



# Australian Guidelines TO REDUCE HEALTH RISKS from Drinking Alcohol

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ISBN Print: 1864963743

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ISBN Online: 1864963808

Published: February 2009

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## Summary

Alcohol has a complex role in Australian society. Most Australians drink alcohol, generally for enjoyment, relaxation and sociability, and do so at levels that cause few adverse effects. However, a substantial proportion of people drink at levels that increase their risk of alcohol-related harm. For some, alcohol is a cause of significant ill health and hardship. In many countries, including Australia, alcohol is responsible for a considerable burden of death, disease and injury. Alcohol-related harm to health is not limited to drinkers but also affects families, bystanders and the broader community.

These 2009 National Health and Medical Research Council (NHMRC) guidelines aim to establish the evidence base for future policies and community materials on reducing the health risks that arise from drinking alcohol. The guidelines communicate evidence concerning these risks to the Australian community to allow individuals to make informed decisions regarding the amount of alcohol that they choose to drink.

Research since the previous edition of the guidelines in 2001 has reinforced earlier evidence on the risks of alcohol-related harm, including a range of chronic diseases and accidents and injury. The new guidelines take a new approach to developing population-health guidance, which:

- goes beyond looking at the immediate risk of injury and the cumulative risk of chronic disease, to estimating the overall risk of alcohol-related harm over a lifetime
- provides advice on lowering the risk of alcohol-related harm, using the level of one death for every 100 people as a guide to acceptable risk in the context of present-day Australian society
- provides universal guidance applicable to healthy adults aged 18 years and over (Guidelines 1 and 2) and guidance specific to children and young people (Guideline 3) and to pregnant and breastfeeding women (Guideline 4).

## GUIDELINE 1

### *Reducing the risk of alcohol-related harm over a lifetime*

The lifetime risk of harm from drinking alcohol increases with the amount consumed.

**For healthy men and women, drinking no more than two standard drinks on any day reduces the lifetime risk of harm from alcohol-related disease or injury.**

Guideline 1 (page 39) is based on modelling that provides information on the lifetime risk of harm from drinking, from a chronic disease or through accident or injury. The modelling shows that:

- for both men and women, the lifetime risk of death from alcohol-related disease or injury remains below 1 in 100 if no more than two standard drinks are consumed on each drinking occasion, even if the drinking is daily
- every drink above this level continues to increase the lifetime risk of both disease and injury
- drinking less frequently over a lifetime (eg drinking weekly rather than daily), and drinking less on each drinking occasion, reduces the lifetime risk of alcohol-related harm.

There is little difference between men and women in the risk of alcohol-related harm at low levels of drinking. However, at higher levels of drinking, the lifetime risk of alcohol-related disease increases more quickly for women and the lifetime risk of alcohol-related injury increases more quickly for men.

Age is an important determinant of health risks related to alcohol. Harm from alcohol-related accident or injury is experienced disproportionately by younger people; for example, over half of all serious alcohol-related road injuries occur among 15–24-year-olds. Harm from alcohol-related disease is more evident among older people.



## GUIDELINE 2

### *Reducing the risk of injury on a single occasion of drinking<sup>1</sup>*

On a single occasion of drinking, the risk of alcohol-related injury increases with the amount consumed.

**For healthy men and women, drinking no more than four standard drinks on a single occasion reduces the risk of alcohol-related injury arising from that occasion.**

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Each drinking occasion also contributes to the lifetime risk of alcohol-related harm.

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Guideline 2 (page 51) is based on evidence suggesting that:

- as more alcohol is consumed on a single occasion, skills and inhibitions decrease while risky behaviour increases, leading to a greater risk of injury during or immediately after that occasion
- while women reach a given blood alcohol concentration with a lower amount of alcohol, on average, men take more risks and experience more harmful effects
- drinking four standard drinks on a single occasion more than doubles the relative risk of an injury in the six hours afterwards, and this relative risk rises even more rapidly when more than four drinks are consumed on a single occasion.

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<sup>1</sup> A single occasion of drinking refers to a sequence of drinks taken without the blood alcohol concentration reaching zero in between.

### GUIDELINE 3

#### *Children and young people under 18 years of age*

For children and young people under 18 years of age, not drinking alcohol is the safest option.

- A** Parents and carers should be advised that children under 15 years of age are at the greatest risk of harm from drinking and that for this age group, not drinking alcohol is especially important.
- B** For young people aged 15–17 years, the safest option is to delay the initiation of drinking for as long as possible.

Guideline 3 (page 57) is based on an assessment of the potential harms of alcohol for young people, as well as a range of epidemiological research, which show that:

- drinkers under the age of 15 years are much more likely than older drinkers to undertake risky or antisocial behaviour connected with their drinking
- risky behaviour is more likely among drinkers aged 15–17 years than older drinkers; if drinking does occur in this age group, it should be at a low risk level and in a safe environment, supervised by adults
- alcohol may adversely affect brain development and lead to alcohol-related problems in later life.

## GUIDELINE 4

### *Pregnancy and breastfeeding*

Maternal alcohol consumption can harm the developing fetus or breastfeeding baby.

- A For women who are pregnant or planning a pregnancy, not drinking is the safest option.**
- B For women who are breastfeeding, not drinking is the safest option.**

Guideline 4 (page 67) is based on an assessment of the evidence on the potential harms of alcohol for the developing fetus and for young babies during the breastfeeding period. The level of risk is:

- highest when there is high, frequent maternal alcohol intake
- likely to be low if a woman has consumed only small amounts of alcohol (such as one or two drinks per week) before she knew she was pregnant or during pregnancy
- more likely to be related to neurodevelopmental abnormalities than prematurity, miscarriage, still birth or reduced birth weight at low levels of maternal alcohol consumption
- individually variable as it is influenced by a wide range of maternal and fetal characteristics.

The evidence also shows that alcohol may adversely affect lactation, infant behaviour (eg feeding) and psychomotor development of the breastfed baby. As Australian and international guidelines recommend breastfeeding for the first six months, advice is provided for women who choose to drink in this period (see page 80).

### ***Further issues to consider***

There are a number of additional factors that influence the risk of alcohol-related harm, including:

- specific situations where alcohol has the potential to endanger life; for example, when drinking is combined with activities such as driving, operating machinery or supervising children
- groups that can be at increased risk if they drink alcohol; for example, young adults (18–25 years), older people (60+ years), people with a family history of alcohol dependence, and people who use drugs illicitly
- people who may need to seek professional advice about drinking; for example, people taking medication, people with alcohol-related or other physical conditions, and people with mental health conditions.

These situations and groups are discussed in more detail in Appendix A1.

### ***Making decisions about personal risk***

Choices about alcohol are part of wider considerations that include factors related to each person's lifestyle and health and also depend on contextual and individual circumstances. However, people who choose to drink more than the guideline levels should understand that they will, on average, increase their risk of harm to a level that is higher than that for a person who chooses not to drink or to drink at a lower level.

When making decisions about drinking levels and patterns, people should also take into account the fact that drinking can adversely affect others, and in some circumstances may be against the law.

These guidelines are concerned with risks to health, and not with moral or normative standards about drinking. Various groups in Australian society differ about what they consider to be 'responsible' drinking, and about when they consider drinking to be appropriate or acceptable. There is a need for continuing public debate about these standards of conduct.

# Introduction

This is a revised edition of the National Health and Medical Research Council's (NHMRC) evidence-based alcohol guidelines. The previous edition, *The Australian Alcohol Guidelines: Health Risks and Benefits*, was issued by the NHMRC in 2001 (NHMRC 2001 guidelines). As part of its regular review of guidelines, and following consultation with the Australian Government Department of Health and Ageing (DoHA), the former NHMRC Health Advisory Committee (HAC) undertook to review and update the guidelines in 2006–07 and appointed an expert working committee to guide the redevelopment process. Committee members are listed in Appendix A2.

Research since 2001 has reinforced previous evidence on the health risks from drinking alcohol. In addition, recent research has taken an analytic approach to the health risks of drinking, resulting in new findings regarding the risks of alcohol-related harm over a lifetime.

Consequently, these revised guidelines are significantly different from the last edition. Major changes include the following:

- the guidelines introduce the concept of progressively increasing risk of harm with the amount of alcohol consumed, rather than specifying 'risky' and 'high risk' levels of drinking above guideline levels
- the number of guidelines has been reduced and the text simplified to provide two universal guidelines for healthy adults, one guideline for children and young people, and one guideline for pregnant or breastfeeding women. Appendix A1 sets out further issues to consider for certain groups and situations
- the guidelines for healthy adults are based on calculations that estimate the cumulative lifetime risk of alcohol-related injury or disease associated with many drinking occasions (Guideline 1) and the immediate increase in risk of injury from drinking on a single occasion (Guideline 2), both of which are lower than the NHMRC 2001 guideline levels for men, with Guideline 1 also lower for women
- the guidelines for children and young people, and for women during pregnancy and breastfeeding, are both more conservative than the comparable NHMRC 2001 guidelines, with advice to consider not drinking to eliminate alcohol-related risk in these situations.

## Scope of the guidelines

These *Australian Guidelines to Reduce Health Risks from Drinking Alcohol* (the guidelines) are intended to form the evidence base for developing future policies and materials. They aim to establish the basis of clear guidance for Australians on reducing their risk of harm from drinking alcohol.

The guidelines provide a resource for a range of groups including health professionals, professional organisations and industry. They will also inform policy makers, planners, decision-makers, and those responsible for providing alcohol, who have a broader responsibility to the community.

### KEY POINT

The intended role of the guidelines is as a technical document, although members of the public wanting to make decisions about their own drinking may also be interested in reading them. A range of plain-English booklets and other resources will be produced to help individuals, families and community groups to make choices based on the guidelines.

**The guidelines focus on reducing health risks from drinking.** The following are *not* included as they go beyond the scope of scientific advice:

- detailed information about the adverse economic and social effects of alcohol consumption
- recommendations about legal or other regulatory processes associated with alcohol
- detailed recommendations in relation to specific health conditions
- standards of conduct associated with alcohol
- the role of the health service (including general practice) in alcohol assessment, referral and treatment.

As detailed recommendations relating to specific health conditions are beyond the scope of these guidelines, specialist professional organisations and societies are encouraged to develop additional guidelines to meet such needs.

## Evidence base for the guidelines

The guidelines are based on scientific evidence from the following sources:

- a literature review of epidemiological studies on a broad range of alcohol-related issues, with a focus on meta-analyses and systematic reviews of prospective cohort and other epidemiological studies of alcohol's causative role in different health conditions (see page 103)
- use of a modelling approach based on single episode data from major epidemiological studies to estimate a level of drinking that leads to a lifetime risk of death from alcohol-related injury or disease of less than 1 in 100 people with that drinking pattern (Rehm et al 2008; Taylor et al 2008)
- re-examination of a range of existing datasets to estimate levels of drinking on a single occasion associated with harm
- investigation of the risk of being hospitalised for injury at different frequencies of drinking specific amounts on an occasion, using the Victorian Admitted Episode Database
- analysis of adult respondents (aged 18+) in the *2004 National Drug Strategy Household Survey* (AIHW 2005) concerning gender patterns in hazardous behaviour and delinquent behaviour.

A summary of the evidence is included for each of the guidelines. An overview of the guideline development process, including the methodology for the literature review, is presented in Appendix A3. Appendix A5 contains more detailed technical information about the risk analyses supporting the guidelines.

## Quality of the evidence

All guidelines are based on the best available evidence. There is much evidence on alcohol-related disease, including many epidemiological studies and, in recent years, several major meta-analyses. There is less evidence available on the risk of alcohol-related injury. The most comprehensive data are on patients who present to emergency departments for alcohol-related injuries. Studies and surveys are generally based on self-reported data on the amount drunk, which are known to be frequently underestimated.

There are no prospective trials of key areas of interest as these would be both unethical and inappropriate. Most evidence is observational, except for some experimental studies (eg driving simulation studies).

Guideline 1 is based on a modelling approach that uses the best available data and relates specifically to alcohol-related disease and injury. This narrow focus is necessary because of the nature of the available evidence. However, it does not take into account the consequences of drinking on others,

particularly violence and anti-social behaviour. While these consequences are of as much concern to the community as the health effects of drinking, there is little evidence available on them.

The advice in the guidelines cannot be ascribed levels of evidence ratings as occurs with other NHMRC guidelines, due to the analytic approach taken in their development. However, the nature of the evidence forming the basis of each guideline is described in detail, as is the methodology for the modelling.

The guidelines identify limitations in our understanding and controversy in the evidence. The scientific evidence will continue to develop and change and the guidelines will need to be regularly reviewed and updated as significant new evidence emerges.

## Structure of the guidelines

This document is organised into the following sections:

- **Summary** – presents key information from the guidelines, for easy reference
- **Background** – provides contextual information about the guidelines and includes an overview of the health effects of alcohol and drinking patterns in Australia
- **Key concepts underpinning the guidelines** – discusses the basic principles behind the guidelines, including a discussion of risk, lifetime risk and patterns of drinking
- **Guidelines 1 to 4** – presents each drinking guideline with a rationale and explanation of the underpinning scientific evidence
- **Appendices** – contain additional information about the working committee, the guideline development process, the methodology underpinning the risk analyses and further issues for consideration.



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## Key terms used in these guidelines

For the purposes of these guidelines, the following definitions are used:

**Risk** – a person's risk of experiencing an adverse health outcome is the probability of the person developing that outcome in a specified time period

**Lifetime risk** – the accumulated risk from drinking either on many drinking occasions, or on a regular (eg daily) basis over a lifetime. Lifetime risk of death is a common outcome used for measuring risk from exposures to hazardous substances

**Relative risk** – the risk of harm in drinkers relative to the risk of harm in non-drinkers. Note that the relative risk on its own does not give any information about the absolute risk of harm

**Absolute risk** – the actual risk of injury or disease from drinking

**Harm** – adverse health outcomes; in this context harm includes disease and/or injury resulting from consumption of alcohol

**Standard drink** – the Australian standard drink contains 10g of alcohol (equivalent to 12.5 mL of pure alcohol)

**Drinking occasion/single occasion** – a sequence of drinks taken without the blood alcohol concentration reaching zero in between. This might include a drink at home over dinner, or at a specific event, such as a party, and can include drinking spread across more than one context or venue

**Regular drinking** – repeated drinking occasions over a period of time – eg drinking daily, or every weekend, over many years

**Harmful drinking** – drinking at levels that are likely to cause significant injury or ill health

**Immediate effects** – the effects of drinking either during or after an occasion of drinking, lasting until the blood alcohol concentration returns to zero

**Cumulative effects** – the effects of many drinking occasions over time.

A number of well-known terms are difficult to define or pejorative and are avoided wherever possible in these guidelines. In particular, levels of drinking are defined in many different ways and are often difficult to quantify. However, as many of these terms are used in the literature, they may be mentioned in the discussion of the evidence. These include 'binge-drinking', 'risky drinking', 'heavy drinking' and 'problem drinking'.

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## Background

Most Australians have tried alcohol at some time in their lives, and many drink at levels that have few adverse effects. However, any level of drinking increases the risk of ill-health and injury. Alcohol is responsible for a considerable burden of death, disease and injury in Australia. Drinking is a major factor in much of the injury resulting from road crashes and other accidents, and in social problems such as violence, family breakdown and child abuse and neglect. As such, alcohol-related harm is not restricted to individual drinkers but has relevance for families, bystanders and the broader community.

### How much do Australians drink?

The *2007 National Drug Strategy Household Survey* (AIHW 2008) indicated that the majority of Australian adults have tried alcohol, and many continue to drink throughout life:

- around 90 per cent have tried alcohol in their lifetime
- over 83 per cent have consumed an alcoholic drink in the past 12 months.

This means that at one end of the range:

- about 10 per cent of Australian adults have never had a full serve of alcohol and about 17 per cent have not consumed alcohol in the past year.

While at the other end:

- around 8 per cent drink daily
- around 41 per cent drink weekly.

Both in terms of hours and places of sale, and in terms of price relative to income, alcohol has become much more readily available over the past two decades in most economically developed countries, including Australia (Loxley et al 2004). This includes the greater availability of alcohol through new outlets such as supermarkets and via extended trading hours.

The mean volume of alcohol consumed has remained relatively stable since 1991, but there have been important changes in the patterns of consumption. Preferences in beverage type have shifted towards spirits and pre-mixed drinks, especially among younger drinkers (White & Hayman 2004), and there is an increased level of informality in drinking styles, such as drinking directly from the container.

## Reasons for drinking

People use alcohol for a wide range of reasons and in different social and cultural contexts. They may drink for sociability, cultural participation, religious observance or as a result of peer influence. They may also drink for pleasure, relaxation, mood alteration, enhanced creativity, intoxication, addiction, boredom, habit, to overcome inhibitions, to escape or forget or to 'drown sorrows'. These reasons are likely to be closely related to age, culture and socioeconomic grouping.

Harmful alcohol use affects a wide range of people, regardless of race, cultural background, education, religion, gender or age. Reasons for drinking at harmful levels are varied and may be complex.

## Risk of alcohol-related harm

In the NHMRC 2001 guidelines, the risk of accidents and injuries occurring immediately after drinking was labelled as 'short-term risk', while the risk of developing alcohol-related diseases from regular drinking over a lifetime was labelled as 'long-term risk'. From 2001, data and information collection systems about alcohol consumption in Australia changed to reflect this approach. Therefore, in this section, drinking levels are referred to in terms of the NHMRC 2001 guideline levels, which state that more than six standard drinks in any one day for men or more than four for women increases the risk of accident or injury (short-term harm); and more than four standard drinks per day/twenty-eight per week for men, or more than two per day/fourteen per week for women increases alcohol-related disease risks (long-term harm).

The *2007 National Drug Strategy Household Survey* (AIHW 2008) found that:

- almost half (48 per cent) of Australians reported drinking alcohol at or below NHMRC 2001 guideline levels for short-term harm; however, about one-third (35 per cent) of people drank above NHMRC 2001 guideline levels on at least one occasion in the 12 months before the survey
- most Australians (72 per cent) reported drinking at or below NHMRC 2001 guideline levels for long-term harm; however, about 10 per cent of people reported drinking above NHMRC 2001 guideline levels.

### ***Children and young people***

The 2002 national survey on the use of alcohol by Australian secondary school students (White & Hayman 2004) found that experience with alcohol was high among secondary school students. Alcohol consumption became more common as age increased:

- by the age of 14, around 90 per cent of students had tried alcohol
- at the age of 17, around 70 per cent of students had consumed alcohol in the month before the survey
- the proportion of students drinking in the week before the survey increased with age from 19 per cent of 12-year-olds to a peak of 50 per cent among 17-year-olds.

Rates of drinking above NHMRC 2001 guideline levels among 14–19 year-olds are similar to the rates for the general population – about 9 per cent drink above guideline levels for long-term harm and 39 per cent drink above guideline levels for short-term harm (AIHW 2008). Among people in the 20–29 year age group, about 60 per cent drink above NHMRC 2001 guideline levels for short-term harm and about 16 per cent drink above guideline levels for long-term harm (AIHW 2008).

Among school students aged 16–17 years who report drinking in the past week, there has been a slight increase in numbers drinking above NHMRC 2001 guideline levels for accidents and injuries (White & Hayman 2004). This may be because of changes in the type of alcohol young people are drinking. The 2002 survey found that among male adolescent drinkers, the proportion consuming beer decreased while consumption of spirits, in either their un-pre-mixed or their pre-mixed form, increased. Among adolescent female drinkers, the proportion drinking pre-mixed spirits as opposed to un-pre-mixed spirits increased significantly (White & Hayman 2004).

### ***Older people***

Although older people tend to consume less alcohol during any one session than younger people, they are more likely to drink every day. About 11 per cent of people aged 60 years or more drink above NHMRC 2001 guideline levels for short-term harm; and about 6 per cent drink above NHMRC 2001 guideline levels for long-term harm (AIHW 2008).

### ***Pregnant women***

Recent data show that 59 per cent of Australian women drank alcohol at some time during their pregnancy (Colvin et al 2007). Furthermore, 14 per cent reported drinking five or more drinks on an occasion in the three months prior to pregnancy (Colvin et al 2007). However, many women choose to

abstain from alcohol some time during pregnancy — 58 per cent during the first and second trimester and 54 per cent in the third trimester (Colvin et al 2007). In the first trimester, 15 per cent of women surveyed drank above the NHMRC 2001 guidelines and this proportion decreased to 10 per cent in the second and third trimesters (Colvin et al 2007). In a recent national survey, “34 per cent of women had drunk alcohol during their last pregnancy and 24 per cent indicated they would drink in a future pregnancy, despite knowledge of the adverse effects of alcohol and the fact that 78 per cent believed that reducing or ceasing alcohol intake in pregnancy may benefit their baby” (Peardon et al 2007).

### **Population groups**

The *2004 National Drug Strategy Household Survey* provided information on levels of drinking within certain population groups (AIHW 2005):

- people whose main language spoken at home was English were more likely to drink alcohol than those whose main language spoken at home was not English
- people living in regional or remote areas were more likely to drink at higher levels compared with people in major cities and towns
- Aboriginal and Torres Strait Islander peoples were less likely to drink alcohol than other Australians but more likely than other Australians to drink at levels above the NHMRC 2001 guidelines if they did drink:<sup>2</sup>
  - approximately 50 per cent of Aboriginal and Torres Strait Islander people drink above NHMRC 2001 guideline levels for short-term harm (compared with about 34 per cent of other Australians)
  - approximately 20 per cent of Aboriginal and Torres Strait Islander people drink above NHMRC 2001 guideline levels for long-term harm (compared with about 10 per cent of other Australians).

### **Workforce**

Recent research that examined alcohol consumption patterns among the Australian workforce indicated 44 per cent drank above NHMRC 2001 guideline levels at least occasionally (Berry et al 2007). This level of consumption was more prevalent among particular occupational groups. Young workers, workers in blue-collar occupations, and workers employed in the hospitality, agriculture, manufacturing, construction, and retail industries

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<sup>2</sup> Aboriginal and Torres Strait Islander people provide a compelling example of the link between socioeconomic status and health, with evidence that social factors are responsible for the adverse health and social impacts of drug use, particularly alcohol, among Indigenous Australians (Loxley et al 2004). These issues are discussed in detail in the Department of Health and Ageing report *Alcohol Treatment Guidelines for Indigenous Australians* (available at [www.alcohol.gov.au](http://www.alcohol.gov.au)).

were identified as groups more likely to drink at harmful levels (Pidd et al 2006a; 2006b; Berry et al 2007).

In addition, the burden of harm associated with the alcohol use of Australian workers is not restricted to those traditionally defined as ‘heavy’ or ‘problem’ drinkers. It has been estimated that in 2001, nearly 2,700,000 work days were lost due to workers’ alcohol use, at a cost of \$437m (Pidd et al 2006a; 2006b). Workers who drank at ‘risky’ and ‘high risk’ levels infrequently or occasionally accounted for more than half this alcohol-related absenteeism (Pidd et al 2006a; 2006b; Roche et al 2008).

### KEY POINTS

- Surveys show that most Australian adults drink alcohol; about half do so at levels that put them at higher risk of alcohol-related harm in the short-term, and about one-quarter at levels that put them at higher risk of alcohol-related harm in the long-term.
- At all ages, greater proportions of the population drink above levels that increase their risk of short-term harm than at levels that increase their risk of alcohol-related harm in the longer term.

## How much do Australians think they can drink without harm?

The *2004 National Drug Strategy Household Survey* (AIHW 2005) explored awareness of health risks related to alcohol consumption among Australian men and women. The survey found that:

- 44 per cent of men and 65 per cent of women had the perception that they could drink one to two standard drinks every day for many years without adversely affecting their health
- perceptions regarding short-term risk were closely linked to NHMRC 2001 guidelines levels, with 30.6 per cent of men perceiving that they could drink five to six standard drinks on a single occasion before putting their health at risk and 40.1 per cent of women perceiving they could drink three to four standard drinks on a single occasion before putting their health at risk
- perceptions concerning levels of consumption in the longer term varied significantly between men and women, for example, 35.6 per cent of men perceived that they could consume three to four standard drinks per day before putting their long-term health at risk, while only 13 per cent of women had this perception.

Figures 1 and 2 show perceptions of drinking levels for short-term and long-term risk, respectively.

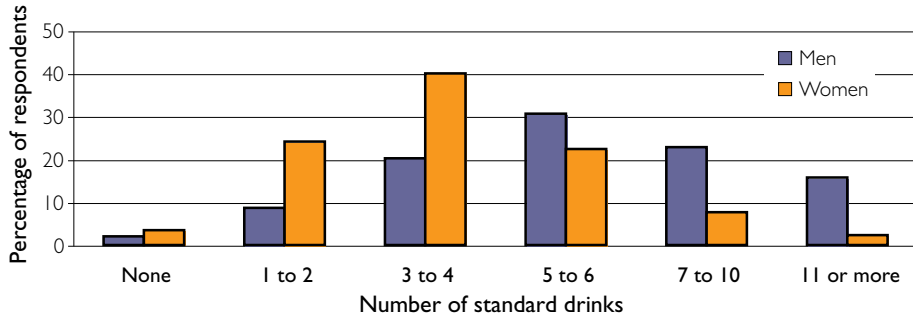


Figure 1 Perception of the number of standard drinks an adult could drink on a single occasion before increasing the risk of short-term alcohol-related harm

Source: AIHW 2005

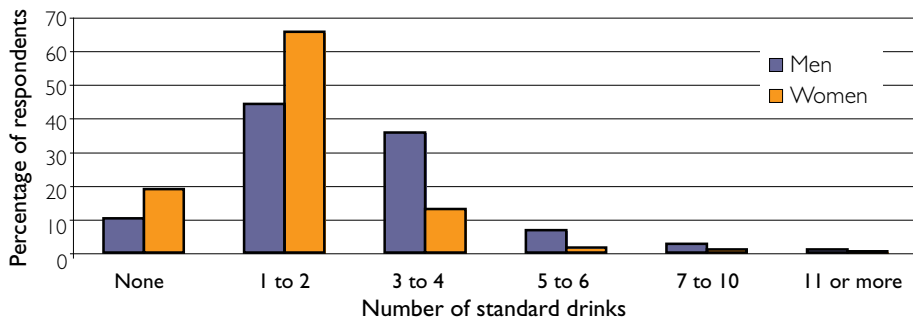


Figure 2 Perception of the number of standard drinks an adult could drink per day before increasing the risk of long-term alcohol-related harm

Source: AIHW 2005.

## How do Australian guidelines compare with international recommendations?

Australian guidelines on drinking will be viewed in the context of similar overseas guidelines. Figure 3 shows recommended levels of alcohol consumption in Organisation for Economic Cooperation and Development (OECD) countries where standard drinks are defined in grams of ethanol (which have been converted here to Australian standard drinks). These are based on a variety of types of guidance from the different countries, some of which are currently under review. Some of the recommendations provide a range rather than a single limit and some provide weekly rather than daily maximums. The Figure shows the diversity of guidelines in use, but also that the universal guideline for adults (see Guideline 1) falls within the range of recommendations elsewhere.

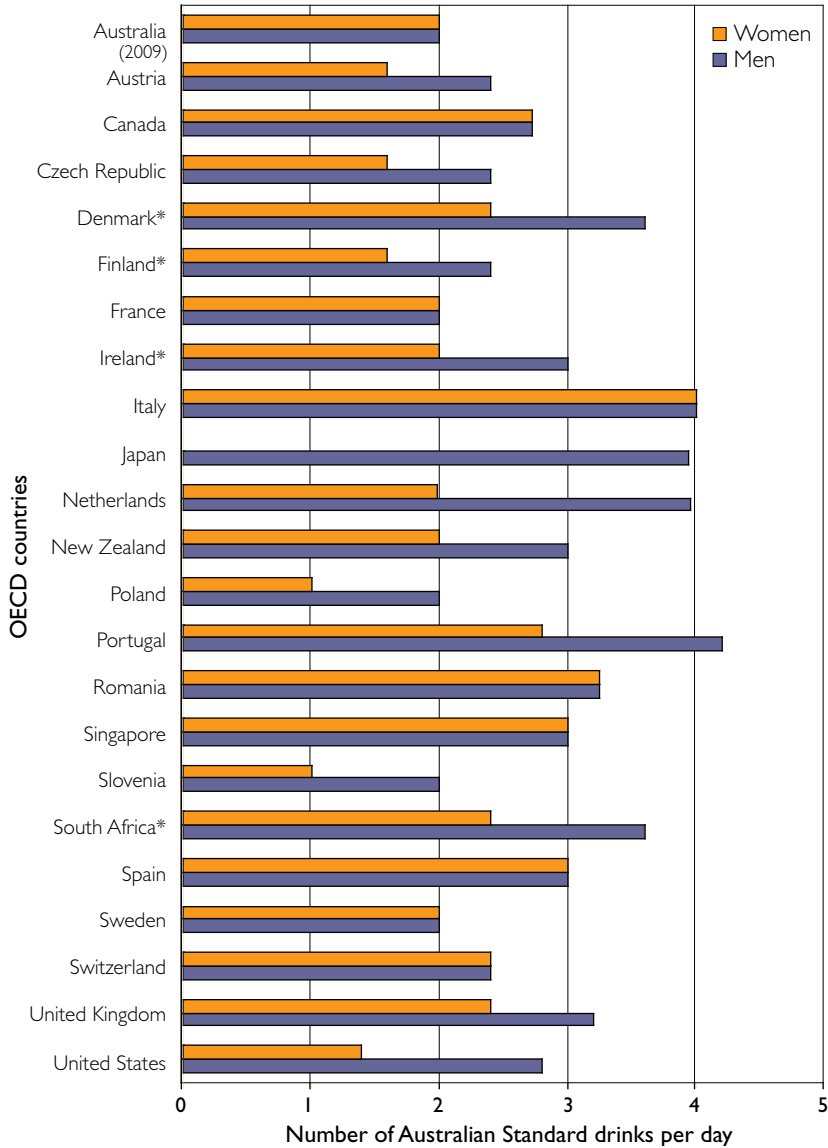


Figure 3 Recommendations on level of alcohol consumption in OECD countries

Notes: \* Converted from weekly recommendation.

Where a range is given, the maximum has been included in this Figure.

Source: International Center for Alcohol Policy (ICAP) 2007.



While these figures give an indication of the diversity of guidelines in use, they should not be taken out of context. Many of the guidelines set a maximum weekly amount of alcohol (which precludes drinking the maximum level each day) and some guidelines recommend alcohol-free days. Also, as with these Australian guidelines, many specifically exclude certain groups from drinking the full amount shown above – for example, pregnant women or people who are on medication. A summary of policies on alcohol use in pregnancy in Australia and other English-speaking countries, published in 2007, highlights the lack of consensus about the relationship between levels of alcohol consumption in pregnancy and harm to the fetus and the difficulty in developing evidence-based guidelines (O’Leary et al 2007).

## Effects of alcohol at the individual level

Consumption of alcohol has both immediate and cumulative effects. Alcohol-related harm in individuals arises not only from the quantity of alcohol consumed but also from a complex interaction between the age and experience of drinkers, their social environment, their genetics and general health.

### Individual variability

There is significant variability in biological responses to alcohol, determined by factors such as sex, body size and composition, age, experience of drinking, genetics, nutrition and individual metabolism. There are also social determinants of variability, with clustering of risk-taking behaviours (eg smoking and harmful drinking) in some people and differences in the risk of harm depending on setting (eg there is a greater risk of harm if the drinker has to travel after drinking).

Due to individual variability, **there is no amount of alcohol that can be said to be safe for everyone.** People’s perception of how much alcohol they can ‘handle’ can lead them to believe that they are able to drink more without harm. There is always some risk to the drinker’s health and social well-being, although there are ways to minimise the risks.

Factors that affect susceptibility to alcohol include the following:

- *Sex* – the same amount of alcohol leads to a higher blood alcohol concentration (BAC) in women than in men, as women tend to have a smaller body size, a lower proportion of lean tissue and smaller livers than men. On the other hand, the higher level of risk-taking behaviour among men means that, over a lifetime, male risks exceed female risks for a given pattern of drinking (see Guideline 1)

- *Age* – in general, younger people are less tolerant to alcohol, and have less experience of drinking and its effects. In addition, puberty is often accompanied by risk-taking behaviours (see Guideline 3). Later in life, as people age, their tolerance for alcohol decreases and the risk of falls, driving accidents and adverse interactions with medications increases (see Section B of Appendix A1)
- *Mental health* – people who have, or are prone to, mental health conditions (eg anxiety and depression, schizophrenia) may have worse symptoms after drinking. Alcohol can also trigger a variety of mental health conditions in people who are already prone to these conditions (see Section C of Appendix A1)
- *Other health conditions that are made worse by alcohol* – people who already have health conditions caused or exacerbated by alcohol, such as epilepsy, alcohol dependence, cirrhosis of the liver, alcoholic hepatitis or pancreatitis, are at risk of the condition becoming worse if they drink alcohol (see Section C of Appendix A1)
- *Medication and drug use* – alcohol can interact with a wide range of prescribed and over-the-counter medications, herbal preparations and illicit drugs. This can alter the effect of either the alcohol or the medication and has the potential to cause serious harm to both the drinker and others (see Section C of Appendix A1)
- *Family history of alcohol dependence* – people who have a family history of alcohol dependence (particularly among first-degree relatives) have an increased risk of developing dependence themselves (see Section B of Appendix A1).

## Metabolism of alcohol

Alcohol usually starts to affect the brain within about five minutes of being swallowed. The BAC reaches its peak about 30–45 minutes after the consumption of one standard drink (10g alcohol). Rapid consumption of multiple drinks results in a higher BAC because the liver has a relatively fixed rate of metabolism regardless of how many drinks are consumed.

It generally takes about an hour for the body to clear one standard drink, although this varies from person to person. The rate of this metabolism depends on several factors including liver size, body mass and composition, and alcohol tolerance (Edenberg 2007). Differences in the speed of alcohol metabolism between people are also related to individual variation in the genes that control expression of alcohol-metabolising enzymes in the liver (Whitefield & Martin 1994; Li et al 1998; Edenberg 2007).

Eating when drinking alcohol slows the increase in BAC as food in the stomach reduces the speed at which alcohol is absorbed into the bloodstream. However, activities such as drinking coffee, having a cold shower, vomiting or exercise do not reduce BAC. After a very heavy drinking occasion, it takes many hours for the BAC to return to zero.

## Immediate effects

The most obvious and immediate effects of alcohol are on the brain, beginning with feelings of relaxation, wellbeing and loss of inhibitions. However, as the intake of alcohol increases, these effects are counterbalanced by less pleasant effects, such as drowsiness, loss of balance, nausea and vomiting, as well as the other harmful effects described below.

The range of normal pathophysiological reactions to alcohol begin with dampening of the brain's arousal, motor and sensory centres, which reduces reactions to stimuli and affects coordination, speech, cognition and the senses. Alcohol can also affect the pituitary gland, suppressing the production of anti-diuretic hormone. This causes the kidneys to fail to reabsorb an adequate amount of water and results in dehydration.

There is evidence that drinking decreases cognitive performance, even at low levels of consumption. The first potentially adverse effect of alcohol consumption is loss of fine motor skills and inhibitions. A BAC of about 0.05g/100mL (or 0.05 per cent) is the legal limit for driving in Australia, which was based on controlled studies testing driving skills (Transport and Road Research Laboratory 1987). As more alcohol is consumed and the BAC rises, performance and behaviour deteriorate progressively.

If the BAC reaches a high enough level, it can lead to life-threatening events such as unconsciousness and, eventually, inhibition of normal breathing. This may be fatal, particularly as the person may vomit and can suffocate if the vomit is inhaled.

As well as effects on the body, the amount of alcohol consumed on a single occasion increases the risk of accidents and injury during and immediately after drinking. Every additional drink significantly increases the risk of injury and death for the drinker and may place others at risk of harm as well. Adolescents and younger adults are particularly vulnerable and drinking in this group can form part of a pattern of risk-taking behaviour (Chrikritzhs et al 2003; Loxley et al 2004). The evidence in this area is discussed in Section B of Appendix A1.

Alcohol consumption also increases the likelihood and extent of aggressive behaviours and reduces the cognitive or verbal capacity to resolve conflicts,

thereby increasing the likelihood of physical violence (eg fights and assaults) (Hingson et al 2001; Giancola 2002; Jewkes et al 2002; Moraes & Reichenheim 2002; Howard et al 2003; Borges et al 2004ab; Cherpitel et al 2004a; Ogle & Miller 2004; Haggard-Grann et al 2006; Mousavi & Eshagian 2005; Borges et al 2006).

## Cumulative effects

Alcohol consumption has been associated with a range of diseases that may cause death and adverse effects that reduce quality of life.

A detailed discussion of the adverse long-term effects of alcohol on health is beyond the scope of these guidelines. Briefly, they can be summarised as follows:

- *Cardiovascular disease* – The effect of alcohol on the cardiovascular system is complex. Alcohol can raise blood pressure and increase the risk of arrhythmias, shortness of breath, some types of cardiac failure, haemorrhagic stroke and other circulatory problems. Low levels of alcohol raise high-density lipoprotein cholesterol and reduce plaque accumulations in arteries. Alcohol can also have a mild anti-coagulating effect
- *Cancers* – Alcohol is increasingly associated with a raised risk of cancer: a recent report by the International Agency for Cancer Research (Baan et al 2007) found convincing evidence that alcohol is carcinogenic to humans, being causally related to cancers of the oral cavity, pharynx, larynx, oesophagus, liver, colorectum and female breast
- *Diabetes* – The relationship between alcohol consumption, insulin sensitivity, type 2 diabetes mellitus and the metabolic syndrome that clinically precedes it, is not clear (Hulthe & Fagerberg 2005). However, alcohol affects the management of diabetes in a number of ways (see Section C of Appendix A1)
- *Nutrition-related conditions* – alcohol consumption is linked to malnutrition, Wernicke-Korsakoff syndrome, folate deficiency, Vitamin A depletion and pellagra (NHMRC 2001)
- *Overweight and obesity* – although the epidemiological data on the relationship between alcohol intake and being overweight or obese are not clear, alcohol adds kilojoules to the normal diet and may increase energy intake and fat storage further by increasing appetite and displacing fat and carbohydrate oxidation (NHMRC 2003a). The absolute amount of alcohol consumed, drinking frequency and genetic factors all influence an individual's tendency to gain weight (Suter 2005)

- *Risks to unborn babies* – alcohol enters the bloodstream of the fetus when the mother drinks and can cause a range of birth defects and growth and developmental problems, comprising Fetal Alcohol Spectrum Disorder, which may persist into adulthood. Alcohol also enters the breast milk. The evidence in this area is discussed in more detail under Guideline 4
- *Liver diseases* – alcohol consumption is the most common cause of cirrhosis of the liver, and drinking alcohol over many years can cause cirrhosis in the absence of other causes (NHMRC 2001). The presence of conditions such as hepatitis B or C increases the effects of alcohol in contributing to development and course of cirrhosis
- *Mental health conditions* – there is growing evidence that alcohol increases the risk of highly prevalent mental health conditions such as depression and anxiety in some people, and may affect the efficacy of antidepressant medication (Loxley et al 2004):
  - alcohol dependence increases the risk of having major depression one year later, and equally, the presence of major depression elevates the risk of having an alcohol dependence disorder one year later
  - the lifetime prevalence of major depressive disorders in people seeking treatment for alcohol dependence is around 40 per cent
  - the co-occurrence of major depression and alcohol-use disorders increases the risks of both violence and suicidal behaviour
- *Tolerance* – the immediate effects of alcohol on the brain are often less apparent in people who drink regularly, as they acquire a degree of tolerance. Tolerance occurs in part because the liver becomes more efficient at breaking down alcohol. The person learns to cope with, and compensate for, the deficits induced by alcohol. Despite this tolerance, the long-term effects remain damaging, particularly as the drinkers who have greater tolerance for alcohol are likely to be those who experience higher blood alcohol levels more frequently
- *Dependence* – alcohol is an addictive drug and regular use can result in alcohol dependence. Alcohol dependence is a complex phenomenon. In brief, it refers to situations where a person feels a strong need to drink so that 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 at high-risk 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 tremulousness and anxiety. Alcohol is strongly linked with anxiety and depression in those with alcohol dependence, and this increases the risks of violence and self-harm. Alcohol dependence is a major risk factor for suicide

- *Long-term cognitive impairment* – the relationship between chronic harmful alcohol consumption and cognitive impairment is well established (Friend et al 2005; Liappas et al 2005; Rosenbloom et al 2005; Glass et al 2006). Drinkers who consume alcohol at harmful levels exhibit negative structural and metabolic brain changes, and have an increased risk of dementia (Gilchrist et al 2005; Gazdzinski et al 2005)
- *Self-harm* – Harmful drinking is a major risk factor for suicide and suicidal behaviour in both males and females across the lifespan (Cherpitel et al 2004b; Kaminer et al 2006; Kolves et al 2006; Sher 2006).

### **Potential health benefits of drinking**

At low levels of consumption, alcohol has some health benefits in certain age groups – many studies, including meta-analyses (Holman et al 1996; Corrao et al 2000; Corrao et al 2004; Di Castelnuovo et al 2006) have suggested that drinking reduces the risk of some cardiovascular and cerebrovascular disorders; specific studies have found reductions in cardiovascular disease (particularly in middle-aged and older males) and ischaemic stroke (in women after menopause) (eg English et al 1995; Holman et al 1996). The large Kaiser Permanente Study (Klatsky & Udaltsova 2007) found a clear protective association for cardiovascular disease. The extent of these risk reductions is uncertain.

Other studies have found differing results:

- some recent studies have found no significant cardioprotective or all-cause associations (Fillmore et al 2006; Fillmore et al 2007)
- a systematic review (Fuchs & Chambless 2007) and other recent studies (Baglietto et al 2006; Stockwell et al 2007; Friesema et al 2008) have suggested that the cardioprotective or all-cause effect may have been overestimated.

The body of evidence suggests that most of the potential cardiovascular benefit of alcohol may be achieved by drinking within the levels recommended in Guideline 1. For example, it has been reported that the benefits can be achieved with an intake of half a standard drink per day (Bagnardi et al 2004; WHO 2007; Lewis et al 2008).

It should also be noted that the potential cardiovascular benefits from alcohol can also be gained from other means, such as exercise or modifying the diet.

## Burden of alcohol-related disease and injury

Worldwide, alcohol caused 3.7 per cent of all deaths (2.1 million) and 4.4 per cent of the total burden of disease<sup>3</sup> in 2001 (WHO 2007). In Australia:

- Alcohol is second only to tobacco as a preventable cause of drug-related death and hospitalisation (English et al 1995; Mathers et al 1999; Higgins et al 2000; Ridolfo & Stevenson 2001):
  - between 1992 and 2001, more than 31,000 deaths were attributed to risky or high-risk alcohol consumption, defined by the NHMRC 2001 Guidelines<sup>4</sup> (Chikritzhs et al 2003)
  - in the eight years between 1993–94 and 2000–01, over half a million completed hospital episodes were associated with alcohol (Chikritzhs et al 2003)
- While the number of emergency department presentations caused by alcohol is unknown, it is likely to account for a large proportion of all presentations (McLeod et al 2000; Poynton et al 2005; Watt et al 2004; 2006)
- Alcohol accounts for 13 per cent of all deaths among 14–17-year-old Australians – it has been estimated that one Australian teenager dies and more than 60 are hospitalised each week from alcohol-related causes (Chikritzhs et al 2004)
- Alcohol is also a significant contributor to premature death and hospitalisation among older Australians – among 65–74-year-olds, almost 600 die every year from injury and disease caused by drinking above the NHMRC 2001 guideline levels, and a further 6,500 are hospitalised (Chikritzhs & Pascal 2005)
- Although most surveys show that Aboriginal and Torres Strait Islander people are less likely than the general population to drink, alcohol-attributable injury and disease are particularly high among this group (DHS 1994). The rate of alcohol-attributable death among Indigenous Australians is about twice that for the non-Indigenous population (Chikritzhs et al 2007), with a particularly strong association apparent between alcohol use and suicide among some Aboriginal and Torres Strait Islander people (Hunter et al 1999; Tatz 1999).

<sup>3</sup> 'Burden of disease' is the cumulative effect of a broad range of harmful health consequences on a community.

<sup>4</sup> That is, more than six standard drinks in any one day for men or more than four for women; or more than twenty-eight standard drinks per week for men, or more than fourteen per week for women.

### ***Alcohol-related disease and injury***

Alcohol consumption accounted for 3.3 per cent of the total burden of disease and injury in Australia in 2003; 4.9 per cent in males and 1.6 per cent in females (Begg et al 2007). This compared with a contribution of 7.8 per cent for tobacco smoking, 7.5 per cent for high body mass, 7.6 per cent for hypertension and 6.6 per cent for physical inactivity (Begg et al 2007). However, the dataset used to estimate alcohol consumption may underestimate its contribution to the true burden of disease and injury. This is borne out by the higher published figures for New Zealand (10 per cent for men and 4 per cent for women) (Connor et al 2005).

Drinking alcohol has been associated with injuries in many settings, including motor vehicle and bicycle accidents, incidents involving pedestrians, falls, fires, drowning, sports and recreational injuries, alcohol poisoning, overdose, suffocation, inhalation of vomit, assault, violence, and intentional self-harm (Chikritzhs et al 2000; Ridolfo & Stevenson 2001; Chikritzhs et al 2003).

- For Australian men, about one-third (33 per cent) of motor vehicle deaths and one-quarter (25 per cent) of motor vehicle injuries have been attributed to alcohol consumption; for women the figures are 11 per cent in each case (Ridolfo & Stevenson 2001). For pedestrians, alcohol accounted for 40 per cent of male and 17 per cent of female deaths; and 37 per cent of male and 6 per cent of female hospitalisations (Ridolfo & Stevenson 2001).
- Alcohol use is significantly associated with episodes of deliberate self-harm (McCloskey & Berman 2003), with about one-third of all self-inflicted injuries and suicides linked to alcohol consumption in men (32 per cent) and women (29 per cent) (Ridolfo & Stevenson 2001). Longitudinal data show that the prevalence of alcohol use around the time of a deliberate self-harm episode has increased for both males and females over the past two decades (Haw et al 2005; O'Loughlin & Sherwood 2005).



## KEY POINTS

- The main causes of alcohol-related deaths are road trauma, cancer, and alcoholic liver cirrhosis (Loxley et al 2004).
- Among people aged 15 to 34 years, alcohol is responsible for the majority of drug-related deaths and hospital episodes, causing more deaths and hospitalisations in this age group than all illicit use of drugs, and many more than tobacco.
- Alcohol-related harm during or immediately after drinking is experienced disproportionately by younger people, while cumulative alcohol-related harm is more evident among older people.

## Social and economic consequences

### *Social consequences*

These guidelines have been modelled only on adverse health effects of alcohol. However, the effects of alcohol consumption go beyond diseases, accidents and injuries to a range of adverse social consequences, both for the drinker and for others in the community. These consequences include harm to family members (including children) and to friends and workmates, as well as to bystanders and strangers.

Concerns to the community that are associated with alcohol use include noise, litter, offensive behaviour, vandalism, aggression, petty crime, assault and road safety issues (Loxley et al 2004). Many of these social consequences can result in affront, violence or injury to others.

Alcohol is significantly associated with crime (Loxley et al 2004), with studies suggesting that alcohol is involved in up to half of all violent crimes and a lesser but substantial proportion of other crimes. There appear to be multiple contributory and causal mechanisms, including the characteristics of the drinker, the effects of alcohol, the drinking environment, and the cultural expectations surrounding alcohol and violence. There is also a link between drinking and domestic violence. In men who are already predisposed towards domestic violence, alcohol increases the risk of violence. Alcohol consumption also increases the risk of being a victim of domestic violence.

The most visible and measurable effects of drinking on others, including children, result from accidents and injury during or after drinking occasions. When families have to deal with a relative's alcohol dependence, violence, injury or even death, these serious consequences can cause great suffering.

Among respondents to the *2004 National Drug Strategy Household Survey* (AIHW 2005), 21.9 per cent reported that they had been verbally abused in the past 12 months by a person affected by alcohol, 11.8 per cent said that they had been in a frightening situation, and 3.7 per cent stated that they had been physically abused.

Regular use of alcohol by adults is considered acceptable by more than three-quarters of Australians (AIHW 2008). However, about one-third (33 per cent) of people consider alcohol to be the most serious concern for the general community (AIHW 2005), and about 10 per cent think alcohol is the drug most likely to be associated with a 'drug problem' (AIHW 2008).

Most Australians support more severe penalties for drink driving, stricter laws for serving intoxicated people, and strict monitoring of late-night licensed premises. However, only 24 per cent of people support an increase in the price of alcohol (AIHW 2008).

### ***Economic consequences***

In terms of government responses to alcohol problems, treatment and care in the health system is only part of the story. For example, of total government expenditure on alcohol problems in Scotland in 2002–03, only 23 per cent was estimated to be in health services, with 20 per cent in welfare services and 57 per cent in police and emergency services (Scottish Health Economics Unit 2004).

The costs accrue not only to government health and welfare systems, but also to industry through absenteeism (Roche et al 2008), premature retirement, and impaired or lost productivity (Rehm et al 2007a). It has been estimated, for example, that alcohol cost the Australian community about \$15.3 billion in 2004–05, when factors such as crime and violence, treatment costs, loss of productivity and premature death were taken into account (Collins & Lapsley 2008). These figures are recognised to be conservative, as the cost of alcohol-related absenteeism alone has recently been estimated at \$1.2 billion per year, using self-report data from the 2001 National Drug Strategy Household Survey (Pidd et al 2006a; 2006b).

## Key concepts underpinning the guidelines

A number of concepts underpin these guidelines:

- the Australian Standard drink, which is used to quantify the alcohol consumed and calculate the ensuing risk
- calculations of risk that investigate both immediate and lifetime risk across a range of patterns of drinking
- a population health approach, which aims to provide a range of advice to improve health and wellbeing across the community.

### The Australian standard drink

The notion of a standard drink is used widely internationally, but the definition varies from country to country. These guidelines use the Australian standard drink, which is defined as containing 10g of alcohol (equivalent to 12.5mL of pure alcohol).

Where possible, in discussing the evidence, these guidelines:

- define amounts of alcohol in grams
- define levels of drinking precisely using the Australian standard drink
- include quantitative descriptors where descriptive terms such as ‘heavy’, ‘moderate’ or ‘light’ are used.

A serving of alcohol frequently differs from a ‘standard drink’, often being larger. For example, for table wine, a standard drink corresponds to 100mL of wine, whereas a typical serve may be 150mL.

In Australia, all bottles, cans and casks containing alcoholic beverages are required by law to state on the label the approximate number of standard drinks they contain. Table 1 provides a rough guide.

Table 1 Numbers of Australian standard drinks in common containers of various alcoholic beverages

Alcoholic beverage	Standard drinks
<b>Low strength beer (2.7% alcohol)</b>	
1 can or stubbie	0.8 standard drinks
285mL glass	0.6 standard drinks
425mL glass	0.9 standard drinks
slab of 24x375mL cans or stubbies	19 standard drinks
<b>Mid strength beer light beer (3.5% alcohol)</b>	
1 can or stubbie	1 standard drink
285mL glass	0.8 standard drinks
425mL glass	1.2 standard drinks
slab of 24x375mL cans or stubbies	24 standard drinks
<b>Full strength beer (4.9% alcohol) (includes diet beer)</b>	
1 can or stubbie	1.4 standard drinks
285mL glass	1.1 standard drinks
425mL glass	1.6 standard drinks
slab of 24x375mL cans or stubbies	34 standard drinks
<b>Wine (9.5%–13% alcohol)</b>	
100mL glass	1 standard drink
Average restaurant serving (150mL)	1.4–1.6 standard drinks
750mL bottle	7 to 8 standard drinks
4-litre cask	36 to 43 standard drinks
<b>Spirits (37%–40%)</b>	
1 nip (30mL)	1 standard drink
700mL bottle	22 standard drinks
<b>Pre-mixed spirits (5%–7% alcohol)</b>	
1 can (375mL)	1.5–2.1 standard drinks
1 bottle (275mL)	1.1–1.5 standard drinks

It can be difficult to translate standard drinks into real-life situations, and people commonly underestimate what they drink. There are no common glass sizes used across all public drinking environments, or in private homes. Most glasses hold more than one standard drink. The problem is compounded when large containers (jugs, casks, flagons) are shared, when glasses are topped up by another person, when the composition of mixed drinks is not known (eg cocktails or punch at a party), and when pre-mixed spirit drinks contain variable amounts of alcohol per bottle or can.

Graphics representing the number of standard drinks in a range of alcoholic beverages are given in Appendix A6.

## Concepts of risk

### Definition and calculation of risk

These guidelines use the epidemiological definition of risk, which is similar to but more precise than the everyday use of the word. In epidemiology, a person's risk of experiencing an adverse health outcome is defined as the probability of the person developing that outcome in a specified time period. The specified time period may be short (eg a few hours after drinking) or long (eg after five years or over a lifetime).

As well as focussing on the effects of alcohol during and immediately after drinking, these guidelines introduce the concept of lifetime risk of alcohol-related disease or injury as a result of drinking alcohol regularly over a lifetime.

The guidelines for healthy adults (Guidelines 1 and 2) are based on calculations that estimate:

- the risks of developing alcohol-related diseases as a result of drinking at specific levels on a regular basis over a lifetime, compared with not drinking (Guideline 1)
- the cumulative lifetime risk of death from injury associated with many drinking occasions, compared with not drinking (Guideline 1)
- the immediate increase in the risk of injury associated with drinking a defined amount of alcohol on a single drinking occasion, compared with not drinking (Guideline 2).

Figure 4 illustrates these concepts of risk. The concept of lifetime risk and how this is applied in these guidelines is outlined in the discussion below. The methods used to estimate risks of alcohol-related harm are discussed in detail in Appendix A5.

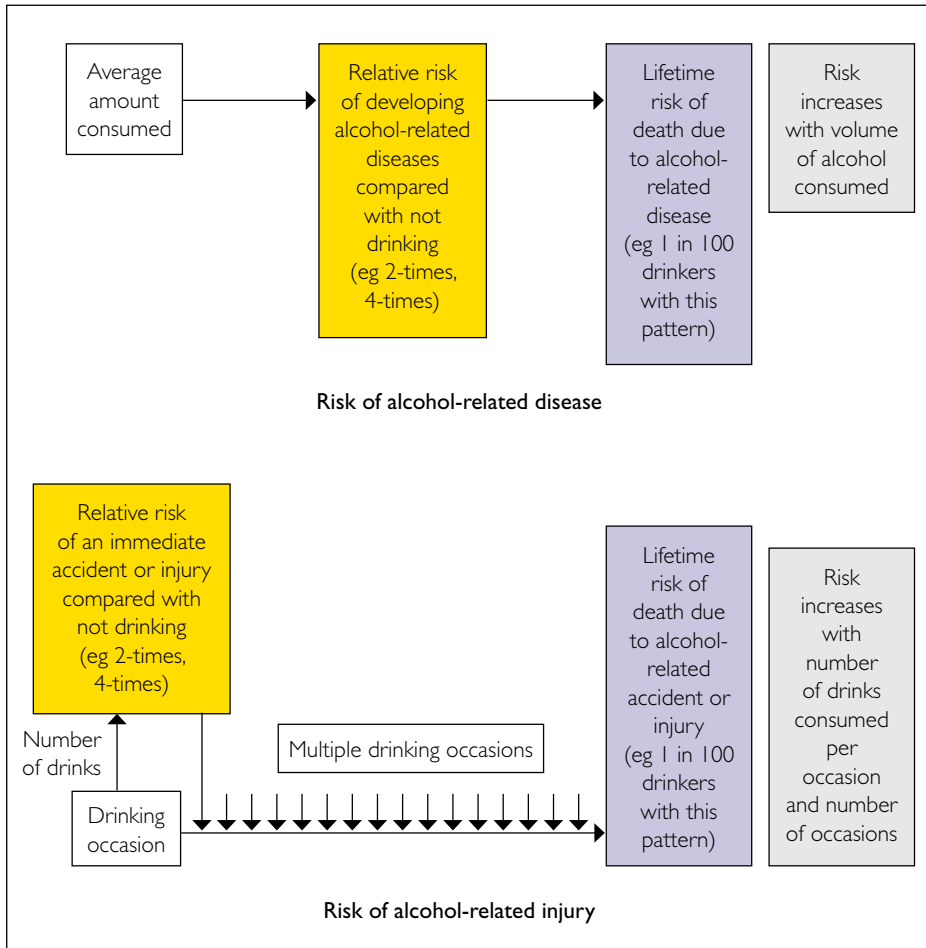


Figure 4 Alcohol-related risks for different patterns of drinking

### Lifetime risk

Lifetime risk is a commonly used standard for evaluating the risk associated with exposure to a particular substance or situation, for instance, in evaluating what are acceptable levels of environmental poisons or food additives. The arbitrary limit often used for environmental toxins has been a risk of death of 1 in 1,000,000: that is, that the chance of death attributable to a given level of exposure over a lifetime should be no more than one in a million. This standard is used in Australia for contaminants of drinking water (NHMRC 2004).

A child drinking tap water is not choosing to take on a risk of poisoning. For such involuntary risks, the threshold of acceptable risk is therefore set

very low. However, for behaviours that are seen as voluntarily adopted, such as driving a car, higher risks are routinely accepted. For example, the lifetime risk of dying in a traffic accident associated with driving 10,000 miles a year in the US has been calculated to be about 1 in 60 (Walsh 1996). From this perspective, at least some of the risks from drinking alcohol can be seen as voluntarily assumed by the drinker. On the other hand, there are harms from drinking that are not voluntarily assumed; in particular, harm to people other than the drinker. Drinking alcohol is thus a mixed case in terms of whether the associated risks are voluntary.

Judgements about the acceptability of risk presuppose that there is some benefit in undertaking the risky activity in question. However, people do not just judge risk against benefits. Characteristics such as control, familiarity, immediacy of the harm, and the catastrophic or chronic nature of the harm or benefit, all influence individual perceptions of what constitutes 'high' and 'low' risk.

The fact that risk is perceived as multi-dimensional, and judged according to its characteristics and context, makes it difficult to convey concepts of risk at a population level. The NHMRC decided on a lifetime risk of dying from alcohol-caused disease or injury of 1 in 100 (ie one death for every 100 people) as the basis for guidance as to what could be seen as an acceptable risk from drinking in the context of present-day Australian society. Guideline 1 in general aims to keep drinking below that risk level for the drinker. This may be seen as too high or too low a risk by the individual drinker. This report also presents tables and figures that show how the risk of harm varies, for those who wish to guide their drinking by another level of risk.

## Patterns of drinking

The lifetime risk is associated with patterns of drinking as well as the number of standard drinks consumed on each occasion of drinking, and is also influenced by factors such as gender, age and body size.

'Patterns of drinking' describes how people drink and the circumstances in which they drink. Patterns of drinking may refer to several aspects of drinking behaviour, including the frequency of drinking occasions, variations in drinking over time and the number and characteristics of 'risky' drinking occasions. It also includes the settings where drinking takes place, the activities associated with drinking, the personal characteristics of the drinkers and their drinking companions, the types of beverage consumed, and the clusters of drinking norms and behaviours often referred to as 'drinking cultures' (Rehm et al 1996).

A drinking occasion refers to a sequence of drinks taken without the blood alcohol concentration reaching zero in between. This might include a drink at home at the end of the day or over dinner, or at a specific event, such as a party, night out, visit to the pub, a family or business event or other function. It may also include drinking spread across more than one context or venue, for instance on a ‘Friday night out’.

Every drinking occasion contributes to the lifetime risk of harm from alcohol. The number of drinking occasions over a lifetime varies widely, depending on the frequency of occasions and the span of years over which alcohol is consumed.

Table 2 shows the relationship between numbers of drinking occasions and some possible lifetime drinking scenarios. For example, drinking once or twice a year for ages 18 to 70 amounts to around 100 drinking occasions in a lifetime, while drinking most days for the same period would amount to around 20,000 drinking occasions.

Table 2 Examples of lifetime drinking patterns

Drinking occasions	Examples of lifetime drinking patterns
100	One or two times a year for ages 18–70 Approximately once a month for ages 18–25
500	Approximately once a month for ages 18–25 then decreasing to 3–4 times a year to age 70
1000	Approximately once a week for ages 18–25 years decreasing to once per month to age 70
5000	Approximately twice a week for ages 18–70 years
10 000	Every other day for ages 18–70 years
20 000	Most days for ages 18–70 years

Figures in Guideline 1 (pages 43 and 45 to 47) clearly show the increase in risk of harm associated with lifetime drinking patterns involving greater numbers of drinking occasions.



## Population health approach

These are population health guidelines and focus on understanding factors affecting health and disease in the community, and on improving health and well-being through priority health approaches. While it is not possible to take account of the full range of individual variation in reactions to alcohol, a range of considerations have been identified to address factors specific to certain population groups:

- *universal guidelines* for healthy Australian adults, which provide advice on levels of drinking to reduce alcohol-related harm both over a lifetime (Guideline 1), and during and immediately after a single drinking occasion (Guideline 2)
- *specific guidelines* for children and young people (Guideline 3), and for pregnant and breastfeeding women (Guideline 4).

Further issues for consideration for specific groups of adults at an increased risk (such as young adults, older people, people with a family history of alcohol dependence or people who use drugs illicitly), for people with physical or mental conditions made worse by alcohol, and for specific situations in which the acute effects of alcohol can endanger the lives of the drinker and others are discussed in Appendix A1.

## Guideline 1: Reducing the risk of alcohol-related harm over a lifetime

### GUIDELINE 1

The lifetime risk of harm from drinking alcohol increases with the amount consumed.

**For healthy men and women, drinking no more than two standard drinks on any day reduces the lifetime risk of harm from alcohol-related disease or injury.**

This guideline applies to healthy men and women aged 18 years or over.<sup>5</sup> The guideline does not represent a 'safe' or 'no-risk' drinking level; nor does it set a prescribed level or absolute upper limit of intake. It identifies a level of drinking at which the risk of alcohol-related harm remains low (compared with not drinking) over a lifetime, in terms of both risk of death from alcohol-related disease and risk of hospitalisation or death from alcohol-related injury.

The lifetime risk of alcohol-related harm is associated with *patterns* of drinking as well as *levels* of drinking and is also influenced by biological and social factors. Further reductions in the lifetime risk of alcohol-related disease or injury can be achieved by reducing the number of occasions of drinking across a lifetime, for example through regular alcohol-free days.

At levels of alcohol consumption recommended here (two standard drinks or less on any day), there is little difference in the risk of alcohol-related harm for men and women over a lifetime. At higher levels of consumption, the risk of alcohol-related disease increases more quickly for women and the risk of alcohol-related injury increases more quickly for men.

### Guideline rationale

This guideline takes a new approach to estimating the risks of harm from drinking and developing population-health guidance about reducing these risks. It focuses on the lifetime health risks from drinking alcohol, and

<sup>5</sup> Special precautions for children and young people under 18 years of age and women who are pregnant or breastfeeding are given in Guidelines 3 and 4 respectively.

considers the volume of drinking over time. The risk of chronic disease from drinking is considered in terms of the cumulative risk over a lifetime. Consideration of the risk of injury takes into account the risk from drinking on a single occasion and how the risk accumulates across multiple drinking occasions.

This guideline is based on a review of the evidence and calculation of risks. The basis and quality of the evidence for lifetime risk of alcohol-related disease and injury are discussed on page 41 and page 43, respectively. The findings are summarised on page 42 (for disease) and on pages 45 to 48 (for injury). The methodology for the literature review is presented in Appendix A3. The process of modelling is outlined in Appendix A5.

The model used to estimate lifetime risk of alcohol-related harm is suited to the population health approach taken in these guidelines. Lifetime risks of both disease and injury were calculated separately, as detailed below. As the model is extrapolated from population event-related data, it does not take into account individual variability (eg body weight) and does not necessarily include all relevant factors (eg evidence on alcohol and colorectal cancer that emerged after the analysis was done).

Further difficulties inherent in establishing advice on reducing risk of alcohol-related harm include the following (Rehm et al 2008):

- the focus of relevant studies is generally on health risks, without consideration of alcohol-attributable social harm
- there is a range of potential harms from alcohol consumption and the risk curve for each is different (Rehm et al 2003; Rehm et al 2004)
- almost all of the risk curves have no apparent threshold and they may also vary with the circumstances of drinking and characteristics of the drinker
- for some specific disorders, protective effects from drinking are likely and these have their own risk curves
- existing guidelines tend to differentiate between gender but not age, although harms from a different level or pattern of drinking may vary more by age than gender.

Despite these limitations, the model provides a liberal estimate and a recalculation would be unlikely to lead to a higher guideline level.

## Alcohol-related disease

### Basis and quality of the evidence

There is a large body of literature, dating back several decades, measuring the risks and benefits of alcohol for a range of chronic diseases and disorders. However, a variety of methods for measuring alcohol were used in these studies, making it difficult to compare results. Until the mid-1990s, there was little systematic work undertaken to put the studies together so as to describe the overall dose-response relationship between alcohol consumption and the risk of disease. In the past 10 years, however, several studies (eg Corrao et al 2004; Ridolfo & Stevenson 2001) have combined the data from the major epidemiological studies worldwide to derive the overall impact of alcohol on developing or dying from alcohol-related diseases and conditions.

The modelled analysis (Rehm et al 2008) included those chronic conditions where accepted epidemiological criteria have shown a causal and detrimental effect of alcohol consumption (Rehm et al 2003; Baan et al 2007). The following process was used to calculate lifetime absolute risk of death from each of these diseases as a result of drinking alcohol:<sup>6</sup>

- calculation of the relative risks of developing each disease, from drinking one standard drink to ten standard drinks per day (in one standard drink intervals compared with people who do not drink alcohol)
- estimation of the alcohol-attributable fraction for diseases by number of standard drinks per day
- calculation of the absolute risk of dying from alcohol-attributable disease categories within one year and derivation of the absolute age-specific one-year risks for all chronic disease, separately for men and women.

At low levels of consumption, alcohol may have health benefits for some age groups (see page 25). As the evidence concerning the potential benefits is uncertain, a decision was made not to incorporate these into the modelling. Where there was no detrimental effect, or a beneficial effect, the relative risk was recorded as 1.0. It is acknowledged that recalculation of the model with estimated benefits included may have slightly altered results.

While it uses the best available information, this approach has some limitations.

- Life expectancy and the absolute risk of mortality from diseases are both based on one year of Australian data (from the WHO Global Burden of Disease project for the year 2002) and may therefore change over time. However, as these parameters do not change much in the short term, this should not bias the outcome.

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<sup>6</sup> Details of the relative risks, alcohol-attributable fractions and calculations are given in Appendix A5.

- The results also assume that the relative risks are the same in Australia as in the countries where the studies included in the meta-analyses have been done (predominantly the United States and the United Kingdom). However, the effects of alcohol on alcohol-related diseases are mainly based on biological mechanisms, and there is no reason to believe that this will be different in different countries that have a similar genetic make-up. The relative risks from the meta-analyses are clearly estimates, though, and the real risks may fluctuate to a certain degree, although applying different relative risk estimates from different authors to the data did not change the results noticeably (Rehm et al 2007b).
- The underlying meta-analysis may be affected by biases concerning the definition of abstainers as different studies have different definitions of this group.

## Summary of the evidence

As the average volume of alcohol consumption increases, the lifetime risk of death from alcohol-related disease increases. For both sexes, the lifetime risk of death from alcohol-related disease more than triples when consumption increases from two to three standard drinks a day. At higher levels of drinking, large differences by gender are seen, with the risk for women being significantly higher than that for men. The risk for women also increases faster with increased consumption than for men. At one standard drink, the lifetime risk for women is lower than for men. At ten standard drinks a day, the lifetime risk for women is almost 1 in 10, twenty-five times the risk at two standard drinks; for men, there is an almost twelve-fold increase and the risk is 6 in 100.

Figure 5 shows the lifetime risks of alcohol-related disease deaths per 100 people who drink at each level (in terms of the number of standard drinks consumed per day).

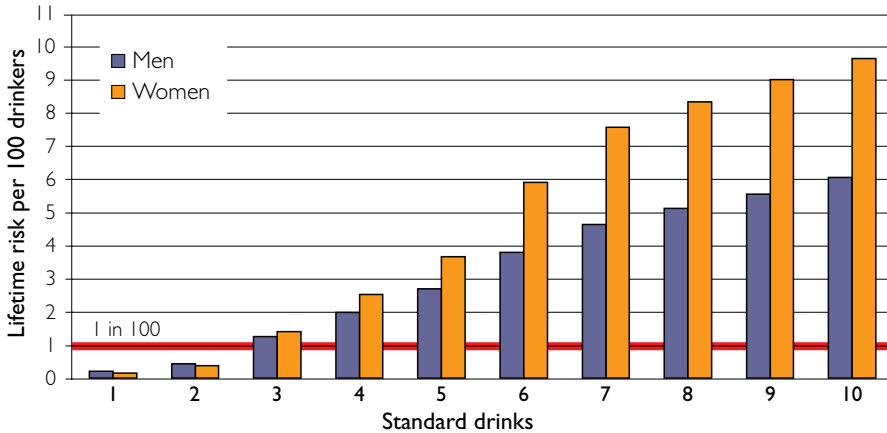


Figure 5 Lifetime risk of death from alcohol-related disease per 100 drinkers, by number of standard drinks per occasion, Australia 2002

Source: Rehm et al (2008)

For people who regularly drink two standard drinks per day, the lifetime risk of death from an alcohol-related disease is about 0.4 in 100 people with that drinking pattern. Above that level, the risk increases with the number of drinks per day, and is above 1 in 100 at three drinks per day. The risk increases more sharply for women than for men.

## Alcohol-related injury

### Basis and quality of the evidence

#### *Lifetime risk of death from alcohol-related injury*

A modelled analysis (Rehm et al 2008) was used to determine the accumulated lifetime risk of death from injury categories for which alcohol has an accepted causal effect (based on established epidemiological data: English et al 1995; Rothman & Greenland 1998). Lifetime risk was calculated for an increasing number of drinks per occasion and for various numbers of drinking occasions over a lifetime. Alcohol-attributable injury deaths per drinking occasion, and lifetime risk, were calculated using four main steps:<sup>7</sup>

- calculation of overall gender- and age-specific risk of fatal injury per day for each category of injury without the impact of alcohol (ie baseline risk)

<sup>7</sup> Details of the modelling are given in Appendix A5.

- calculation of the residual risk of death due to alcohol-related injury that would have occurred in Australia without any involvement of alcohol in the year 2002
- estimation of the increased risk of injury after drinking a specific number of drinks compared to not drinking and of the absolute risk of having a fatal injury after consuming a specific number of drinks, taking into account the fraction of a day for which the risk is increased for that number of drinks
- incorporation into the model of the risk from alcohol consumption on multiple occasions based on the number of drinking occasions, the risk of injury death given the number of lifetime drinking occasions and the risk of injury death for one drinking occasion per year, depending on the number of drinks per occasion.

The estimates are conservative in that the increases in risk are based on studies of non-fatal injuries. The relevant literature indicates that injuries tend to be more severe when alcohol is involved, and thus the relative risks and proportion of alcohol-related injuries are larger for fatal compared with non-fatal injuries (Rehm et al 2004). On the other hand, basing these estimates on emergency department studies may have led to an overestimate of the effects, because people who attend emergency departments with injuries do not represent the general population. They may be characterised as more risk-taking, and thus the risk for alcohol in this population may be higher than in the general population. Unfortunately, data are not available to assess this potential effect.

It is also possible that some of the methodologies employed in studies assessing the risk of injury from different levels of alcohol may overestimate risk, as self-reported levels of the amount drunk are often underestimated (Stockwell et al 2004). Some methodological studies have suggested that:

- case-crossover studies into the association between alcohol and injury may be confounded by recall bias
- social desirability bias may be significant in survey-type alcohol consumption questionnaires (Embree & Whitehead 1993; Stockwell et al 2004)
- legal or other issues may encourage patients to minimise their reported consumption before an injury arising from a vehicle accident (or conversely, to overestimate consumption to excuse socially unacceptable behaviour resulting in violence-relating injuries).

### Risk of hospitalisation for alcohol-related injury

Methods similar to those applied to the injury analysis were used in investigating the risk of hospitalisation for alcohol-related injury. The risk of being hospitalised for injury at different frequencies of drinking specific amounts on an occasion was investigated using data for hospitalised injuries from the Victorian Admitted Episode Database for fiscal year 2001–2002. The database includes only cases admitted to a hospital; that is, it does not include cases treated in an emergency department without admission to the hospital. The data can include more than one ‘admission’ for a single injury, in case of transfers to another hospital; and some hospitalised injuries result in death and will also be counted in the injury deaths. These factors are small; thus the proportion of hospitalisations analysed here for injury that ends in death is 1.1 per cent. Around Australia there will be variations between the rate of injuries and the rate of hospitalisations, for example between urban and remote settings due to access of hospitals.

### Summary of the evidence

For both men and women, the lifetime risk of death or hospitalisation due to alcohol-related injury increases with the frequency of drinking occasions. The model incorporated a range of patterns of drinking, based on the number of drinking occasions (see page 35).

### Risk of death from alcohol-related injury

Figures 6 and 7 show the risk curves for different frequencies of drinking the specified amounts.

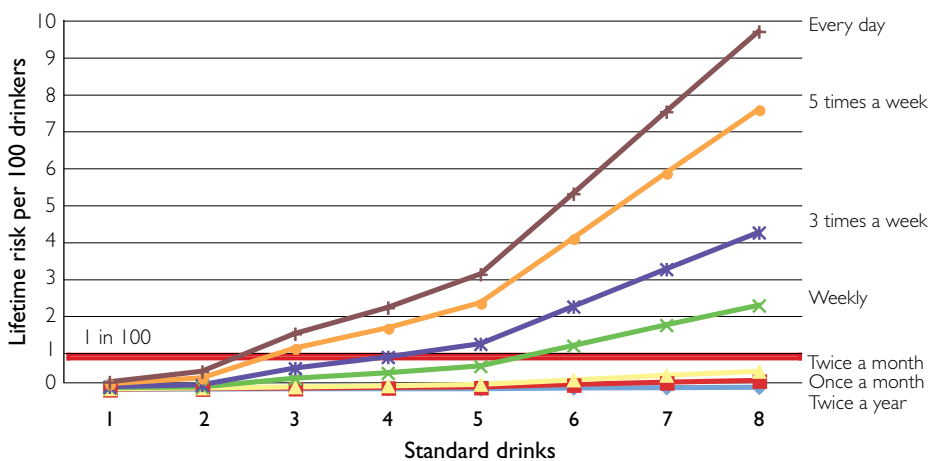


Figure 6 Lifetime risk of death from alcohol-related injury per 100 male drinkers, by number of standard drinks per occasion and frequency of occasions



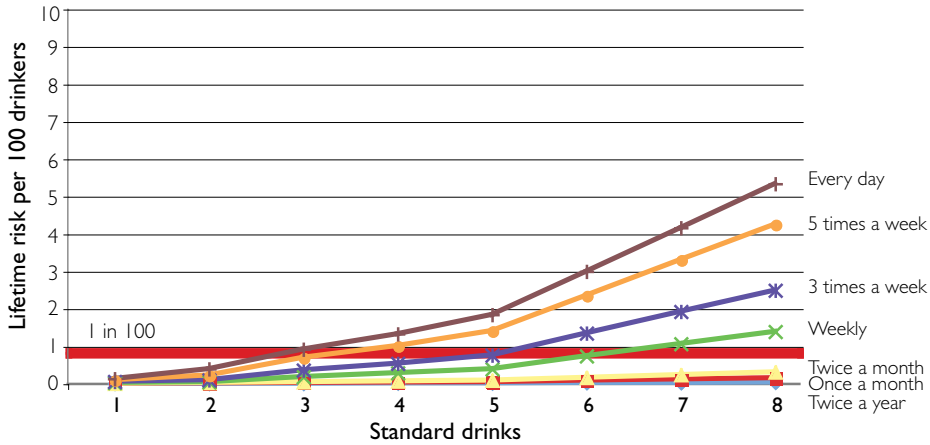


Figure 7 Lifetime risk of death from alcohol-related injury per 100 female drinkers, by number of standard drinks per occasion and frequency of occasions

The figures show that:

- for both men and women, risk of death increases with frequency of drinking
- risks of death for men are higher than those for women at all levels of drinking
- the risk of death from injury remains below 1 in 100 for both men and women if they always drink two drinks or less on an occasion, even if the occasions are every day.

Men are at a higher risk per day than women because injury mortality *per se* is higher among men than among women, based on higher rates of risk behaviour at a given level of drinking by men. However, both men and women show similar patterns of increasing risk of injury mortality as both the lifetime drinking occasions and the number of drinks consumed increase.

### Risk of hospitalisation due to alcohol-related injury

Investigation of the risk of being hospitalised for injury at different frequencies of drinking specific amounts on an occasion showed that the lifetime chance of injury related to drinking is an order of magnitude higher than the lifetime chance of death from injury except at the most harmful drinking levels. The ratio of the odds of hospitalisation to the odds of death from injury was much higher at lower amounts of drinking (eg two standard drinks or less on a day) than at higher amounts of drinking. This reflects that the chances of dying from an alcohol-related injury that is serious enough to require hospitalisation increases for higher levels of drinking.

Figures 8 and 9 show the lifetime chances per 100 of being hospitalised for alcohol-related injury with different lifetime frequencies of drinking occasions at different numbers of standard drinks per occasion. The curves are somewhat more linear than the curves for deaths from injury.

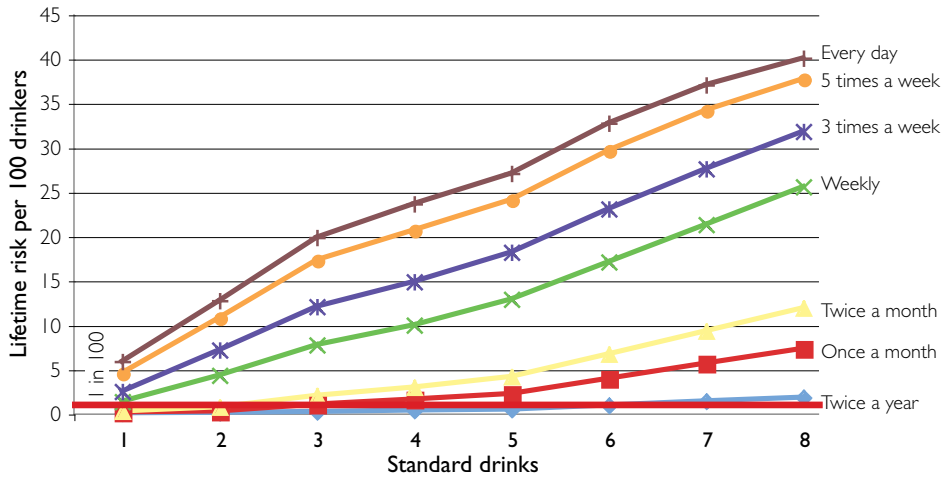


Figure 8 Lifetime risk of hospitalisation for alcohol-related injury per 100 male drinkers, by number of standard drinks per occasion and frequency of occasions

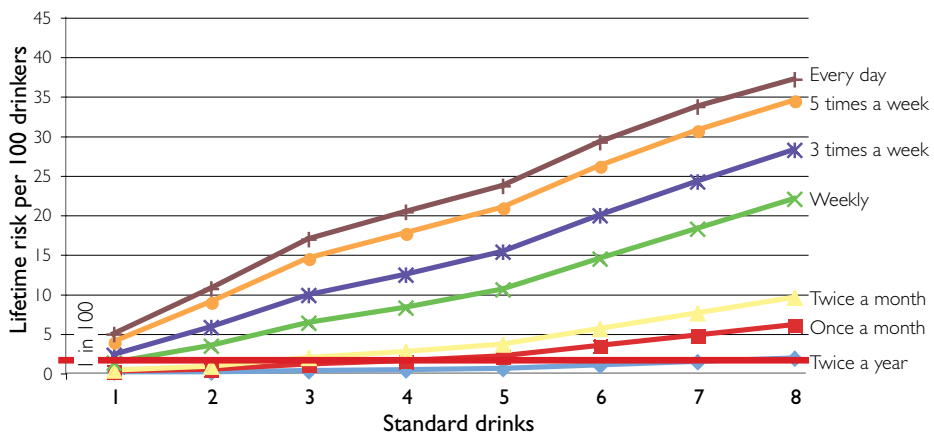


Figure 9 Lifetime risk of hospitalisation for alcohol-related injury per 100 female drinkers, by number of standard drinks per occasion and frequency of occasions

The figures show that:

- for both men and women, the risk of hospitalisation for alcohol-related injury increases with frequency of drinking
- risks of being hospitalised for men are higher than those for women at all levels of drinking
- when drinking occasions are frequent (eg nearly every day) the lifetime risk of hospitalisation for alcohol-related injury is approximately 1 in 10 for both men and women if they always drink two drinks or less on an occasion.

## Lifetime risk of alcohol-related harm

The modelling described above found that consumption of alcohol at any level increases the risk of harm. It also provides convincing evidence that drinking at levels higher than two standard drinks on any day is associated with increased risks of alcohol-related injury, disease and death. Table 3 shows the lifetime risk of alcohol-related death for drinking daily. Table 4 shows the risk for drinking weekly. A more detailed version of this information, including data for other frequencies of drinking, is included in Appendix A5.

Note that the risks of death from alcohol-related disease and of death from injury for a given pattern are additive; therefore, for example, the total risk of death from drinking four drinks daily is estimated at 4.2 in 100 for men and 3.8 in 100 for women.

Table 3 Lifetime risk of alcohol-related death, drinking a certain amount daily

Men			
Number of standard drinks	Total risk of alcohol-related death	Death from alcohol-related disease	Death from injury
1	0.4 in 100	0.2 in 100	0.2 in 100
2	0.9 in 100	0.4 in 100	0.5 in 100
3	2.8 in 100	1.3 in 100	1.5 in 100
4	4.2 in 100	2.0 in 100	2.2 in 100
5	5.8 in 100	2.7 in 100	3.1 in 100
6	9.0 in 100	3.8 in 100	5.3 in 100
7	12.2 in 100	4.7 in 100	7.5 in 100
8	14.8 in 100	5.1 in 100	9.7 in 100

Table 3 Lifetime risk of alcohol-related death, drinking a certain amount daily (cont.)

<b>Women</b>			
<b>Number of standard drinks</b>	<b>Total risk of alcohol-related death</b>	<b>Death from alcohol-related disease</b>	<b>Death from injury</b>
1	0.3 in 100	0.2 in 100	0.1 in 100
2	0.8 in 100	0.4 in 100	0.4 in 100
3	2.3 in 100	1.4 in 100	0.9 in 100
4	3.8 in 100	2.5 in 100	1.3 in 100
5	5.5 in 100	3.7 in 100	1.8 in 100
6	8.9 in 100	5.9 in 100	3.0 in 100
7	11.8 in 100	7.6 in 100	4.2 in 100
8	13.7 in 100	8.4 in 100	5.3 in 100

*Note:* The figures in this table represent the risks above the baseline (not drinking).

Figures have been rounded to one decimal place and therefore may not add up.

Table 4 Lifetime risk of alcohol-related death, drinking a certain amount weekly

<b>Men</b>			
<b>Number of standard drinks</b>	<b>Total risk of alcohol-related death</b>	<b>Death from alcohol-related disease</b>	<b>Death from injury</b>
1	0.1 in 100	0.1 in 100	<0.1 in 100
2	0.2 in 100	0.1 in 100	0.1 in 100
3	0.6 in 100	0.3 in 100	0.3 in 100
4	0.8 in 100	0.3 in 100	0.4 in 100
5	1.1 in 100	0.4 in 100	0.6 in 100
6	1.7 in 100	0.5 in 100	1.2 in 100
7	2.4 in 100	0.6 in 100	1.7 in 100
8	3.0 in 100	0.7 in 100	2.3 in 100

Table 4 Lifetime risk of alcohol-related death, drinking a certain amount weekly (cont.)

<b>Women</b>			
<b>Number of standard drinks</b>	<b>Total risk of alcohol-related death</b>	<b>Death from alcohol-related disease</b>	<b>Death from injury</b>
<b>1</b>	<b>0.1 in 100</b>	0.1 in 100	<0.1 in 100
<b>2</b>	<b>0.2 in 100</b>	0.1 in 100	<0.1 in 100
<b>3</b>	<b>0.5 in 100</b>	0.3 in 100	0.2 in 100
<b>4</b>	<b>0.7 in 100</b>	0.4 in 100	0.3 in 100
<b>5</b>	<b>0.9 in 100</b>	0.6 in 100	0.4 in 100
<b>6</b>	<b>1.4 in 100</b>	0.7 in 100	0.7 in 100
<b>7</b>	<b>1.9 in 100</b>	0.8 in 100	1.1 in 100
<b>8</b>	<b>2.3 in 100</b>	0.9 in 100	1.4 in 100

*Note:* The figures in this table represent the risks above the baseline (not drinking).

Figures have been rounded to one decimal place and therefore may not add up.

The tables highlight that:

- drinking less frequently over a lifetime (drinking weekly rather than daily) considerably reduces the risk of alcohol-related harm
- drinking less on occasions when drinking does occur also considerably reduces the risk over a lifetime, both of alcohol-related disease and of injury
- increasing consumption from two to four standard drinks daily increases the lifetime risk of death from alcohol-related injury by more than four-fold for men and more than three-fold for women
- increasing consumption from two to four standard drinks daily increases the lifetime risk of death from alcohol-related disease by five-fold for men and more than six-fold for women.

## Guideline 2: Reducing the risk of injury on a single occasion of drinking

### GUIDELINE 2

On a single occasion of drinking, the risk of alcohol-related injury increases with the amount consumed.

**For healthy men and women, drinking no more than four standard drinks on a single occasion reduces the risk of alcohol-related injury arising from that occasion.**

Each drinking occasion also contributes to the lifetime risk of alcohol-related harm.

This guideline applies to healthy men and women aged 18 years or over.<sup>8</sup> It does not represent a 'safe' or 'no-risk' drinking level; nor does it set a prescribed level or absolute upper limit of intake.

Data that differentiate between men and women concerning the risk of alcohol-related injury on a single occasion of drinking<sup>9</sup> give different results depending on the criterion.

On average, women reach a given blood alcohol concentration with a lower amount of alcohol than men. However, on average, men take more risks than women at a given level of drinking, and thus most emergency department presentations for alcohol-related injuries involve men.

<sup>8</sup> Special precautions for children and young people under 18 years of age and women who are pregnant or breastfeeding are given in Guidelines 3 and 4 respectively.

<sup>9</sup> A drinking occasion is defined as a sequence of drinks taken without the blood alcohol concentration reaching zero in between. This can be at home or at an event, but also includes drinking spread across more than one context or venue.

## Guideline rationale

A primary emphasis in developing the *Australian Guidelines to Reduce Health Risks from Drinking Alcohol* was calculating the lifetime risk of alcohol-related harm. However, in view of growing community concern about harm arising from single occasions of drinking, particularly drinking among young people, the NHMRC undertook to also set a guideline on reducing the risk of injury on single occasions of drinking.

This guideline is based on review of the available evidence. The basis and quality of the evidence for the guideline are discussed below. The findings are summarised on pages 54 to 56. The methodology for the literature review is presented in Appendix A3. The process of deriving harm scores for adult drinking behaviours is outlined in Appendix A5.

There are a number of difficulties in establishing advice on reducing the risk of alcohol-related injury on a single occasion of drinking:

- evidence concerning the risk of alcohol-related injury is largely based on self-reporting which may lead to underestimation or overestimation of effects
- variability in terms of individual metabolism and timing of drinking on a single occasion affect the blood alcohol concentration (BAC) from a given number of drinks
- the tendency towards hazardous behaviour or delinquent behaviour varies between individuals and with age and gender
- the setting in which the drinking takes place can affect the level of risk of injury (eg if travel is necessary after drinking).

## Basis and quality of the evidence

### *Available published studies*

The association between alcohol and injury has not been thoroughly examined as, until recently, prospective studies did not focus on amounts consumed on a single occasion of drinking, and few controlled studies of drinking events and injury were undertaken. The literature review for these guidelines identified relevant published studies as follows:

- studies of emergency department presentations for alcohol-related injury (Stockwell et al 2002; Vinson et al 2003; Borges et al 2004a; Cherpitel et al 2004a; Watt et al 2004; Spurling & Vinson 2005; Borges et al 2006; Gmel et al 2006) (these were also used as the basis for the modelling of lifetime risk of alcohol-related injury; see Guideline 1)

- studies into the effects of alcohol on cognitive performance (Tagawa et al 2000; Howland et al 2001; Marinkovic et al 2001; Verster et al 2003; Marinkovic et al 2004; Schweizer et al 2004; Easdon et al 2005; Moulton et al 2005; Schweizer et al 2006; Breitmeier et al 2007)
- studies investigating the relationship between BAC and injury severity (Li et al 1997; Porter 2000; Borges et al 2004a; Cherpitel et al 2004a; Johnston & McGovern 2004; Borges et al 2006; Watt et al 2006)
- studies that differentiate between the alcohol-related risks of injury for men and women (McLeod et al 1999; Mumenthaler et al 1999; Bergman & Kallmen 2002; Seale et al 2002; Worrall et al 2002; Delva et al 2004; Flanagan et al 2004; Geisner et al 2004; Parry et al 2005).

A variety of methods have been used for assessing the risk of injury from different levels of alcohol consumption and definitive methods of determining risk have not been established. As discussed under Guideline 1 (see page 39), it is possible that some of the methodologies employed may overestimate risk. However, these studies provide the best available evidence on which to base calculations for risk of alcohol-related injury.

### ***Re-analysis of existing emergency department datasets***

To assess the association between alcohol-related injury and levels of drinking on a single occasion, the following datasets from peer-reviewed, published studies were also re-analysed by the original authors of the papers, using reported alcohol use prior to injury categorised in terms of single-drink increments:

- a large Australian dataset (n=1,770) on emergency department admissions for alcohol-related injury (Stockwell et al 2002)
- emergency department data from the Gold Coast (n=488) (Watt et al 2004)
- data from the WHO 10-site international study of emergency department alcohol-related admissions (n=4,320) (Borges et al 2006).

Across the three different data sets, a substantial similarity was found in terms of the risk curve associated with an emergency department presentation for an alcohol-related injury, with all studies showing a greater increase in risk of injury after four standard drinks on a single occasion than after lower numbers of drinks. However, comparison with results from breath-tests in the WHO study (WHO 2004) suggest that under-reporting by injury patients of how much they had consumed may have produced the apparent threshold of around four drinks.



### ***Analysis of adult drinking behaviours***

An analysis of adult respondents (aged 18+) in the *2004 National Drug Strategy Household Survey* (AIHW 2005) concerning gender patterns in hazardous behaviour and delinquent behaviour was also undertaken to inform the guidelines. The methodology for the analysis is discussed in Appendix A5.

## **Summary of the evidence**

### ***Emergency department presentations***

The modelled analysis used to identify the lifetime risk of injury for Guideline 1 (Rehm et al 2008) is based on studies of the acute effects of alcohol in people who present to emergency departments with injuries (Vinson et al 2003; Borges et al 2004a; Cherpitel et al 2004b; Watt et al 2004; Spurling & Vinson 2005; Watt et al 2005; Borges et al 2006; Gmel et al 2006). These studies calculated the relative risk of injury after drinking specific numbers of drinks, compared with not drinking, and show an increase in risk of injury by between about two and 10 times, depending on the amount of alcohol consumed. A new meta-analysis found that the relative risk of injuries other than in motor vehicles is doubled after three drinks, compared with not drinking (Taylor et al 2009). The studies also showed that the risk of injury increased more for people whose level of consumption varied significantly from time to time, and was particularly high for those who occasionally drank much more than their usual amount.

Using these data as a basis, studies show a greater increase in risk of injury after four standard drinks on a single occasion than after lower numbers of drinks.

### ***Cognitive performance***

There is evidence indicating that drinking decreases cognitive performance, even at low levels of consumption. The acute effects of alcohol increase with the amount consumed, along with the risk of adverse outcomes.

Studies into the effect of alcohol on cognitive performance have found that:

- as the blood alcohol level increases, cognitive function and psychomotor performance decrease rapidly (Easdon et al 2005); consumption of less than two standard drinks potentially results in effects that increase risk of injury (Tagawa et al 2000; Howland et al 2001; Marinkovic et al 2001; Marinkovic et al 2004; Moulton et al 2005; Breitmeier et al 2007) and driving ability is impaired at blood alcohol levels of about 0.05 per cent, a level reached after two or three standard drinks (Tagawa et al 2000)

- substantial impairment can exist well after alcohol has been metabolised and passed from the body (Schweizer et al 2004; Schweizer et al 2006); such temporary impairment, and its attendant risk, is the result of a 'hangover' effect (Verster et al 2003).

### ***Injury severity***

Studies into the relationship between BAC and injury severity had mixed results, for example:

- two studies (Li et al 1997; Johnston & McGovern 2004) reported that injury severity increased proportionate to BAC
- a case-control study (Watt et al 2006) found that patients who drank alcohol above 'low-risk' levels or who drank beer in the six hours before being injured were significantly more likely to sustain serious rather than minor injuries
- in contrast, analysis of a state trauma database (Porter 2000) found no significant association between BAC and fatal injuries, and a trend towards decreased injury severity with the presence of alcohol.

Several studies have shown a higher proportion of violence-related injuries among those drinking before the incident than for non-alcohol-related injuries (Cherpitel et al 2004a, Borges et al 2004a, Borges et al 2006).

### ***Gender differentiation***

Data that differentiate between alcohol consumption levels for men and women provide contrasting results and, as a consequence, the issue of whether or not to set different guideline levels for men and women is contentious.

- *Blood alcohol levels* – because of both body weight and body composition, women on average attain a given blood alcohol level with a lower amount of alcohol than men. However, experimental studies of differences in post-drinking performance give a mixed picture (Mumenthaler et al 1999).
- *Chances of injury at a given blood-alcohol level* – emergency department studies show that most injuries involve men rather than women, and approximately two-thirds of all patients with an alcohol-related injury are men. Men's behaviour when drinking is, on average, more risky than women's at a given level of drinking. Modelling on US data shows that the relative risk of driver fatality is particularly high for young male drivers who have been drinking – at 0.05–0.08 BAC, the relative risk is 14.3 for males aged 16–20 years, compared with 4.6–5.3 for older males and for females of all ages (Zador et al 2000).

- *Drinking pattern and risk of injury in a defined period* – the analysis of the 2004 National Drug Strategy Household Survey (AIHW 2005) investigated the risk associated with drinking events, and gender differences or similarities in this, from several perspectives (see Appendix A5; pages 131 to 135). With respect to hazardous behaviour connected to drinking, women have a lower risk of hazardous behaviour while drinking at each level of maximum number of drinks, and this is generally true in all age groups. On an index of hazardous behaviour per litre, men showed a higher average score than women at all ages. Age was a much more important differentiator than gender on this measure.

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The available evidence on single occasion drinking suggests that:

- any consumption of alcohol increases the risk of injury on a single drinking occasion
  - having four drinks on a single occasion more than doubles the relative risk of an injury in the six hours afterwards
  - the relative risk rises more rapidly above the level of four drinks on an occasion
  - each drinking occasion contributes to the lifetime risk of alcohol-related injury and disease, as noted in the discussion of Guideline 1
  - the lifetime risk of death from injury remains below 1 in 100 for both men and women if they always drink two drinks or less on an occasion, even if the occasions are every day
  - the lifetime risk of hospitalisation from injury is about 1 in 10 for men and 1 in 12 for women with a drinking pattern of four drinks on an occasion about once a week.
-

## Guideline 3: Children and young people under 18 years of age

### GUIDELINE 3

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For children and young people under 18 years of age, not drinking alcohol is the safest option.

- A Parents and carers should be advised that children under 15 years of age are at the greatest risk of harm from drinking and that for this age group, not drinking alcohol is especially important.**
- B For young people aged 15–17 years, the safest option is to delay the initiation of drinking for as long as possible.**

This guideline applies to children and young people up to 18 years of age and provides guidance for parents and carers, as well as for young people themselves, about the safest option to prevent alcohol-related harm during these years.

Guideline 3 is based on an assessment of the potential harms of alcohol for this age group, as well as a range of epidemiological research that indicates that alcohol may adversely affect brain development and be linked to alcohol-related problems later in life. However, this evidence is not conclusive enough to allow definitive statements to be made about the risks of drinking for young people.

As a result, as for adult drinking, it was not possible to set a 'safe' or 'no-risk' drinking level for children and young people. The safest option for children and younger people is not to drink at all and the safest option for older teenagers (15–17-year-olds) is to delay the initiation of drinking for as long as possible.

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This guideline does not advocate that young people drink or that adults provide them with alcohol, but that if drinking does occur it should be at a low-risk level and in a safe environment, supervised by adults. Drinking to intoxication is particularly risky in this age group.

Serving drinks to young people under the age of 18 years by parents, carers or other adults may be subject to legislation. Supervision of drinking by young people should take account of local legislation.

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## Guideline rationale

This guideline is based on evidence showing that the risks of accidents, injuries, violence and self-harm are high among drinkers aged under 18 years. Drinkers under 15 years of age are much more likely than older drinkers to experience risky or antisocial behaviour connected with their drinking, with the rates also somewhat elevated among drinkers aged 15–17 years. In addition, the evidence suggests that earlier initiation of drinking is related to more frequent and higher quantity alcohol consumption in adolescence, and these patterns are in turn related to the development of alcohol-related harms in adolescence and adulthood.

This guideline is based on review of the evidence as summarised on pages 58 to 68.

Evidence specific to young adults up to the age of 25 is discussed in Section B of Appendix A1.

## Quality of the evidence

The main evidence in this area is epidemiological, principally longitudinal studies, cohort studies and survey reports of drinking prevalence, associated risk-taking behaviours and adverse outcomes in children and younger people.

There have been very few human studies on the effects of alcohol on brain development in young people, with most information being drawn from studies of animal models. Human studies are largely limited to young people with alcohol-use disorders. The lack of evidence on the impact of alcohol on young people is partly because this is a developing area and methods to examine these risks have only recently become available, and partly because the endpoints are in many cases quite subtle, and may not have been detected by previous research studies.

A secondary analysis was undertaken to derive harm scores from 2004 *National Drug Strategy Household Survey* data (Livingston & Room, in press), in order to assess more accurately the potential harms of alcohol in this age group, and to make guideline recommendations.

## Summary of the evidence

### *Risk of injury and self-harm*

Rates of drinking at harmful levels by 12–17-year-olds have doubled in the past two decades (White & Hayman 2007). Drinking contributes to the three leading causes of death among adolescents – unintentional injuries, homicide and suicide (Stephens 2006; Miller et al 2007).

Between 1993 and 2001:

- 28 per cent of all alcohol-related injury deaths and more than one-third (36 per cent) of alcohol-related injury hospitalisations were sustained by young people aged 15–29 years (Chikritzhs et al 2003)
- about half (54 per cent) of all serious road injuries involved young people (see Figure 10).

Alcohol consumption as an adolescent or young adult is also associated with physical injury, risky sexual behaviour, adverse behavioural patterns and academic failure (Bonomo et al 2001; Zhang et al 2002; Abbey et al 2003ab; Barnett et al 2003; Howard et al 2003; Vinson et al 2003; Johnson & Stahl 2004; Swahn et al 2004; Coleman & Cater 2005; Kebede et al 2005; Mora-Rios et al 2005; Boyd et al 2006; Davis et al 2006; Dye & Upchurch 2006; Fallu et al 2006; French & Maclean 2006; Kaysen et al 2006; Shepherd et al 2006).

There have been a number of studies into alcohol and violence in teenagers. For example:

- a nationwide survey of teenagers in Finland showed that 13 per cent of 14-year-olds, 41 per cent of 16-year-olds and 62 per cent of 18-year-olds reported being under the influence of alcohol during a violent incident (Mattila et al 2005)
- an American cohort study found 11 per cent of youths reported being influenced by alcohol during a fight and were also significantly more likely than their sober counterparts to injure others or sustain an injury during the fight (Kodjo et al 2004).

The prevalence of risk-taking behaviours increases in adolescence and the likelihood of injury increases further still when alcohol is also involved.

- A recent study of high-school students found a strong dose–response relationship between ‘binge-drinking’ and risky behaviours, including riding in a car with an intoxicated driver and using illicit drugs (Miller et al 2007).
- A register-based retrospective cohort study of 334,000 adolescents in Sweden showed that alcohol was significantly associated with the severity of motorcycle-related injury (Zambon & Hasselberg 2006).
- The United States National Youth Survey (Swahn & Bossarte 2007) showed that alcohol use in adolescents, and particularly in pre-teenagers, is a strong predictor of both suicidal ideation and completed suicide for both males and females.

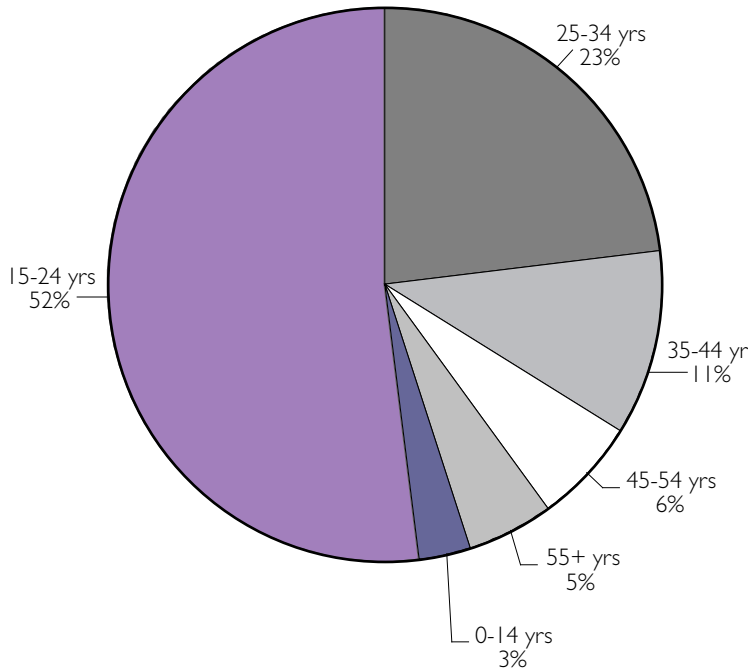


Figure 10 Overall age distribution among alcohol-related serious road injuries occurring in Australia (excluding Victoria), 1990–97

Source: Chikritzhs et al (2000).

A particular concern is the increase in adolescent risky sexual behaviour when alcohol is involved (Coleman & Cater 2005).

- Results from a prospective cohort study in the United States show that use of alcohol decreases the likelihood that adolescents will use a condom, especially at their first sexual experience (Dye & Upchurch 2006). However, diary studies of young adults show that when young people expect to get drunk, they plan for sexual activity that may occur (Leigh et al 2008).
- Adolescents who drink alcohol are at risk of sexual coercion (Davis et al 2006). A controlled trial in which 180 youths were given either an alcoholic beverage or a placebo and asked to give their opinion about a hypothetical sexual situation showed that intoxicated participants viewed the woman in the vignette as more aroused, and the man as more justified in his attempts to force the woman to have intercourse with him (Abbey et al 2003a).

Death by alcohol overdose in adolescents is also a risk, due to their generally smaller physique, preference for drinking spirits and lower alcohol tolerance. A recent emergency-department surveillance study found that 56 per cent of poisonings admissions were due to acute alcohol intoxication (Cheng et al 2006). Although alcohol is often used by depressed and suicidal adolescents as a means of self-harm or an attempt on their life, unintentional alcohol poisoning is also common (Cheng et al 2006).

### ***Effect on brain development***

Although the prevalence of alcohol-use disorders in younger people is high, there are few human studies that show the effects of alcohol consumption on brain development during this period. Much of the information about the effect of alcohol on brain development is drawn from studies of animal models. Animal research has shown that young animals are more sensitive to ethanol-induced disruptions in brain plasticity and are also less sensitive to cues that serve to moderate alcohol intake (Spear 2004).

In a study of adolescent and adult rats, Crews et al (2000) found that the effects of a four-day alcohol 'binge' were worse for adolescent rats. Although brain damage was found in both age groups, substantial frontal lobe deterioration was observed only in adolescent rats.

Young people with alcohol-use disorders display significant and detrimental changes in brain development compared with their non-alcohol-using peers. Studies have shown that significant changes in brain structure accompany heavy drinking:

- alcohol-abusing adolescents tend to have smaller pre-frontal cortices and white matter volumes – an effect more pronounced for males than females (De Bellis et al 2005)
- white matter structural irregularities and reduced hippocampal volumes have also been observed (Brown & Tapert 2004)
- hippocampal function is uniquely responsive to alcohol during adolescent development and may be more sensitive to neurotoxicity during this period (White & Swartzwelder 2004).

Adolescent drinking is associated with diminished retrieval of verbal and non-verbal material, and poorer performance on attention-based testing (Brown & Tapert 2004). Brown and Tapert (2004) suggest that physiological effects of alcohol withdrawal over the teen years contribute to deterioration in cognitive functioning in visuospatial tasks. Cognitive impairment is also common in young adults with alcohol dependence (Kelly & Witkiewicz 2003).



## ***Mental health***

Alcohol use, especially when initiated at a young age, elevates the risk of many mental health and social problems (Brown & Tapert 2004). The existence of psychiatric comorbidities in adolescents who abuse drugs is common, especially for conditions such as depression, anxiety, bipolar disorder, conduct disorder and attention-deficit/hyperactivity disorder (Turner & Gil 2002; Brown & Tapert 2004; Cheng et al 2006; Deas & Brown 2006; Cargiulo 2007).

The nature of the relationship between alcohol use and mental health in adolescence is somewhat reciprocal. Youths with certain mental health disorders are more likely to initiate alcohol use and accelerate their use throughout adolescence (Brown & Tapert 2004). In turn, alcohol use may contribute to poor mental health.

In a random sample of more than 27,400 college students from across the United States, Weitzman (2004) found that students with poor mental health were likely to report frequent, heavy and heavy episodic drinking and were also more likely to drink with the intent of getting drunk. In a similar study, Geisner et al (2004) found that the association between psychological distress and negative drinking consequences was greater for male college students than females.

One of the major complications of adolescent alcohol use is self-harm, having suicidal thoughts and suicide (Miller et al 2007). Alcohol-use disorders, in conjunction with major depression, represent an especially high-risk profile for adolescent suicidal behaviour and completed suicide (Sher 2006). In addition, adolescents with alcohol-use disorders tend to complete suicide at a greater rate than those without alcohol problems (Sher 2006). It has been suggested that adolescents who use drinking as a method of coping are more likely to suffer from depression, precipitating heavy drinking, which is itself predictive of suicidal behaviour (Windle 2004).

## ***Age of first drinking***

Several studies indicate that initiating alcohol use at an early age increases the likelihood of later adverse physical and mental health conditions (Hemmingsson & Lundberg 2001; Hingson et al 2003; Guilamo-Ramos et al 2004; Toumbourou et al 2004; Wells et al 2004; Jefferis et al 2005).

There is some evidence to suggest that the later adolescents delay their first alcoholic drink, the less likely they are to become regular consumers (Australian Institute of Family Studies 2004). In addition, various studies have shown that:

- those who first became drunk by 19 years are more likely to be alcohol dependent and heavy drinkers in later life (Hingson et al 2003)

- drinking status at 16 years is a predictor of negative alcohol outcomes as a young adult (Wells et al 2004)
- teens who were drinking by 14 years were more likely to experience alcohol dependence than their peers who did not drink until they were over 21 years old (Hingson et al 2006; Toumbourou et al 2004)
- both age of drinking onset and feeling drunk during first alcohol experience increased the odds of problem drinking into adulthood (Warner et al 2007) and this level of risk was higher in men than in women (Pitkanen et al 2005).

The way in which adolescents are introduced to alcohol may affect future drinking patterns. An American cross-sectional survey of adolescents (Foley et al 2004) found that provision of alcohol to adolescents by their parents or adult relatives at home reduced the level of drinking. However, providing alcohol to adolescents at a party was associated with a two-fold risk of 'binge-drinking' (Foley et al 2004).

Australian longitudinal studies have demonstrated that regular drinking in adolescence is an important risk factor for the development of abusive, dependent (Bonomo et al 2001) and risky (Toumbourou et al 2004) patterns of use in young adulthood (Australian Institute of Family Studies 2004).

### Self-reported harm scores

Given the inconclusive nature of the epidemiological evidence in this area, a secondary analysis was undertaken in order to provide a basis for the guideline recommendations (Livingston & Room, in press). The analysis considered the level of harm caused by a given level of drinking for different age groups, using data from the *2004 National Drug Strategy Household Survey* (AIHW 2005).

These data were used to derive harm scores from the series of questions on self-reported problems from drinking. The harm scores were calculated as the harm score divided by the volume of drinking, and by the number of days per year on which five or more standard drinks were consumed. These are presented as ratios, with the male indexes at 40–44 years set as 1.0. Two separate scores were calculated: a hazardous behaviour score and a delinquent behaviour score. The methodology for the calculation of the scores is discussed in Appendix A5.

Figure 11 gives results for the hazardous behaviour score, showing that the scores tend to be higher for 12–14 year-olds than for any other age group. From age 15 on, the scores are fairly steady until age 40, after which they tend to decline.

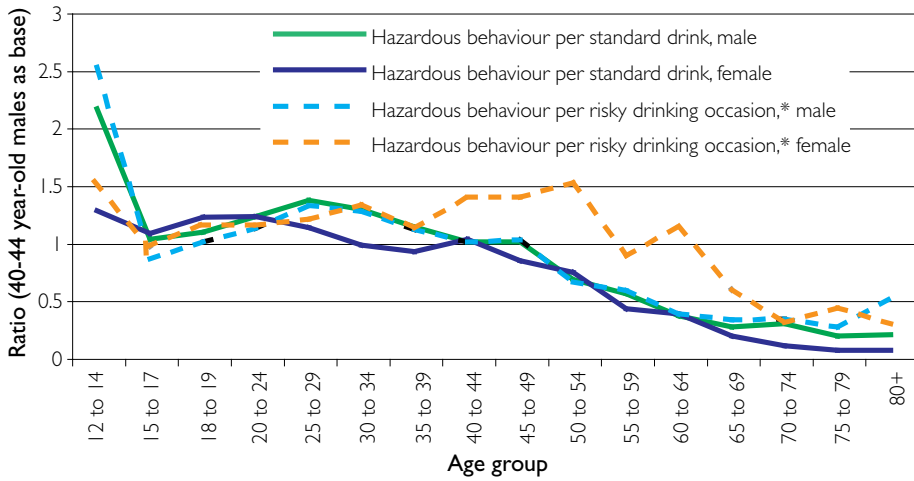


Figure 11 Hazardous behaviour score per volume of alcohol consumed and occasions drinking five or more drinks, by age and gender; National Drug Strategy Household Survey, 2004

*Note:* \*Risky drinking occasion refers to an occasion where five or more standard drinks are consumed. This also refers to Figure 13.  
The varying results for older females per drinking occasion reflect the small base of older women drinking five or more standard drinks.

Figure 12 shows results for the delinquent behaviour score. These indexes are much higher for 12–14-year-olds than for any other age group, and at 15–17 years are still several times higher in both genders than for 40–44-year-olds; after this there is a long and steady decline in the indices with increasing age.

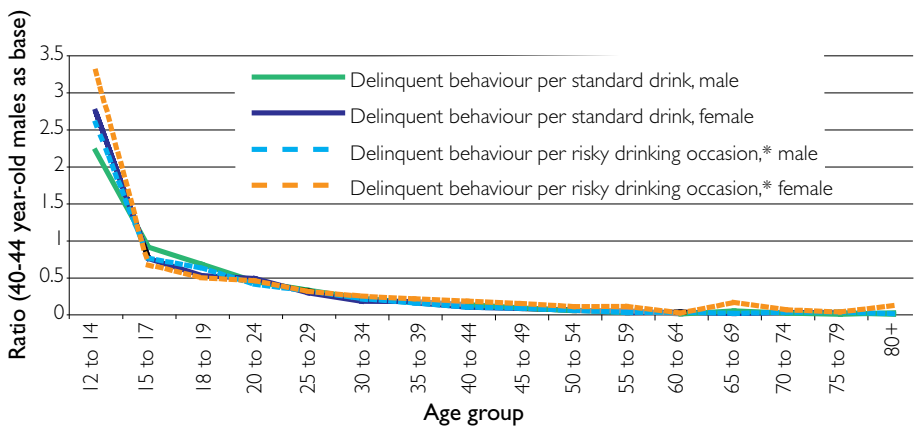


Figure 12 Delinquent behaviour score per volume of alcohol consumed and occasions drinking five or more drinks, by age and gender; National Drug Strategy Household Survey, 2004

Generally, the harm indexes are similar for males and females at a given age, although the hazardous behaviour indexes are slightly higher for males at ages 12–14, and the delinquent behaviour indexes slightly higher for females at ages 12–14 and for males at ages 18–19, compared with the same gender at 40–44 years.

The analysis also considered the pattern of overall harm scores per volume of drinking among the minority of drinkers at each age who reported drinking no more than one or two drinks on any occasion:

- the proportion limiting their drinking in this way was 22 per cent among 12–14-year-olds, fell as low as 7 per cent among 18–19-year-olds, and then gradually rose to 39 per cent among 75–79-year-olds—in this minority of drinkers keeping their drinking at low levels, the drinking-related harm scores were low
- however, the patterning of the overall harm index by age again showed a much higher value among 12–14-year-olds, and a somewhat higher value among 15–17-year-olds, than among older respondents (Figure 13).

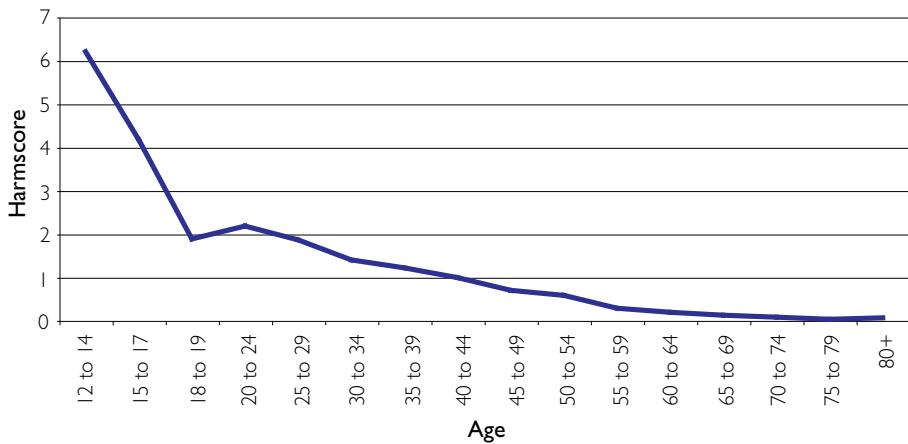


Figure 13 Harm per volume for drinkers drinking less than three standard drinks per session

Note: Ratio with 40–44 as base.

## KEY POINTS

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Taken together, the results of the secondary analysis suggest that:

- Drinkers under the age of 15 years are much more likely than older drinkers to experience risky or antisocial behaviour connected with their drinking, providing the basis for Guideline 3A.
- The rates of risky behaviour are also elevated among drinkers aged 15–17 years, warranting the recommendation for caution (Guideline 3B).
- To a lesser extent, young adults up to the age of 25 also show a propensity for greater harm per unit of alcohol than older people (see Section B of Appendix A1).

## Guideline 4: Pregnancy and breastfeeding

### GUIDELINE 4

Maternal alcohol consumption can harm the developing fetus or breastfeeding baby.

**A For women who are pregnant or planning a pregnancy, not drinking is the safest option.**

**B For women who are breastfeeding, not drinking is the safest option.**

This guideline applies to women who are pregnant, are planning a pregnancy, or are breastfeeding. It is based on an assessment of the evidence concerning potential harms of alcohol for the developing fetus and for young babies during the breastfeeding period. Apart from adverse pregnancy outcomes, harms to the mother from alcohol consumption during pregnancy are not discussed as the available evidence is limited.

As the risks from maternal alcohol consumption in pregnancy and during lactation differ, these are considered separately.

#### ***Pregnancy***

Guideline 4A is based on systematic reviews of the literature and prospective cohort studies. However, the complexity of the issue makes development of policy and provision of definitive advice difficult. Maternal alcohol consumption can result in a spectrum of harms to the fetus. Although the risk of birth defects is greatest with high, frequent maternal alcohol intake during the first trimester, alcohol exposure throughout pregnancy (including before pregnancy is confirmed) can have consequences for development of the fetal brain. It is not clear whether the effects of alcohol are related to the dose of alcohol and whether there is a threshold above which adverse effects occur (RCOG 2006). However, variation in effects can be due to the stage of development of the fetus at the time of exposure and to individual characteristics of the mother.

This uncertainty is reflected in policy regarding alcohol use in pregnancy within Australia and overseas (O'Leary et al 2007). Most policies stress that 'heavy' drinking poses the greatest risk; that the timing of exposure is important; and that not all 'heavy' drinkers will have an affected child. However, several policies emphasise that a safe level has not been established and conclude that not drinking is the safest option.

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A 'no-effect' level has not been established, and limitations in the available evidence make it impossible to set a 'safe' or 'no-risk' drinking level for women to avoid harm to their unborn children, although the risks to the fetus from low-level drinking (such as one or two drinks per week) during pregnancy are likely to be low. A conservative, public health approach has therefore been taken in recommending that 'not drinking alcohol is the safest option' for pregnant women and women planning a pregnancy. This decision was not based on the fact that substantial new evidence had emerged since the previous guidelines were published, but on limitations of the existing evidence.

Women who drank alcohol before they knew they were pregnant or during their pregnancy should be reassured that the majority of babies exposed to alcohol suffer no observable harm. The risk to the fetus from low level drinking is likely to be low. Women who find it difficult to decrease their alcohol intake will require support and treatment. It is important that they are referred to the appropriate services.

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### ***Breastfeeding***

There is a lack of good quality evidence from human studies regarding the effects of maternal alcohol consumption on lactation, infant behaviour and development. As a result, as for pregnancy, it was not possible to set a 'safe' or 'no-risk' drinking level for breastfeeding women. Guideline 4B therefore takes a conservative approach and advises not drinking as the safest option.

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It is acknowledged that an abstinence message may discourage breastfeeding. For this reason, although women who are breastfeeding are advised that 'not drinking alcohol is the safest option', practical guidance regarding minimising the risk to lactation and to the breastfed infant is also provided for mothers who choose to drink.

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### **Quality of the evidence**

The best evidence to support this guideline on alcohol during pregnancy was provided by systematic reviews of the literature (Makarechian 1998; Polygenis 1998; Testa et al 2003; Henderson et al 2007a; Henderson et al 2007b), as well as other reviews (O'Leary 2004;) and prospective cohort studies published subsequent to these reviews, which provide high level evidence for the risks of alcohol consumption during pregnancy. However, interpretation of the published research is hampered by methodological problems, including:

- difficulties inherent with under-reporting of alcohol intake and accurate documentation of the quantity, timing and frequency of alcohol intake during pregnancy

- use of variable definitions for low, moderate and high levels of maternal alcohol intake
- failure or inability to identify and adjust for potential confounding factors such as maternal age and parity, body composition, nutrition, polydrug use, cigarette smoking, socio-economic status and education, and maternal and fetal genetics
- short duration of follow-up, loss to follow-up, or evaluation of only limited outcomes in exposed children
- difficulties in comparing studies from different countries and settings due to differences in how alcohol consumption is measured and reported
- the focus of some studies on high-risk population groups, the findings of which may not be applicable to Australia or Australians
- publication bias, in which studies with positive results are both more likely to be submitted and accepted for publication.

The best evidence to inform the guideline on the effect of alcohol intake on breastfeeding and the breastfed infant comes from a systematic literature review (Giglia & Binns 2006). However, the authors note the limitations of the evidence from human studies and the consequent difficulty in providing definitive advice to breastfeeding women about the effects of alcohol on lactation, infant behaviour and development.

## Pregnancy

### Rationale

The rationale for this guideline is based on review of the evidence as summarised on pages 71 to 77.

- Most Australian women consume alcohol once a month or more, with 18 per cent of women aged 18–23 years having five or more drinks on one occasion, once a week or more (Young & Powers 2005).
- Rates of drinking during pregnancy are high, with recent Australian surveys reporting rates of 47 per cent in a national survey (Wallace et al 2007) and 59 per cent in a West Australian study (Colvin et al 2007). Of respondents to the West Australian survey, 15 per cent drank above NHMRC 2001 guideline levels<sup>10</sup> during the first trimester (Colvin et al 2007).

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<sup>10</sup> More than seven standard drinks in a week and/or more than two standard drinks on any one day.



- Drinking levels in the period before pregnancy are also high. In the West Australian survey, 14 per cent of respondents reported drinking five or more standard drinks on a typical occasion during this period (Colvin et al 2007). As many pregnancies are unplanned (47 per cent in the West Australian survey), many fetuses may inadvertently be exposed to alcohol before pregnancy is confirmed.
- Between 19 and 44 per cent of Aboriginal women drink alcohol in pregnancy (Zubrick et al 2005; Zubrick 2006; Hayes 2001) and between 10 and 19 per cent drink at harmful levels (Zubrick et al 2006; Hayes 2001).
- High-level and/or frequent intake of alcohol in pregnancy increases the risk of miscarriage, stillbirth and premature birth (O'Leary 2004).
- Alcohol crosses the placenta and nearly equal concentrations in the mother and fetus can be attained. Alcohol is a teratogen (O'Leary 2002) and exposure of the fetus to alcohol may result in a spectrum of adverse effects, referred to collectively as fetal alcohol spectrum disorders (FASD). Of these, fetal alcohol syndrome (FAS) has been described in children exposed to high levels of alcohol in utero as a result of either chronic or intermittent maternal alcohol use (Lemoine et al 1968; Jones et al 1973; Hoyme et al 2005; Astley & Clarren 2000). These children have characteristic facial abnormalities (and often a range of other birth defects), impaired growth and abnormal function or structure of the central nervous system. The diagnosis may not be evident at birth. However, not all children exposed to alcohol during pregnancy are adversely affected, or affected to the same degree. Expression of FAS appears to depend on other factors including (O'Leary 2004): the timing of alcohol intake in relation to the stage of fetal development; the pattern and quantity of alcohol consumption (dose and frequency); and socio-behavioural risk factors (maternal age/duration of drinking, low socio-economic status, race, genetic differences, polydrug use).
- A number of alcohol-related birth defects (ARBD) and alcohol-related neurodevelopmental disorders (ARND) have also been described following exposure to alcohol during pregnancy and can be included, with FAS, under the umbrella term of Fetal Alcohol Spectrum Disorders (FASD) (Hoyme et al 2005; Astley & Clarren 2000). Although children with ARND do not have birth defects, they have significant developmental, behavioural and cognitive problems similar to children with FAS.
- People with FASD experience lifelong problems, including learning difficulties and disrupted education, increased rates of mental illness, drug and alcohol problems and trouble with the law (Streissguth et al 2004).

- The effects of alcohol exposure on fetal development occur throughout pregnancy (including before the pregnancy is confirmed), with the developing fetus being most vulnerable to structural damage during the first three to six weeks of gestation (O’Leary 2004). Effects also vary depending on the dose of alcohol and the pattern of consumption. The most serious of the adverse pregnancy outcomes occur when pregnant women consume high levels of alcohol frequently.
- Recent data indicate that women are likely to be receptive to the advice included in this guideline. In a recent survey of 1,103 Australian women of child-bearing age (Peadon et al 2007):
  - 80 per cent agreed that pregnant women should not drink alcohol
  - 99 per cent said information about the effects of alcohol on the fetus should be readily available
  - 97 per cent said health professionals should ask women about their alcohol use in pregnancy and 97 per cent said they should provide advice about alcohol use in pregnancy
  - 91 per cent said that health professionals should advise women who are pregnant or thinking of becoming pregnant to give up drinking alcohol.

## Summary of the evidence

### *Adverse effects of alcohol on pregnancy, fetal, neonatal and early childhood outcomes*

Several authors have tried to establish a safe lower level of drinking to prevent adverse effects of alcohol consumption in pregnancy on fetal, neonatal and early childhood outcomes.

- In a systematic review, with meta-analysis, of case-control and cohort studies (n=130,810 pregnancies), no increase was found in fetal malformations at or soon after birth with moderate maternal alcohol consumption (between 2.4 and 16.8 standard drinks per week) in the first trimester compared with an intake below this level (Polygenis et al 1998). One limitation of this review is the wide range of intake by ‘moderate’ drinkers. Furthermore, 16 of the 24 studies identified as potentially relevant were excluded because of problems with data quality.
- A review of eight studies into the effects of moderate alcohol consumption in pregnancy on rates of spontaneous abortion, stillbirth and premature birth (Makarechian et al 1998) found that, compared with women who did not drink, women who drank between two and 14 standard drinks per week had a significantly higher rate of miscarriage (OR 1.51; 95% CI 1.20–1.89).

- A systematic review, with meta-analysis, of mental development in infants following different levels of prenatal alcohol exposure (<1 drink per day, 1–1.99 drinks per day, and 2 or more drinks per day) found a significant, negative, linear (dose-dependent) impact on mental development in children aged 12–13 months at an average intake of two or more drinks per day (Testa et al 2003). However, only seven of 24 studies identified were included due to inadequate reporting of raw data or of the amount and timing of alcohol consumption, or lack of a control group. A further limitation of this review is that the amount of alcohol consumed was not specified, only the number of drinks. The authors concluded that ‘because the literature is neither large nor conclusive, and because of heterogeneity in measurement, analysis and samples, caution is urged in interpreting results’.
- The National Perinatal Epidemiology Unit in the UK has recently published two high quality systematic reviews (Henderson et al 2007a; 2007b). The reviews were based on a thorough search of the literature, used strict inclusion criteria for studies (including acceptable methodological quality), and papers were critically appraised using established criteria.
- The first review, which included 46 studies, addressed the effects of low to moderate prenatal alcohol exposure (less than 12g alcohol, or about one standard drink, per week) on pregnancy outcomes (Henderson et al 2007a). In five of the eight studies reporting on miscarriage, an increased rate was observed in the exposed group; however two of these studies had methodological limitations, including failure to adjust for confounding factors. Overall, there was no convincing evidence that low-moderate maternal alcohol intake conferred an increased risk of miscarriage, still birth, prematurity, intrauterine growth restriction (or small for gestational age at birth) and birth defects, including FAS.

Limitations in the studies included in the review include the lack of accurate data on alcohol intake (many studies used a daily or weekly average and did not document the pattern of drinking); lack of focus of studies on low-moderate alcohol intake; potential for publication bias; and lack of prospectively collected information on timing of exposure and hence potential for recall bias. Most of the studies came from the United States and there was lack of consistency in findings between countries, which may reflect different drinking patterns or under-reporting of drinking. Due to the heterogeneity of the included studies, meta-analysis was not performed. The authors concluded that it ‘is difficult to determine whether there was any adverse effect on pregnancy outcome associated with low-moderate levels of prenatal alcohol consumption’ and that the paucity and inconsistency of available evidence ‘preclude the conclusion that drinking at these (low-moderate) levels during pregnancy is safe’.

In the second systematic review (Henderson et al 2007b), the fetal effects of prenatal 'binge-drinking' (defined in most studies as six standard drinks on a single occasion) in women who were pregnant or trying to become pregnant were examined. Outcomes of interest included prematurity, miscarriage, stillbirth, birth weight, gestation, intrauterine growth retardation, birth defects including FAS, and neurodevelopmental outcomes. In the 14 included papers, there was no consistent evidence that 'binge-drinking' influenced rates of these outcomes, with the exception of neurodevelopmental outcomes. Individual studies suggest that disinhibited and delinquent behaviour, reduction in verbal IQ, and learning difficulties and poor educational performance are more common in children whose mothers 'binge'. Furthermore, the risk increased with higher alcohol intake and greater frequency of 'binges'. Many of the included studies had methodological weaknesses and different definitions for 'binge-drinking' were used (eg some studies included women who had a 'binge' on a single occasion and others included only women who 'binged' throughout pregnancy).

A further limitation of both this and the earlier review (Henderson et al 2007a) is the short duration of follow-up of exposed children in the included papers. Alcohol-related neurodevelopmental disorders, for example, may not be diagnosed until school age. The study authors conclude that, despite lack of good evidence of harm from heavy drinking in human studies, the animal literature suggests that the appropriate public health message may be 'recommending pregnant women to avoid binge-drinking'. The reviewers note that, when pregnant women who do not drink regularly report having had isolated episodes of heavy drinking, they should be reassured that the evidence for risk of harm is minimal.

Several papers on pregnancy outcomes have subsequently been published.

- A case-control study (n=552) found an average intake of 1.2 standard drinks per day increased the risk of low birth weight (OR 2.67; 95% CI 1.39–5.12). This effect was greater in smokers but was not observed in infrequent drinkers (Mariscal et al 2006). Another case-control study (n=555), found that small for gestational age (as opposed to low birth weight) was associated with intake of more than 3.6 standard drinks per day during pregnancy and the effect was more marked with exposure in the first trimester (OR 2.67; 95% CI 1.39–5.12) than in the second or third trimester (Chiaffarino et al 2006). Limitations of these two studies included reliance on retrospective, self-reporting of alcohol intake.

- A prospective cohort study from early pregnancy (n=7,141) found no association between alcohol consumption and small for gestational age or preterm birth (Jaddoe et al 2007). However, an average intake of 1.2 standard drinks per day before pregnancy was confirmed as associated with low birth weight (OR 4.81; 95% CI 1.10–21.08). Although this is a large, population-based study, information on alcohol consumption was missing in 14 per cent of women enrolled.

Other recent studies document neurological effects from alcohol, including reduction in nerve-conduction velocity and amplitude (de los Angeles Avaria et al 2004); a dose-dependent decrease in visual acuity in infants (Carter et al 2005); and a dose-dependent reduction in size of the frontal cortex (but not of other brain structures) at intakes of two to six standard drinks per day (Wass et al 2001), a finding consistent with impairment of executive function, working memory and attention observed in children with FASD.

### FETAL ALCOHOL SPECTRUM DISORDERS IN AUSTRALIA

Australian studies indicate:

- continuing occurrence of FAS, with many children in foster care and many with an affected sibling suggesting missed opportunities for prevention (Elliott & Bower 2004; Elliott et al 2007)
- a likely under-ascertainment of FAS due to a lack of knowledge among health professionals of the condition and criteria for its diagnosis (Payne et al 2005; Elliott et al 2006)
- higher rates of FAS in some Indigenous communities compared with non-Indigenous communities (Bower et al 2000; Harris & Bucens 2003; Elliott et al 2007)
- an identified need for research into the association between low to moderate alcohol consumption and fetal harm (O'Leary et al 2007)
- a lack of data on rates of, and need for research on, ARBD and ARND in Australia.

### *Alcohol exposure and outcomes in childhood and adolescence*

There are no published systematic reviews on the effect of alcohol exposure in pregnancy and outcomes in childhood and adolescence, including growth, educational attainment, drug and alcohol dependence, mental health problems, employment and problems with the law. Limitations in published

studies include difficulties in accurately documenting the quantity, timing and frequency of alcohol intake in pregnancy, and failure to identify and adjust for potential confounding factors. Recent prospective cohort studies indicate that:

- Compared with controls, children exposed prenatally to more than 1.4 standard drinks per day in utero had deficits in working memory and executive function but not impulsivity or sustained attention at 7.5 years of age (Burden et al 2005a; Burden et al 2005b) – the effect, which increased with increasing alcohol consumption, was most marked for numeracy tasks and persisted after controlling for IQ. In both studies, adverse effects were more pronounced in children born to mothers aged 30 years or more.
- Prenatal exposure to three or more drinks per week had a significant deleterious effect on children's verbal and non-verbal learning and memory score at 10 years of age (Richardson et al 2002) and with deficits in learning and short- and long-term memory, but only in the verbal domain, in the same cohort at 14 years of age (Willford et al 2004) – in both age groups problems with learning and memory were seen in light drinkers (average of less than 4.2 standard drinks per week).
- There was no association between alcohol consumption of less than ~1 standard drink per day in early pregnancy and intellectual ability, learning and attention at 14 years of age (O'Callaghan et al 2007) – drinking more than five drinks on one or more occasion was associated with decreased cognitive ability with a linear relationship between outcome and frequency of heavy drinking (five or more drinks per occasion).
- Abnormalities of motor coordination demonstrated in children exposed to alcohol prenatally persist into adulthood only in children with other features of FASD (Connor et al 2006) – however, measures used to assess motor coordination may not have been sufficiently sensitive or motor problems may represent developmental delay, which normalises with increasing age.
- Among women of lower socioeconomic status an association between low-to-moderate prenatal alcohol use (less than one drink per day) in the first and second trimesters independently predicted poor teacher rating of overall school performance at 10 years of age (Goldschmidt et al 2004). Deficits in reading comprehension and teachers' rating of poor school performance were significantly associated with second-trimester heavy drinking (four or more drinks per occasion).
- Prenatal alcohol exposure to about 1.4 standard drinks per week independently predicted lower intelligence scores and specific academic deficits, especially in mathematics, in adolescents with a low socioeconomic status who did not have dysmorphic features associated with FAS (Howell et al 2006).

- Children aged 6–7 years with any prenatal alcohol exposure scored higher for externalising (aggression, delinquency) and internalising (anxiety, depression, withdrawal) behaviours after adjusting for confounders (Sood et al 2001) – the odds of delinquent behaviour were significantly higher in children who had been exposed to any level of alcohol compared with non-exposed controls. The adverse effects on behaviour were dose-related and were evident at low levels of exposure, on average 0.6 standard drinks per day.
- There is no difference in any of the global indices of intellectual function or in language development in children exposed to ‘binge-drinking’ (5–6 drinks per occasion) in the first trimester compared with controls; however, exposed children displayed greater social disinhibition than controls (Nulman et al 2004).
- Adolescents aged 14 years, exposed to an average of three or more drinks per week in the first trimester, had a decreased weight, height, head circumference and skin-fold thickness compared with controls (exposed to less than three drinks per week) (Day et al 2002) – there was a dose-response relationship between growth deficit and prenatal alcohol exposure; effects on growth were detectable at average intakes of below one drink per day; and effects were most marked with exposure in the first trimester.

### ***Prenatal alcohol exposure and alcohol disorders in adulthood***

New evidence suggests that prenatal alcohol exposure may increase the risk of alcohol dependence in adolescence (Alati et al 2006) and at 21 years of age (Baer et al 2003). Follow-up of 2,555 youth (35 per cent) from a large Australian birth cohort study (n=7,223) suggests the risk of developing early-onset alcohol abuse disorder (at between 13 and 17 years of age) was higher in those exposed to three or more standard drinks in early pregnancy than in those exposed to less alcohol after adjusting for confounders and excluding those with a family history of alcohol problems (Alati et al 2006). The risk of developing late-onset alcohol abuse disorder (between ages 18 and 21 years) was higher with exposure in early pregnancy. Maternal alcohol consumption in pregnancy did not vary between the sample studied and the group lost to follow-up, suggesting that attrition did not substantially bias the results.

### ***Genetic effects***

Genetics has a role in determining the effects of alcohol on the developing fetus, some genotypes conferring increased risk of harm and others providing protection (Jacobson et al 2006). This factor, if unknown, contributes to the difficulty in estimating risk to the fetus in an individual pregnancy. Metabolic

rates, risk of reacting adversely to alcohol metabolites and the biochemical and inflammatory response to alcohol at a cellular level are all influenced by genetic factors.

## Practical advice

While there is convincing evidence linking chronic or intermittent high level alcohol intake with harms, including adverse pregnancy outcomes and FASD, there remains uncertainty about the potential for harm to the fetus if a woman drinks low levels of alcohol during pregnancy. It is important that all women of child-bearing age are aware, before they consider pregnancy, of both this uncertainty and the potential risks of harm, so they can make informed decisions about drinking in pregnancy. Health professionals should highlight that:

- the risk is higher with high alcohol intake, including episodic intoxication
- the risk appears to be low with low level intake
- it is impossible to determine how maternal and fetal factors will alter risk in the individual.

The high rates of drinking in Australian women, including pregnant women, and the high rates of unplanned pregnancy suggest that, regardless of policy, many fetuses will be inadvertently exposed to alcohol. Assessment of women who have consumed alcohol before knowing that they were pregnant should include appraisal of how much alcohol was consumed and at what stage in the pregnancy. Efforts should be made not to induce unnecessary anxiety for isolated episodes of drinking. Women who drank alcohol before they knew they were pregnant or during pregnancy should be reassured that the risk to the fetus is likely to be low if they had drunk at low risk levels. Women who remain concerned should seek specialist medical advice. Health professionals who are uncertain how to advise pregnant women seeking information concerning the potential for alcohol-related harm should seek expert advice from specialist medical services.



## ADVICE FOR WOMEN WHO ARE PREGNANT OR PLANNING A PREGNANCY

- Not drinking alcohol is the safest option.
- The risk of harm to the fetus is highest when there is high, frequent, maternal alcohol intake.
- The risk of harm to the fetus is likely to be low if a woman has consumed only small amounts of alcohol before she knew she was pregnant or during pregnancy.
- The level of risk to the individual fetus is influenced by maternal and fetal characteristics and is hard to predict.

## Breastfeeding

### Rationale

The rationale for this guideline is based on review of the evidence as summarised on pages 79 to 81.

- Internationally and in Australia it is recommended that infants are exclusively breastfed for the first six months of life and that breastfeeding (in addition to complementary foods) is extended into the second year of life (WHO 2003; NHMRC 2003b).
- Although 75.6 per cent of Australian infants are exclusively breastfed on discharge from hospital following birth, only 12 per cent of infants are exclusively breastfed at 6 months of age and only 19 per cent of infants are receiving any breast milk at 12 months of age (Scott et al 2006). It is therefore important to have a policy that will not discourage women from breastfeeding.
- Alcohol enters the breast milk and may persist in the milk for several hours after alcohol consumption (Table 5) (Ho et al 2001; Giglia & Binns 2006). Alcohol adversely affects lactation, infant behaviour (eg feeding, arousal) and psychomotor development of the breastfed baby (Giglia & Binns 2006).
- Analysis of the 2001 National Health Survey found that, although most breastfeeding women drink at low levels (up to two standard drinks per week), 17 per cent were drinking more than 7 standard drinks per week. This proportion was significantly higher than in the 1995 survey (13 per cent) (Giglia & Binns 2008).

- Qualitative research has shown that breastfeeding mothers are generally unaware of the effects of alcohol on breastfeeding performance and development of the infant (Giglia & Binns 2007).
- Women who consumed alcohol at levels of more than two standard drinks per day were almost twice as likely to discontinue breastfeeding before the infant was 6 months old than women who drank below this level (Giglia et al 2008).

## Summary of the evidence

The effect of alcohol consumption by breastfeeding mothers on milk production (lactogenesis), breast milk and infant blood alcohol concentrations, and the breastfeeding infant, have been described in a thorough systematic review of research from 1990–2005 by Giglia and Binns (2006). The reviewers found limited research on the effect of alcohol on lactation and on breastfed infants, most studies having been conducted in animal models. They note that the lack of high quality evidence limits our ability to give women definitive advice. They also comment that an abstinence message may discourage women from breastfeeding and thus provide practical advice to minimise risk to the infant.

The reviewers found that consumption of two standard drinks or more per day during lactation was associated with:

- decreased lactational performance (in terms of the milk ejection reflex, milk production by the mother and milk consumption by the baby)
- earlier cessation of breastfeeding
- deficits in infant psychomotor development
- disrupted infant sleep-wake behavioural patterns.

Table 5 shows the length of time after drinking alcohol before a zero level of alcohol will be reached in the breast milk of an average woman of a given bodyweight (Ho et al 2001). It should be noted that the times given in the table are estimates of the time it will take for alcohol to disappear from breast milk and that the actual time will vary for each individual woman.

Table 5 Time taken for alcohol to be cleared from breast milk (hours: minutes)

Maternal weight (kg)	Australian standard drinks						
	1	2	3	4	5	6	7
50	1:51	3:43	5:35	7:27	9:18	11:11	13:03
59	1:42	3:26	5:09	6:52	8:36	10:19	12:02
66	1:37	3:15	4:53	6:31	8:10	9:48	11:26
70	1:33	3:07	4:41	6:15	7:50	9:24	10:57

*Notes:* Time is calculated from the beginning of drinking. Assumptions made: alcohol metabolism is constant at 15 mg/dL; height of the women is 162.56 centimetres.

**Example 1:** For a 40.8 kg woman who consumed three standard drinks in 1 hour, it would take 8 hours 30 minutes for there to be no alcohol in her breast milk, but for a 95.3 kg woman drinking the same amount, it would take 5 hours 33 minutes.

**Example 2:** For a 63.5 kg woman drinking four standard drinks starting at 8:00pm, there would be a zero level of alcohol in her breast milk 9 hours 17 minutes later (ie at 5:17am).

*Source:* Giglia & Binns 2006 (adapted from Ho et al 2001).

### Practical advice

Breastfeeding mothers should be advised that not drinking is the safest option and, specifically, to consider not drinking alcohol during the first month after delivery until breastfeeding is well established. For women who choose to drink after this time, advice should be provided on a recommended maximum level of consumption (eg two standard drinks or less in any one day), the length of time that alcohol is excreted in the breast milk and the optimal timing of breastfeeding in relation to intake. The option of expressing prior to consuming alcohol could also be discussed (Giglia & Binns 2006).

## ADVICE FOR BREASTFEEDING MOTHERS

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- Not drinking alcohol is the safest option.
- Women should avoid alcohol in the first month after delivery until breastfeeding is well established.
- After that:
  - alcohol intake should be limited to no more than two standard drinks a day
  - women should avoid drinking immediately before breastfeeding
  - women who wish to drink alcohol could consider expressing milk in advance.

## Appendices

### A1 Further issues to consider

Guidelines 1 and 2 apply to most healthy adults. However, there are some additional factors that should be considered, including:

- situations in which the acute effects of alcohol can endanger the lives of the drinker and others, because of the effects of alcohol on performance in those undertaking or supervising risky activities (see Section A)
- specific groups within the population who are at increased risk if they drink (see Section B)
- specific groups within the population who should seek professional advice about their risks if they drink (See Section C).

This appendix outlines the underlying evidence for these factors. Although the advice given for these issues is brief, they are important to consider.

#### A Situations where drinking increases the immediate risk of harm

Alcohol consumption affects performance and the acute effects of drinking can endanger the lives of the drinker and/or others. Therefore, in some situations, not drinking is the safest option.

**This includes:**

- when taking part in recreational or occupational activities that require a high level of attention, psychomotor skills and concentration (eg driving, water activities, snow sports, flying an aircraft or operating heavy machinery)
- when supervising others who are taking part in such activities
- when supervising children.

## Acute effects of alcohol

As discussed under Guideline 2, there is evidence indicating that drinking decreases cognitive performance, even at low levels of consumption. The acute effects of alcohol increase with the amount consumed, along with the risk of adverse outcomes.

There is also evidence that raised Blood Alcohol Concentration (BAC) affects performance in those undertaking risky and/or complex activities.

- The results of studies examining impairment at low BAC in the range of 0.01 to 0.05 are mixed, depending on how impairment and performance are measured (Howland et al 2000; Fell & Voas 2006; Howland et al 2006; Breitmeier et al 2007). However, while blood alcohol levels up to 0.05 per cent may not significantly impair psychomotor performance, they invoke a level of drowsiness sufficient to impair performance and increase motor vehicle crash risk (Banks et al 2004; Barrett et al 2004; Barrett et al 2005). This is compounded if alcohol is combined with other drugs such as cannabis (Lamers & Ramaekers 2001; Kuypers et al 2006).
- A meta-analysis (Fell & Voas 2006) found that the relative risk of being involved in a fatal crash as a driver is 4–10 times greater for drivers with a BAC between 0.05 and 0.07, compared with drivers with a BAC of zero.
- Maritime cadets showed significant decreases in their ability to concentrate and plan, diminished concentration and accuracy, and an increased risk disposition at a BAC of 0.02 (Ritz-Timme et al 2006).
- An American study found that in 44 per cent of all watercraft-related drownings, the deceased had a positive blood alcohol reading of 0.05 or higher (Browne et al 2003).

Impairment persists even after BAC has returned to normal (Wiese & Shlipak 2000; Kim et al 2003; Barrett et al 2004; Prat et al 2008). A recent study on maritime cadets found that residual effects were found on some complex performance tasks (Rohsenow et al 2006), and a further study found that off-the-job drinking was associated with workers' injury compensation claims (Ragland et al 2002).

The effects of alcohol in risky situations are increased by interactions between alcohol and illicitly used drugs (see Section B) and between alcohol and prescribed and over-the-counter medications (see Section C).

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## Driving and BAC

Australian State and Territory laws allow a BAC of up to 0.05 while driving for full licence holders, zero for learner drivers, and between zero and 0.02 for provisional drivers (depending on the State or Territory). Those who operate commercial aircraft, public or heavy vehicles, commercial vessels, machinery, and mobile plant or farm equipment must observe blood alcohol levels required by their employer's company policy as well as those required by law. For most adults, drinking no more than two standard drinks on an occasion will keep the BAC below 0.05.

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## B People who should be aware that they have an increased risk

Specific population groups can be at increased risk if they drink alcohol.

**These include:**

- young adults aged 18–25 years
- older people aged over 60 years
- people with a family history of alcohol dependence
- people who use drugs illicitly.

## Young adults

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Young adults up to the age of 25 should be aware that they are at particular risk of harm from alcohol consumption, due to a greater risk of accidents and injuries, a lower alcohol tolerance than older adults, and an increased risk of cognitive impairment and alcohol dependence in later life. Young adults are advised to drink within the low-risk guideline levels, and to take steps to minimise their risk of accidents and injury.

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The issues for young adults are similar to those for adolescents (see Guideline 3), in particular:

- like adolescents, young adults continue to be greater risk takers than older adults, but still have poorly developed decision-making skills – factors that are reflected in the high levels of injuries sustained by this age group

- alcohol affects brain development in young people; thus, drinking, particularly heavy drinking, at any time before brain development is complete (which is not until around 25 years of age) may adversely affect later brain function.

In addition, young adults are also the adult age group most likely to take mood-altering drugs (AIHW 2008).

### ***Risk taking and injuries***

The elevated rate of 'high-risk' drinking in the young adult age group is due to young people being more likely to drink a large amount of alcohol in a short space of time, typically on weekends. These drinking patterns are reflected in the types of harm, which typically include drink driving and violence.

Studies show that of the 35 per cent of all injuries presenting to emergency departments that are alcohol-related (Humphrey et al 2003):<sup>11</sup>

- two-thirds are sustained by males under 30 years of age (Borges et al 2004a, Cherpitel et al 2004a, Gmel et al 2006)
- females aged less than 30 years are the group most likely to be the victims of alcohol-related violence (Cherpitel et al 2004a).

Use of alcohol by young adults compounds the likelihood of high-risk behaviours that are common to this age group (Watt et al 2004; Miller et al 2007). Observational studies have shown that alcohol increases the prevalence of unsafe sex behaviours (Coleman & Cater 2005; Lin et al 2005; Abbey et al 2006; Dye & Upchurch 2006). A number of randomised controlled trials have shown that drinking increased participants' likelihood of making risky choices about sexual activity (Maisto et al 2002; Maisto et al 2004; Testa et al 2006), although Leigh et al (2008) showed that young people prepare for safe sex when they know that they will be drinking. Young people who drink alcohol are also at risk of sexual coercion (Abbey et al 2003a; 2003b; Davis et al 2006; Farris et al 2007).

Young people have a significantly lower tolerance to alcohol and relative inexperience at performing certain tasks that require attention and psychomotor coordination, placing them at increased risk of alcohol-related harm. For example, although the risk of motor-vehicle accidents increases proportionate to BAC, younger drivers at a given BAC are at greater risk than older drivers at the same blood alcohol level, due to their relative inexperience and lower alcohol tolerance (Mayhew et al 1986; Zador 1991; Harrison & Fillmore 2005).

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<sup>11</sup> These studies do not report specifically on young adults under 25 years of age.



Different States and Territories in Australia have varying limits for blood alcohol levels permissible in novice drivers. This reflects the complexity of the scientific evidence on drinking and driving and the concern of Governments to minimise risk to young drivers and others using the road. Individuals must be aware of the legal limits allowable for drivers; this information can be found on relevant RTA websites.

## REDUCING THE RISK OF HARM

Planning and taking precautions before drinking that may help young adults to reduce risk include:

- assigning a designated driver
- not drinking if risky activities are planned
- preparing for possible sexual encounters
- selecting less risky venues for drinking.

### ***Cognitive effects***

During adolescence and young adulthood, the human brain is sensitive to injury from alcohol and is less able to respond to physiological cues to stop drinking (Spear 2004). Studies have shown significant and detrimental effects in brain structure when heavy drinking is engaged in during this period. Young adults who drink heavily tend to have smaller prefrontal cortices and white matter, structural abnormalities of white matter and reduced hippocampal volumes (White & Swartzwelder 2004; De Bellis et al 2005). These structural changes lead to a diminished ability to retrieve verbal and non-verbal material and poorer performance in attention-based tests (Brown & Tapert 2004). Cognitive impairment is also common in young adults with alcohol dependence (Kelly & Witkiewicz 2003).

### ***Mental health***

Alcohol use elevates the risk for a number of mental health and social problems in young adults (Brown & Tapert 2004). The existence of psychiatric comorbidities in young people who drink heavily is common, especially for conditions such as depression, anxiety, bipolar disorder, conduct disorder and attention-deficit/hyperactivity disorder (Turner & Gil 2002; Brown & Tapert 2004; Chen & Storr 2006; Deas & Brown 2006; Cargiulo 2007).

Large American cross-sectional studies have shown that college students suffering from social anxiety disorders report frequent heavy and heavy episodic drinking, and drinking with the intention of getting drunk (Geisner et al 2004; Weitzman 2004). Drinking heavily, in combination with depression, is a significant predictor of suicidal ideation, self-harm and suicide in young people (Windle 2004; Miller et al 2007; Sher 2006).

### **Other factors**

- *Cardiovascular protection* – alcohol has no immediate benefit for young people and young adults in protecting against heart disease, as few people show clinical signs of atherosclerosis below 40 years of age. The exception to this is a small number of people with a strong family history of atherosclerotic heart disease in young adulthood (Femia et al 2006). However, it is not yet known whether a regular pattern of drinking is an advantage for any young adult in reducing the 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 in young adulthood.
- *Chronic disease* – young people with conditions such as type 1 diabetes, liver disease and epilepsy require early education and continuing guidance about the effects of alcohol on their disease.
- *Developmental disability* – young adults with developmental disabilities may need special guardianship provisions around the consumption of alcohol, to protect them from harm.

## **Older people**

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Light to moderate alcohol consumption in older adults may lower the risk of several chronic conditions. However, for some older adults, drinking alcohol increases the risk of falls and injuries, as well as some chronic conditions. Older people are advised to consult their health professionals about the most appropriate level of drinking for their health.

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Although drinking volume declines with age, drinking alcohol remains an enjoyable aspect of a healthy lifestyle for many older adults. Population-based studies estimate that approximately 40 per cent of males and 30 per cent of females aged over 60 years drink at a moderate level (Ganry et al 2001; Breslow et al 2003; Breslow & Smothers 2004; Aira et al 2005). The decline in alcohol consumption in the older population is primarily associated with the onset of health problems (Moos et al 2005).

### ***Benefits of light to moderate drinking for older people***

There are a number of studies that suggest that light to moderate alcohol consumption (one to two drinks per day) may convey some health benefits to older adults, including:

- reduced bone loss in males and females (Baron et al 2001; Bakhireva et al 2004; Mukamal et al 2007)
- reduced risk of cardiovascular conditions such as heart failure (Bryson et al 2006), stroke (Mukamal et al 2005) and atherosclerosis (Mukamal et al 2003a; Hougaku et al 2005; Mattace-Raso et al 2005).

Data from prospective cohort studies and cross-sectional surveys also suggest that light to moderate alcohol consumption may protect against cognitive impairment and dementia in older adults (Bond et al 2001; Huang et al 2002; Mukamal et al 2003b; Bond et al 2004; Cassidy et al 2004; den Heijer et al 2004; Hajat et al 2004; Ganguli et al 2005; Deng et al 2006; Reid et al 2006; McDougall et al 2006; McGuire et al 2007).

A prospective observational study found that among women undergoing menopausal transition, moderate alcohol consumption was one of a range of protective factors contributing to women's perceptions of general and emotional well-being, but that the effects of alcohol were different depending on the women's other lifestyle patterns (Alati et al 2007).

### ***Risks of drinking for older people***

For some older people, drinking poses a risk (Fink et al 2002). Older people are more vulnerable to the effects of alcohol due to changes in their body composition, decreased metabolic capacity, the presence of co-morbid conditions and the medications that regulate these conditions (Aira et al 2005). A population-based study of adults aged over 75 years found that among those using alcohol at any level, almost 87 per cent also regularly used medications that had potentially adverse interactions with alcohol (Aira et al 2005).

Alcohol can increase the risk of falls, motor vehicle accidents and suicide in elderly people (Margolis et al 2002; Akechi et al 2006; Sorock et al 2006):

- Alcohol and medication, either alone or in combination, can increase falls-related injury risk (Fletcher & Hirdes 2005).
- A prospective cohort study showed that the risk of falls and fall-related injuries was increased for older adults who drank more than 18 standard drinks per week (Pluijm et al 2006). Another longitudinal study found that consumption of 14 or more drinks per week was associated with an increase in falls risk, although the study collected data once per year, which may not be sufficient (Mukamal et al 2004).

Drinking and driving is also a concern, as older drivers are at greater risk of motor vehicle crashes, particularly at intersections (Grabowski et al 2004; Mayhew et al 2006). A large prospective cohort study of older women found that crash risk was significantly increased for those participants who had fallen in the previous year, while vision status and medical diagnoses were not significantly associated with crash risk (Margolis et al 2002).

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## People with a family history of alcohol dependence

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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. Anyone with first or second-degree relatives with alcohol dependence should consider reducing their drinking to below Guideline 1 and 2 levels and also discuss their alcohol intake with a health professional.

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Family history is a strong predictor of developing an alcohol-use disorder. The children of alcoholic parents are at significantly greater risk of dependence than those of non-alcoholic parents (Haber et al 2005; Capone & Wood 2008).

### *Social and environmental factors*

Social and environmental factors, such as being exposed to a family culture that accepts heavy drinking, may contribute to development of dependence in the children of heavy drinkers (Hingson et al 2003). Social environments and routines, such as tobacco smoking, can contribute to increased drinking frequency and hence lead to dependence (Barrett et al 2006; Gruzca & Bierut 2006).

### *Genetics*

Genetic factors play a very important role in the complex interaction between an individual and his or her response to alcohol. The influence of genetic variability on risk of dependence and risk of tissue injury from alcohol intake is only partly understood at present. Some individuals inherit variations of the genes that encode the enzymes that regulate alcohol metabolism (alcohol dehydrogenase [ADH], aldehyde dehydrogenase [ALDH] and microsomal P4502E1 [CYP2E1]) (Gemma et al 2006). This may influence their susceptibility to alcohol dependence (Ilveskoski et al 2001; Hasin et al 2002, Petrakis et al 2004) and alcohol-related liver disease.

Other individuals inherit variations in the genes that encode the hormones of the hypothalamic–pituitary axis. These hormones are involved in regulating reward systems, which are heavily implicated in alcohol dependence (Dai et al 2002ab; Dai et al 2005). Candidate genes for alcohol preference have

been studied in animal models and human studies suggest familial risk of alcohol dependence can be influenced by genes encoding DRD2 and ANKK1 (Bauer et al 2007; Carr et al 2007; Dick et al 2007). While the studies are of great interest it is not possible at this stage to use genetic testing as a means of identifying individuals at increased risk of alcohol dependence or alcohol-related tissue damage with the degree of accuracy that clinicians or individuals desire. The contribution of any one gene to these risks appears small in real life situations.

### ***People who use drugs illicitly***

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There is a range of documented adverse outcomes from illicit use of drugs, and consuming alcohol together with illicit use of drugs can have dangerous or lethal consequences.

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Drugs such as cannabis, methamphetamines, ecstasy, cocaine and heroin are increasingly used with alcohol, placing users at greater risk of harm. Many studies have reported that any level of alcohol consumption is a significant predictor of non-fatal and fatal drug overdose (eg Kaye & Darke 2004; Coffin et al 2007). However, people who use drugs illicitly are typically unaware of the potentiating effects of alcohol and illicit drugs (Dietze et al 2005; Neira-León et al 2006). There are no recommended safe levels of illicit drug use when combined with alcohol as dosage levels for illicit drugs are unpredictable.

- As alcohol is a central nervous system depressant, mixing central nervous system depressant drugs (eg heroin) with alcohol compounds the risk of fatal overdose from respiratory depression.
- Combining alcohol with central nervous system stimulants such as cocaine, methamphetamines and ecstasy is also risky and any perceived benefit is overridden by increased risks of dehydration, cardiac arrhythmias and fits.

### ***Cannabis***

The combined use of alcohol and cannabis is dangerous when driving or engaging in other activities requiring motor skill and judgment. Like alcohol, cannabis has been shown to impair psychomotor performance and perception, and several studies have shown that the combined use of alcohol and cannabis is associated with a higher risk of motor vehicle crashes (O’Kane et al 2002, Ramaekers et al 2006, Appenzeller et al 2005).

In a driving simulation study, the National Highway Traffic Safety Administration (2000) found that:

- the effects of 0.04g/dL of alcohol and moderate doses of marijuana are greater in combination than for either drug alone

- aspects of driving performance, especially reaction time, visual search frequency and ability to perceive or respond to changes in relative velocity of other vehicles were negatively affected by the combination of alcohol and cannabis.

### ***Methamphetamines and ecstasy***

Combining methylenedioxyamphetamine (MDMA or ecstasy) and alcohol is common, especially in young adults. Several studies have shown that use in combination does not mitigate many effects of alcohol, so that driving or taking part in other risky activities is especially dangerous (Kuypers et al 2006; Ramaekers & Kuypers 2006; Ramaekers et al 2006). Driving simulation studies have found that:

- MDMA moderated the impairing effects of low-dose alcohol (equivalent to BAC 0.06 mg/mL) on road-tracking performance, but could not overcome alcohol-related impairment on other aspects of driving (Kuypers et al 2006).
- The stimulant effects of MDMA were never sufficient to overcome alcohol-induced impairment of impulse control or risk-taking behaviour (Ramaecker & Kuypers 2006).

### ***Cocaine***

A recent systematic literature review found that combining any dose of alcohol and cocaine induces greater-than-additive effects on heart rate and 30 per cent greater blood cocaine concentrations (Pennings et al 2002). The systematic review included studies that used retrospective data to suggest that combining alcohol and cocaine leads to formation of cocaethylene, a compound that may potentiate greater cardiotoxic effects than the use of either drug alone (Pennings et al 2002). An Australian study found that the combination of alcohol and cocaine is more common in users who are socially integrated, rather than users at societal margins (Shearer et al 2007).

### ***Heroin***

The synergistic effects of alcohol and heroin also increase the risk of non-fatal and fatal overdose. Heroin and alcohol are both central nervous system depressants that, at high doses, suppress breathing through their action on the neurotransmitters glutamate and gamma-amino butyric acid (GABA). Alcohol decreases the excitatory effect of glutamate, while heroin increases the inhibitory effect of GABA, resulting in a reduced respiration rate. For these reasons, the concomitant presence of alcohol and heroin has been described in many forensic examinations of heroin overdose victims (Coffin et al 2007, Darke et al 2007).

### *Illicit use of legal drugs*

Taking prescription medications that have not been prescribed for the user brings additional risk when these medications are combined with alcohol (see Section C).

## C People who should seek professional advice about drinking

A range of people may need to seek professional advice about drinking, because of the possibility of interactions and harmful effects.

**This includes:**

- anyone taking medication, either over-the-counter or prescription
- people with alcohol-related or other physical conditions that can be made worse or affected by alcohol
- people with mental health conditions.

### People taking medications

Alcohol may interact with prescribed and over-the-counter medications and thus increase their potential adverse effects or reduce their effectiveness. People taking medications (including herbal preparations) should check with their doctor or pharmacist about possible harmful interactions between their medications and alcohol and read any information on alcohol interactions included in the packaging. Temporary or permanent abstinence from alcohol may be necessary, particularly for people taking multiple medications.

Alcohol interacts with many other drugs, including prescription and over-the-counter medications and herbal preparations (Weathermon & Crabb 1999; Izzo & Ernst 2001; Koski et al 2005; Pringle et al 2005). Alcohol can exert direct effects on the absorption of medications, change the way medications are metabolised, or interfere with the effect of the medication at its site of action (Weathermon & Crabb 1999).

The effects of combining alcohol and medication depend on the type and dosage of medication, the volume of alcohol consumed, and also on personal factors, such as genetics, gender and comorbid health conditions (Weathermon & Crabb 1999). Commonly prescribed classes of medications,

such as benzodiazepines, barbiturates, opiates, analgesics, antidepressants, antibiotics, antihistamines, anti-inflammatories and hypoglycaemic agents have known interactions with alcohol (Brunton et al 2006). Interactions between alcohol and medications have serious implications for people undertaking activities requiring concentration, such as driving a motor vehicle or operating heavy machinery, as discussed in Section A.

Some herbal medicines and prescription drugs, and other products such as mouthwash, contain alcohol.

There is a potential for severe, rarely fatal drug interactions with alcohol secondary to altered drug metabolism as is seen in the case of paracetamol and alcohol in chronic alcoholics (Riordan & Williams 2002; Krahenbuhl et al 2007).

## **People with physical health problems that are made worse or affected by alcohol**

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Drinking leads to poorer outcomes for people with certain diseases and conditions, including alcohol-related diseases. Anyone having treatment for any of these conditions, or any other problem that might be made worse or affected by alcohol, should discuss their alcohol intake with a health professional. In many instances, temporary or permanent abstinence may be necessary.

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### ***Alcohol-related diseases***

For people who have serious diseases directly related to alcohol consumption, such as cirrhosis of the liver, alcoholic pancreatitis, alcohol-related brain damage and alcohol dependence, any further drinking may aggravate the condition, causing immediate problems or worsening of the prognosis in the longer term. In many instances, temporary or permanent abstinence may be necessary. For some people with these conditions, however, a planned program of limited drinking under the supervision of a health professional may be an appropriate option.

### ***Diabetes***

People with diabetes may need to take special precautions with drinking and should discuss alcohol use with a health professional.

Alcohol interferes with the action of insulin, insulin secretagogues and glucagon, thereby increasing the risk of hypoglycaemia in people with type 1 or 2 diabetes who take these medications (ADA 2007; CMP Medica 2007). As alcohol and hypoglycaemia have independent but additive effects on



cognitive function, it is recommended that people with diabetes abstain from alcohol if they plan to drive (Cheyne et al 2004).

Alcohol worsens medical conditions associated with diabetes, such as liver disease and advanced neuropathy (ADA 2007; Tolman et al 2007).

There is limited research into alcohol and self-management behaviours but one study (Ahmed et al 2006) found an inverse association between drinking and diabetes self-management behaviours, even at a low intake of one drink per day.

Caloric restriction, which may involve reducing alcohol intake, can be important in reducing overweight as part of diabetes management (ADA 2007).

### ***Other conditions affected by alcohol***

People with other conditions not related to alcohol may also need to seek medical advice about drinking, as alcohol may worsen the condition or interfere with treatment.

- *Infectious diseases* – heavy alcohol consumption may impair immune function, leading to an increased risk of infections including skin and respiratory infections. Those who contract infections tend to have poorer outcomes (Sulis 2003).
- *Liver diseases of any form* – alcohol intake can increase the severity of hepatitis C, non-alcoholic fatty liver and other drug-induced liver injury. Reducing alcohol intake can restrict the severity of liver injury in those with other liver disorders (Ostapowicz et al 1998, 2001; Marcellin et al 2008).
- *Sleep disorders* – alcohol causes interruptions to normal sleep patterns, in particular the later, heavier part of the sleep cycle (Castaneda et al 1998). While alcohol may induce sleep in the short term, it leads to increased arousal and wakefulness several hours after consumption (Castaneda et al 1998; Peppard et al 2007). Sleep disruption and chronic sleep deprivation can increase the risk of injury, disrupt cognitive processes and trigger a variety of mental health conditions (Castaneda et al 1998; Williamson and Feyer 2000; Drummond et al 2006).
- *Sexual dysfunction* – alcohol use can cause or exacerbate a range of sexual problems in males and females. At low levels, alcohol can reduce inhibition and increase sexual desire (Cheng et al 2007); however, beyond the level of the guidelines, the depressive effects of alcohol are apparent (Bacon et al 2003; Arackal & Benegal 2007). In females who consume alcohol heavily, the likelihood of heavy or irregular menstrual periods, spontaneous abortion and infertility is greater when alcohol is consumed above the guideline levels (Bradley et al 1998).

## People with mental health conditions

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Drinking can lead to poorer outcomes for people who have a mental health condition, whether it is a high-prevalence condition such as depression or a low-prevalence condition such as schizophrenia. Anyone at risk of, or under treatment for, a mental health condition should discuss their alcohol intake with a health professional. Recommendations about drinking will vary depending on the presenting mental health condition and medication regimes. In many instances, temporary or permanent abstinence may be necessary. Carers can encourage people with a mental health condition to stay within guideline levels, or to abstain if necessary.

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Alcohol plays a complex role in the development and progression of mental health conditions.

- People with, or at risk of, a mental health condition are more likely to use alcohol than those without (Kessler et al 1997).
- Drinking can promote the development of mental health conditions in at risk people (eg those prone to depression and/or anxiety) (Degenhardt et al 2001; Cornelius et al 2003; Nunes & Levin 2004; Clark et al 2003; Currie et al 2005; Haynes et al 2005).
- Alcohol use is associated with a high prevalence of several mental health conditions, ranging from common conditions such as social phobias and anxiety disorders to less common conditions such as uni- and bipolar disorder and schizophrenia (Kranzler et al 1996; Merikangas et al 1996; Hodgins et al 1999; Burns & Teesson 2002; Schuckit 2006).
- There is a high degree of comorbidity between individuals with anxiety, depressive and schizophrenic diagnoses (Baker & Velleman 2007).

### *Social phobias and anxiety disorders*

The literature surrounding alcohol use and anxiety disorders is large and complex; however, it has been shown repeatedly that individuals with social phobias are much more comfortable in social situations if they have used alcohol (Abrams et al 2002; Lehman et al 2002; Lewis & Vogeltanz-Holm 2002; Thomas et al 2003). While a small amount of alcohol may induce short-term stress relief, it does not address the root cause. There is considerable evidence that repeated use of alcohol to dampen stress may increase anxiety levels and lead to a degree of dependence on alcohol, especially in vulnerable people (Kushner et al 2000; Carrigan & Randall 2003; Thomas et al 2003).

In the hours after drinking, increased anxiety and depression are common (Kushner et al 2000). People who depend upon alcohol are more likely to have mood and anxiety disorders, and vice versa, and for these people the depressive effects of alcohol are of particular consequence (Hakko et al 2005; Goldstein et al 2006).

### ***Depression***

Although most of the epidemiological literature suggests an association between alcohol use and depression, the exact nature of the relationship is unclear. This may in part be due to the manner in which both consumption and depression are measured (Graham et al 2007). In a longitudinal study, Haynes et al (2005) found that excessive alcohol consumption was not associated with the onset of anxiety and depression, but that abstinence was associated with lower depressive risks. A meta-analysis found that a higher proportion of people diagnosed with alcohol dependence also suffers comorbid mental health conditions than people who suffer a mental health condition and are also alcohol-dependent (Jane-Lopis & Matytsina 2006). In a population-based survey, Graham et al (2007) found no data to support the notion that light to moderate drinking (defined by low frequency or low volume) protects against major depression.

A meta-analysis of 35 epidemiological studies showed that alcohol problems are more common in people who are depressed than in the general population (Sullivan et al 2005). The meta-analysis reported a median prevalence of current or lifetime alcohol problems in depressed patients of 16 per cent and 30 per cent, respectively, compared with 7 per cent and 16–24 per cent current or lifetime alcohol problems in the general population (Sullivan et al 2005). Furthermore, the study found that heavy alcohol use was associated with worse outcomes in terms of depression course, self-harm and suicide risk, social functioning and health care use.

### ***Bipolar disorder***

Alcohol use is also high in patients with bipolar disorder (Goldstein et al 2006). Patients who suffer from bipolar disorder and alcohol dependence have significantly reduced quality of life compared with patients with bipolar disorder only (Singh et al 2005). A study in a psychiatric population by Goldstein et al (2006) found that even if alcohol consumption volume was low (measured using definitions of the Canadian Alcohol Guidelines and Khavari Alcohol Test), it remained associated with measures of illness severity in both male and female patients. It is thought that the adverse effects of alcohol on bipolar disorder may occur over a range of consumptions, rather than being confined to alcoholics or heavy drinkers (Goldstein et al 2006).

## **Schizophrenia**

There is a well-established comorbidity between schizophrenia and heavy alcohol use, although the certainty of the association between alcohol use as an independent risk factor for schizophrenia is not clearly defined. Studies of patients with schizophrenia have reported a wide range of prevalences for heavy drinking, with younger males more likely to suffer from alcohol dependence (Cantor-Graae et al 2001). A recent Swedish study found that heavy alcohol use was the most common type of substance abuse among people with schizophrenia (Cantor-Graae et al 2001). It is thought that heavy alcohol use, especially early in the illness, determines the earlier onset of schizophrenia and may increase the severity of common psychotic symptoms, such as hallucination and unusual content of thought (Mauri et al 2006; Mohamed et al 2006).

## **Other problems**

- *Alcohol dependence* – People who use alcohol to cope with their mental health conditions have a tendency to become dependent (Kushner et al 2000; Carrigan & Randall 2003; Thomas et al 2003). 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 (Lynskey 1998; Kushner et al 2000).
- *Interaction with medications* – in addition to all its other effects, alcohol – even at low levels (one or two drinks a day) – can interact adversely with most of the medications commonly prescribed for treating mental health conditions, including antidepressants, benzodiazepines and muscle relaxants (Castaneda et al 1998; Weathermon & Crabb 1999; Koski et al 2005).

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Recommendations about drinking for people with mental health conditions will vary depending on the presenting mental health condition and medication regimes. For those with an active mental health condition, drinking alcohol may have a negative impact on mental health and lower the efficacy of antidepressant medication. In some instances, for stable patients, the recommendations for the general population may be applicable, while for others a more cautious approach will be required.

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## A2 Committee membership and terms of reference

### Membership, NHMRC Review of the Australian Alcohol Guidelines Working Committee

Professor Jon Currie (Chair)	Department of Addiction Medicine St Vincent's Hospital, Melbourne, VIC NHMRC National Health Committee member
Professor Steve Allsop	National Drug Research Institute Curtin University of Technology, WA
Professor Robert Batey	Centre for Drug and Alcohol NSW Health Department
Dr Ngiare Brown	Menzies School of Medical Research Charles Darwin University, NT (until August 2007)
Mr Bruce Clark	Consumer representative
Professor Charlotte de Crespigny	School of Nursing and Midwifery Flinders University, SA
Professor Elizabeth Elliott	University of Sydney Children's Hospital, Westmead, NSW Australian Paediatric Surveillance Unit
Professor Ann Roche	National Centre for Education on Training and Addiction Flinders University, SA
Professor Robin Room	School of Population Health, University of Melbourne Director, AER Centre for Alcohol Policy Research, Turning Point Alcohol and Drug Centre, VIC
Associate Professor Ted Wilkes	National Drug Research Institute Curtin University of Technology, WA (from December 2007)
<b>Corresponding members</b>	
Dr Michael Bolton	Damascus Health Service, QLD
Professor Margaret Hamilton	Multiple and Complex Needs Panel, VIC (until August 2007)

<b>Observers</b>	
Ms Jennie Shortt Ms Kellie Fixter Ms Sarah Murray	Australian Government Department of Health and Ageing
Dr Utz Mueller	Food Standards Australia New Zealand
<b>Technical writers</b>	
Dr Janet Salisbury	Biotext Pty Ltd (Phase 1: Evidence synthesis and consultation draft)
Ms Elizabeth Hall and Ms Jenny Ramson	Ampersand Health Science Writing (Phase 2: Final guidelines)

The work on this project was managed by the Evidence Translation Section, Health Evidence and Advice Branch, National Health and Medical Research Council.

### Terms of reference

1. The Review of the Australian Alcohol Guidelines Working Committee (the Working Committee) will oversee and provide expertise in the review of the Australian Alcohol Guidelines. The review should take into account, but not be limited to, the following:
  - the *Australian Alcohol Guidelines: Health Risks and Benefits* (2001)
  - the best available current scientific evidence
  - comments provided by the broader community through public consultation
  - the needs of health service providers
  - any other relevant matter.
2. The Working Committee will provide regular reports on the progress of guideline development.
3. The Working Committee will provide the NHMRC CEO with a draft report for the CEO to seek advice from Council.
4. The NHMRC will engage a consultant to assist the Working Committee in this task.

## A3 Process report

In October 2001, NHMRC issued the *Australian Alcohol Guidelines: Health Risks and Benefits*. These guidelines were developed by the NHMRC in collaboration with the Population Health Division (PHD) of the Australian Government Department of Health and Ageing (DoHA), with funding from DoHA.

It is NHMRC policy that all guidelines are reviewed every five years.

In 2006–07, again in collaboration with PHD of DoHA, the former NHMRC Health Advisory Committee commenced an update of the guidelines. The guidelines were finalised under the auspices of the National Health Committee (NHC), a principal committee of NHMRC.

The aim of the updated *Australian Guidelines to Reduce Health Risks from Drinking Alcohol* (the guidelines) is 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 guidelines are also intended to provide an evidence base to 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.

### Working Committee

An expert Working Committee, chaired by Professor Jon Currie, was appointed to guide the redevelopment of the guidelines (see Appendix A2). Representatives from DoHA and from Food Standards Australia New Zealand attended Working Committee meetings as observers.

### Literature review

In preparation for the update of the guidelines, the NHMRC commissioned a systematic literature review, which analysed and synthesised the best available current evidence on a range of topics to be covered by the guidelines. The reviewers searched EMBASE.com (a composite database including MEDLINE and EMBASE) and the Cochrane Library from 2000 to early 2007 for all papers relating to alcohol, alcohol use, alcohol consumption, drinking, intoxication, problem drinking and related terms. Only papers describing human studies were included. This provided a potential 223,153 articles from EMBASE.com, 74 Cochrane reviews, 208 other articles from the Cochrane Library and 6,637 clinical trials from the Cochrane Central Register of clinical trials. These articles were then used as the basis for subject-specific searches.

Subject-specific searches were also carried out in the Project Cork database and other databases relevant to specific topics. Additional references included references in the bibliographies of publications identified in the search and references supplied by the Working Committee and based on their expertise in specific areas.

The subject areas addressed by the reviewers were:

- adolescents and young adults
- the elderly
- people with a family history of alcohol abuse
- differences between men and women
- Aboriginal and Torres Strait Islander people
- occupational groups
- women who are pregnant or breastfeeding
- alcohol dependence
- abstinence.

The literature review also looked at the health outcomes associated with alcohol consumption.

For each subject area, abstracts of all the identified articles were retrieved and reviewed for relevance. At this stage, reviewers excluded papers published before 2001, as well as off-topic papers, duplicates and papers that were not about human research or were not research studies (eg letters, editorials).

The remaining papers were grouped by study type (systematic reviews, randomised controlled trials, prospective cohort studies and other observational studies) and the relevant data were extracted by two reviewers and tabulated. A brief overview of each subject area was prepared.

The Working Committee and technical writers subsequently further analysed selected studies, as required.



Table 6 Major systematic reviews and meta-analyses informing these guidelines

Review	Subject area	Study details
<b>Guideline 1</b>		
Corrao et al 1999	12 alcohol-related neoplasm and non-neoplastic diseases/injuries	Literature searched from 1996 to 1998  Meta-regression models used to evaluate linear and non-linear effects of alcohol intake on relative risk
Corrao et al 2000	Coronary heart disease	Literature searched from 1966 to 1998  51 studies included in meta-analysis
Corrao et al 2004	15 major alcohol-related neoplasms and non-neoplastic diseases	Literature searched from 1966 to 1998  240 studies included in the analysis  Fixed and random effects models used to evaluate linear and non-linear effect of alcohol intake
Di Castelnuovo et al 2002	Vascular risk	Literature search of published studies to 2001  23 studies reporting data on wine and 22 studies reporting data on beer included in analysis  General variance-based methods used to evaluate effects of beer and wine consumption on vascular risk
Di Castelnuovo et al 2006	Alcohol dosing and mortality	Literature searched to December 2005  Meta-analysis included 34 prospective studies
Fell & Voas 2006	BAC driving limits	Review of 14 studies

Review	Subject area	Study details
Fillmore et al 2006	Coronary heart disease mortality	Literature searched from 1950s to mid 2004  54 prospective mortality studies included  Modelling to accommodate for misclassification bias for abstainer category (ie distinction made between never-drinkers and former drinkers)
Gmel et al 2003	All-cause mortality	Literature searched until 2000  Precision-weighted pooling of relative risks; precision-weighted hierarchical analysis
Reynolds et al 2003	Stroke	Literature searched from 1966 to 2002  35 observational studies included
White et al 2007	All-cause mortality	Analysis of published systematic reviews (Corrao et al 1999, 2000) and mortality data for England and Wales 1997
<b>Guideline 4</b>		
Giglia & Binns 2006	Lactation	Literature searched from 1990 to 2005  53 studies reviewed
Henderson et al 2007a	Pregnancy outcome	Literature searched from 1970 to 2005  Review included 46 studies
Henderson et al 2007b	Prenatal "binge" drinking	Literature searched from 1970 to 2005  Review included 14 observational studies
Makarechian et al 1998	Spontaneous abortion, stillbirth and premature birth	Literature searched from 1966 to 1993  Meta-analysis included eight case-control or cohort studies

Review	Subject area	Study details
Polygenis et al 1998	Fetal malformations	Literature searched from 1966 to 1998  Of 24 studies that met inclusion criteria, only seven had extractable data
<b>Review</b>		
Testa et al 2003	Infant mental development	Literature searched from 1973 to 2000  10 studies included in meta-analysis
<b>Further issues to consider</b>		
Cheng et al 2007	Erectile dysfunction	Meta-analysis of 11 cross-sectional studies and two cohort studies
Izzo & Ernst 2001	Interactions between herbal medications and prescribed drugs	Literature searched from 1966 to 2000  41 case reports or case series and 17 clinical trials identified
Nunes & Levin 2004	Depression	Literature searched from 1970 to 2003  Meta-analysis included 14 studies
Pennings et al 2002	Cocaine	Literature searched from 1963 to 2001  Analysis included 14 prospective and retrospective studies
Sullivan et al 2005	Depression	Literature searched from 1980 to 2002  Analysis included 35 studies

## Modelling studies

### *Lifetime risk*

In developing the guidelines, the NHMRC considered modelling which had been commissioned by DoHA on lifetime risk-modelling studies of injuries, and of alcohol-related diseases. The aim was to estimate the risk of death from injury and alcohol-related diseases for Australians during their lifetime. Details of these models are shown in Appendix A5.

### *Harm scores*

To help with differentiating recommendations for particular age groups, the NHMRC considered a further modelled assessment of the amount of harm caused by a given level of drinking, based on the *2004 National Drug Strategy Household Survey* (AIHW 2005). The harm score was created from the series of questions on self-reported problems from drinking.

## Public consultation

Public consultation on the draft guidelines was undertaken from 13 October 2007 to 11 December 2007. The public consultation was advertised in the major Australian newspapers and on the NHMRC website. Invitations were also forwarded to key stakeholders and those with a known interest in alcohol. One hundred and sixty-two submissions were received from a variety of stakeholders including individuals, government agencies, health organisations, health professionals and the alcohol industry. The Working Committee met on 14 December 2007 and 15 February 2008 to consider the submissions. All submissions received during the consultation were taken into consideration. Several submissions raised concerns about the evidence underpinning the draft guidelines.

Specific concerns were raised about:

- men and women having the same low-risk drinking guideline
- the concept of lifetime risk, combining immediate and long-term harm and the calculations used to determine risk
- the focus on low-risk drinking and the absence of differentiation of risk at higher levels of drinking
- the inclusion of the condition 'If drinking does occur...' in the guideline relating to young people
- the stringency of the 'no drinking is the safest option' guideline for pregnant and breastfeeding women.

On the basis of this feedback, the Working Committee undertook to review and clarify the evidence base, and to provide further clarification of the risk analysis. The Working Committee considered that the evidence base did not support a more stringent form of Guideline 3, recommending not drinking at all for young people aged 15–17 years.

A list of the submissions is at Appendix A4.

## Peer review

Throughout the public consultation process, the NHMRC received a number of comments regarding the validity of some of the journal articles referenced in the draft guidelines. All parties were provided with copies of particular references when requested. However, and in response to a number of public submissions and to ensure that all current and best available literature and evidence were used, the draft guidelines underwent a peer review by four independent international experts in the field of alcohol and two Australian health economists. Peer review reports were provided by:

- Ms Margaret Geddes Chartres, New Zealand
- Mr David B. Cooper, England
- Professor Colin Drummond, England
- Dr Tom Greenfield, USA
- Professor Philip Clarke, health economist, Australia
- Professor Jim Butler, health economist, Australia.

The peer review reports were positive overall and remarked on the strength and thoroughness of the document. The guidelines were identified as a well constructed, broad and comprehensive document that would be a helpful contribution for those wishing to brief themselves on the current evidence or develop guidelines in specific areas. The guidelines were described as well written in an accessible style for the intelligent layman. The reports acknowledged and praised the use of the most up-to-date modelling technique and evidence base and an excellent literature search.

The peer reviews also acknowledged that the guidelines were in advance of most other countries in terms of their scope and consideration and handling of the complexities. Particularly welcome was the emphasis on risk, and the clear guidance that low level drinking is not safe, but rather lower risk than heavier drinking.

Some of the suggestions in the peer review reports included a need to clarify some definition and adjust referencing. More substantive comments, particularly from the two health economists, required an explanation of why the ratio for risk of injury or disease was presented as 1 in 100 persons. This issue was clarified in the final guidelines. Other issues such as some minor wording amendments for Guideline 3, further information on the costs to the health care sector, and alcohol-free days were also considered.

The draft guidelines were revised in accordance with relevant recommendations made in the peer reviewers' reports.

### **Additional peer review by Australian expert panel**

An additional peer review was conducted by a panel of Australian epidemiological, research and guideline experts. The review panel overall reported positively on the content and evidence of the guidelines. However, some suggestions for amendment were made including a need for clearer explanation of the modelling, the calculations and decisions for the guideline recommendations. The panel members also commented that more explicit definitions of some terms were needed. The draft guidelines were revised in accordance with the suggested amendments.

Following the completion of the amendments, the review panel and the Working Committee were provided with the revised draft guidelines for final comments. Minor changes were suggested and made accordingly.

### **Independent review**

The draft guidelines underwent an independent review against the NHMRC key criteria for assessing public health guidelines. All key criteria were satisfied in this review.

### **Endorsement**

The guidelines were considered by the National Health Committee and subsequently provided to the Council of NHMRC for consideration. The Council of NHMRC considered the guidelines on 6 February 2009 and again out of session between 19 and 24 February 2009 and provided its advice to the Chief Executive Officer (CEO). The CEO was pleased to accept Council's advice and agreed to issue the guidelines under Section 7(1a) of the *National Health and Medical Research Council Act 1992*.

## Dissemination and implementation

Dissemination involves making guidelines accessible, advertising and distributing them widely. Dissemination of the guidelines included distribution to:

- State and Territory health departments
- Individuals and organisations who participated in the consultation process.

The guidelines are available in PDF and Word Formats from the NHMRC website at [www.nhmrc.gov.au/publications/index.htm](http://www.nhmrc.gov.au/publications/index.htm). In addition, any interested organisations or individuals will be able to order copies from:

### National Mailing and Marketing

Phone: (02) 6269 1000

PO Box 7077

Canberra Mailing Centre

ACT 2610

The guidelines will be provided to the Department of Health and Ageing for their consideration.

## Review

In line with NHMRC policy, these guidelines will be reviewed within five years of publication.

## A4 Submissions to the public consultation

Name	Organisation
	Australian Health Ethics Committee
	Dietitians Association of Australia, ACT
	Family Drug Help at SHARC, Victoria
	National Drug Research Institute, Western Australia
	Alcohol in Pregnancy Research Team Telethon Institute for Child Health Research, Western Australia
	Victorian Alcohol and Drug Association
Ms Nola Adams	State Corresponding Secretary Women's Christian Temperance Union Western Australia
Mr Roger Adamson	Private Submission
Mr Birsen Aktuna	Private Submission
Mr Ian Albrey	Private Submission
Ms Fay Alford	President Foster Care Association of Western Australia
Professor Robert Ali	Chair Royal Australasian College of Physicians Australasian Chapter of Addiction Medicine
Ms Lisa Amir	Mother and Child researcher La Trobe University
Ms Huguette Anglem	Private Submission
Ms Himani Arora	Private Submission
Dr David Atkinson	Medical Educator and Medical Coordinator KAMSC and Rural Clinical School of WA
Ms Clare Avoledo	Private Submission
Mr David Axworthy	Executive Director School Support Programs Department of Education and Training Government of Western Australia
Mr Ron Bagatol	Pharmacist Member, TGA Advisory Committee on Medicines and Pregnancy
Ms Kim Balkovic	Private Submission



Name	Organisation
Ms Margaret Banks	Chief Executive Department of Employment, Education and Training, NT
Mr Stuart Baulk	Affiliate Research Fellow Centre for Sleep Research Adelaide Institute for Sleep Health, SA
Ms Josephine Baxter	Executive Officer Drug Free Australia South Australia
Mrs Baynton	Private Submission
Ms Nerryn Bennett	Private Submission
Ms Anne Bergen	Secretary National Women's Christian Temperance Union of Australia
Mr Colin Berry	Private Submission
Ms Sonia Berton	Chief Executive Officer Alcohol Related Brain Injuries Australian Services
Dr James Bishop	Chief Cancer Officer and Chief Executive Officer NSW Cancer Institute
Ms Anna Blaszczak	Private Submission
Mr Peter Boland	Private Submission
Ms Katie Booth	Dietician/Nutritionist Diabetes Australia
Ms Kirsten Braun	Health Information Officer Women's Health Queensland Wide
Mr Gordon Broderick	Executive Director Distilled Spirits Industry Council of Australia Victoria
Mr Graham and Ms Leela Brown	Private Submission
Ms Rosemary Bryant Ms Elizabeth Foley	Executive Director Director; Policy Royal College of Nursing, Australia
Mr Nicholas Buff	Private Submission
Ms Mary Cabrall	Private Submission
Mr Donald Cameron	State Director People Against Drink Driving, NSW
Ms Kate Carnell	Chief Executive Officer Australian General Practice Network
Ms Heather Ceravolo	Ceravolo and Red Earth Wines, South Australia

Name	Organisation
Ms Ellen Chandler	Director WCTU Drug Free Lifestyles Victoria
Ms Kathy Chapman	National Program Manager Cancer Council, NSW
Mr Suman Chaturvedi	Private Submission
Ms Peta Chubb	Convenor; Public Health Standing Committee Home Economics Institute of Australia ACT
Mr Mike Coleman	Drug and Alcohol Services Salvation Army
Mr Peter Corden	Private Submission
Mr David Crosbie	Chief Executive Officer Mental Health Council of Australia
Ms Ruth Cross	Private Submission
Mr Elwin Currow	Private Submission
Ms Anne Davis	School of Nursing University of Western Sydney NSW
Mr Joseph Docherty	Private Submission
Mr Clyde Dominish	Chairman Coalition on Alcohol and Drug Education Inc NSW
Mr Keith and Ms Tracy Duley	Private Submission
Ms Neroli Endacott	Private Submission
Mr Paul Evans	Director; Government, Regulation and Community Affairs Lion Nathan Pty Ltd
Ms Fiona Feary	Private Submission
Mr Kimble Fillingham	General Manager TAFE Business TAFE NSW
Mr Brian Flanagan	Strategic Communications and Policy Officer Alcohol and Other Drugs Council Australia
Ms Wendy Flesser	Private Submission
Mr Hugh and Ms Janie Frith	Private Submission
Mr Amara Ganesh	Private Submission

Name	Organisation
Ms Roslyn Giglia	Associate Lecturer in Epidemiology School of Public Health Curtin University of Technology, Western Australia
Ms Sukalpa Goldflam	Private Submission
Dr Karleen Gribble	Adjunct Research Fellow School of Nursing University of Western Sydney
Mr Paul Grogan	Private Submission
Mr Peter Grose	Private Submission
Dr Nitin Gupte	Private Submission
Ms Trish Guy	Acting Nutrition Service Manager Sanitarium Health Food Company
Dr Paul Haber Dr Katherine Congrave Dr Nicholas Lintzeris Dr Carolyn Day	Drug Health Services, Royal Prince Alfred Hospital NSW
Ms Irena Harper	Private Submission
Ms Cheryl Harrison	Private Submission
Mr Tony Harrison	Assistant Commissioner South Australian Police
Henderson et al	Journal article
Mr David Hillman	President Australian Sleep Association
Mr William Hodge	Manager Community Health City of Stonnington, Victoria
Mr Brett Johnson	Private Submission
Ms Pauline Jollow	Private Submission
Professor Ross Kalucy	Director Emergency Mental Health Southern Adelaide Health Service
Associate Professor Maarten Kamp	Chair Medical Education and Scientific Council Diabetes Australia
Dr David Kault	Private Submission
Mr David Kavanagh	Private Submission
Mr Mark Kelly	President Victorian Association of Drink and Drug Driver Services

Name	Organisation
Ms Susan Killion	Senior Executive Australian Institute of Health and Welfare
Mr Gary Kirby	Director Prevention and Workforce Development Drug and Alcohol Office Government of Western Australia
Ms Melanie Knight	Private Submission
Mr Chris Kyriacou	Trustee Life Eternal Properties Australia Pty Ltd
Ms Kelly Langdon	Private Submission
Mr Hugh Lantzke	Private Submission
Mr Chris Lee	Sahaja Yoga Meditation
Dr Jim Lemon	National Drug and Alcohol Research Centre
Ms Judith Lenartas	Private Submission
Mr Gary Liddle	Chief Executive Vic Roads
Mr Stephen Ling	Nurse Practitioner Drug and Alcohol John Hunter Hospital NSW
Mr Simon C. Lord	Private Submission
Dr Vivienne Mak	Consultant Psychiatrist Monash Medical Centre Victoria
Ms Lorraine Major	Director Major Egg Trading Western Australia
Ms Gabrielle Marlow	Private Submission
Mrs M. Martin	President Women's Christian Temperance Union of Western Australia
Ms Rosemary McClean	Policy and Program Advisor Australian Drug Foundation
Mr David McGrath	Director Mental Health and Drug and Alcohol Programs NSW Health
Mr Greg Meoliker	Private Submission

Name	Organisation
Ms Sue Miers	Spokesperson The National Organisation for Fetal Alcohol Syndrome and Related Disorders Inc South Australia
Ms Anita Moorhead	CMC, Lactation Royal Women's Hospital, Melbourne
Ms Anant More	Private Submission
Ms Greta More	Private Submission
Ms Kate Mortensen	Manager Lactation Resource Centre Australian Breastfeeding Association Victoria
Mr Alan Naper	Private Submission
Ms Claire Nicholson	Private Submission
Ms Colleen O'Leary	Research Associate Telethon Institute for Child Health Research Western Australia
Rev Brian O'Loughlin	Private Submission
Ms Gillian Patankar	Private Submission
Mr Tobias and Ms Pilat Patterson	Private Submission
Mr Will Patterson	Director of Public Health Australian Medical Association, Western Australia
Dr Ken Pidd	Deputy Director National Centre for Education and Training on Addiction
Dr Priscilla Pyett	Senior Research Fellow Onemda, VicHealth Koori Health Unit Victoria
Mr Harish Rajak	Private Submission
Dr Adrian Reynolds	Department of Health and Human Services, Tasmania
Mr Maurice Rickard	Manager Public Health Policy Australian Medical Association
Mr Bruce Ridge	Private Submission
Ms Christina Ridge	Private Submission
Ms Alma Ries	Community Health Nurse
Mr Troy Robinson	Private Submission
Ms Anne Russell	Russell Family Fetal Alcohol Disorders Association Inc Queensland

Name	Organisation
Ms Elizabeth Russell	Russell Family Fetal Alcohol Disorder Association Inc Queensland
Ms Lilia Safina	Private Submission
Ms Lynda Scott	Nursing Director Royal College of Nursing Australia
Ms Dolly Singh	Private Submission
Mr Kamlesh Singh	Private Submission
Mr Sunil Sivarajah	Private Submission
Ms Stephanie Slaven	Health Promotion Advisor Alcohol Healthwatch New Zealand
Ms Lorna Smith	Private Submission
Ms Alison Sneddon	Private Submission
Ms Vimala Sridhar	Private Submission
Professor Fiona Stanley	Director Telethon Institute for Child Health Research Western Australia
Dr Rima Staugas	Chief Executive Children, Youth and Women's Health Service Government of South Australia
Dr Tim Stockwell	Director Centre for Addictions Research University of Victoria, Canada
Mr Stephen Strachan	Chief Executive Winemakers' Federation of Australia South Australia
Ms Marianne Sturm	Private Submission
Mr Roscoe Taylor	Director; Population Health Tasmanian Department of Health and Human Services
Ms Kate Teasdale	Policy Officer Winnunga Nimmityjah Aboriginal Health Service ACT
Mr Wyeson Teo	Private Submission
Mr Graham Thomas	Cost Positive Economics NSW
Ms Sherry Thompson	Executive Director Early Childhood and Statewide Service SA Department Education and Children's Services

Name	Organisation
Ms Fran Thorn	Secretary Department of Human Services, Victoria
Ms Jennifer Tindall	Project Officer Hunter New England Population Health NSW
Dr Christine Tippet	President Royal Australian and New Zealand College of Obstetricians and Gynaecologists
Dr Melike Topaloglu	Private Submission
Mr Brian Vandenberg	Senior Program Advisor Tobacco Control and Alcohol Harm Reduction Unit VicHealth Victoria
Mr Surender Vasudev	Private Submission
Ms Lyn Vasuveda	Private Submission
Ms Sarah Venner	Drug and Alcohol Services South Australia IGCD Fetal Alcohol Spectrum Disorder Working Party
Dr Barbara Vernon	Executive Officer Australian College of Midwives
Mr Gino Vumbaca	Executive Director Australian National Council on Drugs
Ms Jill Waddell	Medical Affairs Project Officer Heart Foundation
Mr Chris Watters	Executive Director Queensland Government Liquor Licensing Division
Ms Maxine Whitnell	Private Submission
Dr Beverly Wood	Consultant in food, nutrition and dietetics
Dr Jeannette Young	Chief Health Officer Division of the Chief Health Officer Queensland Health
Ms Leonie Young	Chief Executive Officer beyondblue Victoria

The submissions that were given permission to be released publicly can be found on the NHMRC website at [www.nhmrc.gov.au/publications](http://www.nhmrc.gov.au/publications).

## A5 Estimating risk of alcohol-related disease and injury

As well as review of the published evidence (see Appendix A3), Guideline 1 is based on the following analyses:

- use of a modelling approach based on single episode data from major epidemiological studies to estimate lifetime risks of death from alcohol-related disease or injury from different patterns or levels of drinking
- analysis of the harms of alcohol at different ages and for different genders (harm scores) using data from the *2004 National Drug Strategy Household Survey* (AIHW 2005) (this also formed the basis for Guideline 3).

The methodologies for these analyses are discussed below.

### Estimating lifetime risk of alcohol-related disease and injury

As part of the standard five-yearly review of the NHMRC alcohol guidelines, a series of analyses were undertaken with the aim of measuring and specifying the level of risk attached to particular levels or patterns of drinking. These analyses, which were commissioned by the Department of Health and Ageing, informed the review committee's decision-making concerning cut-points for the guidelines. The analyses have been summarised in a peer-reviewed article (Rehm et al 2008).

Modelling lifetime injury mortality risk required estimation of risk based on the amount of alcohol consumed on a drinking occasion (drinking pattern), and then combining this information with assumptions on the number of such occasions and with age, gender, and injury mortality data. Chronic disease mortality risk, on the other hand, was based on average daily volume to generate lifetime chronic disease mortality risk for each age group, gender, and disease category.

The specific steps that were taken for disease and injury are outlined in the sections below.

### Identification of causal conditions

As a basis for estimation of both chronic disease and injury mortality risk, all relevant meta-analyses were reviewed to identify which chronic disease/injury categories were causally related to alcohol, using the approach of the World Health Organization (WHO) Comparative Risk Analysis (Ezzati et al 2004; Rehm et al 2004). The results of this are shown in Table 7.



Table 7 Chronic disease and injury categories causally related to alcohol, and alcohol-attributable fraction/relative risk source

Cause of death	GBD codes	Source
<b>Chronic diseases</b>		
Lip, oral and pharyngeal cancer	W 061	Corrao et al (1999)
Oesophageal cancer	W 062	Corrao et al (1999)
Liver cancer	W 065	Corrao et al (1999)
Breast cancer	W 069	Corrao et al (1999)
Alcohol-use disorders	W 086	Own calculations based on NESARC <a href="http://niaaa.census.gov/">http://niaaa.census.gov/</a>
Hypertensive diseases	W 106	Corrao et al (1999)
Ischaemic heart disease	W 107	Corrao et al (2004)
Ischaemic stroke	W 108	Corrao et al (1999)
Haemorrhagic stroke	W 108	Corrao et al (1999)
Cirrhosis of liver	W 117	Corrao et al (1999)
<b>Injury</b>		
Road traffic accidents	W 150	Ridolfo & Stevenson (2001)
Poisoning	W 151	World Health Organization (2002)
Falls	W 152	Ridolfo & Stevenson (2001)
Fire	W 153	Ridolfo & Stevenson (2001)
Drowning	W 154	Ridolfo & Stevenson (2001)
Other unintentional injuries	W 155	World Health Organization (2002)
Suicide	W 157	Ridolfo & Stevenson (2001)
Violence (homicide)	W 158	World Health Organization (2002)
Other intentional injuries	W 160	World Health Organization (2002)

Source: Rehm et al (2008)

## Calculation of lifetime risk

Because risk cannot usually be calculated directly, it is estimated from age-specific mortality rates, where the mortality rate is the number of deaths in a population divided by the total amount of time that the population is observed.

If the mortality rate is constant within a small time interval such as within the 50–54 year age group, the risk is equal to the mortality rate multiplied by the time at risk. For example, in 2004 in Australia, the mortality rate for breast cancer in the age group 50–54 years was 34.1 per 100,000 women per year and the risk that a 50-year-old woman would die from breast cancer in the next five years is  $34.1 \times 5 / 100000 = 0.0017 = 0.17\%$  (or 1 in 590).

The lifetime risk of death due to a specific cause (eg alcohol consumption or motor vehicle accidents) is estimated as:

$$\text{Lifetime risk} \cong \sum MR_i \times t_i$$

where  $\cong$  means approximately equal,  $MR_i$  is the mortality rate for that cause for the  $i^{\text{th}}$  age group (eg 30 to 44) and  $t_i$  is the number of years in that age interval (eg  $t_i = 15$  for the age group 30 to 44). The symbol  $\sum$  indicates that the products  $MR_i \times t_i$  are summed over all age groups to the chosen upper age limit for the expected lifetime. The approximation is good if the lifetime risk is less than about 15%. This formula assumes that the current mortality rates would apply throughout the lifetime of someone born today.

## Determination of alcohol-attributable lifetime risk for chronic disease

The modelled analysis commissioned for the guidelines calculated alcohol-attributable lifetime risk for chronic conditions for different levels of average consumption. The following steps were used.

### *Calculation of total risk*

As for injury categories, absolute, one-year, age, gender, and disease-specific overall risks were generated using data from studies identified through review of meta-analyses. Alcohol-attributable fraction (AAFs) were then computed based on average daily alcohol consumption levels and applied to the total risk to generate alcohol-attributable risks as outlined below.

### *Calculation of the consumption-specific alcohol-attributable fraction*

This step had two parts – the first was to compute the relative risk for different average daily consumption levels. For this analysis, the relative risk from 10g per day (one drink) to 100g per day on average (10 drinks) was

calculated, using lifetime abstainers as the reference category. The source of the information used to derive the risks associated with alcohol consumption is given in Table 8. For age groups over 60, the relative risk for chronic disease mortality tends to decrease with age, so this was accounted for based on the work of Klatsky and Udaltsova (2007). The results of these calculations are shown in Table 9 for people aged 15–60 years.

Table 8 Conditions caused by alcohol consumption and included in the analyses

Cause of death	Source
Lip, oral and pharyngeal cancer	Corrao et al 1999
Oesophageal cancer	Corrao et al 1999
Liver cancer	Corrao et al 1999
Breast cancer	Corrao et al 1999
Hypertensive diseases	Corrao et al 1999
Ischemic heart disease	Corrao et al 2004
Ischemic stroke	Corrao et al 1999
Haemorrhagic stroke	Corrao et al 1999
Cirrhosis of liver	Corrao et al 1999
Alcohol-use disorders	Calculated from NESARC data (Grant et al 2003)

Table 9 Adverse relative risks for disease conditions by categories of drinking, ages 15–60

Disease conditions	Relative risks (by standard drinks per day) <sup>a</sup>									
	1	2	3	4	5	6	7	8	9	10
Lip, oral and pharyngeal cancer	1.31	1.67	2.08	2.53	3.02	3.53	4.06	4.58	5.09	5.57
Oesophageal cancer	1.33	1.72	2.18	2.69	3.26	3.88	4.52	5.19	5.85	6.51
Liver cancer	1.08	1.15	1.23	1.31	1.40	1.48	1.56	1.65	1.73	1.81
Breast cancer	1.08	1.17	1.26	1.36	1.47	1.58	1.71	1.85	1.99	2.15
Hypertensive diseases	1.15	1.33	1.53	1.77	2.04	2.35	2.71	3.12	3.6	4.15

Disease conditions	Relative risks (by standard drinks per day) <sup>a</sup>									
	1	2	3	4	5	6	7	8	9	10
Ischaemic heart disease <sup>b</sup>	*	*	*	*	*	*	*	1.01	1.03	1.13
Ischaemic stroke <sup>b</sup>	*	*	*	1.12	1.4	1.73	2.04	2.21	2.12	1.72
Haemorrhagic stroke	1.16	1.35	1.57	1.82	2.12	2.46	2.86	3.32	3.86	4.48
Cirrhosis of liver	1.21	1.45	1.72	2.02	2.35	2.71	3.1	3.51	3.94	4.38
	1.32	1.73	2.25	2.89	3.68	4.64	5.8	7.17	8.80	10.69

Notes: <sup>a</sup> Figures are for men (top figure) and women (bottom figure); if only one relative risk is given, the risk did not vary significantly by gender.

<sup>b</sup> \* = no detrimental effect compared to abstainers (ie beneficial effects are not taken into account in this calculation).

As the main objective of the analysis was to determine the lifetime risk of drinking alcohol for chronic disease and acute injury outcomes, only the adverse health effects of alcohol were considered. While there is a substantial literature on protective effects of light drinking for ischemic heart disease and some other conditions (Rehm et al 2003; 2004; Ashley et al 2000), the extent of the effects is contested (eg Fillmore et al 2006). It appears that most of any beneficial effect can be gained at a low level of drinking, for instance a drink every second day (Di Castelnuovo et al 2006; WHO 2007) – well below the level of any likely low-risk drinking guideline. Where there was no detrimental effect, or a beneficial effect, the relative risk was recorded as 1.0.

The second part of this step was to generate an AAF for each age, gender, and disease category based on the relative risk and prevalence (Walter 1976; 1980) for all chronic disease categories except for alcohol-use disorder, based on the following formula. The AAFs used in subsequent calculations are given in Table 10.

$$AAFi = P * (RRi - 1) / [P * (RRi - 1) + 1]$$

Where:

- i: level of drinking (ie 10g pure alcohol per day... 100g pure alcohol per day)
- P: prevalence of alcohol consumption. For this, since we were only dealing with drinkers at a defined level of drinking (10 g/day, 20 g/day etc), we used 100 per cent prevalence, assuming all drinkers drink in the same quantity at 10g, 20g, 30g... 100g per day
- RRi: relative risk for drinking level i

Table 10 Alcohol-attributable fractions for chronic disease mortality, by standard drinks per day

1	2	3	4	5	6	7	8	9	10
<b>Lip, oral and pharyngeal cancer</b>									
23.5	40.0	51.9	60.5	66.9	71.7	75.4	78.2	80.4	82.1
24.7	41.9	54.0	62.9	69.4	74.2	77.9	80.7	82.9	84.6
<b>Oesophageal cancer</b>									
14.6	27.1	37.7	46.7	54.3	60.7	66.1	70.7	74.6	77.9
<b>Laryngeal cancer</b>									
7.0	13.3	18.9	23.9	28.4	32.4	36.0	39.2	42.1	44.7
<b>Breast cancer</b>									
7.4	14.2	20.6	26.4	31.9	36.9	41.5	45.9	49.9	53.6
<b>Hypertensive diseases</b>									
13.3	24.8	34.8	43.4	50.9	57.4	63.1	68.0	72.2	75.9
<b>Ischaemic heart disease</b>									
*	*	*	*	*	*	*	1.0	2.9	11.5
<b>Ischaemic stroke</b>									
*	*	*	10.9	28.3	42.0	51.0	54.8	52.8	41.8
<b>Haemorrhagic stroke</b>									
13.9	25.9	36.2	45.1	52.8	59.3	65.0	69.9	74.1	77.7
<b>Cirrhosis of liver</b>									
17.3	31.0	41.8	50.5	57.5	63.2	67.8	71.5	74.6	77.1
24.4	42.2	55.5	65.4	72.8	78.4	82.7	86.1	88.6	90.6

*Note:* Where two AAFs are given, the first applies to men; the second to women. If only one AAF is given, attributable risk did not vary significantly by gender.

*Source:* Rehm et al (2008)

### Determining risks of alcohol-use disorder

Alcohol-use disorder is, by definition, 100 per cent attributable to alcohol (ie it would disappear completely if alcohol was not present). Therefore, the issue here was not determining the portion of risk that was alcohol attributable, but rather to estimate the risk of developing alcohol-use disorder at a given level of drinking. For this, survey data were used. The US-based

National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) data were used (sample size = 43, 093 persons 18 years and older, response rate = 81 per cent) (for more information, please see the NESARC website at: <http://niaaa.census.gov/>). The risk of alcohol-use disorder by age and gender based on average daily drinking that year (from Rehm et al, 2007b) was derived and modelled for daily consumption categories corresponding to Australian standard drinks (10g/day – 100 g/day) (see Figure 14; similar figure for women not shown), and AAFs of all alcohol-use disorder deaths for different levels of drinking were based on these risk estimates.

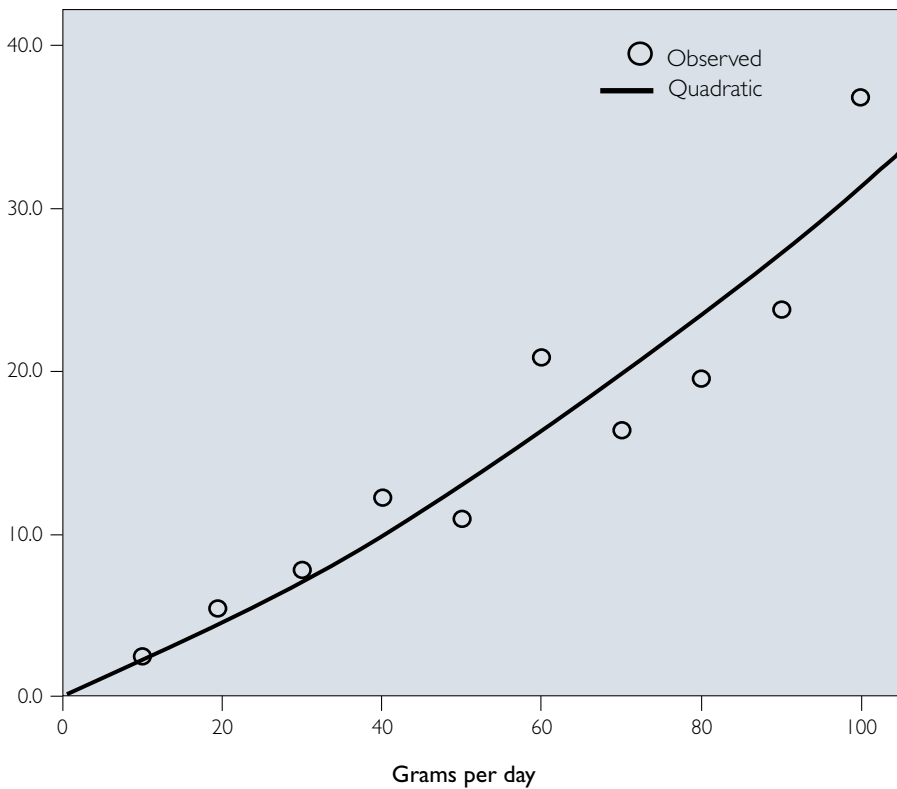


Figure 14 Risk for alcohol-use disorder (men) by average level of alcohol consumption

*Note:* Calculations based on NESARC data using quadratic regression.

*Source:* Rehm et al (2008)

### ***Combination of total risk and alcohol-attributable fraction***

To obtain the one-year, alcohol-attributable risk for each category, the total risk and the computed AAF for each age, gender, and disease category were combined.

The year 2002 was selected as the reference year for the determination of absolute risk for chronic disease. For each chronic disease category on the list, the age-specific death rates were calculated separately for men and women. The following age categories were used: 15–29, 30–44, 45–59, 60–69, 70–79 and 80 and older.

To obtain the lifetime risk, age-gender-disease one-year risks were multiplied by the time spent in each age category; ie for women aged 30–44, their one-year alcohol-attributable risk would be multiplied by 15, since they would have spent 15 years in this ‘risk-block’. For those aged 20–29, it would have been multiplied by 10 years. For those in the oldest age category, their risks were multiplied by the residual life expectancy (obtained from WHO 2004). So, for women 80+, their one-year was multiplied by three (83 year average life expectancy for Australian women – 80 years). Finally, the risks were added up across age groups and disease categories.

### ***Results***

Lifetime risk of chronic disease mortality shows a different relationship with alcohol than does injury mortality. This relationship graphically looks much more linear than the lifetime risk of injury death. Figure 5 (see page 33) shows that the lifetime risk of chronic disease death increases as average daily volume increases. What is most striking in this graph is the higher risk among women compared to men at higher daily volume levels. The risk for women also increases faster with increased consumption than for men. At 10 grams per day, lifetime risk for women is actually lower than that for men, but increases to over 50 per cent higher (96 vs 60 per 1,000) at 100 grams. Overall, risk increases by about 10 per cent with each 10-gram (1 drink) increase in alcohol consumption. The risks for men and women are quite similar at average daily volume levels below 40 grams per day, while at higher levels of drinking large differences by gender are seen.

### ***Determination of lifetime injury mortality risk***

Alcohol-attributable injury deaths per drinking occasion, and lifetime risk, were calculated using the following steps.

### Calculation of the baseline risk

Age and sex-specific mortality rates for 2002 were obtained from the WHO Global Burden of Disease project for the year 2002 (WHO 2002). To obtain the rates that would be observed in the absence of alcohol consumption (ie the baseline rates), these mortality rates were multiplied by the age/sex/injury-specific alcohol-attributable fractions (AAF) and the resulting number subtracted from the total rates (ie baseline rate = total population rate – total population rate x AAF). The AAFs were Australia-specific and obtained from Ridolfo & Stevenson (2001) or the WHO Comparative Risk Analysis (Western Pacific A Region) (Rehm et al 2004; WHO 2002) (see Table 7). The AAFs used in the calculations are given in Table 11.

#### EXAMPLE

In 2002, there were 286 deaths from road traffic accidents in men aged 30–44 out of a total population of 2,204,725 men in that age group; thus the total rate was  $(286/2204725) \times 1,000 = 0.130$  deaths per 1,000 man years in this age group. Multiplying this by the AAF (0.305) and subtracting this from the total rate gives the baseline rate:  $0.130 - (0.130 \times 0.305) = 0.090$  deaths from road traffic accidents per 1,000 man years for men aged 30–44 years.

Table 11 Alcohol-attributable fractions for injury mortality, by age group, gender and injury type

15–29 Yrs		30–44 Yrs		45–59 Yrs		60–69 Yrs		70–79 Yrs		80+ Yrs	
M	F	M	F	M	F	M	F	M	F	M	F
<b>Road traffic accidents</b>											
30.5	10.7	30.5	10.7	30.5	10.7	30.5	10.7	30.5	10.7	30.5	10.7
<b>Poisoning</b>											
29.0	23.0	16.0	15.0	16.0	15.0	16.0	15.0	8.0	7.0	8.0	7.0
<b>Falls</b>											
22.0	14.0	22.0	14.0	22.0	14.0	12.0	04.0	12.0	04.0	12.0	4.0



15–29 Yrs		30–44 Yrs		45–59 Yrs		60–69 Yrs		70–79 Yrs		80+ Yrs	
<b>Fire</b>											
44.0	44.0	44.0	44.0	44.0	44.0	44.0	44.0	44.0	44.0	44.0	44.0
<b>Drowning</b>											
34.0	34.0	34.0	34.0	34.0	34.0	34.0	34.0	34.0	34.0	34.0	34.0
<b>Other unintentional injuries</b>											
29.0	23.0	29.0	23.0	24.0	19.0	24.0	19.0	24.0	19.0	24.0	19.0
<b>Suicide</b>											
32.3	28.5	32.3	28.5	32.3	28.5	32.3	28.5	32.3	28.5	32.3	28.5
<b>Violence (homicide)</b>											
27.0	27.0	27.0	27.0	27.0	27.0	27.0	27.0	27.0	27.0	27.0	27.0
<b>Other intentional injuries</b>											
20.0	20.0	20.0	20.0	20.0	20.0	20.0	20.0	10.0	10.0	10.0	10.0

Source: Rehm et al (2008).

### **Estimation of the relative risk for injury immediately following an occasion of alcohol consumption**

Estimation of the relative risk for injury following an occasion of drinking was based on data from the WHO 10-site international case-crossover study of emergency department admissions for injury (N = 4,320, 91 per cent response rate) (Borges et al 2006), in which patients presenting with an injury were asked about their alcohol consumption in the six hours prior to the injury and for the same period the previous week. This study reported relative risks corresponding to the consumption of numbers of a standard drink. As the standard drink was defined as 16 mL of pure alcohol (as opposed to the Australian Standard Drink, which is defined as about 10g of alcohol – equivalent to 12.5mL of pure alcohol), the emergency department data were modelled and the relative risk converted to Australian standard drinks for up to five drinks based on the model, and used Borges et al (2006) for over this amount (see Table 12 for a comparison of the original and converted data).

Table 12 Relative risk data for Australian standard drinks

Before conversion (12.6g standard drinks)		After conversion (Australian 10g standard drinks)	
Number of drinks	OR	Number of drinks	OR
0	1.0	0	1.0
1	3.3	1	1.9
2.5	3.9	3	3.8
4.5	6.5	5	5.6
>6	10.1	7+	10.1

OR = odds ratio (used as an estimate of relative risk)

Source: Borges et al (2006).

### Combination of baseline risk and alcohol-based relative risk

The relative risks from Borges et al (2006) were for a single occasion of drinking and can be combined with the baseline rates to estimate the risk of dying from injury within a short time after such an occasion.

When a person drinks one drink, for example, he or she is not at risk for an entire day (24 hours). Rather, risk should be based on the average time it takes for the liver to metabolise a certain number of drinks and thus was modelled based on work from the National Institute for Alcohol Abuse and Alcoholism (NIAAA 1997). Generally, for 1, 3, 5, and 7 drinks, risk periods of 30 minutes, two hours, three hours and 4.8 hours were used.

#### EXAMPLE

If a 35-year-old man consumes three standard drinks on one occasion, based on Table 12, his risk of dying in a road traffic accident is 3.8 times greater than if he had not been drinking. This relative risk applies for three hours, after which it returns to 1. His risk of dying in a road traffic accident within three hours if he had not been drinking would be equal to the baseline rate multiplied by the time at risk, which is  $(0.0000103) * 3 = 0.0000309$  per 1000 (or about 1 in 32 million) and his risk after drinking would be  $0.0000309 * 3.8 = 0.000117$  per 1000 (or about 1 in 8.5 million).

As the objective of this analysis was to estimate the excess lifetime risk attributable to repeated occasions of drinking over a lifetime, it was necessary to assume that the risk of injury following an occasion of drinking was independent of the risk following any other occasion of drinking. That assumption allowed the calculation of lifetime risks for various patterns of consumption over a lifetime using the rules of probability.

$$\Pr(\text{Death} | N) = 1 - \left[ 1 - \frac{\Pr(\text{death})_d}{i_d} \right]^N$$

Where:

N = the number of lifetime drinking occasions (eg, 50, 100, 500, 1,000, 5,000, 10,000, or 20,000). These correspond to drinking once, twice, and ten times per year; about twice per month, twice per week, about four times per week, and about every day, respectively.

Pr(Death | N) = the risk of injury death (per 1000) given N lifetime drinking occasions

Pr(death)<sub>d</sub> = the risk of injury death for one drinking occasion per year; depending on the number of drinks per occasion. This is the baseline risk (from Step 2) multiplied by the standard drink-specific relative risks (from Step 3)

*i<sub>d</sub>* = the 'risk period'. This is the time period in which the person is at risk of an alcohol-attributable injury death. When a person drinks one drink, for example, he or she is not at risk for an entire day (24 hours). Rather, risk should be based on the average time it takes for the liver to metabolise a certain number of drinks and thus was modelled based on work from the National Institute for Alcohol Abuse and Alcoholism (NIAAA 1997).

Generally, for 1, 3, 5, and 7 drinks, risk periods of 30 minutes, two hours, three hours and 4.8 hours were used.

Probabilities were computed for each age, gender, disease, and consumption group (eg the lifetime probability of an alcohol-attributable motor vehicle death for 20–29 year old males who drank approximately 2 standard drinks 4 times per week), and added up across disease categories and ages.

### **Results of the analysis**

For both men and women, the absolute lifetime risk of injury increase as the number of drinks per occasion and the number of drinking occasions increase, although men experience about 30 per cent higher risks than women at all levels of drinking, with an even greater difference when the occasions are very frequent. Additionally for both men and women a significant jump in risk was seen at 5,000 occasions (about two drinking occasions per week); the risk increases substantially for each increase in the

number of drinking occasions thereafter. A lifetime risk of 1 in 100 is reached for consumption level of three drinks on 20,000 occasions (about five times a week) for men and daily for women. At frequencies at or below about twice a month (1,000 occasions), risks are much closer together.

Figures 7 and 8 (pages 46-47) show the lifetime risk per 100 of overall injury mortality by number of standard drinks consumed and lifetime drinking occasions among men and women in Australia, 2002.

## Reanalysis of National Drug Strategy Survey

### Derivation of harm scores

Using data from the *2004 National Drug Strategy Household Survey* (AIHW 2005), harm scores from the series of questions on self-reported problems from drinking were derived, with one point assigned for each positive response concerning the past 12 months (Livingstone & Room, in press). An overall harm score was derived, as well as two sub-scores of hazardous behaviours and delinquent behaviours, distinguished from each other in a factor analysis. The items used from the NDSHS were as follows:

- In the past 12 months, did you undertake the following activities while under the influence of alcohol?

#### **Hazardous behaviours**

- Went to work
- Went swimming
- Operated a boat
- Drove a motor vehicle
- Operated hazardous machinery

#### **Delinquent behaviours**

- Created a public nuisance or disturbance
- Caused damage to property
- Stole money, goods or property
- Verbally abused someone
- Physically abused someone.

The harm indexes were calculated as the harm score divided by the volume of drinking, and by the number of days per year on which five or more standard drinks were consumed. These are presented as ratios, with the male indexes at 40–44 years set as 1.0.

### Harmful behaviours while drinking, by sex and age

The harm indexes were used in an analysis of the relative rates of hazardous and delinquent behaviours by age and sex in connection with Guideline 3. The hazardous and delinquent behaviour scores were also used in an analysis of how rates differed for men and women drinking in the same range. This contributed to the Committee’s deliberations on whether guidelines should differ for men and women.

Figure 15 shows for the hazardous behaviour score and Figure 16 for the delinquent behaviour score the average score for men and women drinking who have always in the last year consumed below a certain threshold: below three drinks for the leftmost point, no more than three or four drinks for the next point, then no more than five or six, and lastly those who have drunk seven or more at some time in the last year. It will be seen that the average hazardous behaviour score rises across these categories for both men and women, while the delinquent behaviour score rises substantially only for the last category. Men consistently report a higher average score than women in all comparisons.

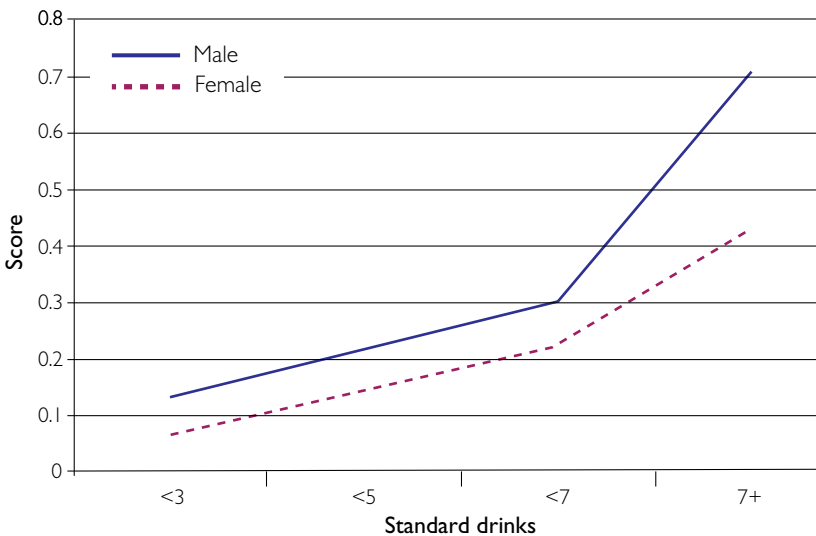


Figure 15 Mean hazardous behaviour score among males and females for drinkers who never drink more than two drinks; those who sometimes drink three or four drinks but never more; those who sometimes drink five or six drinks but never more; those who sometimes drink seven or more drinks, 2004 National Drug Strategy Household Survey respondents aged 18+

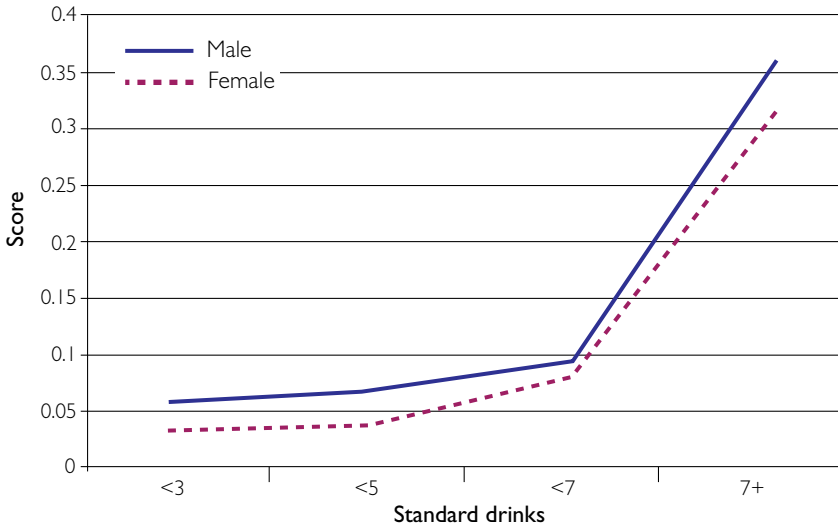


Figure 16 Mean delinquent behaviour score among males and females for drinkers who never drink more than two drinks; those who sometimes drink three or four drinks but never more; those who sometimes drink five or six drinks but never more; those who sometimes drink seven or more drinks, 2004 National Drug Strategy Household Survey respondents aged 18+

The harm per litre reported by males aged 18–29 is set at 1.0, and rates for the other five gender-age categories are expressed in comparison to that. Figure 17 shows the results for an index of hazardous behaviour per litre and Figure 18 for an index of delinquent behaviour per litre. It will be seen that the harm per litre declines with age, particularly for delinquent behaviour and particularly for the oldest age group. Men report slightly more hazardous behaviour per litre than women, while there is little difference between men and women for delinquent behaviour.

A third perspective is in terms of relative harm per drinking occasion of 5+ drinks. This analysis is confined to drinkers (64% of male drinkers, 44% of female) who drank 5+ drinks at some time in the past year. Figure 19 shows the results for an index of hazardous drinking per heavy drinking occasion, and Figure 20 for an index of delinquent drinking per heavy drinking occasion. In this more restricted sample, there is no gender difference for ages 18–29 for either measure, but both indexes are somewhat higher for women than for men in middle age and older age, with a marked difference on the index of hazardous behaviour at older age.

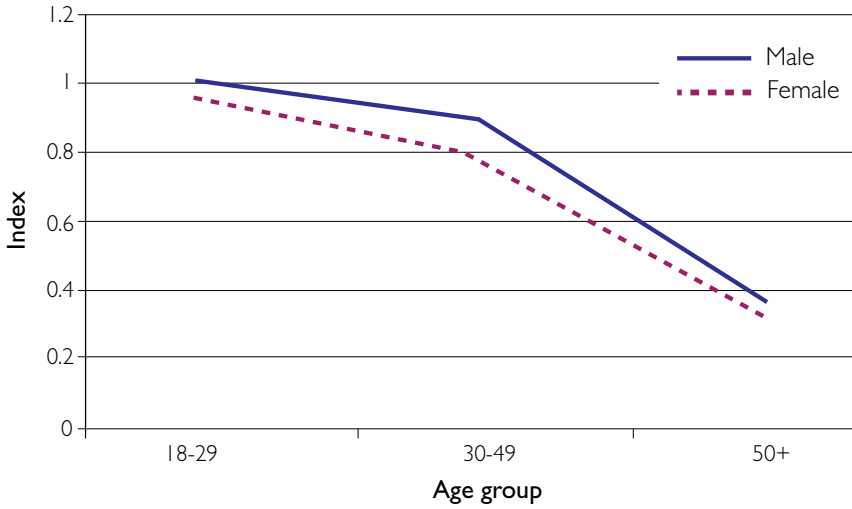


Figure 17 Index of hazardous behaviour per litre of total annual consumption, comparing by gender and age group, males 18–29 set to 1.0

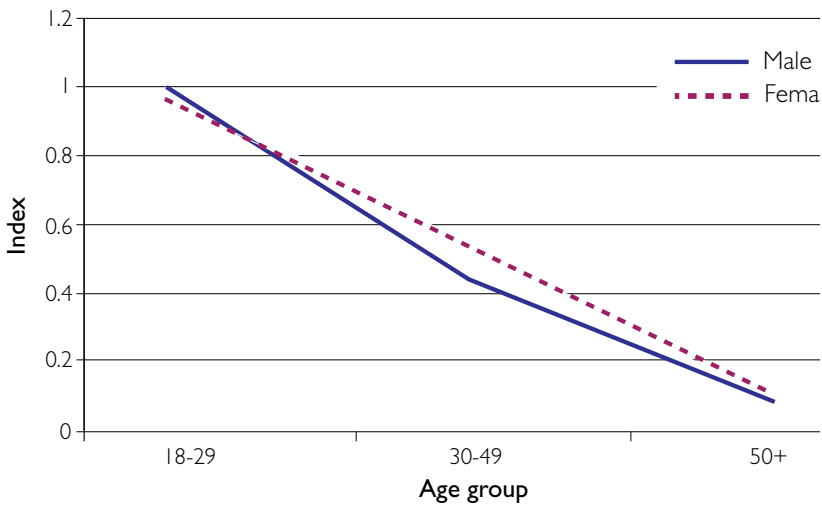


Figure 18 Index of delinquent behaviour per litre of total annual consumption, comparing by gender and age group, males 18–29 set to 1.0

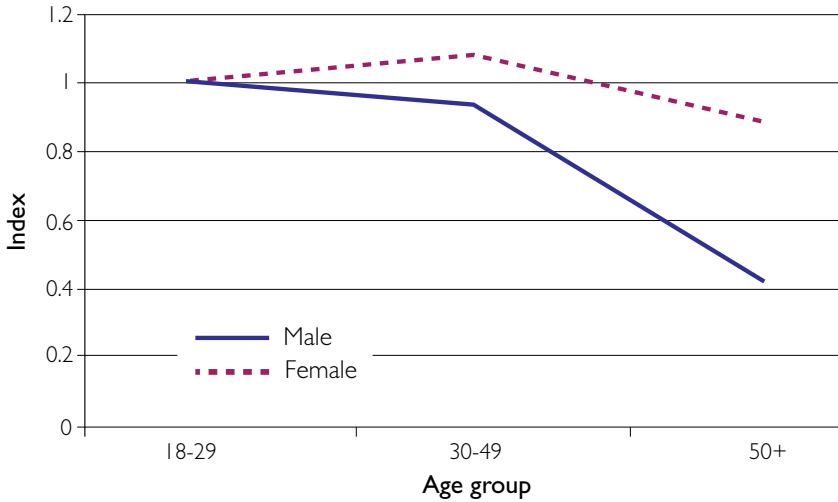


Figure 19 Index of hazardous behaviour per drinking occasion of 5+ drinks, among those drinking five or more drinks in the last year, males 18–29 set to 1.0

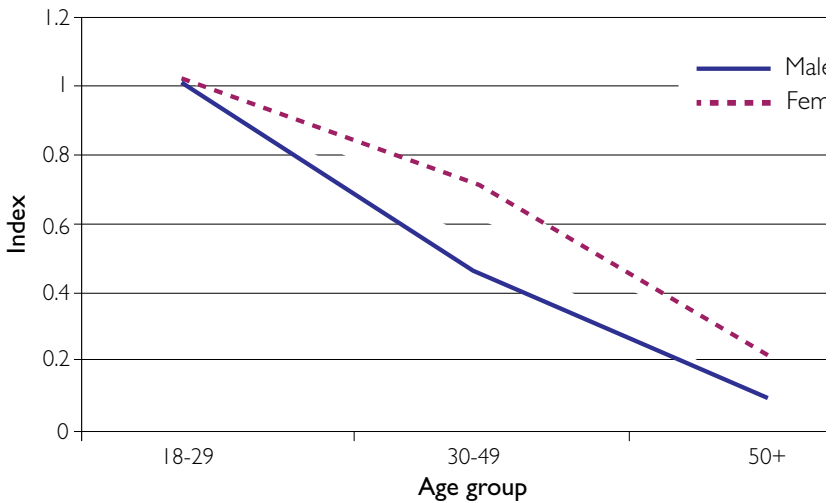


Figure 20 Index of delinquent behaviour per drinking occasion of 5+ drinks, among those drinking five or more drinks in the last year, males 18–29 set to 1.0



## Summary of findings

Putting together the results from these three different ways of comparing harms associated with male and female drinking at specified levels, there is no clear justification on this basis for a differentiation between men and women in guidelines for number of drinks on an occasion. It should be noted, however, that there are clear differentiations by age, with younger adults tending to report more associated harms per litre of drinking.

### Detailed estimates of risks from particular patterns of drinking

Tables 3 and 4 (pages 48–50) give the estimated lifetime risks from drinking at different levels in two particular patterns – weekly and daily. Below is a more detailed table of the estimated risks, for women and for men separately, from drinking to particular levels with different frequencies. The risks, for hospitalisation from injury, for death from injury, and for death from alcohol-related disease, are stated in terms of lifetime risks per 100 drinkers. In using this table, it should be kept in mind that the risks of death from injury and of death from alcohol-related disease are additive. That is, a male who drinks four standard drinks daily has a lifetime risk of over 4 in 100 of death from drinking (2.21 + 1.99).

Table 13 Risks of injury and disease per 100 drinkers, for particular patterns of drinking

Men				
Number of standard drinks	Hospitalisation from injury	Death from injury	Death from alcohol-related disease	Total risk of alcohol-related death
Drinking this amount twice a year (and not otherwise)				
1	0.03	*	*	
2	0.12	*	*	
3	0.22	0.01	0.01	0.02
4	0.34	0.01	0.01	0.02
5	0.49	0.01	0.01	0.02
6	0.94	0.03	0.01	0.04
7	1.39	0.04	0.01	0.05
8	1.83	0.05	0.01	0.06

<b>Men</b>				
<b>Number of standard drinks</b>	Hospitalisation from injury	Death from injury	Death from alcohol-related disease	Total risk of alcohol-related death
<b>Drinking this amount once a month (and not otherwise)</b>				
1	0.13	*	0.01	0.01
2	0.28	0.02	0.02	0.04
3	1.06	0.03	0.03	0.06
4	1.59	0.05	0.03	0.08
5	2.26	0.06	0.04	0.10
6	3.95	0.13	0.05	0.18
7	5.66	0.19	0.06	0.25
8	7.34	0.24	0.07	0.31
<b>Drinking this amount twice a month (and not otherwise)</b>				
1	0.28	0.01	0.02	0.03
2	0.74	0.03	0.03	0.06
3	2.03	0.06	0.05	0.11
4	2.96	0.09	0.07	0.16
5	4.14	0.13	0.09	0.22
6	6.69	0.25	0.10	0.35
7	9.30	0.37	0.12	0.49
8	11.91	0.48	0.14	0.62
<b>Drinking this amount once a week (and not otherwise)</b>				
1	1.33	0.04	0.09	0.13
2	4.30	0.06	0.13	0.19
3	7.72	0.29	0.26	0.55
4	10.00	0.44	0.35	0.79
5	12.87	0.63	0.44	1.07
6	17.08	1.18	0.53	1.71
7	21.28	1.74	0.62	2.36
8	25.56	2.27	0.72	2.99

<b>Men</b>				
<b>Number of standard drinks</b>	Hospitalisation from injury	Death from injury	Death from alcohol-related disease	Total risk of alcohol-related death
<b>Drinking this amount three times in each week (and not otherwise)</b>				
1	2.52	0.07	0.13	0.20
2	7.18	0.12	0.17	0.29
3	12.08	0.57	0.36	0.93
4	14.87	0.87	0.72	1.59
5	18.18	1.23	0.91	2.14
6	23.09	2.24	1.10	3.34
7	27.58	3.25	1.31	4.56
8	31.82	4.24	1.51	5.75
<b>Drinking this amount five times in each week (and not otherwise)</b>				
1	4.59	0.15	0.17	0.32
2	10.80	0.30	0.29	0.59
3	17.30	1.11	0.64	1.75
4	20.69	1.66	1.51	3.17
5	24.13	2.34	1.94	4.28
6	29.68	4.09	2.39	6.48
7	34.17	5.85	2.86	8.71
8	37.74	7.57	3.35	10.92
<b>Drinking this amount daily</b>				
1	5.89	0.20	0.21	0.41
2	12.71	0.48	0.44	0.92
3	19.86	1.50	1.26	2.76
4	23.63	2.21	1.99	4.20
5	27.06	3.11	2.70	5.81
6	32.74	5.29	3.80	9.09
7	37.06	7.51	4.66	12.17
8	40.06	9.69	5.14	14.83

<b>Women</b>				
<b>Number of standard drinks</b>	Hospitalisation from injury	Death from injury	Death from alcohol-related disease	Total risk of alcohol-related death
<b>Drinking this amount twice a year (and not otherwise)</b>				
1	0.03	*	*	
2	0.03	*	*	
3	0.22	*	0.01	0.01
4	0.34	0.01	0.01	0.02
5	0.49	0.01	0.01	0.02
6	0.93	0.02	0.01	0.03
7	1.37	0.02	0.01	0.03
8	1.80	0.03	0.02	0.05
<b>Drinking this amount once a month (and not otherwise)</b>				
1	0.14	*	0.01	0.01
2	0.36	0.01	0.02	0.03
3	1.02	0.02	0.03	0.05
4	1.49	0.03	0.04	0.07
5	2.07	0.04	0.05	0.09
6	3.38	0.08	0.06	0.14
7	4.72	0.12	0.07	0.19
8	6.05	0.15	0.08	0.23
<b>Drinking this amount twice a month (and not otherwise)</b>				
1	0.28	*	0.02	0.02
2	0.78	0.02	0.04	0.06
3	1.87	0.04	0.06	0.10
4	2.62	0.06	0.08	0.14
5	3.56	0.08	0.10	0.18
6	5.53	0.16	0.12	0.28
7	7.51	0.23	0.14	0.37
8	9.49	0.30	0.17	0.47

<b>Women</b>				
<b>Number of standard drinks</b>	Hospitalisation from injury	Death from injury	Death from alcohol-related disease	Total risk of alcohol-related death
<b>Drinking this amount once a week (and not otherwise)</b>				
1	1.26	0.02	0.10	0.12
2	3.41	0.04	0.14	0.18
3	6.29	0.18	0.32	0.50
4	8.21	0.28	0.43	0.71
5	10.5	0.39	0.55	0.94
6	14.40	0.73	0.67	1.40
7	18.22	1.06	0.79	1.85
8	21.99	1.38	0.92	2.30
<b>Drinking this amount three times in each week (and not otherwise)</b>				
1	2.27	0.05	0.12	0.17
2	5.77	0.09	0.20	0.29
3	9.82	0.36	0.38	0.74
4	12.40	0.53	0.92	1.45
5	15.31	0.76	1.18	1.94
6	19.87	1.34	1.46	2.80
7	24.15	1.92	1.75	3.67
8	28.19	2.49	2.05	4.54
<b>Drinking this amount five times in each week (and not otherwise)</b>				
1	3.90	0.09	0.13	0.22
2	8.94	0.22	0.27	0.49
3	14.49	0.69	0.69	1.38
4	17.66	1.01	2.05	3.06
5	20.91	1.41	2.70	4.11
6	26.16	2.35	3.41	5.76
7	30.65	3.31	4.18	7.49
8	34.44	4.25	4.99	9.24

<b>Women</b>				
<b>Number of standard drinks</b>	Hospitalisation from injury	Death from injury	Death from alcohol-related disease	Total risk of alcohol-related death
<b>Drinking this amount daily</b>				
1	4.89	0.13	0.16	0.29
2	10.63	0.39	0.38	0.77
3	16.89	0.91	1.41	2.32
4	20.36	1.32	2.53	3.85
5	23.65	1.84	3.68	5.52
6	29.18	2.99	5.93	8.92
7	33.65	4.16	7.61	11.77
8	37.09	5.33	8.37	13.70

*Note:* The figures in this table represent the risks above the baseline (not drinking).















\* = risk less than 0.005

Frequencies are approximate, translated from number of times in a lifetime: twice a year = 100 occasions; once a month = 500 occasions; twice a month = 1,000 occasions; weekly = 5,000 occasions; three times in each week = 10,000 occasions; five times in each week = 20,000 occasions; daily = 27,375 occasions.

Figures have been rounded to two decimal places.

# A6 Number of standard drinks in various beverages

NUMBER OF STANDARD DRINKS – BEER	
	<b>1.1</b> 285ml Full Strength 4.8% Alc. Vol
	<b>0.8</b> 285ml Mid Strength 3.5% Alc. Vol
	<b>0.6</b> 285ml Low Strength 2.7% Alc. Vol
	<b>1.6</b> 425ml Full Strength 4.8% Alc. Vol
	<b>1.2</b> 425ml Mid Strength 3.5% Alc. Vol
	<b>0.9</b> 425ml Low Strength 2.7% Alc. Vol
	<b>1.4</b> 375ml Full Strength 4.8% Alc. Vol
	<b>1</b> 375ml Mid Strength 3.5% Alc. Vol
	<b>0.8</b> 375ml Low Strength 2.7% Alc. Vol
	<b>1.4</b> 375ml Full Strength 4.8% Alc. Vol
	<b>1</b> 375ml Mid Strength 3.5% Alc. Vol
	<b>0.8</b> 375ml Low Strength 2.7% Alc. Vol
	<b>34</b> 94 x 375ml Full Strength 4.8% Alc. Vol
	<b>24</b> 24 x 375ml Mid Strength 3.5% Alc. Vol
	<b>19</b> 24 x 375ml Low Strength 2.7% Alc. Vol

NUMBER OF STANDARD DRINKS – WINE			
	<b>1.6</b> 150ml Average Restaurant Serving of Red Wine 135% Alc. Vol		<b>8</b> 750ml Bottle of Red Wine 135% Alc. Vol
	<b>1</b> 100ml Standard Serve of Red Wine 13.5% Alc. Vol		<b>43</b> 4 Litres Cask Red Wine 13.5% Alc. Vol
	<b>0.9</b> 60ml Standard Serve of Port 18% Alc. Vol		<b>21</b> 2 Litres Cask Red Wine 13.5% Alc. Vol
	<b>1.4</b> 150ml Average Restaurant Serving of White Wine 11.5% Alc. Vol		<b>7.5</b> 750ml Bottle of White Wine 12.5% Alc. Vol
	<b>1</b> 100ml Standard Serve of White Wine 11.5% Alc. Vol		<b>39</b> 4 Litres Cask White Wine 12.5% Alc. Vol
	<b>1.4</b> 150ml Average Restaurant Serve of Champagne 12% Alc. Vol		<b>19.5</b> 2 Litres Cask White Wine 12.5% Alc. Vol
	<b>7.5</b> 750ml Bottle of Champagne 12.5% Alc. Vol		<b>28</b> 2 Litres Cask of Port 17.5% Alc. Vol



NUMBER OF STANDARD DRINKS – SPIRITS	
	<b>1</b> 30ml High Strength Spirit Nip 40% Alc. Vol
	<b>22</b> 700ml High Strength Bottle of Spirits 40% Alc. Vol
	<b>1.1</b> 275ml Full Strength RTD* 5% Alc. Vol
	<b>1.2</b> 330ml Full Strength RTD* 5% Alc. Vol
	<b>2.6</b> 660ml Full Strength RTD* 5% Alc. Vol
	<b>1.5</b> 275ml High Strength RTD* 7% Alc. Vol
	<b>1.8</b> 330ml High Strength RTD* 7% Alc. Vol
	<b>3.6</b> 660ml High Strength RTD* 7% Alc. Vol
	<b>1</b> 250ml Full Strength Pre-mix Spirits 5% Alc. Vol
	<b>1.2</b> 300ml Full Strength Pre-mix Spirits 5% Alc. Vol
	<b>1.5</b> 375ml Full Strength Pre-mix Spirits 5% Alc. Vol
	<b>1.7</b> 440ml Full Strength Pre-mix Spirits 5% Alc. Vol
	<b>1.4 – 1.9</b> 250ml High Strength Pre-mix Spirits 7% – 10% Alc. Vol
	<b>1.6</b> 300ml High Strength Pre-mix Spirits 7% Alc. Vol
	<b>2.1</b> 375ml High Strength Pre-mix Spirits 7% Alc. Vol
	<b>2.4</b> 440ml High Strength Pre-mix Spirits 7% Alc. Vol

\* Ready-to-Drink

## Acronyms and abbreviations

AAF	alcohol-attributable fraction
ADA	American Diabetes Association
AIHW	Australian Institute of Health and Welfare
ARBD	alcohol-related birth defects
ARND	alcohol-related neurodevelopmental disorders
BAC	blood alcohol concentration
CI	confidence interval
DoHA	Commonwealth Department of Health and Ageing
FAS	fetal alcohol syndrome
FASD	fetal alcohol spectrum disorders
GABA	gamma-amino butyric acid
HAC	Health Advisory Committee
ICAP	International Center for Alcohol Policy
MDMA	methylenedioxymethamphetamine
NHC	National Health Committee
NHMRC	National Health and Medical Research Council
NESARC	National Epidemiologic Survey on Alcohol and Related Conditions (US)
OECD	Organisation for Economic Cooperation and Development
OR	odds ratio
PHD	Public Health Division (DoHA)
RTA	Road Traffic Authority
WHO	World Health Organization

## Glossary

### Terms not used in these guidelines as they are difficult to quantify

A number of terms have been used in reporting studies of drinking and many of these are hard to quantify. For this reason, they are not used in these guidelines other than in the context of discussion of existing studies. These terms include: 'binge drinking', 'heavy drinking', 'problem drinking' and 'risky drinking'.

### Terms used in these guidelines

#### *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 these guidelines. Other forms of alcohol, including methanol, are more toxic to humans than ethanol and are not suitable for human consumption.

#### *Dependence*

Alcohol dependence refers to situations where a person feels a strong need to drink so that 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 at high-risk 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 tremulousness and anxiety.

#### *Drinking occasion*

In these guidelines, a drinking occasion refers to a sequence of drinks taken without the blood alcohol concentration reaching zero in between. This might include a drink at home at the end of the day or over dinner, or at a specific event, such as a party, night out, visit to the pub, a family or business event or other function. It may also include drinking spread across more than one context or venue, for instance on a 'Friday night out'.

#### *Fetal Alcohol Syndrome (FAS)*

FAS is a disorder of permanent birth defects that can occur in the offspring of women who drink alcohol during pregnancy. These defects include growth deficiency, characteristic facial features, and central nervous system damage.

***Fetal Alcohol Spectrum disorder (FASD)***

FASD describes a continuum of permanent birth defects related to a maternal consumption of alcohol during pregnancy, which includes FAS. Other subtypes with evolving nomenclature and definitions based on partial expressions of FAS, including Partial Fetal Alcohol Syndrome (PFAS), Alcohol-Related Neurodevelopmental Disorder (ARND), Alcohol-Related Birth Defects (ARBD), and Fetal Alcohol Effect (FAE).

***Odds ratio***

The odds ratio is a measure of the size of an effect. It is defined as the ratio of the odds of an event occurring in one group to the odds of it occurring in another group, or to a sample-based estimate of that ratio. If the probabilities of the event in each of the groups are  $p$  (first group) and  $q$  (second group), then the odds ratio is:

$$\frac{p/(1-p)}{q/(1-q)} = \frac{p(1-q)}{q(1-p)}$$

An odds ratio of 1 indicates that the condition or event under study is equally likely in both groups. An odds ratio greater than 1 indicates that the condition or event is more likely in the first group. And an odds ratio less than 1 indicates that the condition or event is less likely in the first group. The odds ratio must be greater than or equal to zero. As the odds of the first group approaches zero, the odds ratio approaches zero. As the odds of the second group approaches zero, the odds ratio approaches positive infinity.

***Tolerance***

The immediate effects of alcohol on the brain are often less apparent in people who drink regularly, as they acquire a degree of tolerance. Tolerance occurs in part because the liver becomes more efficient at breaking down alcohol. The person learns to cope with, and compensate for, the deficits induced by alcohol.

## References

Where possible, these guidelines are based on meta-analyses and systematic reviews, which synthesise the results from a number of single studies. These are marked with an asterisk(\*) in the following list and summarised in Table 6.

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