of the brain, suppressing the production of a hormone that keeps the body's fluid reserves in balance. The kidneys fail to reabsorb enough water from the urine, and the body excretes more water than it takes in, leaving the person dehydrated and with a headache.

The immediate and short-term effects on the brain are often less apparent in people who drink regularly at a given level, as they acquire a degree of tolerance which, in some, enables them to drink large amounts with relatively little obvious effect in the short term. This tolerance has two elements:

- metabolic—the liver becomes faster and more efficient at breaking down alcohol; and
- functional—the person learns to cope with, and compensate for, the deficits induced by alcohol. This ability to compensate tends to be associated with, and therefore happens more easily in, familiar drinking environments (Siegal 1999).

Despite this tolerance, the long-term effects remain damaging, particularly as the drinkers who have greater tolerance for alcohol are those who subject themselves to higher blood alcohol levels more frequently. The long-term effects of alcohol are largely related to the level of drinking over time (see page 39).

Stress, sleep and sexual function

Alcohol accompanies and, to some extent, encourages good times, sociability, shared experiences, and personal enjoyment and well-being (Peele 1997), although, ironically, these benefits are so widely accepted as to be somewhat invisible (Roche 1998b). The social and psychological benefits may also include enhanced creativity and a therapeutic value in times of stress. However, while a small amount of alcohol may bring short-term relief from stress, it does nothing to address the cause of the stress. Indeed, there is considerable evidence that sustained drinking increases anxiety levels, and where the stress is ongoing, it may lead to a degree of dependence on alcohol (Kessler et al 1996 1997).

Alcohol is commonly seen as aiding sleep. While it may induce sleep, however, it also leads to increased wakefulness and arousal several hours later, and it aggravates sleep disorders (Castaneda et al 1998).

See GUIDELINE 6

It can also cause or exacerbate a range of sexual problems. In particular, alcohol consumed at risky or high risk levels adversely affects male potency—the ability to get an erection.

Gut and pancreas

In the gut, low levels of alcohol cause acid to be secreted. As levels rise, secretion of pepsin (a digestive hormone) is reduced, the lining of the gut becomes irritated, and diarrhoea may result. The pancreas may become inflamed, sometimes chronically, and this can cause severe pain and a highly unpleasant reaction to alcohol in the future.

Heart and circulation

One or two standard drinks (10–20g alcohol) can affect heart rate, blood pressure, the contraction of heart muscle and its efficiency in pumping blood, and blood flow throughout the body (Friedman 1998). While these actions generally are not considered to be clinically important, the overall effects on blood flow may have some unfavourable implications for some people with cardiovascular disease.

At higher levels, alcohol may precipitate heart arrhythmias, raised blood pressure, shortness of breath, and cardiac failure. Often called 'holiday heart syndrome', this commonly occurs after a few days of somewhat increased alcohol consumption. Stroke is another relatively well described outcome of intermittent heavy drinking, and a significant proportion of strokes occur after a drinking episode above guideline levels.

The relationship of alcohol to heart disease and stroke is discussed on pages 67 and 70, respectively.

Alcohol also causes the blood vessels near the skin to open up so that the blood flow through them increases. This is what causes a flushed face in response to drinking, but it also causes the body to lose heat more rapidly, and this can be dangerous in the cold.

Risky behaviour, injury and death

People who drink alcohol above guideline limits place themselves increasingly at risk of injury, trauma, risky behaviours or assault. Potential types of harm include falls, coercive sexual activity and/or unprotected intercourse (particularly among adolescents), fights and violence, and road trauma. Alcohol is involved in a significant proportion of accidental deaths, including road deaths and drownings, as demonstrated in Table 4, page 32.

See GUIDELINES 1, 2

Heavy drinking is also a major risk factor for suicide and suicidal behaviour among both young people and adults. Given the high and rising suicide rates in young adult men in Australia, this is of particular concern. While data are poor, there appears to be a particularly strong association between suicide and alcohol among some Aboriginal and Torres Strait Islander peoples.

Alcohol-related injury is discussed in more detail on page 65.

LONGER-TERM HEALTH CONSEQUENCES

Alcohol over the longer term is an important cause of ill health in our society. Certain levels and patterns of drinking over time increase the risk of developing a number of disorders. For many of these conditions, there appears to be a threshold effect—that is, a level of drinking below which alcohol is not a risk for that disorder. This, however, varies widely between individuals and population groups. In addition, the effects of alcohol are often compounded and exacerbated by other risk factors, such as smoking, dietary factors, or other drugs.

See GUIDELINE 1

These conditions are discussed in detail later in this document, but, in summary, alcohol at sufficient levels over time increases the risk of developing:

- **Cancer:** Chronic heavy drinking has been shown to be a cause of cancers of the mouth, throat and oesophagus. Smoking in conjunction with heavy drinking hugely increases the risk. There is also evidence that alcohol is associated with an increased net risk of cancer, taking into account the incidence at all cancer sites (see page 72).
- **Cirrhosis of the liver:** This is the most common cause of illness and death related to chronic heavy drinking. Cirrhosis may be caused by alcohol acting alone, and the risk is further increased in the presence of hepatitis B or C, and other conditions affecting the liver (see page 75).
- **Cognitive problems and dementia:** Alcohol in high doses affects the entire brain, and continued high levels of drinking over time lead to cell damage and cell death in the brain. This affects cognitive skills, including memory and reasoning skills. One manifestation of this is Korsakoff's syndrome, which particularly affects recent memory, and for which lack of thiamine in the diet is an important contributing factor. There is, however, some evidence to suggest that light drinking may help to prevent cognitive decline in older age (see page 79).
- **Alcohol dependence:** The debilitating effects and loss of control brought about by alcohol dependence constitute a disorder in their own right. As well as that, there is the role of dependent drinking in causing a range of other conditions. Once a person has become dependent on alcohol, it is much more likely that drinking will continue in a way that could harm both the drinker and others (see page 75).

The situation is more complex in relation to cardiovascular disease—that is, conditions affecting the heart and the blood vessels:

- **Heart disease:** There is good evidence to show that a regular pattern of drinking at a low risk level helps to prevent heart disease from middle age onwards. There is no evidence, however, that heavy drinking (at risky or high risk levels) has any *additional* benefit in relation to heart disease, and any benefits are outweighed by the other risks associated with alcohol. The benefits in preventing heart disease have been demonstrated in people of middle age onwards: studies have not been able to look at younger age groups because heart disease is so rare before middle age. It is not yet known whether low-risk drinking in younger adulthood helps to prevent the onset of cardiovascular disease in later life.
- **Stroke:** Drinking significantly above the recommended guideline levels increases the risk of stroke; but the effect of lower levels of drinking is uncertain, with the possibility that light drinking may protect against some types of stroke (see page 70).

Other long-term effects that may result from risky or high risk drinking include:

- effects on the nerves that supply the arms and legs, leading to loss of sensation, pins and needles, and possibly muscle weakness (peripheral neuropathy);
- weakness of the heart muscle, so the heart does not pump blood as efficiently (cardiomyopathy);
- sexual problems: in particular, alcohol at risky or high risk levels may reduce male potency; that is, men may find it more difficult to get an erection.

SOCIAL CONSEQUENCES

In addition to the health problems resulting from alcohol, certain patterns of drinking have social consequences that affect many members of the community from time to time. These range from minor acts of vandalism or offensive behaviour to far more serious anti-social behaviour which can result in affront, violence or injury to others, including bystanders. The many types and intensities of these incidents make them far more difficult than health consequences to document or measure, but even the more minor ones constitute a considerable public nuisance. A 1995 survey found, for example, that some 20 percent of all incidents of custody were for public drunkenness (Carcach & McDonald 1997, cited in Makkai 1997). There is no doubt that the social amenity of a community is improved if drinking is well managed.

See GUIDELINES 1 & 4

The 1998 National Drug Strategy Household Survey (Adhikari and Summerill 2000) reported that 24 percent of males and 11 percent of females who were surveyed said they had driven a motor vehicle while under the influence of alcohol, while 13 percent of males and 6 percent of females had verbally abused someone. Some 2 percent of the total population had physically abused someone, 3 percent had damaged property, and 1 percent had stolen, with males again predominating in each of these areas.

At the same time, the survey reported that nearly 30 percent of the population aged 14 or over reported being verbally abused in the previous 12 months by someone affected by alcohol, 16 percent of people had been put in fear by someone so affected, and 6 percent had been physically abused. With respect to property crime, 8 percent had property damaged and 4 percent had property stolen by someone affected by alcohol.

Victims of such incidents are themselves more likely to have been drinking. The Drug Strategy Household Survey (Adhikari and Summerill 2000) showed that males, and, in particular, those who themselves were regular drinkers, were more likely to

have experienced alcohol-related crimes than females, although females were significantly more likely to report that they had been put in fear. Other factors (in addition to high alcohol consumption) that make people more likely to be either offenders or victims of alcohol-related aggression, verbal abuse or crime include being young, in the paid workforce, and non-married (Makkai 1998, 1997).

Alcohol is also an important factor in homicide, with 34 percent of offenders and 31 percent of victims being under the influence of alcohol at the time (Carcach 1997, James & Carcach 1997, both cited in Makkai 1997).

It is now widely accepted that alcohol often makes a causal contribution to violence, principally by increasing the likelihood that a dispute or conflict fails to be resolved peacefully by verbal means alone (US Department of Health and Human Services 2000, Stockwell et al 1995, Homel & Clark 1994). Alcohol involvement in violent offences reported to the police is above 90 per cent for incidents that occur between 10pm and 2am in public places (Ireland & Thomenny 1993). Alcohol's causal role in relation to violent behaviour has now been demonstrated by a wide variety of psychological, pharmacological and epidemiological studies (Graham & West, 2001).

Drinking can have a major impact on partners and families. Income available for basic household expenses may be reduced substantially. Family members are often isolated, with little understanding or support from others. This may lead to anxiety, depression and ill-health. Their needs are often neither recognised nor supported by the social and health care support systems.

Furthermore, many inappropriate and harmful patterns of drinking are learned in the family (Sher et al 1997, Barnes et al 1997, Abrams & Niaura 1987, Kandel 1983). Alcohol-related problems in succeeding generations of the one family are not uncommon, with younger family members acquiring particular patterns of consumption from older family members. While a genetic component may, in some instances, contribute to such problems (see page 77), social learning is a major, and often the sole, determining factor (Bandura 1976).

At the same time, families are a key resource in helping people with a drinking problem, and preventing further problems.

Alcohol is involved in about 50 percent of cases of domestic physical and sexual violence, although its role in precipitating or exacerbating violence varies. Where drinking is considerably above guideline levels, it is more likely to be perceived as contributing actively to violence. While alcohol intoxication increases the risk of violence, this should not be interpreted as a socially acceptable 'excuse' for violence in the home. Alcohol dependence also appears to play a significant role in relation to child neglect (English et al 1995).

Women who have alcohol problems often also have marital problems (Blankfield & Maritz 1990), and are less confident about resolving marital disagreement (Kelly et al 2000). Women who are alcohol-dependent report high rates of aggression in their spouses (Miller et al 1989, Miller & Downs 1993). They tend to drink more than their male counterparts do, in response to marital conflict (Olenick & Chalmers 1991).

THE INFLUENCE OF THE DRINKING ENVIRONMENT

The setting or environment in which drinking takes place has a substantial influence on the risk of injury. Physical hazards heighten risk, and the most vulnerable situations are those requiring complex reasoning and motor skills (Eckardt et al 1998).

See GUIDELINE 3

Population surveys have found that high levels of drinking are most likely to occur in particular types of venues (Single & Wortley 1993). Heavy drinkers, for example, report drinking most often in bars. Compared with the levels of harm associated with nightclubs, hotels and taverns, the lower levels of harm that are associated with such settings for drinking as restaurants and clubs have been in part attributed to the nature of the clientele (Stockwell et al 1992). Studies in Australia have confirmed that a high percentage of people involved in public-order incidents (assaults, offensive behaviour, offensive language) had been drinking alcohol within the previous few hours, and incidents were most common around the closing time of bars (Stockwell 1995). An accumulation of research over twenty years demonstrates a link between various environmental factors, and aggression and harm in licensed environments (Graham et al 1996). These include overcrowding, a predominantly young male crowd, boring entertainment, loud music, little provision of food, cheap drinks and high levels of intoxication, combined with aggressive bouncers and confrontational staff, low standards of furnishing and upkeep, and poor ventilation (Homel et al 1992, Graham et al 1996).

Much can be done to reduce the risk. Alcohol in sufficient quantities reduces a person's ability to cope with frustration. It affects perception, cognitive functioning, comprehension and communication skills, increasing the likelihood of conflict while reducing the ability to resolve conflict. It is thus critical to minimise the likelihood of conflict and frustration, not only through the design and management of licensed premises, but at any other public or private occasions that include drinking. Strategies include appropriate training of licensees and security staff, sensitive community policing, sufficient late-night public transport, management of the way people leave the premises to avoid sudden crowding on the streets (Marsh & Kibby 1992), providing non-alcoholic beverages, and providing adequate lighting and ventilation.

Providing food is another important strategy. Recent studies have shown that high risk drinking and harmful outcomes are more common for single males under 25 who have not eaten and who have been drinking at hotels or nightclubs (Stockwell 1995). Eating while drinking is well established as a means of minimising intoxication, and various studies have shown that providing food on site can ameliorate the harm from alcohol (Murgraff et al 1999, Ligon et al 1996 Stockwell et al 1992).

Responsible server programs have become common over the past decade, and have been found to be quite effective, especially if combined with enforcement

(Stockwell 1997, Putnam et al 1993, Saltz 1987). Now mandatory in some states in the United States, server training aims to alter serving practices, particularly with underage customers and people showing obvious signs of intoxication. Training covers the effects of alcohol, applicable laws, how to refuse service to obviously intoxicated patrons, and how to help customers to obtain transport instead of driving. Some, but not all, studies report more interventions with customers after server training than before, and an evaluation of Oregon's mandatory server training policy found that it achieved a statistically significant reduction in traffic crashes in that State (Holder & Wagenaar 1994).

DOES THE TYPE OF DRINK MAKE A DIFFERENCE?

Despite attempts by many investigators to identify differences in the effects of different drinks, relatively few studies have actually found such differences, in relation to either the benefits or the harms of alcohol.

It has been argued that red wine has a particularly protective effect against heart disease, but the literature in general does not support this. A large study in England, for example, found that while wine drinkers had a lower risk of heart attack than beer or spirits drinkers, their risk of death from all causes was lower and the social and life-style advantages they experienced very largely accounted for the differences (Wannamethee & Shaper 1996). The extra health benefit associated with drinking wine may result from differences in patterns of drinking, with wine usually being drunk in small amounts regularly rather than large amounts occasionally (Doll 1997). Overall, the degree of protection that alcohol confers against heart disease from middle age onwards appears to be common to all types of drink, and results from the alcohol content. While there may be other less important factors at play, associated with specific types of beverage, the evidence for such factors is inconclusive.

In relation to the harmful health effects of alcohol, there are few areas where the type of drink is important: it is the amount drunk that is of primary importance. A possible exception is in relation to cancers of the mouth, throat, larynx and oesophagus. Alcohol is involved in causing many of these cancers, probably through direct contact with the mucous membrane lining of the mouth, throat, larynx and oesophagus, and studies suggest that drinks with a higher alcohol concentration pose a higher risk (Jaber et al 1998, Doll et al 1993). One study found an association between 'moderate drinking' and cancer of the throat and oesophagus, but only for beer and spirits, not wine (Grønbæk et al 1998).

There is evidence to suggest that people who drink different types of alcoholic beverages differ in important ways. Studies indicate that, compared with beer or spirit drinkers, wine drinkers are more likely to be older, female, more educated, more affluent, to drink socially at private homes, drink less frequently and less per occasion (Gruenewald et al 1995, Single & Wortley 1993, Berger & Snortum 1985). Wine was found to be most popular for light drinkers, and beer for moderate to heavy drinkers; and most women who drank moderately chose wine, whereas women who drank heavily tended to prefer spirits (Berger & Snortum 1985). Beer drinkers were more likely than wine or spirit drinkers to drink to levels that would

impair driving, and were more likely to drink and drive than those who drank wine or spirits (Berger & Snortum 1985).

Another study found that people presenting to hospital emergency rooms (accident and emergency) with an alcohol-related injury were more likely to have consumed alcoholic beverages other than wine (Cherpitel 1997). A recent Australian study found that the beverages most commonly associated with night-time assaults and alcohol-related injury were the cheaper alcohol products—cask (not bottled) wine, and regular-strength (not low-alcohol) beer—but consumption of all beverage varieties except bottled wine was associated with alcohol-related injury or other short-term harm (Stockwell et al 1998).

Assaults mostly occur in public places and often between individuals who do not know each other, while homicides tend to take place in private contexts, among family members, friends or acquaintances. A Swedish study found that assault rate was related to consumption of beer and spirits in bars and restaurants, while the homicide rate was linked to consumption of spirits in private places (Norstrom 1998).

Although the evidence is limited, distilled alcohol (spirits) appears more likely than brewed alcohol (beer) to evoke aggression (Smart 1996). Such effects, however, have more to do with perceptions and beliefs about the beverages than with their pharmacological properties (Graham et al 1996). While certain patterns of problems appear to be fairly consistently associated with particular beverage types, it remains unclear whether it is the inherent properties of the beverage that are important, or the characteristics of the drinker and the setting.

DIFFERENCES IN SUSCEPTIBILITY

Women

Women are more susceptible to the effects of alcohol than men are, for a number of reasons. Alcohol, when it is absorbed, is distributed throughout the body's total water content; however, women, on average, have a smaller body mass, so that a given amount of alcohol is distributed over a smaller body volume. Furthermore, they have a higher proportion of body fat, and alcohol is not taken up by fatty tissues. The ability to break down alcohol is limited by the size of the liver, and women on average have smaller livers than men.

See Guideline 1

The higher concentration of alcohol in the body leads to a greater degree of intoxication for a given amount of alcohol, and this has serious implications in terms of safety. The culture of 'thinness' and dieting that particularly affects young women, also places them at increased risk of the effects of alcohol. Dieting leads to a disproportionate loss of body fluid, so that a given quantity of alcohol in a person who is dieting leads to a higher concentration of alcohol within the body.

Women's smaller liver size means that they are vulnerable to liver damage and cirrhosis at lower levels of drinking. Alcohol also increases women's risk of breast cancer (see page 73). Given that this is the most common cancer among Australian women, and a significant cause of death, this risk is of great significance in terms of the overall public health impact of alcohol use.

As discussed earlier (see page 35), this increased susceptibility of women was shown clearly in the meta-analysis of studies of mortality in relation to alcohol that was carried out for these guidelines (Lawrence & Holman 1999). The risk of death rises about 10 percent above that of non-drinkers at an average intake of 20 to 29 grams of alcohol per day (2-3 standard drinks), and increases steadily with increasing consumption, to be about 40 percent higher than abstainers at an average daily consumption of 40 to 49 grams (4–5 standard drinks). For men, the risk does not start to rise above that of abstainers until consumption reaches 40 grams (4 standard drinks) or more per day, and even at 60 grams (6 standard drinks), the risk is only a little over 30 percent higher than for abstainers.

Unborn children: alcohol and pregnancy

The evidence on alcohol in pregnancy is discussed in more detail on page 77. In summary, however, when a pregnant woman drinks, the alcohol in her blood stream enters that of her unborn child, and, in sufficient quantities, this can cause problems for the unborn child. The first few weeks after conception are the most vulnerable period.

See Guideline 11

The major concern relates to heavy drinking episodes and occasional peak blood alcohol levels, which increase the risk of miscarriage, low birth weight, cognitive defects and congenital malformations. Drinking within the guideline levels is unlikely to have any significant effect on the child. Good antenatal care, good diet, sufficient fluid intake, and not smoking are all important in containing risk and providing a healthy environment for the unborn child.

Young people (up to about 18 years) and young adults (about 18–25 years)

While alcohol consumption among Australian adults has decreased over the past two decades, the patterns and levels of drinking among your people and young adults are of major concern. More young people are drinking alcohol, drinking at an earlier age, and drinking at increasingly higher levels. As the survey results quoted in Chapter 3 (page 60) show, between one quarter and one third of 14–24 year olds drink in a high risk manner, more than any other section of the Australian community.

See Guidelines 2, 3, 9 & 10

Young people and young adults predominate in figures on alcohol-related injury. Between 1990 and 1997, for example, over half of all serious alcohol-related road injuries (52 percent) were sustained by people aged 15–24 years, and a further 23 percent sustained by young adults in the following age bracket, of 25–34 years (Chikritzhs et al 2000) (see Figure 3).

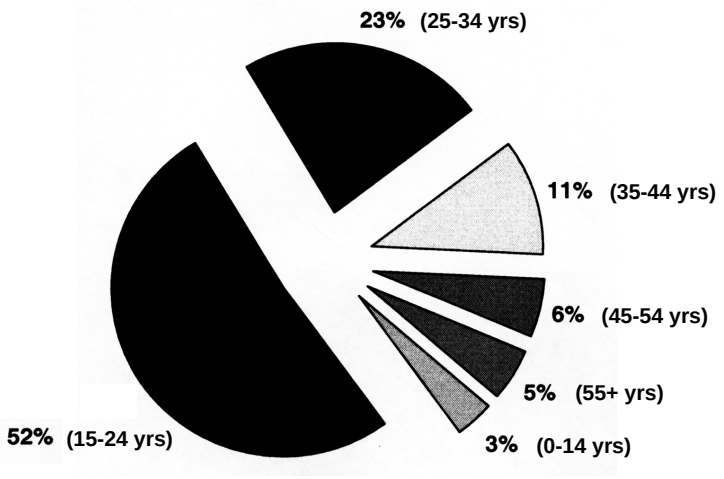

Figure 3: Overall age distribution among alcohol-related serious road injuries occurring on Australian roads (excluding Victoria), 1990–1997

Source: National Alcohol Indicators Bulletin No 2, May 2000

A number of factors work together to place young people at considerably increased risk of harm from drinking.

Children have greater vulnerability to alcohol than adults do. As well as usually being physically smaller, they lack experience of drinking and its effects. They have no context or reference point for assessing or regulating their drinking, and, furthermore, they have built no tolerance to alcohol.

The advent of puberty is often accompanied by a range of risk-taking behaviours as adolescents explore independence, and peer values and norms take on a much greater importance. While this is a normal and generally healthy phase, it can considerably heighten the risk associated with drinking. In later adolescence, risk-taking behaviour is often coupled with potentially dangerous activities such as surf-board riding or abseiling, and, again, alcohol can considerably increase the risk.

In an Australian study, having just finished high school was associated with episodes of very high levels of drinking and intoxication (Zinkiewicz et al 1999). Over 75 percent of males and 60 percent of female school leavers reported getting drunk on most or all days or nights of their 'schoolies week' (Smith & Rosenthal 1997).

Studies in the United States have noted that drinking by college students is associated with automobile crash injury and death, suicide and depression, missed classes and decreased academic performance, loss of memory, blackouts, fighting, property damage, peer criticism and broken friendships, date rape, unprotected sexual intercourse that places people at risk for sexually transmitted diseases, HIV infection and unplanned pregnancy (Williams & Knox 1987).

Injuries caused by accidents or violence are the most common types of harm incurred by young people and young adults as a result of drinking. A recent Australian study of university students found that over one third of those surveyed, reported sustaining an injury as a result of their drinking in the previous 12 months (Roche & Watt 1999). In a study of United States college students presenting at an emergency department with an injury, 16.4 percent of the injuries were alcohol-related (Wright et al 1998). Students in their first year (ie younger students) were at greater risk. People other than the drinkers are also affected. For example, Weschler et al (1994) found that, at colleges with high 'binge' levels, the students who did not 'binge' were more likely than those at schools with low 'binge' levels to experience assaults, property damage, interrupted sleep, unwanted sexual advances, serious quarrels and having to take care of a drunken student.

Risky sexual behaviour and increased vulnerability to coercive sexual activity are also associated with alcohol use. In New Zealand, alcohol misuse was significantly associated with unprotected intercourse and sexual activity before age 16 (Fergusson & Lynskey 1996). In Massachusetts, 44 percent of sexually active teenagers said they were more likely to have sexual intercourse if they had been drinking, and 17 percent said they were less likely to use condoms after drinking (Strunin & Hingson 1992).

The three leading causes of death among adolescents—unintentional injuries, homicide, and suicide—are all associated with alcohol (United States Department of Health and Human Services 1991).

Death by alcohol overdose is also a risk (Poikolainen 1997), and is of particular concern among young drinkers. Their generally smaller physique means that they metabolise alcohol substantially more slowly than a larger individual does, and less efficiently than an adult. In particular, young women metabolise alcohol much less efficiently than males (see page 45). Young Australian drinkers are known to prefer to drink spirits, and these, with their higher alcohol concentration, induce intoxication much more quickly than more dilute forms of alcoholic beverages. This combination of factors suggests that special safeguards are needed for young people.

Strategies that minimise risk for young people are of crucial importance, and learning about drinking and its effects is central in fostering drinking behaviour that minimises or contains risk (Houghton & Roche, 2001). Surveys have found relatively high rates of alcohol-related problems among adolescents in countries where alcohol is officially not available to people of that age. In contrast, cultures that introduce young people to alcohol in a gradual and supported way, within a family setting, tend to avoid the acute problems experienced by many young people

within Anglo-Celtic cultures when they start to drink. At the same time, there is evidence from research in the United States that people who start drinking at a young age, particularly those who start in their early teens, are more likely to be involved in an accident while under the influence of alcohol. This risk, whilst greatest in the years of youth and young adulthood, may persist throughout adulthood (Hingson et al 2000).

A few school programs designed to educate young people about alcohol, to help them to make informed and safe decisions about drinking, have had some limited success in modifying risky behaviours (eg McBride et al 2000, Perry & Kelder 1992). Such programs can have some impact on peer norms among young people. They may, for example, help to establish low-risk drinking or no drinking as normal and acceptable options. In raising awareness, education programs also help to pave the way for other approaches to minimising alcohol-related harm (Roche & Watt 1998).

Alcohol has no immediate benefit for young people and young adults in protecting against heart disease, as very few people show clinical signs of atherosclerosis (hardening of the arteries) below 40 years of age. The one exception to this is a small number of people with a strong family history of atherosclerotic heart disease in young adulthood. It is not yet known whether a regular pattern of drinking is an advantage to young adults in reducing their risk of heart disease in later life. Any potential benefit needs to be weighed against the significant risk of death or injury from other alcohol-related causes, notably trauma, in young adulthood.

Older people

In general, older people tend to drink less than people do in their younger or middle years. Drinking for many, however, remains an important and enjoyable part of a social life that often expands in retirement. There is now considerable research to indicate a positive association between low risk drinking and increased life expectancy (Lawrence & Holman 1999).

See Guideline 8

The proportion of older people in the community is increasing, but data on the effects of drinking in older age groups are scarce, as studies tend to exclude people over about 65 years. A number of factors, however, contribute to an increased susceptibility to harm in relation to alcohol:

- As people move into older age brackets, their tolerance for alcohol tends to decrease. Alcohol, when it is absorbed, is distributed throughout the body's total water content, but the volume of total body water decreases with age, so that a given amount of alcohol produces a higher blood alcohol concentration. This effect is relevant to people aged over about 70 or 75 years, although there is considerable variation from person to person.
- Coupled with this may be a reduced neurological tolerance for alcohol.
- Older people often take regular medication, which may interact with alcohol to cause unpleasant or dangerous side effects, increase the effect of the

alcohol, and/or decrease the effectiveness of the medication. The risk is considerably greater when a number of medications are involved (see page 55).

- Falls are an increasing risk with advancing age, and this risk is also increased by intoxication, with potentially serious and disabling consequences.
- Driving may be influenced by the effects of ageing, particularly visual loss and slowed reaction time. Again, any risk in this area can be increased by alcohol, with the potential to affect others as well as the driver.

At the same time, the benefits of alcohol in preventing heart disease accrue from about 40 years of age for males and 50 for females, and as the risk of heart disease increases with age, so these benefits become more pertinent. The maximum benefit, however, can be gained by a low level of drinking, approximately one to two standard drinks every day for men, and less than one per day for women.

People with a mental health problem and/or sleeping disturbance

Drinking alcohol can have many consequences for people who have or are prone to mental illness or mental health problems. The effects of alcohol depend in part on how much is consumed. In addition, however, the effects may change dramatically in the hours following drinking. After drinking, many people experience positive effects for a short time, while their blood alcohol concentration is rising (Eckardt et al 1998). However, a few hours later or the next morning, even after only a moderate amount of alcohol, it is common to experience increased anxiety and depressed mood (Kushner et al 2000, Eckardt et al 1998).

See Guideline 6

Furthermore, even one or two drink s, while they have been shown to help sleep in the short-term, may disrupt the later part of the sleep cycle, sometimes leading to early morning waking (Castaneda et al 1996). Disruption of sleep can be a trigger for a variety of mental health problems (Castaneda et al 1996).

It has been known for many years that people with mental health problems are at particular risk of experiencing problems relating to alcohol. Similarly, people diagnosed as having significant alcohol dependence are more likely to suffer from mental health problems (Kushner et al 2000, Lynskey 1998).

In particular, there is a strong relationship between alcohol use, anxiety and depression, and drinking is known to significantly increase the risk of suicide in people with depression (Lynskey 1998). While the literature in this area is large and complex (Eckardt et al 1998), it is clear that alcohol can provide temporary relief for people experiencing significant anxiety, and people with significant alcohol dependence are particularly likely to report that alcohol helps to reduce their anxiety (Eckardt et al, 1998). Again, however, the timing of alcohol's effects is crucial. In the longer term, continued drinking over two days or more tends to increase anxiety and depression overall, even though each further drink may temporarily reduce this a little (Kushner et al 2000, Stockwell & Bolderston 1987).

Numerous studies have shown that when people with significant alcohol dependence stop drinking entirely, their mood usually worsens over the first few hours and days, but after two to three weeks it is greatly improved (Kushner et al 2000, Lynskey 1998).

Alcohol intake is also a very significant issue for the treatment of people who suffer from schizophrenia. Not only is risky drinking more common among people with a diagnosis of schizophrenia (Hulse et al 2000), but there is also evidence that even low risk alcohol consumption can worsen the symptoms of this condition as well as interfere with the effectiveness of some standard medications (Castaneda et al 1998). Furthermore, improved treatment outcomes have been achieved when alcohol misuse and the schizophrenia have been tackled in an integrated fashion (Mueser & Kavanagh, 2001).

In addition to all these effects, alcohol, even at low levels (one or two drinks), can interact adversely with most of the medications commonly prescribed for treatment of mental health problems (Castaneda et al, 1996).

In general, people experiencing problems with sleep and/or mental health problems need to be cautious in their use of alcohol. They should discuss with their doctor or pharmacist the possible interactions between alcohol and any medications they may be given. In many instances, the best outcomes may be obtained if the person stops drinking alcohol entirely for a time.

Aboriginal peoples and Torres Strait Islanders

Alcohol has a significant impact on health within many Aboriginal and Torres Strait Islander communities. Problems stem from both volume and patterns of drinking. Drinking within these communities is, in large measure, an expression of and further contributor to severe socio-economic disadvantage and this, in turn, has its deeper roots in the ongoing experience of dispossession, spiritual and cultural dislocation.

At the same time, there is a substantially higher proportion of abstainers among Aboriginal and Torres Strait Islander communities than in the non-Aboriginal population (Commonwealth Department of Health and Human Services 1994). Similarly, some Aboriginal and Torres Strait Islander communities have a far lower level of alcohol-related problems than others.

The adverse effects of high-risk drinking are further exacerbated by smoking, poor nutrition, obesity, poor living conditions, exposure to violence and environmental hazards, and limited access to and use of health and social services. The cumulative impact of all of these factors is reflected in a life expectancy at birth that is, for males, around 17 years less than that of the overall Australian population, with a slightly wider gap for females (Deeble et al 1998). Alcohol, in combination with separation from family, mental health problems and imprisonment, is a significant contributor to the very high rates of suicide in some Aboriginal and Torres Strait Islander communities (eg Hunter 1991, 1999). It has been estimated that, if either unsafe alcohol consumption or unsafe smoking were eliminated, there would be a

considerable increase in Aboriginal life expectancy, and if both were eliminated, the increase would be slightly greater still (an average of 5.9 years for all males and 3.4 years for all females) (Arnold-Reed et al 1998).

The extent of high risk drinking among those who do consume alcohol has presented problems in communicating the principles of drinking guidelines, standard drinks and safe or responsible consumption. There is, however, growing interest in the role of secondary prevention in this endeavour, broadening the present focus on primary prevention and tertiary prevention/rehabilitation (Brady 1995).

In response to their severe problems related to alcohol, Aboriginal peoples and Torres Strait Islanders have established initiatives to encourage non-harmful use, to limit access to alcohol, and to establish 'dry' areas and communities. These are only one small part of major efforts by Aboriginal peoples and Torres Strait Islanders to improve their health status, and their social and economic circumstances.

Levels of drinking among Aboriginal peoples and Torres Strait Islanders, as measured by survey, are summarised on page 62.

People from different cultures

It is difficult to draw many conclusions about patterns of drinking and risk among people from diverse cultural backgrounds living in Australia, given the limited studies available. The data suggest, however, that people whose first language is other than English are more likely than English speaking citizens to be non-drinkers.

It might be expected that people coming to Australia from another country would bring with them the patterns of drinking and risk from that culture, possibly compounded by a sense of isolation or dislocation. People coming from cultures where the consumption of alcohol is generally considered unacceptable (including some Muslim countries) may face particular pressures in the Australian context to increase their consumption. For some groups or individuals, being introduced to a heavy drinking culture may present difficulties.

The children of immigrant parents might be expected to move closer to Australian norms in drinking as in a wide range of other behaviours, opening the potential for conflict within families. This has been found to be the pattern among second generation migrants to the United States (Clark & Cahalan 1974). Others may be advantaged by a culture that enables children to learn about drinking and alcohol gradually, in a supported family environment.

Home brewing is practised within a number of cultures, and this has the potential to create difficulty in monitoring the amount of alcohol consumed, as the alcohol content of these drinks will probably be unknown and variable.

The flushing response is an inherited characteristic seen most often in people of Asian descent. The flushing is caused by a slower metabolism of alcohol, which also results in other side effects as well as reduced tolerance. While in most people,

alcohol within the body is broken down into acetaldehyde, which is almost immediately broken down further, this second step is interrupted in people with the flushing response. Acetaldehyde builds up in the body and rapidly causes unpleasant effects, including nausea and vomiting. Many people with the flushing response choose not to drink for this reason.

Occupational groups

Different occupational groups tend to display different levels and patterns of drinking, suggesting the importance of social and environmental influences in and determining health and social outcomes, and the relevance of occupational settings for interventions to address problem drinking.

National Health Survey data collected in 1989–90 by the Australian Bureau of Statistics (ABS 1991, quoted in Hagen et al 1992), while now over a decade old, provide the most comprehensive information available on occupational and industry differences in Australia. In general, the data showed tradespeople to be the heaviest drinkers, but there were substantial variations within occupational and industry groups, and between men and women in the same occupations. NHMRC guideline consumption levels for low risk of chronic harm were exceeded by:

- almost 25 percent of men working as building tradesmen, waiters and bar staff, construction and mining labourers, and food tradesmen; and
- over 20 percent of women employed as specialist managers (eg. finance, personnel, public policy or sales managers, school principals, etc) or sales representatives.

Percentages for men and women by industry are shown in Table 6 and Table 7.

Hagen et al (1992), in their Victorian study, found indications of consumption levels above NHMRC recommended levels among significant proportions of workers in a range of occupations, including police, forklift operators, truck drivers and metal tradesmen. A study at two mining-related worksites in Western Australia found over 40 percent of workers reported usually drinking above these levels (Midford et al 1997).

Table 6: Top ten industries for men drinking at risky or high risk levels for long-term harm (as specified in these guidelines)

Industry	% drinking above guidelines levels
Fishing and hunting	32.2%
Storage	31.5%
Services to agriculture	27.0%
Restaurants, hotels and clubs	26.4%
Special trade construction	26.1%
Forestry and logging	24.2%
Coal	22.8%
Services to transport	21.7%
General construction	21.5%
Road transport	20.2%

Table 7: Top ten industries for women drinking at risky or high risk levels for long-term harm (as specified in these guidelines)

Industry	% drinking above guidelines levels
Air transport	29.9%
Services to transport	17.0%
Paper, paper products, printing and publishing	14.9%
Wholesale trade	14.7%
Wood, wood products and furniture	14.7%
Electricity and gas	13.6%
Other community services	12.4%
Entertainment and recreational services	12.4%
Metallic minerals	12.3%
Restaurants, hotels and clubs	11.7%

INTERACTIONS WITH MEDICINES AND OTHER DRUGS

Illicit or recreational drugs

The two substances of particular concern in this category are heroin and benzodiazepines. People using heroin are at particular risk if they drink, especially if they engage in heavy bouts of drinking. A high proportion of people who die from heroin overdose also have a high blood alcohol concentration, and the risk of

serious brain damage from overdose is similarly increased. Benzodiazepines in combination with alcohol also pose a significant risk, and the combination of alcohol, heroin and benzodiazepines further increases an already high risk.

Suicide and self-harm are linked both to heavy drinking and to illicit drug use, and drinking and drug use together pose a particular risk. The combination of alcohol with other drugs (particularly cannabis) also poses risks for driving and other complex tasks, with the potential to harm others as well as the drinker.

In the longer term, many people who have used illicit drugs have contracted chronic hepatitis C infection that, in some people, will lead to severe liver disease. Alcohol dramatically increases both the risk and the progression of severe liver disease in these people. The risk increases with the level of drinking (see page 75).

There is also evidence that people who become dependent on one drug are more likely to become dependent on another, including alcohol.

Medications

Alcohol can interact with a wide range of prescribed and over-the-counter medications and herbal preparations, and this can alter the effect of the alcohol and/or the medication. With some medications, interactions may occur even with one drink. Some interactions that cause drowsiness or dizziness with even one drink have serious implications for driving or operating machinery.

See Guideline 7

People taking medications of any type should read the labels and the enclosed pamphlets carefully to check for possible interactions with alcohol. Health care providers should be aware of the possibility of interactions, both in drugs they prescribe and in the medications from other sources that people may be taking. Some people may need to limit their drinking or stop drinking alcohol entirely.

Areas of potential concern include:

- antidepressants and other medications prescribed for the management of mental illness and mood disorders, as they may cause a range of side-effects when combined with alcohol;
- antihistamines, which may cause drowsiness in combination with alcohol;
- benzodiazepines, as they, like alcohol, also depress the central nervous system;
- medicines that already contain alcohol, thereby increasing the amount of alcohol consumed;
- cough medicines that contain antihistamines, codeine and alcohol;
- drugs for high blood pressure or angina, which can cause dizziness when combined with alcohol;

- medications to lessen the risk of blood clotting and stroke, as drinking can affect the control of blood clotting;
- some medications used for epilepsy, arthritis, and diabetes;
- arthritis medicines, which may cause stomach upsets in combination with alcohol;
- some antibiotics.

More information can be found at the MIMS website, <http://www.mims.hcn.net.au>.

3 PATTERNS OF DRINKING IN AUSTRALIA

A HISTORICAL PERSPECTIVE

Patterns of drinking in Australia have their roots in the earliest days of the colony, when males made up a large proportion of the population, and the reliability of demand for rum made it at times the principal currency. In the 1850s, the gold rushes again threw together masses of men without women, and drunkenness was a feature of the diggings. The place of heavy drinking as part of the Australian national myth was further entrenched by the tradition of itinerant bush workers—shearers, drovers, stockmen—and miners. Drinking was a male group activity, part of the tradition of male 'mateship', and 'shouting' a round of drinks—with its further obligation to share in and continue drinking—grew out of this. Hotels, where the drinking was done, were a male domain; women drank little if at all, and at least until the 1960s, public drunkenness was the defining issue in discussion of alcohol problems. The '6 o'clock swill' at hotel closing time only served to reinforce these patterns (Room 1988).

Heavy drinking continued to mark a clear cultural divide between restless mateship and settled family life until well after the Second World War. The temperance movement became a strong force for change, but it also became associated with the caricature of the 'wowser' from the 1880s onwards, and this old association has, until very recently, been an effective deterrent to saying anything negative about alcohol (Room 1988).

Since the 1960s, there has been a gradual change in these patterns. Women have re-entered Australian drinking places, the restaurant industry has been transformed, and alcohol has penetrated into adults' daily routines. Where once it was never drunk with meals, now it often accompanies a meal. The range and sophistication of drinks available has increased enormously, and alcohol, previously available only from hotels, is now sold through a wide range of outlets (Room 1988). People drink at home more often, rather than only in hotels, while hotel environments have improved enormously and are now very much more 'women-friendly' places. There has also been a substantial increase in women's drinking since the 1970s (eg Corti & Ibrahim 1990).

Levels of consumption across the population fluctuated over the years. Consumption of spirits peaked during the gold rushes, but by the end of the 19th century Australia had become a beer drinking country, and per capita consumption of pure alcohol had halved. This fall continued, and per capita alcohol consumption reached its lowest point in 1932, at under 2.5 litres of pure alcohol per annum (Room 1988). By 1981, it had climbed back to 9.7 litres (Higgins et al 2000), at least as high as during the 1850s gold rush era (Room 1988). By the 1990s consumption had fallen slightly again, and between 1992 and 1998 it was relatively stable, fluctuating between 7.6 and 7.8 litres per capita (Australian Bureau of Statistics 2000).

Over three quarters of Australians now report that they drink alcohol. Most of these people drink at low risk levels most of the time, but a high proportion of people also drink, occasionally, at levels that may place them at high risk.

While Australia remains a beer drinking country, wine consumption has increased more than threefold since the late 1940s, and there has been a general trend in the industry towards beverages that are 'lighter' in terms of colour, caloric content, and alcohol content (Australian Bureau of Statistics 2000, Room 1988).

The substantial change in patterns of alcohol consumption over recent years has been paralleled, in the past decade, by an important shift in the public health perspective on drinking, moving from an exclusive focus on the amount consumed to incorporating patterns of consumption. For instance, there is now a greater appreciation of the importance of drinking alcohol in a setting and in a manner that helps to minimise the risk of harm, with a growing emphasis on drinking with food, in a social or family setting, and not drinking to intoxication.

Community attitudes have also changed, and society's permissive acceptance of irresponsible patterns of drinking has declined over the past 20 years. In particular, there is increased acceptance that people who are driving should not drink.

The particular area of concern now is drinking among young people. While a proportion of young people do not drink, many are drinking at an earlier age, drinking heavily more often, and placing themselves at significant risk of harm (see pages 46 and 61).

CURRENT ATTITUDES TO ALCOHOL

The 1995 National Drug Strategy Household Survey (Commonwealth of Australia 1996) looked broadly at the public's perceptions of drugs and their effects. When those who were surveyed were asked to name the drugs they thought of when people talked about 'a drug problem', some 38 percent mentioned alcohol, while 34 percent thought (erroneously) that alcohol was the drug that causes most deaths.

Excessive drinking was seen by 30 percent of the population to be the major drug-related cause for concern to the community. The reasons most frequently cited were:

- road safety and drink driving issues;
- too socially acceptable or widespread;
- causes problems within families;
- innocent people may be harmed;
- unpleasant or irresponsible behaviour, or people lose control;
- violence, aggression or fights;
- domestic violence or child abuse;
- addiction or dependency;

- affects young people, or young people are targeted;
- generally bad for, or a high cost to, the community;
- causes general health problems or deterioration.

Over half the population (54 percent) thought that alcohol was 'okay' to use on a regular basis', and when asked how much an adult male and adult female could drink every day before their health is affected, less than half underestimated the risk, that is, gave amounts more than the NHMRC guideline recommendations.

Research carried out for a National Alcohol Campaign (Shanahan & Hewitt 1999) focused on young people and young adults, involving interviews with teenagers, young adults, parents of teenagers, and teachers. The prevailing attitude across all these groups was that the benefits of alcohol far outweigh the disadvantages, with the perceived benefits including the enjoyment experienced through drinking, its use as a social lubricant to ease awkwardness, and its relaxing qualities. Types of harm associated with drinking included public and domestic violence, property destruction, and both short-term and long-term health problems. The study found far greater concern about social harm than had been expressed in a similar study 12 months earlier.

The vast majority of teenagers felt that more people of their age were drinking too much alcohol, compared with 12 months earlier, though few felt it to be a problem for themselves. There was general concern that teenagers were drinking at a younger age (under 15 years), and that self-inflicted harm, injury and risk-taking behaviour were all increasing, along with sexual abuse and unwanted sexual advances resulting from alcohol use. Young adults (up to 25 years) were also generally thought to be drinking more, and more likely to be in risky drinking environments. Young drinkers, however, had low awareness and understanding of the more severe health problems related to alcohol, particularly the longer-term problems. They also had little understanding of the concept of a standard drink.

DRINKING IN THE AUSTRALIAN POPULATION

Household surveys carried out from 1988 to 1998 showed that, while there was a small increase in the proportion of Australians drinking regularly, there was a significant decline in levels of drinking across the adult population (ie aged 20 years or more) (Commonwealth of Australia 1995, AIHW 1999, Adhikari & Summerill 2000).

The 1988 to 1995 surveys showed evidence of some favourable changes in the drinking patterns of Australian adults. A significant proportion of alcohol-related harms occur as a result of occasional heavy drinking sessions by people who usually drink within guideline levels, and it would seem that prevention strategies have had some effect in changing behaviours in this area. There was also an increase in abstinence over these seven years.

The report of the 1998 survey noted the following changes that occurred in the three years from 1995 to 1998 in the drinking status of Australians population aged 14 and over (AIHW 1999):

- The proportion of people who drank alcohol regularly (ie at least one day per week) increased from 44 percent in 1995 to 49 percent in 1998. In 1998, males were more likely than females to drink regularly (59 percent versus 39 percent).
- The proportion of people who drank alcohol occasionally (ie less than one day per week) decreased from 34 percent in 1995 to 32 percent in 1998. In 1998, females were more likely than males to drink occasionally (39 percent versus 25 percent).
- The proportion of ex-drinkers remained constant at about 10 percent.
- The proportion of people who had never consumed a full glass of alcohol declined from 12 percent to 10 percent.

Patterns of drinking differed with age. In particular, the 1998 survey found that usually drinking above guideline levels, on the days when drinking occurred, was:

- most common in those aged 14 to 19 years (67 percent) and declined in each succeeding decade to 15 percent in those aged 60 years and older;
- much more common in females than males from age 14 to 39 years, slightly more common in males than females from age 40 to 59 years, and more common in women than men over the age of 60 years.

In addition, the 1998 survey found that:

- between the ages of 20 and 59 years, approximately one in every two people was a regular drinker;
- among teenagers, over two-thirds had drunk alcohol within the last 12 months, 30 percent were regular drinkers and 41 percent were occasional drinkers. Males were more likely than females to be regular drinkers (33 percent versus 27 percent).
- from age 20 onwards, most drinkers were regular drinkers. Within a given age group, males were, on average, over 50 percent more likely than females to be regular drinkers.

An analysis of consumption patterns in 1998 showed that almost half of all recent drinkers usually drank 1–2 standard drinks on any one occasion. Male drinkers were more likely than females to drink alcohol every day (14.5 percent versus 6.4 percent).

Nevertheless, a subsequent study based on results of the 1998 survey data found that:

- over half the alcohol consumed in Australia in 1998 was drunk at levels that posed short-term health risks to the drinker, while 39 per cent was drunk at levels that posed a risk to health in the long term;

- 46 per cent of male and 32.5 per cent of female drinkers drank at least once a month at levels that placed them at risk of short-term harm (eg injury) from drinking;
- 9 per cent of male and 9 per cent of female drinkers were at risk of long-term health problems due to alcohol;
- people aged below 25 years had the riskiest drinking patterns (Heale et al 2000).

The authors emphasise that these estimates are very conservative, since the alcohol consumption reported in the 1998 survey accounted for less than half the alcohol sold in Australia in that year.

DRINKING AMONG AUSTRALIAN YOUNG PEOPLE AND YOUNG ADULTS

The figures above make it clear that drinking among young people and young adults is the area of greatest concern.

Estimates by Heale et al (2000), based on the 1998 National Drug Strategy Household Survey data, found that at least two-thirds of the alcohol consumed by people under 25 years of age poses a risk of short-term harm. A smaller but significant amount of this consumption also poses a risk of long-term health problems (Heale et al 2000).

The study found that young adults aged 18 to 24 years were the group most likely to be current drinkers (87.5 percent); that is, they had had at least one drink in the last 12 months. Among these current drinkers:

- over two-thirds of males (69 percent) and over half of females (52 percent) aged 18 to 24 years drank at least once a month at levels that placed them at risk of short-term harm;
- this included 27 percent of males and 13 percent of females who drank at high risk levels, and a further 42 percent of males and 39 percent of females drinking at risky levels (as defined on page 5, Table 1);
- 13.6 percent of male drinkers and 10.1 percent of female drinkers aged 18 to 24 years were drinking at levels that placed them at risk of long-term harm (Heale et al 2000).

While there were fewer current drinkers in the 14 to 17 year age group (62 percent), the numbers in this group drinking at levels that placed them at short-term harm were of considerable concern. Among current drinkers aged 14 to 17 years:

- 41 percent of males and 44 percent of females drank at least once a month at levels that placed them at risk of short-term harm;
- this included 14 percent of males and 10 percent of females who drank at high risk levels, and a further 27 percent of males and 34 percent of females drinking at risky levels (as defined on page 5, Table 1);.

- 4.2 percent of males and 1.6 percent of females were drinking at levels that placed them at risk of long-term harm (Heale et al 2000).

In general, after early adulthood the likelihood of drinking at high risk levels decreases with age. Again, the authors emphasise the very conservative nature of all these estimates, since the alcohol consumption reported in the 1998 survey accounted for less than half the alcohol sold in Australia in that year (Heale et al 2000).

ABORIGINAL PEOPLES AND TORRES STRAIT ISLANDERS

A survey of urban Aboriginal peoples and Torres Strait Islanders (Commonwealth Department of Health and Human Services 1994) showed that, compared with the general urban adult community:

- Aboriginal peoples and Torres Strait Islanders were more likely to be non-drinkers (38 percent versus 28 percent) and to drink less frequently;
- those who drank were more likely to drink at high or very high risk levels (82 percent of drinkers versus 28 percent of drinkers in the general urban population).

A high percentage of non-drinkers were ex-drinkers (22 percent of non-drinkers compared with 9 percent of non-drinkers in the general community). The majority of Indigenous drinkers reported some attempt to reduce their levels of alcohol consumption over the previous year.

DRINKING VENUES

Age and sex are important factors influencing peoples' choice of drinking location (see Table 1). The 1998 National Drug Strategy Household Survey (Adhikari & Summerill 2000) showed that older people were more likely than younger people to prefer drinking in their own home, restaurants or cafes. In contrast, young people aged 14 to 19 years were most likely to drink alcohol at parties or at a friend's house. Licensed premises were preferred more by males than females, with the reverse pattern for restaurants and cafes. More uncommon venues included the workplace (reported by 6 percent of current drinkers, and 10 percent of those aged 20 to 39 years), parks and other public places (4 percent of current drinkers, but 11 percent of young people aged 14 to 19 years), and in motor cars or other vehicles (3 percent of all current drinkers, but 6 percent of those aged 14 to 19 years).

See GUIDELINE 3

Table 8: Where alcohol is usually consumed: proportion of recent drinkers aged 14 years and over by age and sex, Australia, 1998

	Age group						
	14–19	**20–29**	**30–39**	**40–49**	**50–59**	**60+**	**All ages**
	(percent)						
Own home	49.4	68.0	81.6	86.9	85.2	81.2	77.3
Friend's house	54.7	62.5	60.7	52.2	49.6	41.2	54.3
Parties	76.7	66.9	56.5	43.6	41.1	31.1	51.8
Restaurants/cafes	21.1	58.2	60.7	51.4	50.5	43.9	50.7
Licensed premises	42.7	76.0	56.3	47.1	42.5	36.7	52.4
Workplace	3.8	9.7	9.7	5.0	4.5	1.6	6.3
Public places	10.5	4.9	3.3	3.6	2.5	1.6	4.0

Source: Adhikari P, Summerill A (2000) *1998 National Drug Strategy Household Survey: Detailed Findings.* AIHW cat no. PHE 27. Canberra: AIHW (Drug Statistics Series No.6)

Other locations that were cited as common places for drinking, in a survey of young people aged 15 to 17 years, included suburban parks, beaches, and bushland (Shanahan & Hewitt 1999). These latter preferences by very young drinkers usually necessitate off-premise purchases and travel to a particular destination.

Different venues pose different types and levels of risk. In particular, some licensed premises are a concern for both alcohol-related violence and drink-driving (Stockwell 1997).

4 LEVELS OF RISK AND BENEFIT: EVIDENCE FROM THE LITERATURE

ALCOHOL-RELATED INJURY: THE SHORT-TERM RISK

Although it has long been considered that the major consequences of alcohol are its long-term effects, recent studies show that injury and death due to trauma are probably even more important consequences (Single et al 1999b). Drinking alcohol has been associated with risk of injury in many settings, including vehicle and cycling accidents, incidents involving pedestrians, falls, fires, sports and recreational injuries, and violence (United States Department of Health and Human Services 1997, Martin & Bachman 1997, Hurst et al 1994, Freedland et al 1993, Hingson & Howland 1993, Cherpitel 1992, Hingson & Howland 1987, Martin 1982). Presence of alcohol in the body at the time of injury may also be associated with greater severity of injury and less positive outcomes (Li et al 1997, Fuller 1995).

See GUIDELINES 1, 2, 3, 8, 9, 10

The blood alcohol level resulting from an episode of drinking will depend not only on the amount of alcohol and the length of time over which it is drunk, but also on the person's body size and constitution (eg volume of water in the body), their drinking experience, and whether or not the alcohol was taken with food (Kalant & Khanna 1989, Eckardt et al 1998, United States Department of Health and Human Services 1997). Studies suggest that peak blood alcohol concentration is halved for a given quantity of alcohol, if that alcohol is drunk after a meal (Eckardt et al 1998). Two- to three-fold differences also exist as to how rapidly different individuals metabolise and eliminate comparable doses of alcohol (Eckardt et al 1998); and one report cited genetic factors as the most important influence determining individual differences in peak blood alcohol level and alcohol-clearance rate (Martin et al 1985). Because of these factors, blood alcohol concentration can be only approximately determined from number of drinks and timing alone.

Risk of injury starts to increase at relatively low levels of intake—the studies show no threshold—and it increases as the level of intake increases (Cherpitel et al 1995). The risk increases more for people whose level of consumption varies significantly from time to time, and the risk is highest for those who occasionally drink much more than their usual amount (Treno & Holder 1997, Treno et al 1997, Gruenewald et al 1996, Gruenewald & Nephew 1994).

In addition to the risk of accident and injury when blood alcohol levels are raised, recent studies have also indicated that, in areas such as cognitive function, attention span, and reaction time, substantial impairment can exist well after alcohol has been metabolised and passed from the body. Such temporary impairment, and its attendant risk, is the result of a 'hangover' effect (Anderson & Dawson 1999).

Recent Australian research has demonstrated that women have an enhanced risk of sustaining an injury after drinking: at any given level of alcohol intake over six hours it was found that the risk of injury was substantially higher for women than for men (McLeod et al 1999). This evidence suggests that lower levels of drinking should be recommended to women to minimise the short-term risk of harm.

A recent Australian study comparing blood alcohol concentrations of drivers involved in motor vehicle crashes found that crash risk increased 5-fold at 0.05, 25 times at 0.08 and 80 times at 0.15 (Ryan 2000). The study, using police 'Booze Buses' at carefully selected times and places found that, of 8,616 drivers tested, 1.9 per cent were in excess of 0.05. High risk times for excess blood alcohol concentrations were Friday nights and after midnight any day of the week.

While there is good research data that show slightly improved cognitive and psycho-motor performance at very low levels of alcohol consumption, this improvement is seen *only* at very low levels of drinking and performance declines very rapidly and substantially as blood alcohol concentration increases.

Less than two standard drinks have been shown to result in cognitive and psychomotor effects that increase risk of injury, such as effects on reaction time, cognitive processing, co-ordination and vigilance (Eckardt et al 1998, United States Department of Health and Human Services 1997, Kruger et al 1993, Moskowitz & Robinson 1988). One study showed that risk doubled at a blood alcohol concentration of 0.02 (compared with zero) and increased by a factor of 9 for blood alcohol concentrations in the range 0.05 to 0.09 (Zador 1991). A recent review, however, concluded that adverse effects on psychomotor tasks do not generally affect risk until blood alcohol concentrations around 0.04 to 0.05 (Eckardt et al 1998).

The extent of psychomotor impairment varies with the person's level of experience (Preusser et al 1978). While the risk of crash-related injuries increases with the amount of alcohol drunk for all age groups, younger drivers at a given blood alcohol concentration are at much greater risk than older drivers, not only because the young drivers have less tolerance to alcohol, but also because of their relative inexperience in driving (Zador 1991, Mayhew et al 1986, Mayhew et al 1985).

Alcohol can also increase the likelihood and the extent of aggressive behaviour, thereby contributing to risk of injury from violence (both physical and sexual) (Zhang et al 1997, Martin & Bachman 1997, Cherpitel 1994, Norton & Morgan 1989, Martin 1982). It appears to interact with personality characteristics and other factors related to a personal propensity for violence, such as impulsivity (Zhang et al 1997, Lang & Martin 1993). Injury related to violence may also be more closely linked to alcohol dependence, compared with other types of alcohol-related injury (Cherpitel 1997). High blood alcohol levels or high levels of drinking are commonly reported not only in the perpetrators of violence, but also in the victims (Makkai 1997).

The setting or environment significantly influences the level of risk associated with drinking: for example, risk is increased where there are physical hazards, or the person is required to perform complex reasoning and physical tasks (Eckardt et al 1998).

Heavy drinking is also a major risk factor for suicide and suicidal behaviour among both young people and adults (for example, Shaffer et al 1996, 1998, Lesage et al 1994, Andrews & Lesinsohn 1992; all cited in Beautrais 1998). While the available data are poor, there appears to be a particularly strong association between suicide and alcohol among some Aboriginal and Torres Strait Islander peoples (Hunter 1999, Tatz 1999). An extensive review found that excessive alcohol use was very common among adults, particularly men and including older people, who died by suicide (Barraclough 1987).

In summary: The evidence indicates that the amount consumed per occasion, and more specifically, the blood alcohol concentration, is the critical feature in determining risk of injury. Blood alcohol concentrations as low as 0.04 to 0.05 may affect psychomotor skills and increase the risk of injury in circumstances such as driving. However, significant individual differences make it difficult to calculate blood alcohol concentration accurately from number of drinks and time taken to consume them.

The degree of risk also depends upon the setting, the need for high-order physical skills, and the person's experience with the tasks in hand. In relation to driving, younger, less experienced drivers are likely to have lower risk thresholds, at a given blood alcohol level, compared with older drivers.

CARDIOVASCULAR DISEASE: THE RISKS AND BENEFITS OF ALCOHOL

Cardiovascular disease—disease affecting the heart and blood vessels—accounts for more deaths among Australians than any other group of diseases. In this country, ischaemic heart disease is the leading cause of death, while stroke is the third largest cause of death (after cancer), and a major cause of disability.

There has been a great deal of research into the links between alcohol, heart disease and stroke. Heart disease is discussed below, and stroke on page 70. Hypertension (high blood pressure) is a major risk factor for both stroke and heart disease, and its relationship to alcohol is discussed on page 71. Effects of alcohol on the circulation are also likely to influence peripheral vascular disease, and this is discussed on page 72.

Heart disease

While this section uses the general term 'heart disease', it is concerned specifically with ischaemic heart disease. In ischaemic heart disease, there is insufficient blood supply to the heart muscle because of atherosclerosis in the arteries that supply that muscle—the coronary arteries: that is, these arteries become narrowed or blocked by fatty deposits on their walls. This causes angina and heart attack.

Most studies of the effects of drinking measure these in relation to the *amount* people drink over time. Clear indications have emerged over recent years, however, that the *pattern*, as well as the level, of drinking is important in relation to both the risks and the benefits of alcohol. Essentially, the evidence suggests that low to moderate regular drinking, particularly when it occurs with meals, affords partial

protection against heart disease and heart attack for people of middle age and older; while heavy drinking (both episodic and long-term) markedly increases the risk of heart attack.

The evidence supports four main conclusions regarding alcohol and heart disease:

1. *Alcohol appears to provide some protection against heart disease (both illness and death) for both men and women from middle-age onwards. Most if not all of the benefit is achieved with 1–2 standard drinks per day for men, and less than 1 standard drink per day for women.*

   This protective effect has been demonstrated in a large number of studies over three decades (Svardsudd 1998, Doll 1998, Holman et al 1996, Klatsky 1996, English et al 1995, Jackson 1994, Beaglehole & Jackson 1992, Ashley 1982). Only a relatively few studies fail to confirm it (Hart et al 1999, Murray & Lopez 1999, Maskarinec et al 1998, English et al 1995, Coate 1993). However, a recent meta-analysis confirms these levels, and shows that there is significant evidence that harmful effect in relation to heart coronary heart disease begins at 5 standard drinks per day for women and over 11 for men (Corrao et al 2000). Other harmful effects, nevertheless, outweigh this protective effect at very much lower levels of drinking than this (as reflected in Guideline 1 and discussed on page 35).

2. *In countries where the risk of heart disease is high, the protective effect of alcohol against heart disease may be limited to certain population subgroups.*

   The outcome of the protective effect is of primary importance in the age groups in which heart disease is an important cause of death. In Australia, this begins to occur at 40–45 years in men and 45–50 years in women. Most evidence comes from epidemiological studies involving middle-aged or older people in stable social situations. It is not yet known whether earlier low-risk drinking prevents the onset of cardiovascular disease in later life, and it cannot be assumed that the findings translate to other social groups or to younger drinkers. Heart disease is rare before the age of 40 years, except in very uncommon instances where heart attacks at a young age run in a family. Any short-term benefit of alcohol, such as reducing the risk of clot formation in the arteries supplying blood to the heart muscle, is of no benefit to the vast majority of young adults. It is unknown at present whether or not the longer-term benefits of alcohol, in lowering certain blood fats, are of advantage to young adults in reducing their future risk of heart disease at older ages. Any potential benefits of alcohol need to be weighed against increased risk of other outcomes.

3. *Most of the protective effect appears to arise from the alcohol itself, not from other components in the beverage. If specific drinks, such as red wine, do confer additional benefit, this effect is small.*

   This is discussed further on page 44.

4. *Pattern of drinking is important in determining the risks and benefits of alcohol in relation to heart disease.*

   A number of studies suggest that the benefits of alcohol consumption flow from a relatively consistent pattern of drinking (Gruchow et al 1982), and, further, that 'binge drinking' may actually increase the risk of heart attack (McElduff & Dobson 1997).

   Small amounts of alcohol may be particularly helpful if taken with meals, because alcohol with meals reduces the high level of blood lipids found after eating (Criqui & Ringel 1994, Veenstra et al 1990). Moderate alcohol consumption (defined as 40g per day—4 Australian standard drinks) has a beneficial effect on blood lipids (Hendriks et al 1998). Alcohol also has the effect of thinning the blood, but this effect is only present for a period of hours or days after the person has been drinking (Rubin & Rand 1994). A number of mechanisms are involved in producing these effects, so there is a complex relationship between the dose of alcohol and the effect.

   In contrast, both 'heavy drinking' and 'binge drinking' increase the risk of heart attack in the short and the long term. This increased risk has been seen with beer 'bingeing' (defined as six or more bottles per session) (Kauhanen et al 1997b), and it has been suggested as an important cause of sudden death from heart attack in Russia, where such a pattern of drinking is common (Chenet et al 1998).

In summary: There is strong evidence that drinking alcohol reduces the risk of heart disease in people from middle age onwards. This protection is achieved by drinking relatively small amounts of alcohol, with no additional benefit from drinking larger amounts. The benefit is largely attributable to alcohol *per se*, with other constituents of particular beverage types having little or no additional value. Protection is most closely associated with a consistent pattern of drinking small amounts of alcohol. More variable drinking patterns, especially involving large amounts of alcohol, may actually increase the risk of illness and death from heart disease.

Two points should be noted:

- Heart disease is uncommon in young people, who have higher levels of alcohol-related problems. It is therefore *not* recommended that young people try to reduce their cardiovascular risk by drinking, particularly given the high incidence in these age groups of accident and injury related to high risk drinking.
- Older people who are considering reducing their cardiovascular risk through low risk drinking might consider other ways of improving cardiovascular outcome. This can be achieved by stopping cigarette smoking, increasing exercise, improving diet, or taking small quantities of aspirin. A combination of these approaches achieves better results than any one on its own.

See GUIDELINES 8 & 12

Stroke

A stroke results when the blood supply to part of the brain is suddenly disrupted. About 80 percent of strokes are due to sudden blockage of a blood vessel in the brain (ischaemic stroke). The other 20 percent is caused by rupture of a blood vessel, causing bleeding into the brain (haemorrhagic stroke).

The relationship between alcohol consumption and stroke is complex, with alcohol influencing stroke risk in a number of ways. For example:

- There is good evidence to suggest that alcohol increases the risk of high blood pressure (see page 71), which is a major risk factor for both forms of stroke.
- At the same time, alcohol reduces the risk of atherosclerosis (fatty deposits on the walls of blood vessels, causing narrowing), and it is debris from these fatty deposits that most commonly causes ischaemic stroke (Hillbom & Juvela 1996). (This is how alcohol provides some protection against heart disease.)
- Furthermore, alcohol has a complex effect on the clotting of blood. This appears to increase the risk of haemorrhagic stroke, and may also partly explain the decrease in risk of ischaemic stroke at lower drinking levels and the increase in risk in heavier drinkers (Dimmitt et al 1998, Hillbom & Juvela 1996).

A comprehensive meta-analysis of studies looking at the relationship of stroke and alcohol consumption (Holman et al 1996, English et al 1995) found that heavy drinking (at risky and high risk levels) was clearly associated with increased risk of stroke, particularly in women. The meta-analysis concluded that there is substantial evidence that up to two drinks a day for men and one for women does not increase the risk of ischaemic stroke, though it was unclear whether there was actually a protective effect. It also cited two studies indicating that even moderate alcohol consumption may increase the risk of haemorrhagic stroke.

A number of other studies have found similar results: a clear increase in stroke risk for heavy drinkers, and no clear evidence that alcohol protects against stroke in light or moderate drinkers (Single et al 1999). A large United States study, however, found that the risk of dying from stroke was reduced in men who drank between 1 and 4 drinks daily, and in women who drank 1 to 3 drinks daily (Thun et al 1997), and this protective effect was confirmed by a case-control study (Sacco et al 1999).

There is evidence linking heavy drinking with stroke in younger adults. As well as a link between long-term heavy drinking and stroke (You et al 1997), there is also a link between stroke and recent heavy drinking, particularly drinking to intoxication (Haapaniemi et al 1996, Hillbom et al 1995).

In summary: It seems clear that heavy drinking (at risky or high risk levels) is a risk factor for stroke. Evidence concerning the effect of more moderate consumption is less consistent. The weight of evidence suggests that low level alcohol consumption may offer some protection against stroke. However, some studies have either shown no effect or suggested that alcohol increases risk. The pattern of drinking may also be important in determining stroke risk.

Hypertension (high blood pressure)

There have been more than 80 population-based studies of the relationship between alcohol and blood pressure. The large majority have been cross-sectional studies, with few long-term follow-up cohort studies, and most studies have looked at the relationship between drinking and blood pressure *per se*, rather than focussing specifically on the presence or absence of hypertension. At higher levels of drinking (3 or more standard drinks per day), the studies have consistently found that blood pressure is increased in direct proportion to the amount of alcohol consumed, and high blood pressure is more common among people who drink at this level (Beilin et al 1996).

These observations are consistent with the finding, in randomised controlled trials, that heavier drinkers (5 to 6 standard drinks per day) who substantially decrease their drinking (to 1 to 2 standard drinks per day), over a period of one to six months, can achieve a sustained fall in blood pressure. Heavier drinkers tend to comply poorly with medication to lower their blood pressure (Tuomilehto et al 1984), and people who have high blood pressure and who drink are far less likely to achieve satisfactory blood pressure control during regular follow-up (Henningsen et al 1980). Among men drinking 3 or more standard drinks per day, one in five cases of hypertension will be due to alcohol; in women drinking 2 or more standard drinks per day, one in three cases will be due to alcohol. The effects of heavy drinking in raising blood pressure may be an important factor contributing to the increased risk of stroke in heavier drinkers (Puddey et al 1997).

With low level drinking (less than 3 drinks per day), the picture has been less consistent. Some studies have shown an association with raised blood pressure while others suggest that there are lower levels of blood pressure and a decrease in the risk of hypertension with low level drinking. A meta-analysis of 11 studies suggested that such a decrease in risk may only occur in women (Holman et al 1996, English et al 1995), but other reports find a decrease in risk in both men and women depending on which age, race and gender subgroup is studied. Such inconsistencies are more in keeping with a decrease in risk due to the presence of unidentified confounding variables between such subgroups, rather than alcohol itself. An intervention study in which low level drinking was reduced or eliminated would be needed to confirm this suggestion.

The pattern of drinking may also be important. One study of people who drank at a low level showed that blood pressure was more likely to be influenced by the frequency of drinking rather than the amount of alcohol consumed (Russell et al 1991). A study comparing weekend and daily drinkers, all drinking between 21 and 50 standard drinks (210–500 grams alcohol) per week, found that blood pressure was raised by a similar amount in both groups, but while this raised blood pressure was sustained in the group who drank every day, in the weekend drinkers blood pressure fluctuated in response to the drinking pattern (Rakic et al 1998).

In summary: The risk of hypertension increases with heavier drinking, and reducing heavy alcohol consumption will reduce blood pressure. At lower levels of drinking, the picture has not been as consistent, and at these levels, the possible effects of reduced alcohol consumption on blood pressure need to be considered

against evidence that there are potential cardiovascular benefits associated with regular low level drinking (one to two standard drinks per day for men, and less than one drink per day for women) (Corrao et al 2000).

Peripheral vascular disease

If alcohol protects against heart disease which results from atherosclerosis, it is logical to suggest that it may also protect against peripheral vascular disease (disease of the blood vessels, or atherosclerosis, causing narrowing of the arteries in the leg).

A number of earlier studies failed to show any protective effect, but two recent and large studies suggest that such an effect may exist. One showed that people who drank each day and, over a week, consumed 7 or more US standard drinks (14 grams, ie total 98 grams, equivalent to 10 Australian standard drinks) reduced their risk of the disease by about 25 percent (Camargo et al 1997). The other study suggested that the protective effect occurred through increased high density lipoproteins levels, a well-established effect on blood lipids (Zahkari & Wassef 1996).

In summary: The relationship of alcohol consumption to peripheral vascular disease warrants further study.

Other cardiovascular disorders

Consumption of large amounts of alcohol, both on a single occasion and habitually, can adversely effect the structure and function of the heart. In heavy drinkers and people who are dependent on alcohol, this damage manifests itself in conditions such as cardiomyopathy, 'holiday heart' syndrome, disturbances of the heart rhythm, congestive heart failure and sudden death (eg Puddey et al 1999a, Puddey et al 1999b, United States Department of Health and Human Services 1997).

With regard to lower levels of drinking, an extensive review by Freidman (1998) concluded that one to two drinks can affect heart rate, blood pressure, the effectiveness of heart muscle and the amount of blood pumped, and blood flow to regions of the body. Although these actions generally are not considered to be clinically important, their short-term effect on blood flow might be of significance for people with cardiovascular disease

CANCER AND ALCOHOL: EVIDENCE FOR INCREASED RISK

Cancers of the lips, mouth, throat and oesophagus

There is strong, clear evidence to show that alcohol has a role in causing cancers of the lips, mouth, throat, larynx and oesophagus (IARC 1988, Doll et al 1993), most probably through the direct contact between the alcohol and the cells of these areas. Alcohol damages or destroys cells, and the large numbers of replacement cells formed may include some cancerous cells (Garro & Lieber 1992). At the same time, bacteria in the mouth can break down alcohol to form acetaldehyde, and this

can have similar damaging effects on the lining cells of the mouth, throat and oesophagus (Homann et al 1997).

The risk of these cancers is dramatically increased in heavy drinkers who also smoke.

The type of drink may be important (as discussed on page 44). A recent study showed that the damage to cells in the mouth increased in direct proportion to the alcohol concentration (Jaber et al 1998). Another study found that cancers of the throat and oesophagus were associated with 'moderate' drinking of beer and spirits, but not of wine (Grønbæk et al 1998). These results may, however, simply reflect a lower risk if alcohol is accompanied by a meal.

Cancers of the stomach, pancreas and liver

For each of these organs, alcohol and heavy drinking are clearly associated with inflammatory changes, and it appears this may precipitate the development of cancer, particularly in the presence of other cancer-causing factors.

In the case of liver cancer, the link is well established (Doll et al 1993). On the basis of the consistent association between alcohol intake and liver cancer, and the presence of biologically plausible mechanisms, IARC (1988) concluded that alcohol causes liver cancer. English et al (1995) concluded that there was limited evidence of a causal association. Cirrhosis of the liver is a well-documented result of chronic heavy drinking and is known to be associated with cancer. Alcohol may also increase the risk of cancer in other digestive organs through its effect on liver metabolism, by reducing the clearance of other carcinogens from the system (Anderson et al 1996, Chhabra et al 1996).

In the case of cancers of the stomach and pancreas, there is clear evidence that alcohol causes inflammation (gastritis and pancreatitis), but the link with cancer, while probable, is less well documented and understood. (Bode & Bode 1997, 1992, Doll et al 1993)

Bowel cancer

A number of reviews have concluded that there is a clear association between the amount of alcohol consumed and the risk of bowel cancer (cancer of the colon or rectum), but that there is insufficient evidence to show that alcohol causes bowel cancer (English et al 1995, Doll et al 1993, Longnecker 1992, IARC 1988).

More recent studies suggest that alcohol may act as an important risk factor when combined with smoking (Yamada et al 1997) and poor diet, particularly low folate intake (Kato et al 1999, Boutron-Ruault et al 1996).

Breast cancer

In Australia, 10,000 women are diagnosed with breast cancer each year and one in 11 will be diagnosed with the disease by the age of 74 (AIHW 1998). Any factor that may increase breast cancer risk is therefore of considerable concern, even if its contribution to risk is small.

Increased risk of breast cancer has been associated with alcohol use in most epidemiological studies, with the association identified in many countries and many cultures (National Breast Cancer Centre 1999). Most reviewers conclude that there is a linear association between alcohol intake and risk of breast cancer, such that the risk rises steadily with the level of intake (Longnecker 1992, 1994, Longnecker et al 1988).

English et al (1995), in their comprehensive review, found a moderately strong and consistent dose-response association between alcohol intake and breast cancer. They estimated the risk of breast cancer to be 35 percent higher in women who consume three or four standard drinks of alcohol a day, and 67 percent higher with more than four drinks a day, compared with women who drink little or no alcohol.

The temporal relation between alcohol intake and breast cancer is important (Willett & Stampfer 1997). Some evidence suggests that the strongest association is with alcohol use in later years. This suggests that decreasing alcohol consumption in mid- or later life may help to reduce the risk of breast cancer (National Breast Cancer Centre 1999).

A number of different mechanisms have been suggested whereby alcohol might cause breast cancer, including hormonal influences and the level of acetaldehyde circulating in the body (Wright et al 1999), but there is neither definitive evidence nor consensus on this point.

In summary, while there appears to be a modest association between alcohol consumption and increased risk of breast cancer, further studies are needed to clarify and confirm this association.

In summary: cancer and alcohol

There is clear evidence to show that alcohol is associated with an increased risk of cancer overall, and that it is a cause of cancer of the mouth, throat and oesophagus. In addition, the evidence suggests that it may also play a role in other specific cancers. In particular, further research is needed to clarify the possible role of alcohol in relation to breast and bowel cancer.

Unlike cardiovascular disease, there is no evidence that alcohol at any level has any protective effect against cancer.

While there is a clear relationship between cancer and *level* of drinking, little evidence is available on the relationship between cancer risk and specific *patterns* of alcohol intake. The sole exception to this is that prolonged direct contact between drinks of high alcohol-content and the tissues of the mouth, throat and oesophagus seems to pose a higher risk of these cancers, and should be avoided—and this may relate to a lower risk associated to alcohol taken with meals. Beverage differences do not appear to be significant.

CIRRHOSIS OF THE LIVER AND CHRONIC HEPATITIS

In Australia, alcohol consumption is the most common cause of cirrhosis of the liver, and alcoholic cirrhosis is the most common cause of illness and death related to chronic alcohol consumption.

See GUIDELINE 4

While there is no doubt that alcohol is a cause of cirrhosis, it is important to establish whether it alone can cause cirrhosis, or whether it acts together with other contributing factors; and to establish the extent to which other factors may act alone or together to cause cirrhosis.

There is good evidence to show that drinking alcohol over many years can cause cirrhosis in the absence of other causes. One study found that the risk of developing cirrhosis in medically fit men was associated with consumption over many years of more than 16 standard drinks per day (Lelbach 1975). Other studies have found that considerably lower levels of alcohol may cause cirrhosis (Schmidt 1977).

Other individual factors, acting alone, may also cause cirrhosis. These include hepatitis B and C, and the genetic disorders of haemochromatosis, Wilson's disease and alpha-anti-trypsin deficiency. There are also some cases of cirrhosis in which the cause is not identified. There is very good evidence, however, that alcohol can contribute to the development and course of the cirrhosis in people with haemochromatosis, and increasing evidence that it may be important in conjunction with hepatitis B and C infection.

Overall, the evidence suggests that, regarding the liver cirrhosis that occurs within a given community, the degree to which alcohol is responsible for it varies with the per capita alcohol consumption.

For people with hepatitis C, there is substantial evidence confirming that those who drink at risky or high risk levels have poorer health outcomes. Lighter drinking is associated with fewer problems, but there are few data available to suggest a level of consumption associated with acceptably low risk, and no threshold has emerged so far. At the same time, there is no evidence to date to indicate that total abstinence is necessary.

DEPENDENCE

As discussed earlier, alcohol dependence constitutes a condition in its own right, quite apart from the considerable short-term and long-term health consequences for those who drink chronically at high levels. For example, some people who drink very little over the course of a year, and could not by any definition be considered alcohol dependent, sometimes have catastrophic consequences (including death or serious injury from a road crash or violent assault) after drinking significant quantities of alcohol in a short period. On the other hand, some severely dependent

drinkers may still have good health, intact relationships and stable employment. Serious problems may still be a few years down the track.

See Guideline 4

Dependence is not an all-or-none phenomenon, but exists on a continuum from mild to severe. A slight degree of dependence is not uncommon in the Australian population: for example, finding it a little difficult to stop drinking after two or three standard drinks every day (Hall et al 1999, Stockwell et al 1994). Dependence at a low level may not, of itself, be a problem, but there is a tendency in some people for the level of dependence to increase slowly over time.

The symptoms associated with dependence are both physical (including tremors, sweating, anxiety, vomiting) and psychological (including subjective awareness of a compulsion to drink, and continued drinking after severe consequences). For people who are severely dependent, drinking to ease these symptoms gradually begins to control their lives, taking precedence over everything else. It leads to social disintegration, affecting relationships and work, and may be associated with any of the social consequences of drinking already discussed (see page 41).

Brain damage is one of the long-term consequences of the regular heavy drinking that characterises dependence. The resulting cognitive deficit contributes further, and increasingly, to the person's inability to function socially, professionally, and in day-to-day living. Another likely consequence of regular heavy drinking is cirrhosis.

A Canadian study estimated that about 50 percent of the total costs attributed to alcohol involves people who could be classified as alcohol dependent (Single et al 1998, Rehm 1999).

While the risk of dependence is not easy to quantify, it is higher for people drinking more frequently and at higher levels, but very small for people drinking within the guideline limits. One of the earliest and most common signs of dependence is a difficulty with limiting drinking to a moderate amount on any single occasion—a few drinks begin invariably to lead to many (Stockwell et al 1994). Thus, the ability to stay within guideline limits for a single occasion can be an important indication of whether total abstinence might be necessary.

See Guideline 5

Dependence is influenced by many factors, including genetic susceptibility (eg Johnson & Leff 1999). The rewarding and reinforcing effects of drinking may explain part of the progression from low risk to risky or high risk levels of drinking. People who are genetically predisposed generally feel these effects more strongly, and at lower levels of drinking, than people not predisposed (eg Schuckit et al 1997). This is one possible reason why the children, and especially sons, of alcohol-dependent parents are more at risk of becoming alcohol dependent themselves. There are also strong social and environmental reasons why alcohol-related problems can run in families, such as the powerful effects of modelling particular

drinking styles and exposure to a wider culture which is accepting of heavy drinking and intoxication.

One form of alcohol dependence is that of a gradual development of heavy and regular daily drinking that comes to be associated with physical and psychological distress should the drinker attempt to abstain for even a few hours (Edwards et al 1998). The development of such dependence involves physiological adaptation to alcohol's presence in the central nervous system, combined with the drinker learning to keep on drinking to avoid discomfort. Regular periods of not drinking (ie. alcohol-free days) interrupt both the physiological adaptation and the habit development associated with the development of increasing dependence on alcohol (Stockwell 1994).

ALCOHOL AND PREGNANCY

When a pregnant woman drinks, the alcohol in her blood stream enters that of her unborn child, and it is known that, in sufficient quantities, this can cause problems for the child. One of the most vulnerable periods for the unborn baby is probably the first few weeks after conception (Coles 1994, Hannigan et al 1995), including the time before the woman is aware of her pregnancy.

The effects of prenatal alcohol exposure on the physical and nervous system development of the foetus, and on behavioural development in the child, have received a great deal of attention during the past fifteen years. Drinking alcohol during pregnancy has been associated with varying degrees of harm to the unborn child. The most severe types of harm include gross congenital anomalies and a defined syndrome, the foetal alcohol syndrome (FAS), both associated with high levels of drinking. FAS includes characteristic physical abnormalities, growth retardation and neurological dysfunction with developmental delay. Alcohol has also been associated with a variety of more subtle developmental effects.

The research literature in this area is clear that alcohol consumption at high levels during pregnancy (ie at levels above low-risk guidelines for the general population) can contribute to a variety of adverse outcomes in the unborn child, especially where high peak blood alcohol levels are reached (ie if the woman becomes intoxicated),. At these higher levels of drinking, a general dose-response relationship has been established whereby the greater the amount of alcohol consumed , the greater the likelihood of injury. At least four main areas of harm have been identified: foetal death, congenital malformation, growth retardation, and behavioural deficits. Each one of these areas is associated with different toxic dose thresholds (Jacobson & Jacobson 1994): that is, levels of drinking at which the effects start to occurs. These effects have been demonstrated at high levels of consumption, but there are inconsistencies in the research literature pertaining to socio-economic class, cultural context and other maternal characteristics (Abel 1997, Day 1992). That is, social and personal variables also have similar effects on the child, and these effects are difficult to distinguish from the effects of alcohol. For example, women from lower socio-economic backgrounds whose overall health status may be compromised, smokers and older women all appear more vulnerable to the effects of alcohol on the foetus.

The research evidence is less clear about the effect of alcohol when consumed at low to moderate levels (ie within low-risk guidelines for the general population) during pregnancy. The most likely effects at these levels, if effects occur (and the evidence is not clear on this issue), are functional teratogenesis (the production of congenital abnormalities in the developing embryo), and subtle neurobehavioural problems that are not associated with immediately recognisable physical abnormalities. Establishing a lower threshold for risk has proved difficult, and this difficulty supports the emerging evidence that the risk may differ for different effects. With respect to foetal malformation, a recent meta-analysis found no difference in foetal outcomes when the mother drank two or less drinks per day compared with drinking less than two drinks per week (Polygenis et al 1998). But with respect to low birth weight and slowed inter-uterine gestational growth, studies have shown that these are associated with drinking at a level between three drinks per week and two per day (Windham et al 1995), and, in another study, drinking more than three drinks per day (Passaro et al 1996). Further highlighting the complexity of this area, Clarren et al (1987) have suggested that there is no single dose-response relationship for abnormalities in the foetus caused by alcohol, but, rather, that each potential abnormal outcome in brain structure or function, and growth has its own dose-response relationship and period of risk during pregnancy. Single et al (1999a) maintain that larger studies may be required to detect more subtle effects at lower intake levels. It is also theoretically possible that more sensitive instruments may be developed in the future, that could be capable of detecting harm at levels of consumption lower than the levels currently indicated in these guidelines.

In summary: There is clear evidence that alcohol consumption at high levels during pregnancy (ie at levels above low-risk guidelines for the general population), and especially where high peak blood alcohol levels are reached (ie if the woman becomes intoxicated), can contribute to a variety of adverse outcomes in the newborn child. However, the evidence of effects on the foetus of drinking lower levels is less clear-cut. Attempts have been made recently to define dose-response associations and thresholds for drinking during pregnancy in relation to neurological and behavioural deficits in the child. Overall, the most consistent evidence to date identifies *an average* of one standard drink per day as the level below which no discernible evidence has been found for harm to the unborn child (Single et al 1999a).

COGNITIVE SKILLS AND ALCOHOL: RISKS AND POSSIBLE BENEFITS

The relationship between heavy alcohol consumption and cognitive impairment is well established (eg Williams & Skinner 1990). The evidence suggests that people drinking 70 to 84 grams alcohol (7 to 8.5 Australian standard drinks) per day over an extended period of time show some cognitive inefficiencies; people drinking 98 to 126 grams alcohol (10–12.5 Australian standard drinks) per day show mild cognitive deficits; and 140 grams (14 Australian standard drinks) or more per day results in moderate cognitive deficits similar to those found in people with diagnosed alcohol dependence (Parsons & Nixon 1998).

People who regularly drink large quantities of alcohol often have a deficiency in dietary thiamine, a B vitamin, and this causes the neurological disorder, Wernicke-Korsakoff Syndrome (WKS). Wernicke's encephalopathy and Korsakoff's psychosis are the acute and chronic phases, respectively, of the condition, which particularly affects recent memory. An absence of thiamine results in an inadequate supply of energy to the brain, particularly two areas that form part of the limbic system:

- the hypothalamus, which regulates body temperature, growth and appetite, has a role in emotional responses, and controls pituitary functions including metabolism and hormones; and
- the mamillary bodies, where neural pathways connect various parts of the brain involved in memory functions.

There is increasing evidence that the high incidence of this condition in Australia is declining rapidly since the introduction of thiamine fortification in bread in 1991.

In chronic heavy drinkers, the frontal lobes of the brain clearly shrink. This is probably due partly to loss of water and partly to cell death. The lobes may expand again if the person stops drinking, but the evidence of cell death remains in impaired function.

There is very much less information available on the effects of more moderate drinking on the brain's ability to perform tasks, including remembering, reasoning and thinking. While most studies have failed to find any relationship between moderate alcohol consumption and dementia, 'alcohol related dementia' is an established diagnosis that accounts for a substantial part of all dementias in long-term care (Carlen et al 1994).

Other recent studies, however, suggest that moderate alcohol consumption may benefit cognitive function (eg Chick 1999). One study in France, for instance, found a markedly lower incidence of dementia among people drinking 25 to 50 grams of alcohol per day compared with abstainers and people who drank no more than one drink per week (Orgogozo et al 1997). This, the investigators suggested, is possibly because alcohol may have a beneficial effect on blood vessels in the brain. Other studies have found similar effects; but some have found no association between moderate alcohol consumption and cognitive skills.

In summary: It is possible that cognitive decline in older people may be reduced by drinking up to two standard drinks per day; however, better studies are needed, taking account of factors such as education or social adjustment, before coming to more definitive conclusions. In people who drink heavily (significantly above guideline levels), a relationship between alcohol consumption and cognitive impairment is well established.

DOES ALCOHOL PROTECT AGAINST OTHER CONDITIONS?

Diabetes

Some studies have suggested that mild to moderate alcohol consumption is associated with a decreased risk of Type 2 diabetes (Rimm et al 1995, Perry et al 1995, Stampfer et al 1988) while other studies have found no association (Hodge et al, 1993, Feskens and Kromhout 1989). In studies reporting a beneficial effect, the quantity of alcohol associated with this effect has ranged from 20g/week to 60g/ day. Some studies have reported increased incidence of Type 2 diabetes with large quantities of alcohol (Wei et al 2000).

In summary: Any relationship between alcohol consumption and risk of Type 2 diabetes requires further clarification.

Gall stones

Recent studies have confirmed the finding of a previous comprehensive meta-analysis, that there is some evidence that moderate alcohol consumption protects against gall stones (Holman et al 1996, English et al 1995). One study suggests that men who drank 45 g (4.5 standard drinks) or more of alcohol on 5 to 7 days halved their risk of gallstones (Leitzmann et al 1998). Laboratory evidence suggests that this protection occurs through the role of alcohol in cholesterol metabolism and the formation of bile acids.

Two other recent studies, however, have failed to find any relationship between alcohol consumption and gallstones (Sahi et al 1998, Kratzer et al 1997)

In summary: A majority of (but not all) recent studies confirm the finding that alcohol consumption protects against gallstones.

5 RESEARCH QUESTIONS

In compiling these guidelines, the Working Party noted limitations in the evidence base in a number of areas. In most areas there is a need for improved measurement of alcohol intake incorporating more precise measures of average intake (relevant to long-term health problems), and also the pattern of drinking, including amounts consumed per occasion (relevant to short-term health problems). Most epidemiological studies are not designed with a view to developing safe drinking guidelines. The following are some specific topics needing further research, to provide comprehensive evidence on the harms and benefits associated with drinking alcohol:

- acute harms associated with alcohol;
- documentation and measurement of the social harms resulting from alcohol;
- alcohol and older people;
- alcohol and gender;
- alcohol and breastfeeding;
- alcohol and mental health;
- the effect of increasing alcohol consumption to low or moderate levels in abstainers;
- quantification and clarification of risk for dependence in relation to moderate levels of drinking (within existing guidelines) and heavier drinking;
- alcohol-related mortality for specific occupational groups.

APPENDICES

APPENDIX 1: NHMRC WORKING PARTY ON THE REVIEW OF RECOMMENDATIONS REGARDING RESPONSIBLE DRINKING BEHAVIOUR

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APPENDIX 2: GUIDELINE DEVELOPMENT PROCESS

BACKGROUND

In May 1996 the National Drug Strategy Committee requested that the NHMRC guidelines 'Is there a safe level of daily consumption of alcohol for men and women?—Recommendations regarding responsible drinking behaviour' be reviewed.

In September 1998 a working party was formed to consider the above document. Membership included specialists in the field including representation from the National Drug Strategy Committee (full membership details are provided at Appendix One).

It was specifically required that the guidelines be evidence based and informed by the conduct of:

- a review of evidence relating to the level of daily consumption of alcohol for men and women considered to be safe;
- redevelopment of the guidelines through the NHMRC consultation process; and
- development of a plan (including timelines and a resource strategy), in coordination with the National Expert Advisory Committee on Alcohol, for dissemination implementation and evaluation of the guidelines.

A literature review was undertaken by Single and Associates (Canada) to develop the evidence base on the level of alcohol consumption considered to be low-risk for men and women.

PURPOSE OF THE GUIDELINES

The aims of the guidelines are to:

- assist the whole population and sub-groups who may be at greater risk because of their susceptibility, to make informed choices about their drinking and health;
- assist health professionals to give evidence based advice to patients; and
- promote individual and population health and minimise the consequences of alcohol related harm.

THE REPORT DEVELOPMENT PROCESS

On 9 October 1998, the NHMRC called for first stage public submissions on Is there a safe level of daily consumption of alcohol for men and women?—Recommendations regarding responsible drinking behaviour. Twenty-eight written submissions were received and considered by the working party from the following organisations and individuals:

Mr Herschel Mills Baker	Australian Parents for Drug-Free Youth
Dr Alan Coates MD FRACP	Australian Cancer Society
Mr David South	Road Safety Department, VICROADS
Ms Audrey Jackson	The Association of Independent Schools of WA
Mr Fergus Thomson	National Council of Independent Schools' Associations
Mr David Enright	Department of Transport, Tasmania
Mr Ernie Lang	
Ms Mary Johnston	Department of Education, Training and Youth Affairs
Mr Charles Kildruff	SWAT (Society without Alcohol Trauma)
Dr Sandra Hacker	Australian Medical Association
Mr Terry Mott	The Australian Associated Brewers Incorporated
Mr Bob Black	National Epilepsy Association of Australia Inc
Mr Fulvio Penna	Alcohol Advisory Council of Western Australia Inc.
Ms Lily O'Hara	Australian Association of Health Promotion Professionals
Ms Carol McNiven	The Alcohol and other Drugs Council of Australia
Ms Kathy Heinze	
Ms Elizabeth Newman	National Council of Women of Australia
Ms Robyn Charlwood	National Heart Foundation of Australia, Victoria Division
Ms Selena Knowles	Health Department of Western Australia
No name given	Alcohol and other Drugs Program, Health Department of Western Australia
Professor Ian Webster	Australian National Council on Drugs (ANCD)
Mr Ernie Lang	Turning Point Alcohol and Drug Centre
Ms Liz Furler	Population Health Division, Department of Health and Aged Care
Ms Jo Townsend	Territory Health Services

Ms Creina Stockley	The Australian Wine Research Institute (also on behalf of the Winemakers' Federation of Australia)
Mr Romy Lessem and	Diabetes Australia Ms Effie Tsivis APD

Draft recommendations (based on the literature review and first-stage consultation submissions) were made available on 9 November 1999 at The Australian Society for Alcohol and other Drugs (APSAD) workshop. This provided an excellent opportunity to receive valuable comments from the following experts:

Ms Janice Trezise	Department of Health and Human Services (Tasmania)
Mr Gareth Daniels	Northern Rivers Division of GPs
Mr Graeme Denholm	People Against Drink Driving
Mr Jeff Lucas	
Dr Gordon Trinca	Royal Australasian College of Surgeons
Mr Charles Kilduff	SWAT (Society with Alcoholic Trauma Inc)
Mr Donald Cameron	People Against Drink Driving
Mr Terry Mott and Mr Shane Coombe	Australian Associated Brewers
No name given	Medical Advisory Committee, Australian Associated Brewers
Ms Creina Stockley	The Australian Wine Research Institute
Dr Mike MacAvoy and	Alcohol Advisory Council of New Zealand Mr Ian MacEwan

In July 2000, the Health Advisory Committee called for second-stage consultation submissions in which 45 were received from the following individuals and organisations:

Mr John Rogers	
Mr Paul Sullivan	Alcohol Awareness and Family Recovery
Ms Kathy Heinze	
Ms Marilyn Beaumont	Women's Health Victoria
Professor Judith Lumley	Centre for the Study of Mothers' and Children's Health

Mr Peter Bunworth	Inner Eastern Division of General Practice
Ms Jo Townsend	Alcohol and other Drugs Program, Territory Health Services
Ms Margaret Martin	
Ms Anne Bergen	Women's Christian Temperance Union of Victoria
Ms Nola Adams	Women's Christian Temperance Union of WA
Major Brian Watters	Australian National Council of Drugs
Mr Ian Sutton	Winemakers' Federation of Australia
Dr Ki Douglas	Australasian Faculty of Occupational Medicine
Dr Andrew Boyden	National Heart Foundation
Mr Creina Stockley	The Australian Wine Research Institute
No name given	Central Sydney Area Health Service
Ms Leanne Wells	Tobacco and Alcohol Strategies Section, Population Health Division, Department of Health and Aged Care
Mr Rob Bradley	The Royal Life Saving Society Australia
Ms Rosemary McClean	Australian Drug Foundation
Ms Elspeth Sharp	National Council of Women Australia
Mr Tony Ward	
Ms Yvonne Tilley	National Council of Women (Launceston)
Ms Ilse O'Ferrall	Alcohol Advisory Council of Western Australia
Dr Bill Pring	AMA's Public Health and Aged Care Committee
Ms Reyna Dight	Northern Rivers Area Health Service
Mr Alan Barclay	Diabetes Australia
Ms Sue Miers	National Organisation for Foetal Alcohol Syndrome and Related Disorders (NOFASARD)
Ms Victoria Conway	Alcohol and Drug Service, Tasmania
Ms Colleen O'Leary	National Expert Advisory Committee on Alcohol
Mr Neil Roberts MLA	Member for Nudgee
Ms Christine Rogan	Fetal Alcohol New Zealand Trust

Ms Rosemary Bryant	Royal College of Nursing
Dr Nigel Abbott	
Mr Donald Cameron	People Against Drink Driving
Ms Gabrielle FitzGerald	The Royal Australian and New Zealand College of Psychiatrists
Mr Neale Spencer	Road Traffic Authority
Dr Peter d'Abbs	Menzies School of Health Research
Mr Scott Wilson	Aboriginal Drug and Alcohol Council of South Australia
Ms Caroline Fitzwarryne	Alcohol and other Drugs Council of Australia
Dr Di Tibbits	Royal Australian and New Zealand College of
Ms Karyn Hart	Department of Employment, Education, Training and Youth Affairs
Mr Ray Judd	Department of Human Services (Victoria)
Mr Ernie Lang	Turning Point Alcohol and Drug Centre
Mr Terry Mott	National Alcohol Beverages Industry Council Inc (NABIC)
Mr Dominic Nolan	Australian Regional Winemakers' Forum

DISCUSSION OF SUBMISSIONS

The working party developed the scope for the project after considering the issues raised in first stage consultation submissions.

The working party made a number of changes to the draft guidelines and report based on the submissions received from the APSAD workshop and during second stage consultation.

IMPLEMENTATION AND DISSEMINATION

A workshop was held in conjunction with partners in January 2001 to brainstorm implementation and dissemination ideas. Participants discussed strategies and promotional activities that would be effective in disseminating the messages from the Guidelines such as working through intermediaries. The outcome of this workshop was a comprehensive implementation and dissemination paper.

Different media and community strategies will be applied for different target groups. Messages will be formulated in ways that are meaningful and useful to each of the groups specified in the Guidelines. The workshop identified the most

appropriate conduits and settings in which to direct messages to the different target groups. The following approach was agreed:

Guideline number	Conduit or setting
1, 4, 5, 6, 7, 8, 11 & 12	GPs and emergency care settings.
9 & 10	Point of sale, retail and hospitality industry, education, emergency care
3	Local and State government alcohol industry
1 & 2	'All systems' general approach including Aboriginal health workers.

The implementation strategy will be undertaken as part of the National Alcohol Strategy.

APPENDIX 3: EVALUATION OF EVIDENCE

The literature review that underpins these guidelines, *Evidence Regarding the Level of Alcohol Consumption Considered to be Low-Risk for Men and Women,* by Eric Single, Mary Jane Ashley, Susan Bondy, Maureen Dobbins, James Rankin, and Jürgen Rehm (1999a), used the following guidelines from the IARC (1988), which were also used in the NHMRC's publication of *A Guide to the Development, Implementation and Evaluation of Clinical Practice Guidelines* (NHMRC 1999).

GUIDELINES FOR ASSESSING EVIDENCE ON CAUSALITY

Evidence of causality between alcohol consumption and physiological effects (including both harmful and protective effects) was assessed using the following guidelines from the IARC (1988):

1. Sufficient evidence of causality: In the reviewers' opinion, a causal relationship has been established between alcohol consumption and the disease or injury. The evidence indicates that an association (positive or negative) exists between alcohol consumption and the disease or injury in which chance, confounding variables and other bias can be ruled out with reasonable confidence.
2. Limited evidence of causality: An association (positive or negative) has been observed between alcohol consumption and the disease or injury for which a causal interpretation is considered to be credible, but chance, confounding variables or other bias cannot be ruled out with reasonable confidence.
3. Inadequate evidence of causality: The available studies are of insufficient quantity, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal connection.
4. Evidence suggesting lack of causality: There are several adequate studies, covering the full range of levels of alcohol consumption in the population, showing the lack of a relationship (positive or negative) between alcohol consumption and the disease or injury. This assessment is inevitably limited to diseases and injuries, levels of consumption and lengths of observations covered by the available studies; and the possibility of very small risks at the levels of exposure studied can never be excluded.

APPENDIX 4: STRENGTHS AND WEAKNESS OF THE USE OF SURVEYS TO MEASURE ALCOHOL CONSUMPTION

Most of the studies reviewed in the literature review stem from epidemiology, using survey-based estimates of alcohol consumption. These have both strengths and limitations, which vary with the actual survey design.

Survey-based estimates of alcohol consumption can provide evidence on both volume and patterns of drinking. They can provide information on:

- current and lifetime drinking status, identifying current drinkers, former drinkers, and lifetime abstainers;
- the volume of alcohol consumed overall, the average per day or per drinking day, and/or the volume of particular types of drinks; and
- the frequency and volume of high risk drinking.

Surveys permit comparison between sub-groups of interest, and they can enable consumption patterns to be linked with consequences at the individual level, thereby helping to clarify the influence of factors relating to the individual drinker or to the context of drinking.

The accuracy of the data obtained depend critically on the population or 'sample' surveyed: both the size of the sample in relation to the population of interest, and the degree to which it accurately represents this population. The data are also influenced by the length of time covered by the questions. For example, questions covering the past seven days *may* elicit more accurate reporting than asking people to summarise their drinking habits over a longer period, and such questions lessen the tendency for people to ignore occasional heavy drinking days when asked to summarise their 'typical' intake. However, the past seven days may give a false picture of the consumption of irregular drinkers. Many surveys therefore ask about a longer period, typically a month or a year; but this increases the likelihood of recall errors. Current recommendations from the World Health Organization (WHO 2000) recommend the use of questions in national surveys that cover the frequency of drinking varying amounts of alcohol over the past 12 months—known as the Graduated Quantity-Frequency method.

One difficulty in assessing the quantity drunk is the fact that, in many social settings, there is a tendency for a host to top up glasses, or for drinks to be taken from a communal container such as a jug of beer. Furthermore, estimating the size of a drink may be difficult for many survey respondents, with different glass sizes and often with other people mixing drinks.

Drinking that places people at high risk for acute or immediate problems is commonly measured across the population by:

- the number or proportion of drinkers, or of the total population, drinking at a level deemed to be high risk, with a specified frequency over a given period of time: for example, the proportion of drinkers who have drunk more than six drinks on any one day over the past six months;
- the consumption on high risk drinking days : for example, data might be collected on the number (and possibly the sizes) of drinks consumed each day of the past seven days.

These measures must be distinguished from measures of typical daily drinking, which will give a lower figure and are indicative of risk levels for long-term or chronic health problems.

It is also useful to know the context for drinking, because it relates to the consequences of drinking. For example, drinking at home is not likely to result in problems relating to impaired driving. Surveys can therefore ask about where, or with whom, drinking takes place (eg home, the home of friends, or public places), or about the proportion of drinking done in each of these contexts, or how often the person drinks in each context (every day, most days etc). Questions can also be asked specifically about how often the person drinks before driving a car, or engaging in other potentially risky activities.

APPENDIX 5: RECOMMENDED UPPER LIMITS OF DRINKING IN GUIDELINES, POLICY STATEMENTS, JOURNAL EDITORIALS AND STATEMENTS BY LEADING EXPERTS, 1990–

Author/organisation (and reference)	Journal of the American Medical Association (JAMA) (Hwang et al 1999)
Country	US
Date	1999
Drinking level	'moderate drinking' defined as 1–2 drinks/day for men and 1 drink/day for women
Rationale/comments	This is a 1-page fact sheet as part of JAMA's Patient Page. It notes that 'moderate use can have some health benefits'.

Author/organisation (and reference)	National Institute on Alcoholism and Alcohol Abuse (NIAAA) recommended year 2000 alcohol guidelines (Gordis 1999)
Country	US
Date	1999
Drinking level	<2 drinks/day for men under 65; <1 drink/day for women and men over 65
Rationale/comments	In an official comment on US Dietary guidelines, the Director of the NIAAA, Enoch Gordis, advocates informing the public that: • Causality regarding coronary heart disease (CHD) risk reduction is not conclusive • Studies show CHD reduction at levels below moderate • Very serious health consequences quickly accrue at levels of consumption above moderate

Author/organisation (and reference)	Israel Society for the Prevention of Alcoholism (Weiss 1999)
Country	Israel
Date	1999
Drinking level	Not specified
Rationale/comments	15 guidelines are presented on drinking, including avoidance of intoxication, dilution of high alcohol content drinks, drinking with food, drinking slowly, women being more 'careful in drinking' than men, avoidance of drinking when pregnant or taking medications

Author/organisation (and reference)	The John Hopkins White Papers (John Hopkins Medical Institutions 1998)
Country	US
Date	1998
Drinking level	1–2 drinks/day for men and 7 drinks/ week for women if no heart disease or other risk (otherwise 1 drink/day)
Rationale/comments	Nutrition experts from John Hopkins University note 'undeniable' health benefits to moderate alcohol consumption, but they also write that alcohol adds extra calories, can interfere with medication, can cause some types of heart disease, carries the potential for abuse, and may increase breast cancer risk for women. They also recommends that if you don't drink, don't start.

Author/organisation (and reference)	National Stroke Association (NSA 1998)
Country	US
Date	1998
Drinking level	Not specified
Rationale/comments	NSA's Prevention Advisory Board's Stroke Prevention Guideline # 4: 'If you drink alcohol, do so in moderation…one drink each day may actually lower your risk for stroke (provided there is no other medical reason you should avoid alcohol).'

Author/organisation (and reference)	Medical Research Council of Sweden (MRC 1997)
Country	Sweden
Date	1997
Drinking level	'intake should be kept below 10 to 20 g/day'
Rationale/comments	'It is possible that a moderate alcohol intake has certain positive medical effects. However, the causal relationships are not sufficiently clear. Complete or almost complete abstention from alcohol can therefore not be considered a risk'.

Author/organisation (and reference)	Harvard University Cancer Prevention Center (HUCPC 1997)
Country	US
Date	1997
Drinking level	'moderate drinking'=2 or less drinks/day for men; 1 drink/day for women
Rationale/comments	'Recommendations for alcohol intake are complicated by strong evidence that 1 or 2 drinks per day is protective against cardiovascular disease.'

Author/organisation (and reference)	National Heart, Lung and Blood Institute (NHLBI 1997)
Country	US
Date	1997
Drinking level	30 ml of ethanol/day for men, 15 ml for women and lighter weight persons
Rationale/comments	'Such amounts do not raise blood pressure and have been associated with a lower risk for CHD.'

Author/organisation (and reference)	Invited British Medical Journal editorial (Doll 1997)
Country	UK
Date	1997
Drinking level	Up to 4 drinks/day
Rationale/comments	Consumption of small and moderate amounts of alcohol reduces mortality from vascular disease by about one third. Minimum mortality occurs with 2 to 3 drinks/day.

Author/organisation (and reference)	American Cancer Society (ACS 1996)
Country	US
Date	1996
Drinking level	2 drinks/day
Rationale/comments	'Cancer risk increases with the amount of alcohol consumed and may start to rise with intake as few as two drinks a day…Moderate intake of alcoholic beverages has been shown to decrease the risk of CHD in middle-aged adults.'

Author/organisation (and reference)	American Heart Association (Pearson 1996)
Country	US
Date	1996
Drinking level	1 or 2 drinks/day
Rationale/comments	The article notes that 80,000 deaths are prevented each year due to moderate intake of alcohol. Other recommendations include consulting with physician to tailor risks to benefits, and not drinking when operating machinery or motor vehicles.

Author/organisation (and reference)	Pearson and Fuster 1996 (Pearson 1996)
Country	US
Date	1996
Drinking level	Not specified: recommends reducing alcohol consumption to a 'moderate' level
Rationale/comments	Includes decision flow chart for general practitioners on management of vascular disease in which alcohol moderation is included in management of blood pressure.

Author/organisation (and reference)	Centre Alcologico Integrato
Country	Italy
Date	1996
Drinking level	< 40 g/day for men, less for women
Rationale/comments	Although the threshold for low risk drinking is defined in terms of grams in a day, the equivalent number of drinks is not stated.

Author/organisation (and reference)	American College of Cardiology (ACC 1996)
Country	US
Date	1996
Drinking level	Moderate consumption=1–3 drinks/day
Rationale/comments	Moderate drinkers (1–3 drinks/day) 'have a 40% to 50% reduction in coronary artery disease risk compared with individuals who are abstinent.' No alcohol consumption included as a CHD risk factor.

Author/organisation (and reference)	National Institute on Alcoholism and Alcohol Abuse (NIAAA 1995)
Country	US
Date	1995
Drinking level	Not more than 2 drinks/day
Rationale/comments	Recommends physicians advise patients who drink, to do so in moderation and to abstain under certain circumstances.

Author/organisation (and reference)	United States Dietary Association (USDA and USDHHS 1995)
Country	US
Date	1995
Drinking level	2 drinks/day for men and 1 drink/day for women
Rationale/comments	The guidelines state 'if you drink alcoholic beverages, do so in moderation, with meals, and when consumption does not put your or others at risk.'

Author/organisation (and reference)	Royal College of Physicians, Psychiatrists and General Practitioners (Royal College 1995)
Country	UK
Date	1995
Drinking level	21 units/week for men, 14 units/week for women (unit=8g of ethanol)
Rationale/comments	While not recommending that people increase drinking to reduce risk of CHD, the report concludes that moderate drinkers have lower CHD risk.

Author/organisation (and reference)	UK Dept. of Health (UK Dept of Health 1995)
Country	UK
Date	1995
Drinking level	21 units/week for men, 14 units/week for women (unit=8g of ethanol)
Rationale/comments	This UK report on 'Sensible Drinking' notes a significant health benefit from moderate drinking for men over 40 and postmenopausal women, including lower risk of CHD, ischemic stroke and gallstones. A maximal advantage is at 1–2 units/day for men, and significant health risk 'will not accrue' up to 4 units/day for men and 3 units/day for women.

Author/organisation (and reference)	Alcohol Advisory Council of New Zealand, (AACNZ 1995)
Country	New Zealand
Date	1995
Drinking level	<60g/day and 210g/ week for men and <40 g/day and <140 g/week for women
Rationale/comments	Alcohol-free days recommended, and special circumstances are noted when lower limits or abstention is advisable.

Author/organisation (and reference)	Invited editorial in Am J of Public Health (Klatsky and Friedman 1995)
Country	US
Date	1995
Drinking level	Not specified.
Rationale/comments	Light drinkers have 30% to 40% lower CHD risk and 10% lower mortality risk. However, concern about the risks of heavier drinking 'makes it inappropriate to indiscriminately advise non-drinkers to start drinking.'

Author/organisation (and reference)	Letter to the editor of JAMA (Gordis 1995)
Country	US
Date	1995
Drinking level	Not specified
Rationale/comments	In response to the JAMA editorial by Pearson and Terry (below), Enoch Gordis, Director of the NIAAA, cautions against advising abstinent and infrequent drinkers to increase alcohol consumption.

Author/organisation (and reference)	Canadian Centre on Substance Abuse and the Addiction Research Foundation (Ashley et al 1997)
Country	Canada
Date	1994
Drinking level	Men and Women: No more than 2 drinks(27.2g)/day with one day of abstention per week
Rationale/comments	Lower limits also appropriate for persons with low body weight and for inexperienced drinkers. Those who drink less than every day should not increase their consumption, and those whose drinking exceeds two drinks in any day should reduce their consumption of alcohol. All persons who consume alcohol should avoid drinking to intoxication.

Author/organisation (and reference)	Invited editorial in JAMA (Pearson and Terry 1994)
Country	US
Date	1994
Drinking level	More than '1 to 2' ounces/day of ethanol associated with increased health risk
Rationale/comments	Giving patients advice on drinking is described as a 'conundrum', requiring balancing risk of adverse consequences with potential benefits from moderate consumption.

Author/organisation (and reference)	Simon 1994
Country	US
Date	1994
Drinking level	'Low dose' not specified.
Rationale/comments	The author notes that there are circumstances when 'physicians might reasonably prescribe alcohol for responsible people with low high-density lipoproteins (HDL), cholesterol levels or other major coronary artery disease (CAD) risk factors that have failed to respond to lifestyle interventions.'

Author/organisation (and reference)	Friedman and Klatsky 1993
Country	US
Date	1993
Drinking level	Not specified: moderate amounts depend on individual characteristics but 3+ drinks/day is undesirable
Rationale/comments	Some people (those with high risk for CHD but low risk for problem drinking) might benefit from taking up drinking, but this should not be recommended indiscriminately.

Author/organisation (and reference)	NHMRC (1992)
Country	Australia
Date	1992
Drinking level	<40 g/day for men and <20 day for women
Rationale/comments	Also recommends to avoid binge drinking, and that all people should have at least 2 alcohol-free days each week. In addition, special guidelines were detailed for specific situations, such as hazardous situations, when operating machinery or in the context of pregnancy.

Author/organisation (and reference)	Stichting Verantwoord Alcoholgebruik (1991)
Country	Nether-lands
Date	1991
Drinking level	<40 g/day for men and women
Rationale/comments	Abstinence promoted among pregnant women, adolescents, those driving or operating machinery and those who are working or studying. Women and those with low body weight are advised to drink less than the recommended levels.

Author/organisation (and reference)	Editorial in Epidemiology (Ellison 1990)
Country	US
Date	1990
Drinking level	Cautions against a specific amount due to individual differences
Rationale/comments	This editorial, aptly entitled 'Cheers!', cites evidence of CHD reduction from moderate drinking and cautions against the risks of heavier drinking. It concludes that a male without a bleeding tendency or risk of alcohol abuse 'might consider the advantages of washing down his aspirin with a glass of claret.'

APPENDIX 6: ALCOHOL AND DRUG TELEPHONE HELPLINES

National
ADCA (Alcohol and other Drugs Council of Australia)
02 6281 0686

Australian Capital Territory
Alcohol and Drug Information Service
02 6205 4545 (24 hours)

New South Wales
Alcohol and Drug Information Service
(02) 9361 8000 (24 hours) or 1800 422 599 (for callers outside Sydney)

Northern Territory
Amity Community Services
08 8981 8030 or 1800 629 683

Queensland
Alcohol and Drug Information Service
07 3236 2414 or 1800 177 833 (for callers outside Brisbane)

South Australia
Alcohol and Drug Information Service
1300 131 340

Tasmania
Alcohol and Drug Information Service
1800 811 994

Victoria
Alcohol and Drug Direct Line
03 9416 1818 or 1800 136 385 (for callers outside Melbourne)

Western Australia
Alcohol and Drug Information Service
08 9442 5000 or 1800 198 024

REFERENCES

Note: *Indicates references quoted in the Literature Review commissioned by the NHMRC to form the primary evidence base for these guidelines.

*Abel EL (1997) Maternal alcohol consumption and spontaneous abortion. *Alcohol & Alcoholism,* 32, 211–219.

Abrams DB, Niaura RS (1987) Social learning theory. In HT Blane, KE Leonard (Eds) *Psychological Theories of Drinking and Alcoholism* (pp131–178). New York: Guilford Press.

Adhikari P, Summerill A (2000) *1998 National Drug Strategy Household Survey: Detailed Findings.* AIHW cat no. PHE 27. Canberra: AIHW (Drug Statistics Series No.6)

*Ajani UA, Hennekens C, Spelsberg A, Manson JE (1999) Alcohol consumption and risk of type two Diabetes Mellitus. *American Journal of Epidemiology,* 149, S3.

*American Cancer Society (1996) Guidelines on diet, nutrition, and cancer prevention. *Cancer Journal for Clinicians*, 46, 330–332.

*American College of Cardiology (1996) Executive summary: Task force 3 Report. 27th Bethesda Conference. *Journal of the American College of Cardiology*, 27, 961–963.

*Anderson LM, Souliotis VL, Chhabra SK, Moskal TJ, Harbaugh SD, Kyrtopoulos SA (1996) N-nitrosodimethylamine-derived O(6) methylguanine in DNA of monkey gastrointestinal and urogenital organs and enhancement of ethanol. *International Journal of Cancer*, 66, 130–134.

Anderson S, Dawson J (1999) Neuropsychological effects of alcoholic hangover. *South African Journal of Science*, 95, 145–147.

Andrews JA, Lewinsohn PM (1992) Suicidal attempts among older adolescents: prevalence and co-occurrence with psychiatric disorders. *Journal of the American Academy of Child and Adolescent Psychiatry,* 31, 655–662.

Arnold-Reed DE, Holman CD, Coode J, Unwin E (1998) Effects of smoking and unsafe alcohol consumption on Aboriginal life expectancy. (Letter) *Medical Journal of Australia,* 168, 95.

*Ashley MJ (1982) Alcohol consumption, ischemic heart disease and cerebrovascular disease: An epidemiologic perspective. *Journal of Studies on Alcohol,* 43, 869–887.

*Ashley MJ, Ferrence R, Room R, Bondy S, Rehm J, Single E (1997) Moderate drinking and health. Implications of recent evidence. *Canadian Family Physician*, 43, 687–694.

Australian Bureau of Statistics (1991), *1989–90 National Health Survey Summary of Results, Australia.* Catalogue No. 4364.0, Commonwealth of Australia.

Australian Bureau of Statistics (2000) *Apparent consumption of selective foodstuffs, Australia.* Canberra: Australian Bureau of Statistics.

*Australian Institute of Health and Welfare (1999) *1998 National Drug Strategy Household Survey: First Results.* AIHW cat no. PHE 15. Canberra: AIHW (Drug Statistics Series)

Australian Institute of Health and Welfare, Australasian Association of Cancer Registries, & NHMRC National Breast Cancer Centre (1998) *Breast cancer survival in Australian women 1982–1994*. Canberra: Australian Institute of Health and Welfare, Cancer Series No. 9.

Bandura A (1976). *Social Learning Theory*, Prentice Hall.

Barnes GM, Farrell MP, Dintcheff BA (1997) Family socialisation effects on alcohol abuse and related problem behaviors among female and male adolescents. In RW Wilsnack, SC Wilsnack (Eds) *Gender and Alcohol: Individual Perspectives* (pp 156–175), New Brunswick, NJ: Rutgers Center of Alcohol Studies.

Barraclough B (1987) *Suicide: clinical and epidemiological studies*. London: Croom Helm.

*Beaglehole R, Jackson R (1992) Alcohol, cardiovascular diseases and all causes of death: A review of the epidemiologic evidence. *Drug and Alcohol Review,* 11, 290.

Beautrais AL (1998) *Risk factors for suicide and attempted suicide amongst young people.* A report to the National Health and Medical Research Council.

Beilin LJ, Puddey IB, Burke V (1996) Alcohol and hypertension: kill or cure? *Journal of Human Hypertension*, 10 Suppl 2, S1–5.

Berger D, Snortum J (1985) Alcoholic beverage preferences of drinking-driving violators. *Journal of Studies on Alcohol*, 46(3), 232–239.

Blankfield A, Maritz JS (1990) Female alcoholics IV: admission problems and patterns. *Acta Psychiatrica Scandinavica*, 12, 445–450

*Bode C, Bode JC (1997) Alcohol's role in gastrointestinal tract disorders. *Alcohol Health & Research World*, 21, 76–83.

*Bode JC, Bode C (1992) Alcohol malnutrition and the gastrointestinal tract. In: Watson R, Watzl B (Eds) *Nutrition and alcohol*, pp. 403–428. Boca Raton, FL: CTC Press.

*Boutron-Ruault MC, Senesse P, Faivre J, Couillault C, Belghiti C (1996) Folate and alcohol intake: Related or independent roles in the adenoma-carcinoma sequence? *Nutrition & Cancer*, 26, 337–346.

*Bowlin SJ, Leske MC, Varma A, Nasca P, Weinstein A, Caplan L (1997) Breast cancer risk and alcohol consumption: results from a large case-control study. *International Journal of Epidemiology,* 26(5), 915–923.

Brady M (1995) *Broadening the base of interventions for Aboriginal people with alcohol problems.* Technical Report no 29, National Drug and Alcohol Research Centre, University of New South Wales.

Bruun K, Edwards G, Lumio M, Makela K, Pan L, Popham RE, et al (1975) *Alcohol control policies in public health perspective*.Vol 25. Helsinki: Finnish Foundation for Alcohol Studies.

Cahalan D, Cisin IH, Crossley HM (1969) *American drinking practices: a national study of drinking behaviour and attitudes (Monograph 6)*. New Brunswick, NJ: Rutgers Center of Alcohol Studies.

*Camargo CA, Hennekens CH, Gaziano JM, Glynn RJ, Manson JE, Stampfer MJ (1997) Prospective study of moderate alcohol consumption and mortality in US male physicians. *Archives of Internal Medicine*, 157, 79–85.

Carcach C (1997) *Youth as Victims and Offenders of Homicide.* Trends and Issues in Crime and Criminal Justice No 73. Canberra: Australian Institute of Criminology.

Carcach C, McDonald D (1997) *National Police Custody Survey.* Canberra: Australian Institute of Criminology.

*Carlen PL, McAndrews MP, Weiss RT, Dongier M, Hill JM, Menzano E, Farcnik K, Abarbanel J, Eastwood MR (1994) Alcohol-related dementia in the institutionalized elderly. *Alcoholism, Clinical & Experimental Research*, 18, 1330–1334.

*Castaneda R, Sussman N, Levy O'Malley M, Westreich L (1998) A review of the effects of moderate alcohol intake on psychiatric and sleep disorders. In: Galanter M, (Ed.) *Recent developments in alcoholism: The consequences of alcoholism*, Vol 14 edn. pp. 197–226. New York: Plenum Press.

*Chenet L, Leon DA, McKee M, Vassin S (1998) Death from alcohol and violence in Moscow: Socioeconomic determinants. *European Journal of Population*, 14, 19–37.

*Cherpitel CJ (1992) The epidemiology of alcohol-related trauma. *Alcohol Health & Research World,* 16, 191–196.

*Cherpitel CJ (1994) Alcohol and injuries resulting from violence: A review of emergency room studies. *Addiction,* 88, 157–165.

*Cherpitel CJ (1997) Alcohol and violence-related injuries in the emergency room. *Recent Developments in Alcoholism,* 13, 105–118.

*Cherpitel CJ, Tam T, Midanik L, Caetano R, Greenfield T (1995) Alcohol and non-fatal injury in the US general population: a risk function analysis. *Accident Analysis Prevention,* 27, 651–661.

*Chhabra SK, Souliotis VL, Kyrtopoulos SA, Anderson LM (1996) Nitrosamines, alcohol, and gastrointestinal tract cancer: Recent epidemiology and experimentation. *In Vivo*, 10, 265–284.

Chick J (1999) Can light or moderate drinking benefit mental health? *European Addiction Research* 5(2),74–81.

Chikritzhs T, Jonas H, Stockwell, T, Heale P, Dietze P (2001) Mortality and life years lost due to alcohol: a comparison of acute and chronic causes. *Medical Journal of Australia*, 174(6), 281–284.

Chikritzhs T, Stockwell T, Heale P, Dietze P, Webb M (2000) *Trends in alcohol-related road injury in Australia, 1990–1997.* National Alcohol Indicators Project, Bulletin No. 2. National Drug Research Institute, Perth: Curtin University of Technology.

*Clarren SK, Sampson PD, Larsen J, Donnell DJ, Barr HM, Bookstein FL, Martin DC, Streissguth AP (1987) Facial effects of fetal alcohol exposure: Assessment by photographs and morphometric analysis. *American Journal of Medical Genetics,* 26, 651–666.

*Coate D (1993) Moderate drinking and coronary heart disease mortality: Evidence from NHANES I and the NHANES I follow-up. *American Journal of Public Health,* 83, 888–890.

*Coles C (1994) Critical periods for prenatal alcohol exposure. *Alcohol Health & Research World,* 18, 22–29.

Collins D, Lapsley H (1991) *Estimating the economic costs of drug abuse in Australia.* National Campaign Against Drug Abuse Monograph No. 15. Canberra: Commonwealth of Australia.

Collins DJ, Lapsley HM (1996) *The social costs of drug abuse in Australia in 1988 and 1992*. Canberra: Australian Government Publishing Service.

*Commonwealth Department of Health and Human Services (1994) *National drug strategy household survey: Urban Aboriginal and Torres Strait Islander Peoples supplement.* Canberra: Drug Statistics Series.

Commonwealth of Australia (1995) *National drug strategy household survey: Survey report 1995*. Canberra: Commonwealth Department of Health and Human Services.

Corrao G, Luca R, Bagnardi V, Zambon A, Poikolainen K (2000) Alcohol and coronary heart disease: a meta-analysis. *Addiction*, 95(10), 1505–1523.

Corti B, Ibrahim J (1990) Women and alcohol: trends in Australia. *Medical Journal of Australia*, 152(12), 625–32.

*Criqui M, Ringel BL (1994) Does diet or alcohol explain the French paradox? *Lancet*, 344, 1719–1723.

*Day NL (1992) The effects of prenatal exposure to alcohol. *Alcohol Health & Research World,* 18, 238–244.

Deeble J, Mathers C, Smith L, Goss J, Webb R, Smith V (1998) *Expenditure on Health Services for Aboriginal and Torres Strait Islander People*. Australian Institute of Health and Welfare and National Centre for Epidemiology and Population Health, Catalogue No HWE 6, Canberra.

Dimmitt SB, Rakic V, Puddey IB, Baker R, Oostryck R, Adams MJ, Chesternam CN, Burke V, Beilin LJ (1998) The effects of alcohol on coagulation and fibrinolytic factors: a controlled trial. *Blood Coagulation and Fibrinolysis*, 9(1), 39–45.

*Doll R (1997) One for the heart. *British Medical Journal*, 315, 1664–1668.

*Doll R (1998) The benefit of alcohol in moderation. *Drug and Alcohol Review,* 17, 353–363.

*Doll R, Forman D, La Vecchia C, Verschuren PM (1993) Alcoholic beverages and cancers of the digestive tract and larynx. In: Verschuren PM (Ed) *Health issues related to alcohol consumption*, pp. 125–166. Brussels: ILSI Europe.

Eckardt MJ, File SE, Gessa GL, Grant KA, Guerri C, Hoffman PL, Kalant H, Koop GF, Li TK, Tabakoff B (1998) Effects of moderate alcohol consumption on the central nervous system. *Alcoholism, Clinical & Experimental Research*, 22, 998–1040.

Edwards G, Marshall E, Cook C (1997) *The treatment of drinking problems: A guide for the helping professions*. Cambridge University Press.

Edwards GAP, Babor TF, Casswell S, Ferrence R, Giesbrecht N, Godfrey C, Holder HD, Lemmens P, Mäkelä K, Midanik LT, Norström T, Österberg E, Romelsjö A, Room R, Simpura J, Skog O-J (1994) *Alcohol Policy and the Public Good.* New York: Oxford University Press.

*Ellison RC (1990) Cheers. *Epidemiology*, 1, 337–339.

*English DR, Holman CD, Milne E, Winter MJ, Hulse GK, Codde G, Bower CI, Cortu B, de Klerk N, Lewin GF, Knuiman M, Kurinczuk JJ, Ryan GA (1995) *The quantification of drug caused morbidity and mortality in Australia*. Canberra: Commonwealth Department of Human Services and Health.

Favazzi A (1981) Alcohol and special populations. *Journal of Studies on Alcohol*, Suppl 9, 87–98.

Fergusson DM, Lynskey MT (1996) Alcohol misuse and adolescent sexual behaviors and risk taking. *Pediatrics*, 98(1), 91–96.

Feskens EJ, Kromhout D (1989) Cardiovascular risk factors and the 25-year incidence of diabetes mellitus in middle-aged men. The Zutphen Study. *American Journal of Epidemiology,* 130(6):1101–8.

*Freedland E, McMicken D, D'Onofrio G (1993) Alcohol and trauma. *Emergency Medicine Clinics of North America,* 11, 225–239.

*Friedman GD, Klatsky AL (1993) Is alcohol good for your health. *The New England Journal of Medicine*, 329, 1882–1883.

*Friedman HS (1998) Cardiovascular effects of alcohol. In: Galanter S (Ed) *The consequences of alcoholism*, pp. 135–166. New York: Plenum Press.

*Fuller M (1995) Alcohol use and injury severity in trauma patients. *Journal of Addictive Diseases*, 14, 47–53.

*Garro A, Lieber C (1992) Alcohol and cancer. *Annual Review of Pharmacology and Toxicology*, 30, 219–249.

*Gordis E (1995) Should physicians counsel patients to drink alcohol. *Journal of the American Medical Association*, 273, 1415–1416.

*Gordis E Dr Cutberto Garza (1999). Dietary Guidelines Advisory Committee, Letter.

Graham K, Schmidt G, Gillis K (1996) Circumstances when drinking leads to aggression: an overview of research findings. *Contemporary Drug Problems*, 23, 493–557.

Graham K, West P (2001) Alcohol and crime: examining the link. In N Heather, TJ Peters, T Stockwell (Eds.), *International Handbook of Alcohol Problems and Dependence.* West Sussex, U.K: John Wiley & Sons, Ltd.

*Grønbæk DA, Becker U, Johansen D, Tonnesen H, Jensen G, and Sorensen H (1998) Population based cohort study of the association between alcohol intake and cancer of the upper digestive tract. *British Medical Journal*, 317, 844–847.

*Gruchow HW, Hoffman RG, Anderson AF, Barboriack JJ (1982) Effects of drinking patterns on the relationship between alcohol and coronary occlusion. *Atherosclerosis*, 43, 393–404.

Gruenewald PJ, Millar A, Mitchell P (1995) Drinking and driving: explaining beverage specific risks. Paper presented at the Research Society on Alcoholism Annual Meeting, June 15–22, Steamboat Springs Colorado: Research Society on Alcoholism.

*Gruenewald PJ, Nephew T (1994) Drinking in California: theoretical and empirical analyses of alcohol consumption patterns. *Addiction,* 89, 707–723.

*Gruenewald PJ, Treno AJ, Mitchell PR (1996) Drinking patterns and drinking behaviours: theoretical models of risky acts. *Contemporary Drug Problems,* 23, 407–440.

*Haapaniemi H, Hillbom M, Juvela S (1996) Weekend and holiday increase in the onset of ischemic stroke in young women. *Stroke,* 27, 1023–1027.

Hagen R, Egan D, Eltringham A (1992) *Work, Drugs and Alcohol: A Research Report.* Victorian Occupational Health and Safety Commission.

Hall W, Teesson M, Lynskey M, Degenhardt L (1999) The 12-month prevalence of substance use and ICD-10 substance use disorders in Australian adults: findings from the National Survey of Mental Health and Well-Being. *Addiction*, 94, 1541–1550.

*Hannigan J, Abel EL, Spohr H, Steinhaussen H (1995) *Alcohol pregnancy and child development*. Cambridge: Cambridge University Press.

*Hart CL, Smith GD, Hole DJ, Hawthorne VM (1999) Alcohol consumption and mortality from all causes, coronary heart disease, and stroke: results form a prospective cohort study of Scottish men with 21 years of follow up. *British Medical Journal,* 318, 1725–1729.

*Harvard University Cancer Prevention Center (1997) Harvard report on cancer prevention. *Cancer Causes & Control*, 8, S47–S50.

Harwood JH, Fountain D, Livermore G (1999) Economic cost of alcohol and drug abuse in the United States, 1992: a report. *Addiction*, 94 (5), 631–634.

Heale P, Stockwell T, Dietze P, Chikritzhs T, Catalano P (2000) *Patterns of alcohol consumption in Australia, 1998.* National Alcohol Indicators Project, Bulletin No. 3. Perth, WA: National Drug Research Institute, Curtin University of Technology.

*Hendriks JF, Veenstra J, Van Tol A, Groener JE, Schaafsma G (1998) Moderate doses of alcoholic beverages with dinner and postprandial high density lipoprotein composition. *Alcohol & Alcoholism*, 33, 403–410.

Henningsen NC, Ohlsson O, Mattiasson I, Trell E, Kristensson H, Hood B (1980) Hypertension: levels of serum gamma glutamyl transpeptidase and degree of blood pressure control in middle-aged males. *Acta Med Scand,* 207, 245.

Higgins K, Cooper-Stanbury M, Williams P (2000) *Statistics on drug use in Australia 1998.* AIHW cat. no. PHE 16. Canberra: AIHW (Drug Statistics Series).

*Hillbom M, Haapaniemi J, Juvela S, Palomaki H, Numminen J, Kaste M (1995) Recent alcohol consumption, cigarette smoking and cerebral infarction in young adults. *Stroke,* 26, 40–45.

*Hillbom M, Juvela S (1996) Alcohol and the risk of stroke. In: Zakhari S, Wassef M (Eds) *Alcohol and the cardiovascular system*, Research Monograph–31 edn. Bethesda, MD: National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism.

Hingson R, Heerland T, Jamanka A, Howland J (2000) Age of drinking onset and unintentional injury involvement after drinking. *Journal of the American Medical Association*, 284, 1527–1533.

*Hingson R, Howland J (1987) Alcohol as a risk factor for injury or death resulting from accidental falls: a review of the literature. *Journal of Studies on Alcohol,* 48, 212–219.

*Hingson R, Howland J (1993) Alcohol and non-traffic unintended injuries. *Addiction,* 88, 983.

*Hodge AM, Dowse GK, Collins VR, Zimmet PZ (1993) Abnormal glucose tolerance and alcohol consumption in three populations at high risk of non-insulin dependent diabetes mellitus. *American Journal of Epidemiology,* 137, 178–189.

Holder H, Edwards G (Eds) (1995) Research as a basis for rational policies on alcohol consumption. In: *Alcohol and public policy: evidence and issues*. Oxford: Oxford University Press.

Holder H, Wagenaar AC (1994) Mandated server training and reduced alcohol-involved traffic crashes: A time series analysis of the Oregon experience. *Accident Analysis and Prevention,* 26, 89–97.

*Holman CD, English DR, Milne E, Winter MJ (1996) Meta-analysis of alcohol and all-cause mortality: a validation of NHMRC recommendations. *Medical Journal of Australia,* 164, 141–145.

*Homann N, Karkkainen P, Koivisto T, Jokelainen K, Salaspuro M (1997) Effects of acetaldehyde on cell regeneration and differentiation of the upper gastrointestinal tract mucosa. *Journal of the National Cancer Institute*, 89, 1692–1697.

Homel R, Clark J (1994) Predicting and preventing violence in clubs and pubs. *Crime Prevention Studies,* 3, 1–46.

Homel R, Tomsen S, Thommeny J (1992) Public drinking and violence: not just an alcohol problem. *Journal of Drug Issues,* 22(3), 679–697.

Houghton E, Roche AM (eds) (2001) *Learning About Drinking*. Washington DC: Taylor and Francis.

*Howe GR, Decarli A, Kaldor JK, Marubini EMA, Riboli E, Toniolo P (1991) The association between alcohol and breast cancer risk: Evidence from the combined analysis of six dietary case-control studies. *International Journal of Cancer*, 47, 707–710.

Hulse G, Saunders GK, Roydhouse RM, Stockwell TR, Basso MR (1999) Screening for hazardous alcohol use and dependence in psychiatric inpatients using the AUDIT questionnaire. *Drug and Alcohol Review*, 19, 291–298.

Hunter E, Reser J, Baird M, Reser P (1999) *An analysis of suicide in Indigenous communities of North Queensland: the historical, cultural and symbolic landscape*. University of Queensland Department of Social and Preventive Medicine.

Hunter EM (1991) An examination of recent suicides in remote Australia: further information from the Kimberley. *Australia and New Zealand Journal of Psychiatry,* 25, 197–202.

*Hurst PM, Harte WJ, Firth WJ (1994) The Grand Rapids dip revisited. *Accident Analysis Prevention,* 26, 647–654.

*International Agency for Research on Cancer (IARC) (Ed) (1988) *Alcohol drinking*. Lyon: IARC.

*Jaber MA, Porter SR, Scully C, Gilthorpe MS, Bedi R (1998) The role of alcohol in non-smokers and tobacco in non-drinkers in the aetiology of oral epithelial dysplasia. *International Journal of Cancer*, 77, 333–336.

*Jackson R (1994) Cardiovascular disease and alcohol consumption: evidence of benefits from epidemiologic studies. *Contemporary Drug Problems* 5–24.

*Jacobson JL, Jacobson SW (1994) Prenatal alcohol exposure and neurobehavioural development. *Alcohol Health & Research World,* 18, 30–36.

James M, Carcach C (1997) *Homicide in Australia 1989–96.* Research and Public Policy Series No 13. Australian Institute of Criminology, Canberra.

*Johnson JL, Leff M (1999) Children of substance abusers: An overview of research findings. *Pediatrics,* 103, 1085–1099.

*Kalant H, Khanna J (1989) The alcohols. In: Kalant H, Roschlau W (Eds) *Principles of medical pharmacology*, pp. 244–254. Toronto: Decker, Inc.

Kandel DB (1983) Socialisation and adolescent drinking. In O Jeanneret (Ed) *Alcohol and Youth,* Child Health and Development, Vol 2, pp 66–75, New York: Karger.

*Kato I, Dnistrian AM, Schwartz M, Toniolo P, Koenig K, Shore RE, Akhmedkhanov A, Zeleniuch-Jacquotte A, Riboli E (1999) Serum folate, homocysteine and colorectal cancer risk in women: A nested case-control study. *British Journal of Cancer*, 79, 1917–1922.

*Kauhanen J, Kaplan GA, Goldberg DE, Salonen JT (1997) Beer binging and mortality: Results from the Kuopio ischemic heart disease risk factor study, a prospective population based study. *British Medical Journal*, 315, 846–851.

Kelly AB, Halford K, Young RMcD (2000) Maritally distressed women with alcohol problems: the impact of a short-term alcohol-focused intervention on drinking behaviour and marital satisfaction. *Addiction*, 95, 1537–1549.

*Kessler R, Crum R, Warner L, Nelson C, Schulenberg J, Anthony J (1997) Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Archives of General Psychiatry* 54, 313–321.

*Kessler R, Nelson C, McGonagle K, Edlund M, Frank R, Leaf P (1996) The epidemiology of co-occurring addictive and mental disorders: Implications for prevention and service utilization. *American Journal of Orthopsychiatry,* 66, 17–31.

*Klatsky AL (1996) Alcohol, coronary disease, and hypertension. *Annual Review of Medicine,* 47, 149–160.

*Klatsky AL, Friedman GD (1995) Annotation: Alcohol and longevity. *American Journal of Public Health*, 85, 16–18.

Kopp P, Fénoglio P (1999) *Le cout social des drogues licites (alcool et tabac) et illicites*. Unpublished Report, L'Université de Panthéon-Sorbonne (Paris I), France.

*Kratzer W, Kachele R, Mason A, Muche R, Hay B, Wiesneth W, Hill V, Beckh K, Adler G (1997) Gallstone prevalence in relation to smoking, alcohol, coffee consumption and nutrition: the Ulm Gallstone Study. *Scandinavian Journal of Gastroenterology,* 32, 953–958.

*Kruger HP, Utzelmann HD, Berghaus G, Kroj G (1993) Effects of low alcohol dosages: a review of the literature. In: *Alcohol, drugs and traffic safety–T92*, pp. 763–778. Cologne, Germany: Verlag TUV Rheinland.

Kushner M, Abrams K, Borchadt C (2000) The relationship between anxiety disorders and alcohol use disorders: a review of major perspectives and findings. *Clinical Psychology Review*, 20, 149–171.

*Lang A, Martin S (1993) Alcohol-related violence: An individual offender focus. In: *Alcohol and interpersonal violence: Fostering multidisciplinary perspectives*, pp. 221–236. Washington, DC: United States Department of Health and Human Services.

Lawrence DL, Holman CD (1999) Alcohol and all-cause mortality: a meta-analysis. Unpublished paper prepared for the National Health and Medical Research Council.

*Leitzmann MF, Giovannucci EL, Rimm EB, Stampfer MJ, Spiegelman D, Wing AL, Willett WC (1998) The relation of physical activity to risk for symptomatic gallstone disease in men. *Annals of Internal Medicine,* 128, 417–425.

*Lelbach WK (1975) Cirrhosis in the alcoholic and its relation to the volume of alcohol abuse. *Annals of the New York Academy of Sciences,* 252, 85–105.

Lesage AD, Boyer R, Grunberg F, Vanier C, Morissette R, Ménard-Buteau C, Loyer (1994) Suicide and mental disorders: a case-control study of young men. *American Journal of Psychiatry*, 151, 1063–1068.

*Levi F, Pasche C, Lucchine F, La Vecchia C (1996) Alcohol and breast cancer in the Swiss Canton of Vaud. *European Journal of Cancer,* 32A, 2108–2113.

*Li G, Keyl PM, Smith GS, Baker SP (1997) Alcohol and injury severity: reappraisal of the continuing controversy. *Journal of Trauma,* 42, 562–569.

Ligon J, Thyer B, Lund R (1996) Drinking, eating, and driving: Evaluating the effects of partially removing a Sunday liquor sales ban. *Journal of Alcohol and Drug Education*, 42(1), 15–24.

*Longnecker MP (1992) Alcohol consumption in relation to risk of cancers of the breast and large bowel. *Alcohol Health & Research World*, 16, 223–229.

*Longnecker MP (1994) Alcoholic beverage consumption in relation to risk of breast cancer: Meta-analysis and review. *Cancer Causes & Control,* 5, 73–82.

Longnecker MP, Berlin JA, Orza MJ, Chalmers TC (1988) A meta-analysis of alcohol consumption in relation to risk of breast cancer. *Journal of the American Medical Association,* 260(5), 652–6.

Lynskey M (1998) The co-morbidity of alcohol dependence and affective disorders: treatment implications. *Drug and Alcohol Dependence*, 52, 201–209.

Makela K, Room R, Single R, Sulkunen P, Walsh B (1981) *Alcohol, society and the state*. Vol. 1. Toronto: Addiction Research Foundation.

Makkai T (1997) Alcohol and disorder in the Australian community: Part I—Victims. *Trends and Issues in Crime and Criminal Justice* No 76. Canberra: Australian Institute of Criminology.

*Makkai T (1998) Alcohol and disorder in the Australian community: Part II—Perpetrators. *Trends and Issues in Crime and Criminal Justice* No 77. Canberra: Australian Institute of Criminology.

Marsh P, Kibby KF (1992) *Drinking and public disorder*. Oxford: Alden Press.

*Martin N, Perl J, Oakeshott J, Givson J, Starmer G, Wilks A (1985) A twin study of ethanol metabolism. *Behavioural Genetics,* 15, 93–109.

*Martin SE (1982) The epidemiology of alcohol-related interpersonal violence. *Alcohol Health & Research World,* 16, 230–237.

*Martin SE, Bachman R (1997) The relationship of alcohol to injury in assault cases. In: Galanter M (Ed) *Recent developments in alcoholism,* pp. 41–56. New York: Plenum Press.

*Maskarinec G, Meng L, Kolonel LN (1998) Alcohol intake, body weight, and mortality in a multiethnic prospective cohort. *Epidemiology,* 9, 654–661.

Mathers C, Vos T, Stevenson C (1999) *The burden of disease and injury in Australia.* AIHW cat. no. PHE 17. Canberra: AIHW

*Mayhew DR, Donelson AC, Beirness DJ, Simpson MH (1986) Youth, alcohol and the relative risk of crash involvement. *Accident Analysis Prevention,* 18, 273–288.

*Mayhew DR, Simpson MH, Kaye S, Meier G (1985) Alcohol, age, and risk of road accident involvement. In: *Alcohol, drugs and traffic safety–T 83,* pp. 937–947. Washington, DC: National Highway Traffic Safety Administration.

McBride N, Midford R, Farringdon F, Phillips M (2000) Early results from a school alcohol harm minimization study: the School Health and Alcohol Harm Reduction Project. *Addiction*, 95, 1021–42.

McDonnel R, Maynard A (1985) The costs of alcohol misuse. *British Journal of Addiction,* 80, 27–35.

*McElduff P, Dobson A (1997) How much alcohol and how often? Population based case-control study of alcohol consumption and risk of major coronary event. *British Medical Journal*, 314, 1159–1164.

McKibbon KA, Wilczynski N, Hayward RS, Walker Dilks CK, Haynes RB (1995) The medical literature as a resource for health care practice. *Journal of the American Society of Information Science*, 46, 737–742.

McLeod R., Stockwell T, Stevens M, Phillips M (1999) The relationship between alcohol consumption patterns and injury. *Addiction*, 94, 1719–1734.

Midford R, Marsden A, Phillips M, Lake J (1997) Workforce alcohol consumption patterns at two Pilbara mining-related worksites. *Journal of Occupational Health and Safety—Australia New Zealand*, 13(3): 267–274.

Miller BA, Downs WR (1993) The impact of family violence on the use of alcohol by women. *Alcohol Health and Research World*, 17, 137–143

Miller BA, Downs WR, Gondoli DM (1989) Spousal violence among alcoholic women as compared with a random household sample of women. *Journal of Studies on Alcohol*, 50, 533–540.

Moore M, Gerstein D (Eds) (1980) *Alcohol and public policy: beyond the shadow of prohibition.* Washington DC: National Academy Press.

*Moskowitz H, Robinson C (1988) *Effects of low doses of alcohol on driving-related skills: A review of the evidence*. Washington, DC: National Highway Traffic Safety Administration.

Mueser KT, Kavanagh D (2001) Treating comorbidity of alcohol problems and psychiatric disorder. In N Heather, TJ Peters, T Stockwell (Eds), *International Handbook of Alcohol Dependence and Problems,* John Wiley & Sons.

Muir Gray JA (1997) *Evidence Based Health Care: How to Make Health Policy and Management Decisions.* Edinburgh, Churchill Livingston. Cited in Glasziou P, Longbottom H (1999), Evidence-based public health practice, *Australian and New Zealand Journal of Public Health*, 23: 436–440.

Murgraff V, Parrott, A & Bennett, P, (1999) *Risky single-occasion drinking amongst young people: definitions, correlates, policy and intervention: a broad overview of research findings.* Alcohol and Alcoholism, 34(1), 3–14

*Murray CJL, Lopez AD (1996) *The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020*. Cambridge, Harvard University Press.

*Murray CJL, Lopez AD (1999) On the comparable quantification of health risks: Lessons from the global burden of disease study. *Epidemiology,* 10, 594–605.

Nakamura K, Tanaka A, Takano T (1993) The social cost of alcohol abuse in Japan. *Journal of Studies in Alcohol,* 54, 618–625.

National Breast Cancer Centre (1999) *Summary of risk factors for breast cancer.*

*National Health and Medical Research Council (1992) *Is there a safe level of daily consumption of alcohol for men and women*. Canberra: NHMRC.

National Health and Medical Research Council (1999) *A guide to the development, implementation and evaluation of clinical practice guidelines*. Canberra: NHMRC.

*National Heart, Lung and Blood Institute (NHLBI) (1997) *Prevention and treatment of high blood pressure*. 20: H-7, Washington: NHLBI.

*National Institute on Alcohol Abuse and Alcoholism (1995) *The physician's guide to helping patients with alcohol problems*. Cat. No. 95–3769. Washington: NIAAA.

*National Institute on Alcohol Abuse and Alcoholism (1999) NIAAA-recommended year 2000 alcohol guidelines.

*National Stroke Association (1998) *Stroke prevention guidelines*. Washington: National Stroke Association.

Norstrom T (1998) Effects on criminal violence of different beverage types and private and public drinking. *Addiction,* 93, 689–699.

*Norton R, Morgan M (1989) The role of alcohol in mortality and morbidity from interpersonal violence. *Alcohol & Alcoholism,* 24, 565–576.

Olenick NL, Chalmers DK (1991) Gender-specific drinking styles in alcoholics and nonalcoholics. *Journal of Studies on Alcohol*, 52, 324–330.

*Orgogozo JM, Dartiques JF, Lafont S, Letenneur L, Commenges D, Salamon R, Renaud S, Breteler MB (1997) Wine consumption and dementia in the elderly: A prospective community study in the Bordeaux area. *Revue Neurologique,* 153, 185–192.

*Parsons OA, Nixon SJ (1998) Cognitive functioning in sober social drinkers: a review of the research since 1986. *Journal of Studies on Alcohol,* 59, 180–190.

*Passaro K, Little R, Savitz F, Noss J (1996) The effect of maternal drinking before conception and in early pregnancy on infant birthweight. *Epidemiology,* 7, 377–383.

*Pearson TA (1996) Alcohol and heart disease. *Circulation*, 94, 3023–3025.

*Pearson TA (1996) Executive summary. *Journal of the American College of Cardiology*, 27, 957–1047.

*Pearson TA, Terry P (1994) What to advise patients about drinking alcohol: A clinician's conundrum. *Journal of the American Medical Association*, 272, 967–968.

*Peele S (1997) Utilizing culture and behaviour in epidemiological models of alcohol consumption and consequences for Western countries. *Alcohol & Alcoholism,* 32, 51–64.

Perry C, Kelder S (1992) Prevention. In *Annual Review of Addictions Research and Treatment,* Pergamon Press, 453–472.

*Perry IJ, Wannamethee SG, Walker MK, Thomson AG, Whincup PH, Shaper AG (1995) Prospective study of risk factors for development of non-insulin dependent diabetes in middle aged British men. *British Medical Journal,* 310(6979):560–4.

Poikolainen K (1997) *Alcohol Poisoning in Four Nordic Countries: Alcohol Research in the Northern Countries*. Vol. 28, Helsinki: The Finnish Foundation for Alcohol Studies.

*Polygenis D, Wharton S, Malmberg C, Sherman N, Kennedy D, Koren G, Einarson T (1998) Moderate alcohol consumption during pregnancy and the incidence of fetal malformations: a meta-analysis. *Neurotoxicology and Teratology,* 20, 61–67.

*Preusser DF, Ulmer RG, Adams JR (1978) Driver record evaluation of a drinking driver rehabilitation program. *Journal of Safety Research,* 8, 98–105.

Puddey IB, Beilin LJ, Rakic V (1997) Alcohol, hypertension and the cardiovascular system—a critical appraisal. *Addiction Biology,* 2, 159–70.

*Puddey IB, Rakic V, Dimmitt SB, Beilin LJ (1999a) Influence of pattern of alcohol intake on blood pressure in regular drinkers: A controlled trial. *Journal of Hypertension*, 16, 165–174.

*Puddey IB, Rakic V, Dimmitt SB, Beilin LJ (1999b) Influence of pattern of drinking on cardiovascular disease and cardiovascular risk factors—a review. *Addiction*, 94, 649–663.

Putnam S, Rockett IR, Campbell MK (1993) Methodological issues in community-based alcohol-related injury prevention projects. In: Greenfield TK, Zimmerman R (eds.) *Center for Substance Abuse,* Prevention Monograph 14, Rockville, MD: US Department of Health and Human Services.

Rakic V, Puddey IB, Burke V, Dimmitt SB, Beilin LJ (1998) Influence of pattern of alcohol intake on blood pressure in regular drinkers: a controlled trial. *Journal of Hypertension,* 16(2), 165–174.

*Rehm J (1999) Draining the ocean to prevent shark attacks? The empirical foundation of modern alcohol policy. *Nordic Studies on Alcohol and Drugs,* 16, 46–54.

*Rehm J, Ashley MJ, Room R, Single E, Bondy S, Ferrence R, Giesbrecht N (1996b) On the emerging paradigm of drinking patterns and their social and health consequences. *Addiction,* 91, 1615–1621.

Rehm J, Fischer B (1997) Measuring harm: implications for alcohol epidemiology. In: Plant M, Single E, Stockwell T (Eds) *Alcohol: minimising the harm.* London: Free Association Press.

Rice D, Kelman S, Miller L, Dunmeyer S (1990) *The economic cost of alcohol and drug abuse and mental illness 1985.* DHHS Publication No. (ADM) 90, 1694. San Francisco: Institute for Health and Ageing, University of California.

*Rimm EB, Chan J, Stampfer M, Colditz GA, Willett W (1995) Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. *British Medical Journal,* 310, 555–559.

Roche AM (1998a) *The Availability of Alcohol and the Incidence of Harm*. In the Liquor Control Act 1987 Review, Final Report, State Government of Victoria.

Roche AM (1998b) Patterns, paradoxes and ambiguities of consumption. Paper presented to the Kettil Bruun Society Meeting, Perth, 1–5 February.

Roche AM (1999) *Social Issues Pertaining to the Liquor Act Review—Queensland*. A Report Prepared for the National Competition Policy Liquor Review Committee, University of Queensland.

Roche AM, Watt K (1998) *Thinking Drinking: An evaluation of drink smart*. Unpublished Report, QADREC, University of Queensland, July 1998.

Roche AM, Watt KA (1999). From celebration to inebriation: university students and drinking. *Drug and Alcohol Review,* 18:393–403.

Room R (1988) The dialectic of drinking in Australian life: from the Rum Corps to the wine column. *Drug and Alcohol Review*, 7, 413–437.

*Room R (1998) Alcohol and drug disorders in the International Classification of Disease: a shifting kaleidoscope. *Drug and Alcohol Review,* 12, 305–317.

*Rosenqvist M (1998) Alcohol and cardiac arrhythmias. *Alcoholism, Clinical & Experimental Research*, 22, 318S–322S.

*Royal College of Physicians (1995) *Alcohol and the heart in perspective*. London: Royal College of Physicians.

*Rubin R, Rand M (1994) Alcohol and platelet function. *Alcoholism, Clinical and Experimental Research*, 18, 105–110.

*Russell M, Copper M, Frone M, Welte J (1991) Alcohol drinking patterns and blood pressure. *American Journal of Public Health*, 81, 452–457.

Ryan GA (2000) *A road side survey of drinking drivers in Perth, Western Australia.* Report RR94. Road Accident Prevention Research Unit, Department of Public Health, The University of Western Australia.

*Sacco RL, Elkind M, Boden-Albala B, Lin IF, Kargman DE, Hauser WA, Shea S, Paik MC (1999) The protective effect of moderate alcohol consumption on ischemic stroke. *Journal of the American Medical Association,* 281, 53–60.

Sackett D, Rosenberg W, Muir Gray J, Haynes R, Richardson W (1996) Evidence based medicine: what it is and what it isn't. *British Medical Journal*, 312, 71–72.

*Sahi T, Paffenbarger RSJ, Hsieh CC, Lee IM (1998) Body mass index, cigarette smoking, and other characteristics as predictors of self-reported, physician-diagnosed gallbladder disease in male college alumni. *American Journal of Epidemiology,* 147, 644–651.

Saltz R (1987) The roles of bars and restaurants in preventing alcohol-impaired driving: An evaluation of server training. *Evaluation in Health Professions* 10, 5–27.

*Schmidt W (1977) The epidemiology of cirrhosis of the liver: A statistical analysis of mortality data with special reference to Canada. In: Fisher MM, Rankin J (Eds) *Alcohol and the liver*, pp. 1–26. New York: Plenum Press.

*Schuckit MA, Smith TL, Tipp JE (1997) The self-rating of the Effects of Alcohol (SRE) form as a retrospective measure of the risk for alcoholism. *Addiction,* 92, 979–988.

Shaffer D, Gould M, Fisher P, Trautman P, Mourau D, Kleinman M, Flory M (1996) Psychiatric diagnosis in child and adolescent suicide. *Archives of General Psychiatry,* 53, 339–348.

Shanahan P, Hewitt N (1999) *Developmental Research for a National Alcohol Campaign, Summary Report*. Elliott & Shanahan Research. Commonwealth Department of Health and Aged Care.

Sher KJ, Gershuny BS, Peterson L, Raskin G (1997) The role of childhood stressors in the intergenerational transmission of alcohol use disorders. *Journal of Studies on Alcohol,* 58, 414–427.

Siegal S (1999) Drug anticipation and drug addiction: The 1998 H David Archibald Lecture. *Addiction,* 94(8), 1113–1124.

*Simon HB (1994) Patient-directed, nonprescription approaches to cardiovascular disease. *Archives of Internal Medicine*, 154, 2283–2296.

Simons LA, McCallum J, Friedlander Y, Ortiz M, Simons J (2000) Moderate alcohol intake is associated with survival in the elderly: the Dubbo Study. *Medical Journal of Australia* 173, 121–4.

Single E, Ashley MJ, Bondy S, Rankin J, Rehm J (1999a) *Evidence Regarding the Level of Alcohol Consumption Considered to be Low-Risk for Men and Women.* Report Prepared for the Commonwealth Department of Health and Aged Care, Canberra.

Single E, Giesbrecht N, Eakin B (Eds) (1981) Alcohol, society and the state. Vol 2, A social history of control policy in seven countries. Toronto: Addiction Research Foundation.

*Single E, Robson L, Rehm J, Xie X (1999b) Morbidity and mortality attributable to alcohol, tobacco and illicit drug use in Canada. *American Journal of Public Health,* 89, 385–390.

*Single E, Robson L, Xie X, Rehm J (1998) The economic costs of alcohol, tobacco and illicit drugs in Canada, 1992. *Addiction,* 93, 991–1006.

Single E, Wortley S (1993) Drinking in various settings as it relates to demographic variables and level of consumption: findings from a national survey in Canada. *Journal of Studies on Alcohol*, 54, 590–599.

*Singletary KW (1996) Alcohol and breast cancer: Interactions between alcohol and other risk factors. *Alcoholism, Clinical & Experimental Research,* 20, 57A–61A.

Smart RG (1996) Behavioral and social consequences related to the consumption of different beverage types. *Journal of Studies on Alcohol*, 57, 77–84.

Smith AMA, Rosenthal D (1997) Sex, alcohol and drugs? Young people's experience of Schoolies Week. *Australian and New Zealand Journal of Public Health*, 21, 175–80.

Stampfer MJ, Colditz GA, Willett WC, Manson JE, Arky RA, Hennekens CH, Speizer FE (1988) A prospective study of moderate alcohol drinking and risk of diabetes in women. *American Journal of Epidemiology,* 128(3):549–58.

Stockwell T (1994) Alcohol withdrawal: An adaptation to heavy drinking of no practical significance? *Addiction*, 89(11), 1447–1454.

Stockwell T (1995) Alcohol, violence and licensed premises: the nature of the relationship. In T Stockwell (Ed), Alcohol misuse and violence report 5: An examination of the appropriateness and efficacy of liquor licensing laws across Australia. (pp.105–118). Canberra: Australian Government Publishing Service.

Stockwell T (1997) Regulation of the licensed drinking environment: a major opportunity for crime prevention. *Crime Prevention Studies*, 7, 7–34.

Stockwell T, Bolderston H (1987) Alcohol and phobias. *British Journal of Addiction*, 82(9), 971–980.

*Stockwell T, Hawks D, Lang E, Rydon, P (1996) Unravelling the preventive paradox for acute alcohol problems. *Drug and Alcohol Review*, 15, 7–15.

Stockwell T, Masters L, Philips M, Daly A, Gahegan M, Midford R, Philp A (1998) Consumption of different alcoholic beverages as predictors of local rates of night-time assault and acute alcohol related morbidity. *Australian and New Zealand Journal of Public Health*, 22, 237–242.

Stockwell T, Sitharthan T, McGrath D, Lang E (1994) The measurement of alcohol dependence and impaired control in community samples. *Addiction*, 89, 167–74.

Stockwell T, Somerford P, Lang E (1992) The relationship between licence type and alcohol-related problems attributed to licensed premises in Perth, Western Australia. *Journal of Studies on Alcohol*, 53, 495–498.

Strunin L, Hingson R (1992) Alcohol, drugs, and adolescent sexual behavior. *International Journal of the Addictions*, 27(2), 129–146.

*Svardsudd K (1998) Moderate alcohol consumption and cardiovascular disease: is there evidence for a preventive effect. *Alcoholism, Clinical and Experimental Research,* 22, 307S–314S.

*Swanson CA, Coates RJ, Malone KE, Gammon MD, Schoenberg JB, Brogan DJ, McAdams M, Potischman N, Hoover RN, Brinton LA (1997) Alcohol consumption and breast cancer risk among women under age 45 years. *Epidemiology,* 8, 231–237.

*Swedish Medical Research Council (1998) Individual harmful and protective effects of alcohol. *Alcoholism, Clinical and Experimental Research*, 22, 365S–373S.

Tatz C (1999) *Aboriginal suicide is different—Aboriginal youth suicide in New South Wales, the Australian Capital Territory and New Zealand: towards a model of explanation and alleviation*. A report to the Criminology Research Council on CRC Project 25/96–7.

*The Johns Hopkins Medical Institutions (1998) *Nutrition and longevity*. H-2, Baltimore: The Johns Hopkins Medical Institutions.

Thorley A (1980) Medical responses to problem drinking. *Medicine,* 35, 1816–1822.

*Thun MJ, Peto R, Lopez AD, Monaco JH, Henley SJ, Heath CWJ, Doll R (1997) Alcohol consumption and mortality among middle-aged and elderly US adults. *The New England Journal of Medicine,* 337, 1705–1714.

*Treno AJ, Gruenewald PJ, Ponicki WR (1997) The contribution of drinking patterns to the relative risk of injury in six communities: a self-report based probability approach. *Journal of Studies on Alcohol,* 58, 372–381.

*Treno AJ, Holder HD (1997) Measurement of alcohol-involved injury in community prevention: the search for a surrogate III. *Alcoholism, Clinical & Experimental Research,* 21, 1695–1703.

Tuomilehto J, Enlund H, Salonen JT, Nissinen A (1984) Alcohol, patient compliance and blood pressure control in hypertensive patients. *Scandinavian Journal of Social Medicine,* 12, 177–81.

*United Kingdom Department of Health (1995) *Sensible drinking*. H-17, London, UK: Department of Health, Crown Copyright.

United States Department of Health and Human Services (1991) *Healthy People 2000: National health promotion and disease prevention objectives*. NIH publication no. (PHS)91–50212. Washington DC: US Govt Printing office.

*United States Department of Health and Human Services (1997) *Ninth special report to the US Congress on alcohol and health from the secretary of health and human services*. Washington DC: US Department of Health and Human Services, Public Health Service, National Institute of Health, National Institute on Alcohol Abuse and Alcoholism.

United States Department of Health and Human Services (2000) *Tenth special report to the US Congress on alcohol and health from the secretary of health and human services.* Washington, DC: U.S. Department of Health and Human Services, Public Health Service, National Institute of Health, National Institute on Alcohol Abuse and Alcoholism.

*United States Dietary Association and Department of Health and Human Services (1995) *Nutrition and your health: Dietary guidelines for Americans*. H-15, Washington: USDA/USDHHS.

*Veenstra J, Ockhuizen T, van de Pol H, Wedel M, Schaafsma G (1990) Effects of a moderate dose of alcohol on blood lipids and lipoproteins postprandially and in the fasting state. *Alcohol & Alcoholism*, 25, 371–377.

*Wannamethee SG, Shaper AG (1996) Patterns of alcohol intake and risk of stroke in middle-aged British men. *Stroke*, 27, 1033–1039.

Wei M, Gibbons LW, Mitchell TL, Kampert JB, Blair SN (2000) Alcohol intake and incidence of type 2 diabetes in men. *Diabetes Care* 23(1):18–22.

Weiss S (1999) Israeli Alcohol Consumption Guidelines. Personal communication from S Weiss to E Single.

Weschler H, Davenport A, Dowdall G, Moeykens B, Castillo S (1994) Health and behavioral consequences of binge drinking in college. *Journal of the American Medical Association,* 272, 672–7.

Willett WC, Stampfer MJ (1997) Sobering data on alcohol and breast cancer. *Epidemiology,* 8, 225–227.

*Williams CM, Skinner AE (1990) The cognitive effects of alcohol abuse: a controlled study. *British Journal of Addiction,* 85, 911–917.

Williams FG, Knox R (1987) Alcohol abuse intervention in a university setting. *Journal of American College Health*, 36, 97–102.

Williams P (1997) *Progress of the National Drug Strategy: Key National Indicators*. Canberra: Department of Health and Family Services.

*Windham G, Fenster L, Hopkins B, Swan S (1995) The association of moderate maternal and paternal alcohol consumption with birthweight and gestational age. *Epidemiology,* 6, 591–597.

World Health Organization (2000) *International Guide for Monitoring Alcohol Consumption and Related Harm.* Department of Mental Health and Substance Dependence, Noncommunicable Diseases and Mental Health Cluster, World Health Organization, Geneva.

*Wright RM, McManaman JL, Repine JE (1999) Alcohol-induced breast cancer: A proposed mechanism. *Free Radical Biology & Medicine,* 26, 348–354.

Wright S, Norton VC, Dake AD Pinkston JR, Slovis CM (1998) Alcohol on campus: alcohol-related emergencies in undergraduate college students. *Southern Medical Journal*, 91(10), 909–913.

*Yamada K, Araki S, Tamura M, Sakai I, Takahashi Y, Sashihara H, Kono S (1997) Case-control study of colorectal carcinoma in situ and cancer in relation to cigarette smoking and alcohol use. *Cancer Causes & Control*, 8, 780–785.

*You R, McNeil J, O'Malley H, Davis S, Thrift A, Donnan G (1997) Risk factors for stroke due to cerebral infarction in young adults. *Stroke,* 28, 1913–1918.

Zador D, Sunjic S, Darke S (1996) Heroin-related deaths in New South Wales, 1992: Toxicological findings and circumstances. *Medical Journal of Australia*, 164, 204–207.

*Zador PL (1991) Alcohol-related relative risk of fatal driver injuries in relation to driver and age and sex. *Journal of Studies on Alcohol,* 52, 302–310.

*Zakhari S, Wassef M (1996) *Alcohol and the cardiovascular system.* Bethesda, MD: National Institutes on Health, National Institute on Alcohol Abuse and Alcoholism.

*Zhang L, Wieczorek W, Welte J (1997) The nexus between alcohol and violent crime. *Alcoholism, Clinical & Experimental Research,* 21, 1264–1271.

Zinkiewicz L, Davey J, Curd D (1999) Sin and beyond Surfers? Young people's risky behaviour during Schoolies Week in three Queensland regions. *Drug and Alcohol Review,* 18, 279–85.

Note: *Indicates references quoted in the Literature Review commissioned by the NHMRC to form the primary evidence base for these guidelines.

GLOSSARY AND ACRONYMS

AIHW

Australian Institute of Health and Welfare.

alcohol

In this document, refers specifically to *ethyl alcohol* or *ethanol*, the type of alcohol found in drinks intended for human consumption.

atherosclerosis

'Hardening of the arteries'; a disease of the arteries in which fatty and scar-like deposits (*plaques*) of atheroma form on the inner walls of arteries. It is the most common cause of *ischaemic stroke*.

binge

Not a preferred term due to its lack of consistent and specific meaning. Conventionally can refer to either a) occasional bouts of heavy drinking by young and/or non-dependent people, or b) a 'bender' engaged in by an alcohol-dependent person which may last for days or weeks.

BAC

Blood alcohol concentration.

blood alcohol concentration

The quantity of alcohol contained in a given volume of blood, expressed in grams of alcohol per 100 millilitres of blood.

CAD

Coronary artery disease.

cardiovascular

To do with the heart (*cardio*) and the blood vessels (*vascular*). *Cerebrovascular* diseases are a subset of cardiovascular diseases, affecting the blood vessels of the brain, and stroke is caused by cerebrovascular disease.

CHD

Coronary heart disease.

cirrhosis

A permanent change in the architecture of the liver, characterised by necrosis (death of cells) and fibrosis (scarring). Cirrhosis may or may not affect liver function overtly.

cognition

The ability of the brain to perform intellectual tasks, including thinking, planning, recalling, perception and recognition, judgement, language.

dependence

A phenomenon with biological, psychological and social elements, whereby use of a particular drug (eg alcohol) is given priority over other behaviours that were once relatively very much more important to the person. Dependence is not an all or none phenomenon, but exists in degrees along a continuum.

FAS

Foetal alcohol syndrome.

foetal alcohol syndrome

A relatively uncommon cluster of mainly facial abnormalities and poor child development, which has been observed in children born to women drinking heavily during pregnancy.

haemorrhagic stroke

A stroke caused by a bleed either into the brain (intracerebral haemorrhage) or over the surface of the brain (subarachnoid haemorrhage).

high risk

Defines a level of drinking at which there is substantial risk of serious harm, and above which risk continues to increase rapidly.

hypertension

High blood pressure.

IARC

International Agency for Research on Cancer.

intoxication

No consistent or formally agreed definition, however it is usually taken to refer to an elevated blood alcohol concentration such that a person cannot function within their normal range of physical/cognitive abilities. In some instances, levels above 0.05 or 0.08 are taken as a measure of intoxication, but this is not universally agreed upon. In lay terms, it is a subjective feeling, the experience of a substantial effect of alcohol on mood, cognition, and psycho-motor function

ischaemia

An inadequate flow of blood to part of the body due to blockage or constriction of the arteries that supply it.

ischaemic stroke

A stroke caused by blockage of blood vessels.

JAMA

Journal of the American Medical Association.

long-term risk

the level of long-term risk associated with regular daily patterns of drinking, defined by the total amount of alcohol typically consumed per week. This is the way alcohol-related risk has traditionally been assessed.

low risk

Defines a level of drinking at which there is only a minimal risk of harm. At this level, there may be health benefits for some of the population.

meta-analysis

The use of statistical methods to combine and summarise the results of several studies.

NHMRC

National Health and Medical Research Council.

NIAAA

National Institute on Alcohol Abuse and Alcoholism.

pattern of drinking

Refers to aspects of drinking behaviour other than the level of drinking, and includes when and where drinking takes place, the number and characteristics of heavy drinking occasions, activities associated with drinking, personal characteristics of the drinker and drinking companions, the types of drinks consumed, and the drinking norms and behaviours that comprise a 'drinking culture'.

peripheral vascular disease

Disease of the blood vessels (*atherosclerosis*) causing narrowing of the arteries in the leg.

psychomotor

The coordination between the central nervous system (brain) and the muscles of the body.

risk factor

Anything that increases a person's chance of developing a particular disease or condition. Smoking, high blood pressure, and old age, for example, are all risk factors for stroke.

risky

Defines a level of drinking at which risk of harm is significantly increased beyond any possible benefits.

short-term risk

The risk of harm (particularly injury or death) in the short-term, that is associated with given levels of drinking on a single day. These levels assume that overall drinking patterns remain within the levels set for long-term risk, and that these heavier drinking days occur a maximum of three times per week. Outside these limits, risk is further increased.

standard drink

Containing 10 grams of alcohol (equivalent to 12.5 millilitres of alcohol).

stroke

Sudden and unexpected damage to brain cells that causes symptoms in the parts of the body controlled by those cells. It can affect thinking, movement, speech and/or the senses. It happens when the blood supply to part of the brain is suddenly disrupted, either through blockage of an artery in the brain (ischaemic stroke) or bleeding into the brain (haemorrhagic stroke).

threshold

A level of drinking below which drinking is completely safe in relation to a particular disorder, and above which the risk for that disorder starts to increase.

tolerance

The situation, after a person has been exposed to a drug (eg alcohol), when higher quantities of that drug are required to produce a given response. In the case of alcohol, people who drink regularly at a given level gradually show less immediate and apparent effects of alcohol at this level of drinking. This tolerance has a metabolic element, whereby the liver becomes faster and more efficient at breaking down alcohol; and a functional element, whereby the person learns to cope with and compensate for the deficits induced by alcohol.

USDHHS

United States Department of Health and Human Services.

WHO

World Health Organisation.

The National Health and Medical Research Council

The National Health and Medical Research Council (NHMRC) is a statutory body within the portfolio of the Commonwealth Minister for Health and Aged Care, established by the *National Health and Medical Research Council Act 1992*. The NHMRC advises the Australian community and Commonwealth; State and Territory Governments on standards of individual and public health, and supports research to improve those standards.

The NHMRC advises the Commonwealth Government on the funding of medical and public health research and training in Australia and supports many of the medical advances made by Australians.

The NHMRC also develops guidelines and standards for the ethical conduct of health and medical research.

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