National Alcohol Indicators Project

Technical Report No. 1: Alcohol-caused Deaths and Hospitalisations in Australia, 1990-1997

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INTRODUCTION

This is the first report of the National Alcohol Indicators Project (NAIP). The aim of NAIP is to monitor and report on trends in alcohol-related and alcohol-caused harm in Australia, at national, state and local levels. The NAIP is a collaborative project between the National Drug Research Institute (Curtin University of Technology) and Turning Point, Alcohol and Drug Centre Inc. The NAIP is funded by the National Drug Strategy.

This report estimates the burden of alcohol misuse (hazardous/harmful levels as defined by Pols & Hawks in the 1992 NHMRC guidelines) in terms of morbidity and mortality on the 1997 Australian population and documents the trend in deaths and hospitalisations between 1990 and 1997. Patterns across different geographic regions of the country were also examined. Major variables under consideration included the age and sex of the persons dying or being hospitalised as well as their Indigenous status.

Alcohol is a leading cause of death, injury and illness in Australian society. For 1992, the annual cost of alcohol-caused problems to the nation was conservatively estimated at over $4.5 billion – about 2.6 times the cost for illicit drugs (Collins and Lapsley, 1996). Higgins *et al.* (2000) estimated that in 1997 the total number of deaths due to illegal drug use in Australia was 832, while the number of deaths due to tobacco use was 18,224.

This technical report accompanies the NAIP Bulletin No. 1, detailing the methods employed and providing additional results. Future bulletins and technical reports will report on; alcohol-related road injury, violence, per capita alcohol consumption and levels of high-risk alcohol consumption in the community.

METHODOLOGY

This study employed an ecologic design utilising routinely collected data on Australian alcohol-caused deaths and hospitalisations. The methodology centres around an aetiologic fraction approach to determining alcohol-caused morbidity and mortality.

The work undertaken for this report was innovative in that national rates of alcohol-caused harm based on yearly estimates of attributable risk (alcohol aetiologic fractions) were calculated. Since any risk of death or hospitalisation attributable to high-risk alcohol consumption (hazardous/harmful levels, see Pols & Hawks, 1992) is directly influenced by the prevalence of high-risk drinking in a community, it is important that any changes in prevalence over time be incorporated into risk estimates. Therefore, in order to estimate the contribution of alcohol misuse to national numbers of deaths and hospitalisations, the methodology employed in this report utilised estimates of alcohol aetiologic fractions (English *et al*., 1995) subsequently adjusted according to national levels of per capita alcohol consumption. Previous work on alcohol-caused morbidity and mortality generally applied only standard aetiologic fractions (eg English *et al*., 1995) without making adjustments for differences across years as a result of changing prevalence (eg. Jonas *et al*., 1999; Stockwell *et al*., 1996; English *et al*., 1995; Higgins *et al.,* 2000). For individual states/territories estimates of per capita consumption were not available and only trends in wholly alcohol-caused conditions were presented.

SUMMARY OF MAIN FINDINGS

During 1997, 3,290 Australians died from injury and disease caused by high-risk drinking. Seventy percent (2296), of these people were male and most of them died from either alcoholic liver cirrhosis, stroke, road injuries, suicide or alcohol dependence. Among females, the most common alcohol-caused diseases and injury included stroke, alcoholic liver cirrhosis, road injuries, alcohol dependence and assault.

On average, 19 years of life were lost for each person who died prematurely from an alcohol-caused condition. The discrepancy between males and females in years of life lost was also marked with was males who died from alcohol-caused diseases or injuries loosing an average of 21 years and females losing 15 years on average.

Chronic conditions, which arise from long term high-risk drinking (ie, alcoholic liver cirrhosis, alcohol dependence) account for the greatest proportion of all alcohol-caused deaths while acute conditions, generally associated with bouts of intoxication (ie, road injury, assault) contribute to the greatest proportion of all prematurely lost years of life.

People who died from injuries and disease generally associated with bouts of intoxication (acute conditions) were younger and most commonly aged between 15 and 29 years, while older people between the ages of 40 and 74 years were more likely to die from conditions related to long term alcohol misuse (chronic conditions).

Male alcohol-caused death rates declined rapidly between 1990 and 1993 but showed little evidence of decline in the following years. Female alcohol-caused death rates decreased slowly but consistently between 1990 and 1997. Male death rates in particular appeared to follow trends in national per capita alcohol consumption.

In South Australia, Western Australia and the Northern Territory, there were higher rates of alcohol-caused death in non-metropolitan regions than in the capital cities.

Indigenous populations in South Australia, Western Australia and the Northern Territory had higher rates of alcohol-caused death than non-Indigenous populations. Indigenous alcohol-caused death rates were not presented for other states/territories due to a lack of information concerning aboriginality.

Between 1993/94 and 1996/97 overall hospitalisation rates appeared to remain relatively stable with some evidence of a small increase in female admission rates. During 1996/97, high-risk drinking was responsible for 72,302 hospitalisations and 403,795 hospital bed-days in Australia. Similar to alcohol-caused deaths, 70% of people attending hospital for an alcohol-caused condition were male. Male hospitalisations were largely due to falls, alcohol dependence, assaults or road injuries.

CONCLUSIONS AND RECOMMENDATIONS

This research represents an important update and development of previous work quantifying alcohol-caused morbidity and mortality in Australia (English *et al*., 1995).

The main findings show that, in spite of a decline in per capita alcohol consumption, well over three thousand Australians die each year from alcohol-caused disease and injury. These deaths confer a considerable cost to society in both human and economic terms. Similarly, it would appear that the rate of alcohol-caused hospitalisation has been increasing over the period considered which also presents a considerable burden on the community's limited health resources.

The burden of alcohol-caused deaths is distributed unevenly across the population. Males are over-represented in mortality statistics compared to women, as are those living in non-metropolitan regions compared to metropolitan regions and Indigenous Australians compared to non-Indigenous Australians. This has implications for the allocation of resources for the prevention of alcohol-caused death.

The age discrepancy between acute and chronic types of alcohol-caused conditions also has implications for public policy and associated economic costs and benefits. Although, the majority of alcohol-caused deaths arise from chronic conditions, they generally occur among older people and hence the associated premature loss of years of life is far less than that caused by acute conditions (e.g. road injury and assault) which tend to occur among young people. Notably, the bulk of the economic cost of alcohol-caused morbidity and mortality arises from productivity losses due to premature death (Chikritzhs *et al*. 1999; Collins & Lapsley, 1996).

The geographic pattern of hospitalisation does not mirror that for alcohol-caused death and it is assumed this is most likely due to admission practices and bed availability as well as demand. As other alcohol-caused harms are considered by the NAIP it will be of interest to consider the geographic overlap between measures. It may be that some locations with high levels of alcohol consumption and related disease and injury are inadequately resourced.

One of the aims of the NAIP is to produce time and region specific alcohol aetiologic fractions, which play an integral part in accurately estimating alcohol-caused morbidity and mortality. This has been achieved nationally by modelling alcohol aetiologic fractions on changes in estimates of national per capita consumption. Until 1997, it was also possible to obtain per capita consumption data for most states and territories. However, changes to federal legislation have resulted in the cessation of volumetric data collection by most liquor licensing jurisdictions (with the exception of WA, NT and Qld). Since it is not possible to obtain the information necessary for calculating per capita consumption from any other source, (ie cannot be determined from survey data) the current lack of information has directly hindered progress in this area.

As the NAIP progresses, future bulletins will update estimates of alcohol-caused morbidity and mortality. In addition, further evidence of the relationship between

alcohol disease and injury will no doubt continue to progress and lead us to re-asses our understanding of the various relationships and how alcohol-caused deaths and hospitalisations are quantified. Work is currently underway at the AIHW to update and fine tune estimates of relative risk and aetiologic fractions for some sentinel alcohol-related conditions, ie. road injury, stroke, falls and female breast cancer, all of which will be incorporated in future reports.

1 INTRODUCTION

Alcohol is a leading cause of death, injury and illness in Australian society. The annual cost of alcohol-caused problems to the nation has been conservatively estimated at over $4.5 billion – about 2.6 times the cost for illicit drugs (Collins and Lapsley, 1996). Prior to this report, the most recent estimate of alcohol-caused morbidity and mortality in Australia was for 1992 (English et al., 1995). At that time, it was estimated that a total of 3,660 deaths were caused by high-risk drinking, in comparison, the total number of deaths due to illicit drugs was estimated at 488. Higgins *et al.* (2000) estimated that in 1997, 832 deaths were caused by illegal drug use – about 4 times fewer than the number of deaths due to high risk drinking. These costs, in both human and economic terms, highlight the need for accurate and reliable data on alcohol consumption and related harms.

Funded by the National Drug Strategy, the National Alcohol Indicators Project (NAIP) is a nationally coordinated project in which trends in alcohol consumption and related harm in Australia will be tracked and monitored at national, state and local levels. The NAIP is a collaborative project between the National Drug Research Institute (Curtin University of Technology) and Turning Point, Alcohol and Drug Centre Inc.

The aim of the NAIP is to increase awareness of the consequences of alcohol consumption in Australia. The outputs of the NAIP will be a series of bulletins and reports featuring key findings from analyses of available data sources on alcohol consumption and related harm. These data sources include those relating to adult per capita alcohol consumption (including the proportion consumed at high-risk levels) and alcohol-caused road injury, assault, death and hospitalisation. By providing information from a range of sources, the likelihood of deriving accurate conclusions about overall levels and trends in alcohol-caused harm is greatly improved.

The Bulletins emanating from the project will provide detailed statistics on available alcohol-related datasets suitable for presentation to a wide audience. These Bulletins will be supplemented by Technical Reports which will provide, in addition to the statistics presented in the Bulletins, detail on the methods used to generate, analyse and interpret the data. Each Technical Report will endeavour to utilise the latest data

available from the sources employed at the time of analysis and it is anticipated that data will be updated annually.

This Technical Report, the first of the NAIP series, documents population-based rates of death and hospitalisation caused by high-risk alcohol consumption (hazardous and harmful alcohol consumption as defined by Pols & Hawks, 1992) in Australia from 1990-1997. These rates of death and hospitalisation are considered at both national and state levels and are also contrasted between metropolitan and non-metropolitan areas of the country. The rates are also contrasted between Indigenous and non-Indigenous populations where possible. The consequences of high-risk levels of alcohol consumption were chosen because outcomes associated with high-risk drinking generally have a greater application to public health policy and practice (English *et al*., 1995).

The work undertaken for this report was innovative in that national rates of alcohol-caused harm based on yearly estimates of attributable risk (alcohol aetiologic fractions) were calculated. Since any risk of death or hospitalisation attributable to high-risk alcohol consumption is directly influenced by the prevalence of high-risk drinking in a community, it is important that any changes in prevalence over time be incorporated into risk estimates. Previous work on alcohol-caused morbidity and mortality generally applied only standard aetiologic fractions (eg English *et al*., 1995) without making adjustments for differences across years in aetiologic fraction as a result of changing prevalence (eg Jonas *et al*., 1999; Stockwell *et al*., 1996; English *et al*., 1995; Higgins *et al*., 2000). This study has utilised estimates of national per capita consumption to adjust aetiologic fractions to reflect annual levels of prevalence of high-risk drinking for the nation.

2 METHODOLOGY

An ecologic design was used in the present study in which routinely collected data on Australian alcohol-caused deaths and hospitalisations was analysed for trends over time as well as patterns across different geographic regions of the country. Major variables under consideration included the age and sex of the persons dying or being hospitalised as well as their Indigenous status. Where possible, aetiologic fractions were used in order to document the contribution of alcohol to deaths and hospitalisations in Australia and these were based on those calculated by English *et al*. (1995).

2.1 DATA SOURCES

Mortality data was sourced from the Australian Bureau of Statistics (ABS) Mortality Datafile, which is a compilation of details of all Australian deaths obtained from state and territory Death Registries. The ABS Mortality Datafile codes age at death, sex, date of registration of death, date of death, cause of death and place of residence for all cases (according to Australian Standard Geographic Classification systems). Cause of death (both primary diagnosis and any applicable external causes of death, E-codes) is recorded on the Mortality Datafile according to International Classification of Diseases *9th revision*, Clinical Modification (ICD-9-CM). Mortality data analysed in this report covered the calender years 1990 to 1997.

Data on hospitalisations was obtained from the Australian Institute of Health and Welfare's (AIHW) compilation of clinical information on hospital separations (equated here to hospitalisations) occurring within each Australian state/territory. Queensland data was obtained directly from the Queensland Health Department, as the AIHW compilation does not include detailed data on place of residence. This compilation codes information on age at admission, sex, primary cause of admission and place of residence for all cases. Cause of hospitalisation (both primary diagnosis and any applicable external causes) is coded using the ICD-9-CM classification system. Hospitalisation data analysed in this report cover the 1993/94 to 1996/97 fiscal years with the final two years providing the most reliable and thoroughly validated information.

Population statistics for the nation and each state/territory were derived from annual ABS estimates of residential population (ERP) by five year age cohort (0-4yrs, 5-9yrs…) and sex.

National per capita consumption data utilised for estimating change in prevalence of high-risk drinking was obtained from World Drink Trends (1998).

2.2 ALCOHOL AETIOLOGIC FRACTIONS

Alcohol aetiologic fractions define the degree to which alcohol is known to be a causal factor in any particular disease or injury, and are necessary for the derivation of rates of alcohol-caused morbidity and mortality. The aetiologic fractions generated for use in this report are derived from those published by English *et al*. (1995) in their meta-analysis of alcohol-caused morbidity and mortality.

English *et al*. (1995) identified 37 conditions for which there is sufficient evidence to conclude that hazardous/harmful (high-risk) alcohol consumption is a causative factor in the development of disease or occurrence of injury[1] and calculated aetiologic fractions for each of these conditions. Some of these conditions are entirely due to high-risk drinking (ie, wholly attributable to alcohol) while others are only partially caused by high-risk drinking (ie, partially attributable to alcohol). Wholly attributable conditions have an aetiologic fraction of one (eg alcoholic liver cirrhosis, alcoholic gastritis, alcohol abuse) which does not change over time or vary with the level of high-risk consumption in a community. However, in the case of partially attributable fractions (eg assaultive injury, road injuries and stroke), the size of the aetiologic fraction is dependent on two main factors - a) the prevalence of high-risk drinking in the population and b) the magnitude of the association between exposure and disease – the relative risk (RR), or alternatively, an estimate of RR such as the Odds Ratio.

Aetiologic fractions can be estimated directly or indirectly (English *et al*., 1995). The direct method involves attribution of high-risk alcohol consumption to the development of a disease or the occurrence of an injury on the basis of case-series studies. This

[1] English *et al*. (1995) identified 38 conditions attributable to alcohol due to hazardous or harmful consumption. Thirty seven of these were shown to be caused by high-risk alcohol consumption while high-risk consumption was shown to have a protective effect for cholelithiasis.

method, used to determine conditions wholly attributable to alcohol, has also been used for partially attributable conditions (particularly those involving injury). For example, case-series studies have been used to estimate the proportion of all road fatalities that may be reasonably attributed to alcohol on the basis of driver blood alcohol concentration (BAC). This proportion is then used as the aetiologic fraction. However, aetiologic fractions calculated in this way are generally specific to the community from which they were derived and should only be applied to other populations where possible influencing factors are similar. For instance, in the case of road injury, when applying an aetiologic fraction to a non-sampled population, factors such as road conditions, vehicle usage and importantly, alcohol consumption should all be comparatively similar to the community from which the aetiologic fraction was derived.

The preferred method for estimating aetiologic fractions for partially alcohol attributable conditions is the 'indirect' method. Aetiologic fractions derived in this way require knowledge of both the condition-specific Relative Risks (RR), derived from cohort, case-control or cross sectional prevalence studies, and the prevalence of high-risk alcohol consumption in the relevant community, generally derived from population surveys. This information is then combined in a standard equation, *(Equation 1)*, for calculating aetiologic fractions where the exposure variable has several categories (ie low, hazardous, harmful) (Walter, 1976). The partial aetiologic fraction for a particular level of exposure, i, in the general population can be expressed as follows;

(Equation 1)

$$AFi = \frac{p_i(RR_i - 1)}{\sum_{i=0}^{k} p_i(RR_i - 1) + 1}$$

where,

AF_i = population aetiologic fraction for a specific condition at a particular category of exposure (ie, hazardous or harmful), i.

P_i = the estimated prevalence of the i^{th} category of exposure in the total population, and

RR_i = the relative risk (or estimate of relative risk), for the i^{th} category of exposure to the reference category.

English *et al*. (1995) argue that the RR estimates generated from their meta-analysis are typically transferable across communities/regions and across time. However, aetiologic fractions calculated indirectly are also dependent upon the prevalence of high-risk alcohol consumption, which has been shown to vary both across regions and across time. So as to take account of these changes, one of the NAIP objectives is to calculate aetiologic fractions that are temporally and regionally specific.

2.2.1 Estimating year-specific aetiologic fractions

The prevalence of high-risk alcohol consumption is generally measured through population surveys. English *et al*. (1995) used the 1989/90 National Health Survey and the 1989 National Heart Foundation Survey to estimate the prevalence of high-risk alcohol consumption. There have been no equivalent national surveys using similar methodologies since this time (see Donath, 1999). However, from 1989 to the early 1990's per-capita alcohol consumption declined about 10% (World Drink Trends, 1998). In order to adjust the aetiologic fractions for this change in consumption over time, year-specific aetiologic fractions were calculated based on changes evident in per capita consumption since 1989/90 (used as the reference year). Such adjustment is reasonable given that there is a consistent positive linear relationship between mean per capita alcohol consumption and the proportion of individuals consuming at high-risk levels (Edwards *et al.*, 1995). In this report, any change in per-capita consumption over time is assumed to produce a corresponding decline in the proportion of people consuming alcohol at high-risk levels across both sexes and all age groups. No regional adjustment was attempted, as region-specific per-capita consumption estimates were not available at the time of compilation of this report.

The following formula was used to determine change in national per capita alcohol consumption relative to the reference year:

(Equation 2)

$$F = \frac{PCC_x - PCC_{ref}}{PCC_{ref}}$$

where,

F = change factor in per capita consumption from year of interest to reference year,

$\boldsymbol{PCC_{ref}}$ = per capita consumption for reference year (1989/90), and

$\boldsymbol{PCC_x}$ = per capita consumption for year of interest

This change factor *F* obtained through *Equation 2* was then applied to the aetiologic fractions calculated by English *et al*. (1995). Of the 38 fractions calculated by English *et al*. (1995), 11 of the partially attributable conditions were obtained using the direct method. For these conditions the change Factor *F* was applied to the following equation to derive year specific aetiologic fractions:

(Equation 3)

$$AF_x = \frac{\left(F * AF_{ref}\right) + AF_{ref}}{\left(\left(F * AF_{ref}\right) + AF_{ref}\right) + \left(1 - AF_{ref}\right)}$$

where,

$\boldsymbol{AF_x}$ = the estimated alcohol population aetiologic fraction for a specific condition, at a particular level of exposure during the year of interest, and

$\boldsymbol{AF_{ref}}$ = the alcohol population aetiologic fraction for a specific condition at a particular level of exposure for the reference year (1989/90)

For the 15 aetiologic fractions English *et al*.(1995) calculated via the indirect method (conditions where estimates of relative risk were available), adjustments to condition specific aetiologic fractions were made through application of the derived change factor to the prevalences used in *Equation 1*. These adjusted estimates were then contrasted to those obtained through the application of *Equation 3* to the English *et al*. (1995) fractions. The percentage difference between the two sets of estimated fractions was negligible, with an average difference of less than 1% and a median difference of less than 0.5%. Given that the two methods for adjusting the fractions produced largely identical estimates, *Equation 3* was utilised to adjust all aetiologic fractions to ensure consistency across all alcohol-caused conditions.

2.2.2 Aetiologic fraction for elderly falls

English *et al.,* (1995) assigned an aetiologic fraction of 0.38 to all fall injuries regardless of age or sex on the basis of case series studies. Jonas *et al.,* (1999) point out that this fraction greatly overestimates the involvement of alcohol in fall injuries amongst the elderly and, as a consequence, assigned an alcohol aetiologic fraction of zero to those falls in the over 65 age group. This change has significant implications for comparison with English *et al.*'s (1995) work, which showed a large proportion of hospital bed days attributable to elderly falls.

2.3 DERIVING RATES OF ALCOHOL-CAUSED MORBIDITY AND MORTALITY

2.3.1 Data extraction and classification

Cases of death or hospitalisation were extracted from the datasets on the basis of ICD-9-CM codes for the conditions identified by English *et al*. (1995) as being wholly or partially attributable to high-risk alcohol consumption (see Table 2.3a). Cases were assigned to age, sex, regional and ethnic groupings on the basis of information contained in relevant fields included in the datasets. Length of stay (bed days) was also extracted from the hospitalisations dataset. Cases of alcohol-caused death were grouped into calender years on the basis of year of registration of death rather than year of death. However, it should be noted that less than 5% of deaths are registered in a year other than the year of death. Cases of alcohol-caused hospitalisation were grouped into financial year on the basis of year of admission.

Following the identification of cases, deaths and hospitalisations were recoded into sex specific five year age cohorts beginning with ages 0-4. For mortality data the oldest age group was 85+ years while for morbidity data individual ages were only identifiable to 75 years.

Table 2.3a: Conditions identified by English *et al*. (1995) as partially or wholly attributable to high-risk alcohol consumption according to causation grouping

Acute	Chronic	Mixed
Acute pancreatitis	Alcoholic cardiomyopathy[1]	Stroke
Alcohol abuse[1]	Alcoholic dependence[1]	Suicide
Alcoholic beverage poisoning[1]	Alcoholic liver cirrhosis[1]	
Alcoholic gastritis[1]	Alcoholic poly neuropathy[1]	
Alcoholic psychosis[1]	Chronic pancreatitis	
Aspiration[1]	Epilepsy	
Assault	Female breast cancer	
Child abuse	Hypertension	
Drowning	Laryngeal cancer	
Ethanol toxicity[1]	Liver cancer	
Fall injuries	Oesophageal cancer	
Fire injuries	Oesophageal varices	
Gastro-oesophageal haemorrhage	Oropharyngeal cancer	
Low birthweight	Psoriasis	
Methanol toxicity[1]	Unspecified liver cirrhosis	
Occupational and machine injuries		
Other ethanol and ethanol poisoning[1]		
Road injuries		
Spontaneous abortion		

Supraventricular cardiac dysrhythmias

[1]conditions wholly attributable to alcohol consumption

2.3.2 Data manipulation

In order to derive adjusted counts all alcohol attributable deaths and hospitalisations were multiplied out by their year, sex and age specific aetiologic fractions[2]. For the hospitalisation data, aetiologic fractions corresponding to each condition were also applied to numbers of associated bed-days in order to derive adjusted counts of the length of hospital stay due to high-risk alcohol consumption. Data were summed according to the groupings detailed below and crude, age-specific and age-standardised rates were calculated where appropriate from these counts.

Crude rates of alcohol-caused death and hospitalisation were calculated by dividing the counts obtained by the estimated residential population (ERP) relevant to the grouping of interest (eg national, state-specific). Population rates were generated per 10,000 adult persons for males and females.

Crude rates do not account for age-structure within a population (which has been shown to affect levels of alcohol consumption and harm) and are therefore not suitable for inter-regional comparisons (Rothman, 1986). Age-adjustment was undertaken at the national level through the calculation of age-specific rates. These were obtained by dividing the number of alcohol-caused cases in a particular age group by the population of that same age group and multiplying by 10,000.

Age-adjustment was undertaken at other geographic levels through the calculation of age-standardised rates (ASRs) through the direct method using the age structure of the 1997 national population as the reference. This involved the comparison of the age distribution within a defined region with a standard overall age distribution. Rates are then weighted depending on the degree and direction of disparity between these distributions and finally multiplied by 10,000. In this way, differences in age structure between defined populations are removed, allowing direct comparisons between regions.

[2] It should be noted that for some conditions, only overall aetiologic fractions were available and not age or sex specific.

Drinking patterns and alcohol-caused conditions

Different drinking patterns are more likely to be associated with particular alcohol-caused conditions (Jonas *et al*., 1999; Stockwell *et al.,* 1996). Long term regular misuse of alcohol is implicated in conditions such as alcoholic liver cirrhosis while conditions such as road injury and assault are more likely to result from short term episodes of intoxication. In order to capture this distinction, mortality and morbidity cases were categorised into three different categories of alcohol causation, that is, those resulting from: (1) long-term drinking (chronic), (2) short-term episodes of intoxication (acute), or (3) mixed causality (see Table 2.3a). Although few conditions are entirely acute or chronic in nature, the division is useful in delineating between the associated consequences of the two major types of harmful drinking patterns[3]. Notably, both suicide and stroke have equally large acute and chronic components and do not fit neatly into any single category and have been categorised as 'mixed'. Separate rates of alcohol-caused morbidity and mortality were generated according to this categorisation where possible.

2.3.3 Regional comparisons

National estimates were generated on the basis of all cases partially and wholly attributable to alcohol consumption. However, state/territory estimates were only generated on the basis of conditions wholly attributable to alcohol consumption because state-specific aetiologic fractions could not be compiled for those conditions partially attributable to alcohol. Therefore, population rates for individual states are not complete measures of the overall quantification of deaths and hospitalisations but are indicative of trend. At a national level, wholly alcohol attributable conditions account for approximately 37% of all deaths and 31% of all hospitalisations caused by alcohol.

Comparisons between metropolitan and non-metropolitan regions within each state/territory were generated on the basis of the Statistical Division (SD) to which cases were assigned according to the Australian Standard Geographical Classification (ASGC). Specifically, cases with an SD code of '05' in all states except Queensland

[3] Acute and chronic conditions were identified from the Measurement of Alcohol Problems for Policy technical report by Stockwell *et al*.(1997).

were classified as metropolitan, with all other areas considered non-metropolitan. As Queensland did not supply ASGC statistical division information for the hospitalisation data, Rural Remote Metropolitan Area classifications included in the dataset, which also allow the identification of SD code '05', were used.

As only a very small number of persons live in non-metropolitan ACT, only metropolitan rates have been provided.

2.3.4 Indigenous Australians

The ABS Mortality Datafile classifies cases according to whether they are of Indigenous (persons of Aboriginal or Torres Straight Islander decent) or non-Indigenous origin to a publishable standard in Western Australia, South Australia and the Northern Territory. Nevertheless, the annual number of Indigenous deaths is relatively small, with the number attributable to alcohol even smaller. As a consequence, all Indigenous death data from 1991 to 1997 were combined (1990 was omitted due to data limitations) to determine the rate of alcohol-caused deaths among Indigenous Australians in these states. These deaths were also classified according to metropolitan/non-metropolitan regions. In calculating these rates it was assumed that the proportions of Indigenous persons in each region was the same as that evident in 1996 (the only year for which ABS data estimating Indigenous populations at the metropolitan/non-metropolitan level was available).

2.4 CONTROL DIAGNOSES FOR MORTALITY

Variation in the rate of alcohol-caused mortality may merely reflect variation in the overall mortality rates under consideration. In order to examine whether or not this was the case, mortality rates for Australia were also calculated for non-alcohol-caused conditions. Non-alcohol-caused conditions, defined as those that had an aetiologic fraction of zero for all age and sex groups, were employed as controls. For instance, falls among people aged over 65, assigned an aetiologic fraction of zero (see above), were not included as controls since the condition was otherwise associated with high-risk alcohol consumption.

2.5 CALCULATING PERSON-YEARS OF LIFE LOST

Following the identification of reliable alcohol aetiologic fractions it is possible to calculate the number of years of life lost to persons who died prematurely due to high-risk alcohol consumption. For this report, sex specific person-years of life lost (PYLL) were calculated for 1997 for each individual condition, all acute conditions combined, all chronic conditions combined, as well as for all-cause mortality using a life table method originally devised by Chiang (1968) and later described by Hakulinen and Teppo (1976). Essentially, life table methods recognise that for each individual, the likelihood of surviving to an average life expectancy differs as a function of age. Even though persons may survive a condition at a certain age, they remain at risk of succumbing to other remaining causes of death. The life table method therefore takes into account residual mortality from other causes at specified age intervals.

Calculations for total lifetime PYLL usually include sex specific deaths grouped into 5 year age categories (0-4yrs, 5-9yrs….) reaching a maximum of 85 years. Nevertheless, previous researchers have tended to only calculate PYLL to age 69 assuming that average life expectancy in most developed countries (at the time) was around 70-74 years (Holman *et al.*,1988, English *et al.*,1995, Hakulinen and Teppo 1976). However, the average life expectancy between 1994 and 1996 for Australians was 75 years for males and 84 for females. Therefore, it is likely that PYLL calculated to 69 years represents a slight underestimate. However, to maintain comparability with previous national estimates of PYLL due to high-risk alcohol consumption (English *et al.*, 1995), total PYLL from deaths occurring between the ages of 0 and 69 years was calculated[4].

As the formula for calculating PYLL is numerically complex it has not been presented here. For a detailed explanation of the formulae used for calculating PYLL in this report, the reader is referred to English *et al*. (1995) pp.17-19. A software program known as the Rates Calculator (Codde, 1999) was used to calculate PYLL[5].

[4] Estimates of PYLL to 74 years have also been calculated and can be obtained upon request.

[5] The authors gratefully acknowledge the assistance of Dr Jim Codde, Director of Epidemiology and Analytical Services, Western Australian Health Department for the use of the Rates Calculator (1999) and for his helpful advice regarding PYLL calculations.

3 RESULTS AND DISCUSSION

3.1 NATIONAL TRENDS IN ALCOHOL-CAUSED DEATHS AND HOSPITALISATIONS

3.1.1 Alcohol-caused deaths, 1990-1997

Between 1990 and 1997 an estimated average of 2,309 male and 1,013 female deaths were caused every year by high-risk alcohol consumption. On average, the age-standardised death rate of alcohol-caused death was approximately 3.4 deaths per 10,000 adult males and 1.5 deaths per 10,000 adult females. Table 3.1a shows crude and age-standardised rates of deaths caused by high-risk alcohol consumption for adult Australian males and females for the years 1990-1997. For the purpose of comparison, rates of deaths unrelated to high-risk alcohol consumption are shown in parentheses in Table 3.1a.

Table 3.1a shows that between 1990 and 1997 the male alcohol-caused death rate was consistently about 2.3 times greater than that for females. This is not surprising as most data shows that males are the largest consumers of alcohol and are more likely to drink at high-risk levels than females (ABS 1994, English *et al.,* 1995). By comparison, the male and female non-alcohol-caused death rates were similar, with the male rate on average only 20% greater than the female rate. The male and female alcohol-caused rates appeared to converge slightly over the period.

The alcohol-caused death rate was highest in 1990 for both males (4.01 per 10,000) and females (1.75 per 10,000). The female age standardised alcohol-caused death rate declined steadily by a total of 24% between 1990 and 1997. In contrast, the male alcohol-caused death rate decreased by 20.4% between 1990 and 1993, but declined only another 1.3% between 1993 and 1997. The rates of death unrelated to high-risk alcohol consumption declined steadily for both males (14.5%) and females (7.7%) between 1990 and 1997 with no plateau effect evident after 1993 for males. It appears therefore, that the downward trend in overall numbers of male deaths in more recent years has not been reflected in alcohol-caused disease and injury.

Table 3.1a: Estimated national, crude and age standardised death rates for deaths caused by high-risk alcohol consumption per 10,000 adults (15+yrs) by sex, 1990-1997 (Equivalent rates for deaths unrelated to high-risk alcohol consumption in parentheses)

	Males (15+yrs)			Females (15+yrs)		
Year	**N**	**Crude rate/10,000**	**ASR/ 10,000**	**N**	**Crude rate/10,000**	**ASR/ 10,000**
1990	2560 (50410)	3.89 (76.56)	4.01 (86.93)	1093 (40578)	1.63 (60.34)	1.75 (68.21)
1991	2377 (50090)	3.56 (75.07)	3.66 (83.75)	1059 (40518)	1.55 (59.36)	1.64 (66.04)
1992	2351 (52110)	3.48 (77.11)	3.57 (84.58)	985 (43002)	1.42 (62.14)	1.49 (68.10)
1993	2134 (51594)	3.12 (75.55)	3.19 (81.29)	971 (41836)	1.39 (59.77)	1.44 (64.14)
1994	2247 (53285)	3.25 (77.14)	3.30 (81.62)	1020 (43985)	1.44 (62.08)	1.47 (65.53)
1995	2243 (52224)	3.20 (74.62)	3.24 (77.50)	1012 (43586)	1.41 (60.68)	1.43 (62.94)
1996	2294 (53671)	3.23 (75.57)	3.25 (77.11)	985 (45240)	1.35 (62.01)	1.36 (63.26)
1997	2269 (53605)	3.15 (74.38)	3.15 (74.37)	986 (46594)	1.33 (62.94)	1.33 (62.94)

Figure 3.1a shows national trends between 1990 and 1997 in per capita consumption of pure alcohol (World Drink Trends, 1998), rates of death due to high-risk alcohol consumption and non-alcohol-caused death rates for males and females. Per capita consumption declined by 9% between 1990 and 1993 becoming relatively stable over 1994 to 1997.

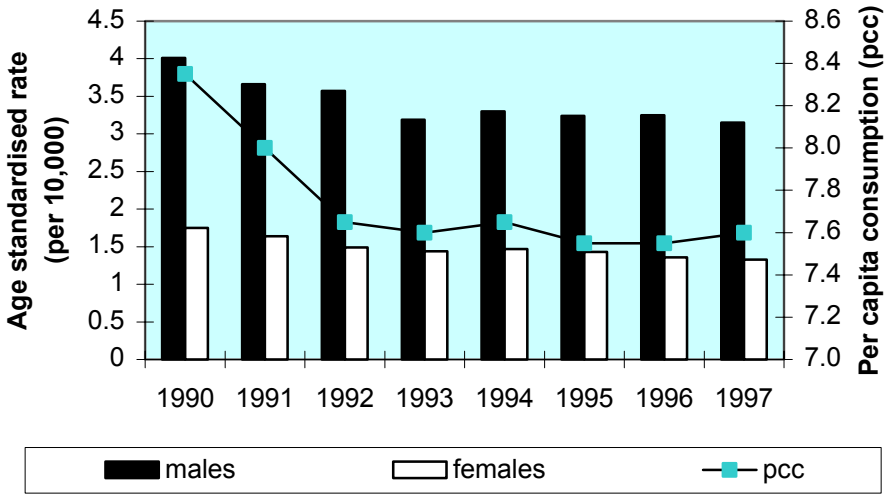


Figure 3.1a: Per capita consumption (PCC) of pure alcohol (litres) and national trends in age standardised rates (per 10,000) of alcohol-caused deaths and non-alcohol-caused deaths for adult (15+yrs) males and females, 1990-1997

The decline in deaths caused by high-risk alcohol consumption parallels the decline in per-capita consumption. This finding may merely be an artefact of the procedures used to adjust for partially alcohol-attributable conditions. In order to examine this issue, crude and age-standardised rates of death for conditions wholly attributable to high-risk alcohol consumption, for which no adjustment procedures were used, were calculated. Table 3.1b shows these rates for the years 1990 to 1997. This Table shows that the wholly attributable conditions follow a similar pattern to per capita alcohol consumption, which suggests that the decline evident in Table 3.1a for all alcohol-caused conditions reflects a real trend that is not an artefact of the method of adjusting aetiologic fractions.

Table 3.1b: Estimated national, crude and age standardised death rates for wholly alcohol attributable conditions per 10,000 adults (15+yrs) by sex, 1990-1997

	Males (15+yrs)			Females (15+yrs)		
Year	**N**	**Crude rate/10,000**	**ASR/ 10,000**	**N**	**Crude rate/10,000**	**ASR/ 10,000**
1990	1014	1.54	1.62	276	0.41	0.43
1991	936	1.40	1.47	269	0.39	0.40
1992	1015	1.50	1.56	234	0.34	0.35

1993	829	1.21	1.26	243	0.35	0.36
1994	914	1.32	1.36	270	0.38	0.39
1995	946	1.35	1.37	263	0.37	0.37
1996	948	1.33	1.35	253	0.35	0.35
1997	959	1.33	1.33	254	0.34	0.34

3.1.2 Alcohol-caused hospitalisations, 1993/94-1996/97

Table 3.1d shows the estimated numbers and age-standardised rates of hospitalisations caused by high-risk alcohol consumption occurring between 1993/94 and 1996/97 for adult males and females (15+yrs). On average there were an estimated 47,410 hospitalisations resulting from high-risk alcohol consumption among males and an estimated 21,338 among females which mirrors the deaths data considered above. In this regard on average the age-standardised rate for males was 2.3 times greater than that for females. Inspection of Table 3.1c suggests a small increase in the number and rate of alcohol-caused hospitalisations over time. This increase, may however, reflect changes in hospital admission practices rather than any real increase in alcohol causation (see Hanlin *et al.*, in preparation).

Table 3.1c: Estimated numbers and age standardised hospitalisation rates for hospitalisations caused by high-risk alcohol consumption per 10,000 Australian adults (15+yrs) by sex, 1993/94-1996/97

Year	Males (15+yrs)		Females (15+yrs)	
	N	ASR/10,000	N	ASR/10,000
1993/94	46521	67.18	20263	28.61
1994/95	46396	66.13	20688	28.81
1995/96	48192	67.79	22003	30.17
1996/97	48533	67.34	22399	30.26

3.1.3 Age-cohort trends

Figure 3.1b shows the number of deaths caused by high-risk alcohol consumption along with age-specific death rates for male and female Australians in 1997 broken down into 5 year age-cohorts (see Appendix A for detailed tables). Age specific alcohol-caused death rates increased with increasing age, peaking from age 60 onwards. There was, however an earlier peak among males aged between 20 and 29 years which reflects high

rates of injury-related deaths among this age group. Female death rates peaked among the older age groups, with the exception of 75 to 79 year olds, where an overall protective effect of stroke was found. Persons aged less than 15 years accounted for about 1% of all alcohol-caused deaths.

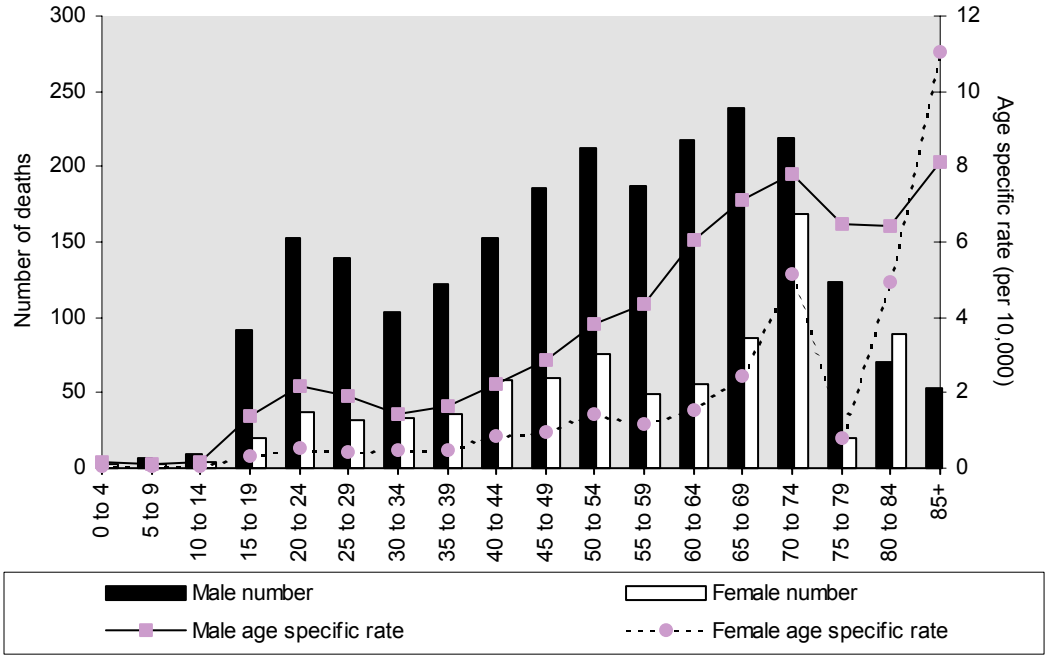


Figure 3.1b: Estimated national numbers and age-specific rates (per 10,000) of alcohol-caused deaths by age group and sex, 1997

Figure 3.1c shows the number of hospitalisations caused by high-risk alcohol consumption along with age-specific death rates for male and female Australians in 1996/97 broken down into 5 year age-cohorts (also see Appendix A). As with the death rates considered above, there was a notable peak in hospitalisation rate among young males aged between 20 and 29. However, in contrast to death rates, the rate of alcohol-caused hospitalisations for males was consistently high over the 20-64 year old age range. The pattern for females was markedly different with a general increase as a function of increasing age with a marked peak evident in the 60-64 year-old age group. Nevertheless, for both sexes, the continuing upward trend noted for deaths after age 64 was not evident in the hospitalisations. Again, hospitalisations among those aged under 15 years only accounted for a very small proportion of all alcohol-caused admissions – less than 2%.

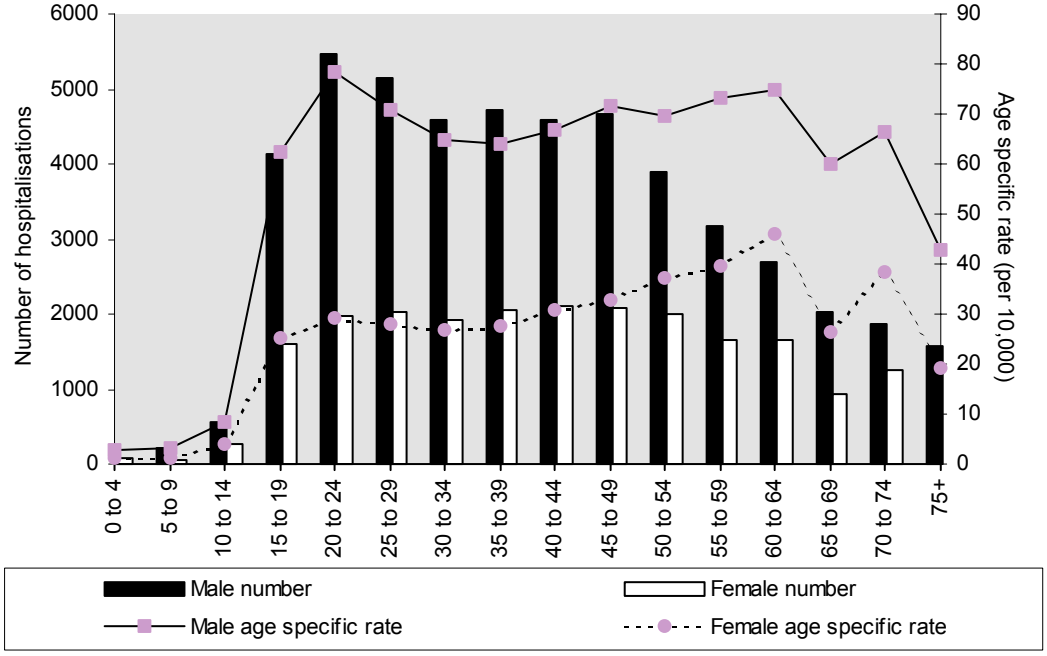


Figure 3.1c: Estimated national numbers and age-specific rates (per 10,000) of alcohol-caused hospitalisations by age group and sex, 1996/97

3.1.4 Trends in deaths and hospitalisations by alcohol-caused condition category

Table 3.1d shows the numbers of deaths caused by high-risk alcohol consumption along with age-standardised rates for males and females in Australia broken down according to type of condition. There were more alcohol-caused deaths from conditions associated with long-term heavy drinking than those associated with episodes of acute intoxication. Strokes and suicides also accounted for a substantial minority of these alcohol-caused deaths.

Table 3.1d: Estimated national numbers and age standardised rates (per 10,000) of alcohol-caused deaths by condition category among males and females

	Chronic		Acute		Stroke		Suicide	
	N	ASR/ 10,000	N	ASR/ 10,000	N	ASR/ 10,000	N	ASR/ 10,000
Males								
1990	1064	1.71	920	1.38	374	0.63	202	0.30
1991	1026	1.61	797	1.18	350	0.57	204	0.30
1992	1078	1.66	750	1.10	328	0.52	194	0.28
1993	946	1.43	693	1.01	317	0.49	178	0.26
1994	975	1.44	741	1.07	336	0.51	195	0.28
1995	1014	1.47	705	1.00	325	0.48	198	0.28
1996	1015	1.44	753	1.06	324	0.46	202	0.28
1997	1061	1.47	668	0.93	312	0.43	228	0.32
Females								
1990	322	0.50	268	0.41	475	0.80	28	0.04
1991	326	0.49	255	0.37	445	0.72	34	0.05
1992	292	0.44	237	0.34	428	0.67	29	0.04
1993	317	0.46	202	0.29	428	0.65	24	0.03
1994	351	0.50	206	0.29	437	0.64	26	0.04
1995	326	0.46	216	0.30	438	0.62	31	0.04
1996	320	0.44	206	0.28	431	0.60	28	0.04
1997	328	0.44	209	0.28	414	0.56	36	0.05

Table 3.1e shows the numbers of hospitalisations caused by high-risk alcohol consumption along with age-standardised rates for males and females in Australia broken down according to type of condition. In contrast to the deaths data considered above, the majority of hospitalisations caused by high-risk drinking were for conditions associated with episodes of acute intoxication. This finding suggests morbidity associated with high-risk alcohol consumption is greatest for episodes of acute intoxication while mortality is greatest for long-term high-risk consumption.

Table 3.1e: Estimated national numbers and age standardised rates (per 10,000) of alcohol-caused hospitalisations by condition category among males and females

	Chronic		Acute		Stroke		Suicide	
	N	ASR/ 10,000	N	ASR/ 10,000	N	ASR/ 10,000	N	ASR/ 10,000
Males								
1993/94	14657	21.49	28770	41.16	2291	3.39	804	1.14
1994/95	13922	20.05	29233	41.42	2332	3.38	909	1.28
1995/96	14423	20.37	30416	42.67	2375	3.37	978	1.37
1996/97	14663	20.35	30407	42.19	2440	3.39	1023	1.42
Females								
1993/94	5590	7.98	11965	16.81	2072	2.94	635	0.88
1994/95	5225	7.34	12491	17.34	2234	3.12	737	1.01
1995/96	5741	7.90	13207	18.09	2246	3.08	808	1.10
1996/97	6158	8.32	13120	17.72	2276	3.08	845	1.14

Figures 3.1d through 3.1h below show age and sex distributions for acute and chronic conditions as well as suicide, stroke and road injury. For the acute conditions, ages 15 through 49 years predominated, with a notable peak between 15 and 29 years. Conversely, chronic conditions were concentrated between the ages of 40 and 74 years, and highest among the 50 to 74 age group. Notably, alcohol-caused road injury and suicide predominate among males aged between 15 and 29 years of age.

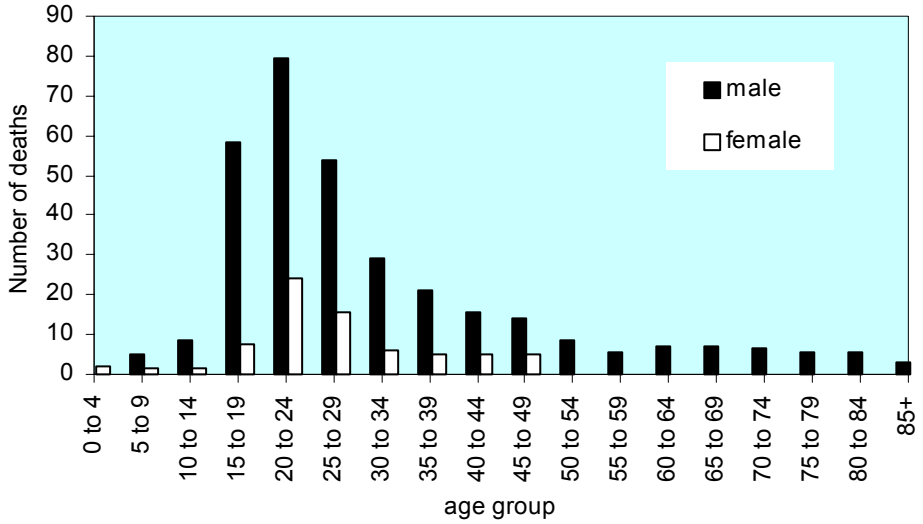


Figure 3.1d: Age and sex distribution among deaths resulting from alcohol-caused road injury, 1997

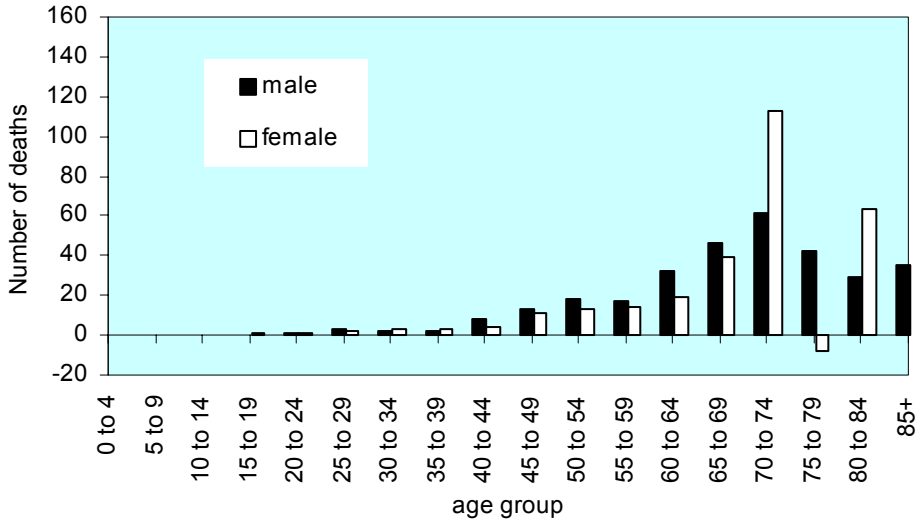
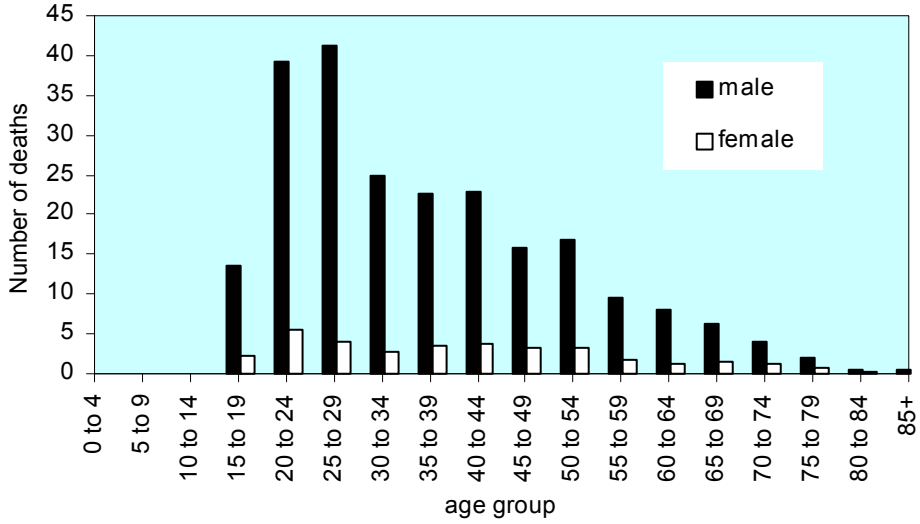


Figure 3.1e: Age and sex distribution among deaths resulting from alcohol-caused stroke, 1997

Evidently, for women aged between 75 and 79 years there were more lives saved than lost to stroke from high-risk alcohol consumption. This arose as a combination of two factors, a) the unique protective effect that a hazardous level of alcohol consumption (2 - 4 drinks per day on average, Pols & Hawks, 1992) impacts on the risk of female stroke and b) the fact that the estimated prevalence of harmful consumption (> 4 drinks



per day, Pols & Hawks, 1992) among 75-79 year old females is close to zero.

Figure 3.1f: Age and sex distribution among deaths resulting from alcohol-caused suicide, 1997

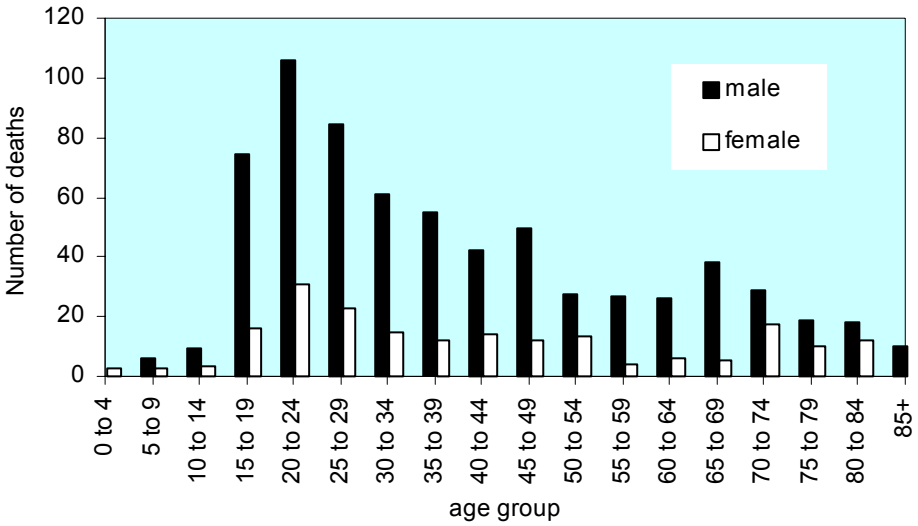


Figure 3.1g: Age and sex distribution among deaths resulting from alcohol-caused acute conditions, 1997

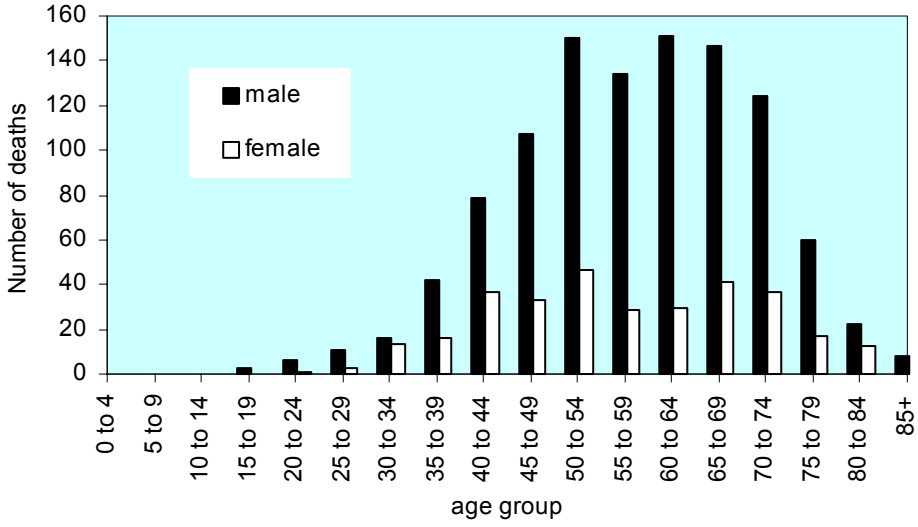


Figure 3.1h: Age and sex distribution among deaths resulting from alcohol-caused chronic conditions, 1997

In summary, section 3.1.3 shows that the rate of alcohol-caused death appears to increase as a function of increasing age, peaking from about age 60 onwards. This peak among older age groups is reflected in the fact that the majority of alcohol-caused deaths were due to conditions associated with long-term heavy drinking. In contrast, hospitalisation rates tended to decline after age 60.

3.2 GEOGRAPHICAL PATTERNS IN ALCOHOL-CAUSED DEATHS AND HOSPITALISATIONS

3.2.1 State-wide trends in alcohol-caused deaths and hospitalisations

State-wide alcohol-caused deaths, 1990-1997

As indicated, state and territory-specific consumption data were not available at the time of compilation of this report. Therefore, only wholly alcohol-caused conditions (e.g. alcoholic liver cirrhosis, alcohol dependence) were used to determine numbers and rates of deaths and hospitalisations at the state and territory level. Raw values of the estimated numbers of alcohol-caused deaths and hospitalisations occurring in each state for available years have been provided in Appendix B.

Table 3.2a shows the rate of deaths wholly attributable to high-risk drinking in all states and territories of Australia for males and females. In all states and territories men had a higher rate of alcohol-caused death than women. There was considerable fluctuation in the rate of deaths in the individual states and territories, which probably reflects the small numbers of wholly alcohol attributable deaths in individual jurisdictions. The Northern Territory consistently maintained the highest alcohol-caused death rate in the country for both sexes.

Table 3.2a: **State-wide age standardised death rates (per 10,000) due to wholly alcohol-caused conditions, males and females, 1990-1997**

	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
Male								
1990	1.85	1.65	1.49	1.26	1.30	1.01	3.56	1.28
1991	1.50	1.51	1.48	1.22	1.30	1.31	3.52	2.08
1992	1.44	1.81	1.48	1.66	1.37	0.98	4.47	1.43
1993	1.42	0.98	1.20	1.41	1.21	1.29	3.84	0.89
1994	1.42	1.34	1.27	1.50	1.23	0.89	2.23	1.32
1995	1.46	1.14	1.30	1.50	1.39	1.34	4.60	1.52
1996	1.38	1.19	1.40	1.42	1.27	1.09	3.84	1.12
1997	1.30	1.26	1.39	1.35	1.31	1.28	4.49	0.90
Female								
1990	0.48	0.36	0.38	0.39	0.50	0.26	3.00	0.39
1991	0.36	0.41	0.37	0.40	0.50	0.65	1.26	0.20
1992	0.42	0.26	0.35	0.22	0.41	0.38	1.44	0.00
1993	0.40	0.28	0.31	0.35	0.48	0.21	1.81	0.27

1994	0.39	0.30	0.33	0.46	0.57	0.37	1.26	0.50
1995	0.44	0.24	0.35	0.36	0.37	0.42	2.61	0.40
1996	0.32	0.28	0.39	0.36	0.52	0.36	1.21	0.12
1997	0.38	0.28	0.31	0.29	0.38	0.25	2.62	0.27

State-wide alcohol-caused hospitalisations, 1993/94–1996/97

Rates of hospitalisations wholly attributable to high-risk drinking in all states and territories of Australia between 1993/94 and 1996/97 have been presented for males and females in Table 3.2b. Male hospitalisation rates exceeded female rates in all jurisdictions and in all years. Rates were consistently lowest in the ACT and highest in Queensland for both males and females over the four years.

There are inherent problems in comparing hospitalisation rates between jurisdictions. Hospital admissions are directly affected by operational limitations such as bed and staff availability, funding and management practices. For example, Victoria was the first state to adopt a 'case mix' system in 1994, in which funding is provided on the basis of services and procedures actually provided by hospitals in terms of the number of cases treated. This change markedly increased the overall number of short stay admissions made in that state (Hanlin *et al*., in preparation). This has direct implications for comparing Victorian hospitalisation rates with all other states/territories. Nevertheless, in spite of this change Victoria had consistently low rates of alcohol-caused hospitalisation.

Table 3.2b: Estimated age standardised rates (per 10,000) for hospitalisations resulting from wholly alcohol-caused conditions by states/territory, males and females, 1993/94-1996/97

	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
Male								
1993/94	29.87	17.70	37.88	20.61	21.53	15.57	19.93	6.18
1994/95	26.37	17.00	36.26	21.02	23.49	14.42	19.92	8.21
1995/96	30.65	16.33	34.50	21.32	22.83	18.36	22.01	6.87
1996/97	30.02	18.13	32.10	19.04	21.77	17.25	22.26	7.12
Female								
1993/94	10.01	7.55	13.23	6.48	8.17	3.73	8.03	3.17
1994/95	8.15	7.32	14.1	7.85	8.38	5.24	7.83	2.94

1995/96	11.14	6.58	12.88	8.01	9.62	6.24	10.01	3.00
1996/97	10.7	8.23	14.4	8.04	9.38	9.66	10.24	3.26

3.2.2 Metropolitan vs non-metropolitan areas

Metropolitan and non-metropolitan deaths, 1990-1997

Table 3.2c shows the total numbers of deaths wholly attributable to high-risk alcohol consumption, along with age-standardised rates, occurring during the period 1990 to 1997 by region for males and females. With the exception of Tasmania, rates of death were highest in the non-metropolitan areas of the states. The Northern Territory had the highest rates of alcohol-cause deaths for both males and females in both metropolitan and non-metropolitan regions. The lowest rates were evident in non-metropolitan Tasmania (1.13 per 10, 0000 adult males) and the ACT (0.28 deaths per 10,000 females).

Table 3.2c: Number and age standardised rate (per 10,000) of deaths wholly caused by high-risk alcohol consumption by metropolitan/non-metropolitan area, Australian states 1990-1997

State	Males (15+yrs)		Females (15+yrs)	
	N	ASR/10,000	N	ASR/10,000
NSW				
metro	1608	1.44	455	0.39
non-metro	1124	1.51	313	0.42
VIC				
metro	1276	1.34	305	0.30
non-metro	561	1.40	125	0.30
QLD				
metro	553	1.34	142	0.33
non-metro	738	1.40	187	0.37
SA				
metro	444	1.33	131	0.36
non-metro	213	1.66	41	0.34
WA				
metro	418	1.19	147	0.40
non-metro	213	1.60	79	0.65
TAS				
metro	69	1.18	28	0.45
non-metro	97	1.13	27	0.30

NT				
metro	57	3.69	26	1.42
non-metro	94	3.99	36	2.34
ACT				
metro	93	1.28	20	0.28

Metropolitan and non-Metropolitan Hospitalisations, 1993/94-1996/97

Table 3.2d shows the total numbers of hospitalisations wholly attributable to high-risk alcohol consumption, along with age-standardised rates, occurring during the period 19993/94 to 1996/97 by region for males and females. As with the deaths data, rates for males were generally highest in non-metropolitan areas (Queensland and Tasmania the exceptions). For females however, the rates were generally higher in the metropolitan areas (South Australia, Western Australia and Northern Territory the exceptions). Western Australia had the greatest disparity between regions of any state.

Table 3.2d: Number and age standardised rates (per 10,000) for hospitalisations resulting from wholly alcohol attributable conditions by state/territory, metropolitan/non-metropolitan and sex, 1993/94-1996/97

State	Males (15+yrs)		Females (15+yrs)	
	N	ASR/10,000	N	ASR/10,000
NSW				
metro	9017	29.85	3499	11.11
non-metro	5753	32.42	1941	10.80
VIC				
metro	4226	16.61	2092	7.78
non-metro	1890	19.31	647	6.57
QLD				
metro	4038	34.80	1824	15.02
non-metro	4654	32.44	1790	12.60
SA				
metro	1312	15.56	613	6.82
non-metro	1023	32.72	348	11.73
WA				
metro	1646	16.32	742	7.04
non-metro	1454	38.39	595	17.06
TAS				
metro	285	19.33	158	10.06

non-metro	355	16.80	143	6.52
NT				
metro	104	15.77	27	5.63
non-metro	222	27.72	82	14.28
ACT				
metro	154	6.79	75	3.06

There was no relationship apparent between levels of alcohol-caused deaths and levels of alcohol-caused hospitalisations for wholly alcohol attributable conditions. For instance, metropolitan Northern Territory had one of the highest male death rates, but it also had one of the lowest male hospitalisation rates. Conversely, while Queensland had average rates of deaths it also had some of the highest hospitalisation rates. It is unclear why hospitalisation and death rates at the regional level are not more similar. It may be that factors such as bed and staff availability, funding and hospital management practices differ substantially between the jurisdictions, affecting admission rates.

3.3 ALCOHOL-CAUSED DEATHS AMONG INDIGENOUS AUSTRALIANS

Table 3.3a shows the number of deaths caused by high-risk alcohol consumption, along with age standardised rates for Indigenous and non-Indigenous Australians in South Australia, Western Australia and the Northern Territory combined between 1991 and 1997. The rate of alcohol-caused deaths among Indigenous Australians was considerably greater than that among non-Indigenous Australians.

Table 3.3a: Total numbers and age standardised death rates (per 10,000) resulting from wholly alcohol-caused conditions for SA, WA and NT combined for Indigenous and non-Indigenous males and females, 1991-1997

	Indigenous Australians (15+yrs)		Non-Indigenous Australians (15+yrs)	
	N	ASR/10,000	N	ASR/10,000
Males	181	10.07	1097	1.28
Females	107	5.44	297	0.34

Tables 3.3b and 3.3c show the rate of deaths caused by high-risk alcohol consumption for Indigenous and non-Indigenous Australians between 1991 and 1997 according to geographic region categories for males (3.3b) and females (3.3c). Regardless of sex or region of residence, rates of alcohol-caused deaths were highest among Indigenous Australians (even to the extent that the female Indigenous death rate exceeded the male non-Indigenous death rate).

The age-standardised rate of alcohol-caused death among Indigenous males was higher in non-metropolitan than metropolitan WA and SA. However, in the NT alcohol-caused death rates among Indigenous males were similar in both metropolitan and non-metropolitan regions. This probably reflects the absence of a large metropolis in the NT in comparison to the other states, such that the actual population living in non-metropolitan NT is larger than that living in metropolitan areas. However, for both SA and the NT, rates of alcohol-caused deaths among Indigenous females were higher in metropolitan regions with rates higher in non-metropolitan areas in WA. The absence of any clear patterns here, however, probably reflects the small raw numbers of Indigenous alcohol-caused deaths.

Recent research indicates that both Indigenous and non-Indigenous Territorians have significantly higher levels of alcohol consumption than for the nation as a whole (Gray & Chikritzhs, 1999). The consequences of these high-rates of consumption are reflected in the high rates of wholly alcohol-caused deaths among both Indigenous and non-Indigenous people in the Northern Territory between 1991 and 1997.

Table 3.3b: Age standardised alcohol-caused death rates (per 10,000) for SA, WA and NT by metropolitan/non-metropolitan regions for Indigenous and non-Indigenous males and females, 1991-1997

State	Indigenous Australians (15+yrs) ASR/10,000		Non-Indigenous Australians (15+yrs) ASR/10,000	
	metro	non-metro	metro	non-metro
SA				
males	8.94	11.46	1.32	1.50
females	6.39	3.91	0.33	0.26
WA				
males	10.65	12.43	1.12	1.16
females	4.78	7.25	0.36	0.33

NT				
males	8.31	8.56	3.27	2.30
females	8.34	3.78	0.77	1.18

3.4 SUMMARY STATISTICS ON ALCOHOL-CAUSED DEATH AND HOSPITALISATION

A summary of national data on alcohol-caused deaths for 1997 and hospitalisations for the 1996/97 financial year is presented in Table 3.4a. As a point of comparison, the estimates of deaths and person-years of life lost calculated by English *et al*. (1995) for 1992 are presented in parentheses.

Any comparison of the data obtained in the current study with that generated by English *et al*. (1995) must be made in the context of the methodological differences between the studies. For example, English *et al*. (1995) attributed 34% of all falls, regardless of age, to high-risk alcohol consumption. However, in accord with practice used in recent research (Jonas *et al*., 1999) in the current study, fall injuries and deaths among people aged 65 years and over were not attributed to alcohol. This method reduces the numbers of fall injuries substantially such that only 19% of all female alcohol-caused bed days were comprised of fall injuries compared to 57% estimated by English *et al.*(1995).

In addition, there were differences in the aetiologic fractions used in this study compared to those used by English *et al.*(1995) who calculated fractions based on 1989/90 estimates of high-risk alcohol consumption. They then applied those fractions to morbidity and mortality records occurring in 1992[5]. It has since become evident that between 1989 and 1992 there was a substantial decline in alcohol consumption across Australian (World Drink Trends, 1998). For this report, aetiologic fractions for conditions partially caused by high-risk alcohol consumption were adjusted to reflect annual levels of prevalence of high-risk drinking. It is therefore likely that earlier estimates of morbidity and mortality for 1992 were overestimated. Indeed, comparison of the Figures obtained in section 3.1 suggests that this was the case for deaths data (differences in total numbers of deaths of 7% and 15% for males and females respectively). This means that differences between estimates for 1992 taken from English *et al*. (1995) and 1997 which appear in Table 3.4a are probably larger than was

actually the case. The differences in hospitalisation rates are only small for males (2%) but substantial for females (22%) which reflects the high prevalence of fall injuries among females aged over 65.

Table 3.4a: Estimated number of deaths[a] (all ages), person-years of life lost[a], hospitalisations [b]and bed-days[b] caused by high-risk alcohol consumption in 1997, 1996/97 by sex, condition and acute/chronic/mixed (Estimates for 1992 deaths and PYLL taken from English et al., 1995, in parentheses)

	Males				Females			
Condition[c]	**Deaths**	**PYLL[d]**	**Hosp.**	**Bed-days**	**Deaths**	**PYLL[d]**	**Hosp.**	**Bed-days**
Acute								
Falls[e]	35 (153)	992 (1053)	8378	32699	6 (172)	145 (239)	5147	24149
Assault	84 (88)	2919 (2502)	6505	19921	40 (50)	1462 (1678)	2049	5961
Road injuries	343 (429)	13751 (15884)	6108	36651	75 (89)	3423 (3799)	1681	8346
Occup. And mach.Inj.	4 (5)	127 (110)	1301	2866	0 (0)	3 (6)	303	817
Fire injuries	24 (32)	446 (474)	568	4005	11 (18)	203 (138)	178	1823
Drowning	61 (58)	1909 (1630)	64	199	12 (17)	325 (417)	29	65
Child abuse	0 (0)	10 (11)	35	161	0 (1)	31 (43)	37	185
Alcohol abuse	10 (12)	309 (367)	2596	6388	3 (3)	96 (28)	1576	4184
Alcoholic psychosis	40 (36)	358 (135)	2457	40951	11 (5)	35 (28)	669	7387
Alcoholic gastritis	2 (2)	44 (2)	856	2070	2 (1)	40 (2)	250	536
Aspiration	34 (56)	498 (722)	285	1189	23 (26)	116 (214)	234	1030
Ethanol toxicity	30 (0)	1118 (0)	10	20	7 (0)	301 (0)	27	73
Alc. Beverage. pois.	0 (7)	0 (169)	274	581	0 (0)	0 (0)	194	343
Other. eth./meth.pois.	2 (0)	87 (0)	44	116	0 (0)	0 (0)	22	63
Acute pancreatitis	20 (18)	126 (125)	886	5939	18 (18)	57 (70)	525	3877
Gastro-oesoph. haem.	1 (1)	0 (0)	366	994	0 (0)	0 (0)	197	572
Supr. card. Dysrhyth.	5 (4)	22 (12)	629	1672	9 (7)	5 (3)	388	1389
Spontaneous abortion	0 (0)	0 (0)	0	0	0 (0)	0 (0)	6	8
Low birthweight	0 (0)	1 (0)	3	55	0 (0)	1 (0)	5	57
Methanol toxicity	0 (0)	0 (0)	0	0	0 (0)	0 (0)	0	0
Sub-total	*695 (901)*	*22743 (23196)*	*31366*	*156476*	*218 (407)*	*6246 (6665)*	*13517*	*60865*

Table 3.4a: (Cont.)

	Males				Females			
Condition[c]	**Deaths**	**PYLL[d]**	**Hosp.**	**Bed-days**	**Deaths**	**PYLL[d]**	**Hosp.**	**Bed-days**
Chronic								
Alc. Liver cirrhosis	539 (572)	8313 (6719)	2468	18887	144 (148)	2795 (1823)	754	6767
Alcohol dependence	204 (159)	3300 (2086)	9054	59279	53 (26)	1035 (339)	3989	26015
Alc. poly neuropathy	0 (0)	0 (0)	23	171	0 (0)	0 (0)	9	69
Alc. Cardiomyopathy	98 (136)	1343 (1309)	132	822	11 (12)	138 (118)	14	51
Female breast cancer	0 (0)	0 (0)	0	0	51 (54)	715 (579)	371	1810
Epilepsy	19 (17)	509 (399)	995	3488	12 (10)	285 (172)	735	2965
Hypertension	19 (19)	167 (83)	211	836	19 (19)	49 (38)	206	1000
Oesophageal varices	2 (1)	28 (5)	339	843	0 (0)	0 (0)	134	362
Chronic pancreatitis	8 (10)	107 (87)	1054	5470	5 (2)	44 (0)	462	2907
Cholelithiasis	-1 (-1)	-4 (-7)	-465	-1714	0 (-1)	-2 (-4)	-653	-2070
Oropharyn.cancer	48 (59)	576 (480)	348	3295	7 (8)	61 (49)	47	413
Oesophageal cancer	46 (47)	485 (343)	185	1364	8 (10)	47 (37)	40	306
Laryngeal cancer	28 (35)	280 (228)	167	1338	3 (3)	20 (14)	15	147
Liver cancer	50 (44)	540 (339)	128	875	15 (13)	119 (76)	33	283
Psoriasis	0 (0)	0 (0)	31	94	0 (0)	0 (0)	9	28
Sub-total	*1061 (1098)*	*15675 (12071)*	*14670*	*95049*	*328 (304)*	*5309 (3241)*	*6165*	*41052*

Table 3.4a: (Cont.)

	Males				Females			
Condition[c]	**Deaths**	**PYLL[d]**	**Hosp.**	**Bed-days**	**Deaths**	**PYLL[d]**	**Hosp.**	**Bed-days**
Mixed								
Stroke	312 (313)	2236 (1473)	2440	21122	414 (397)	1783 (1425)	2276	22125
Suicide	228 (208)	7836 (6249)	1023	3993	36 (33)	1149 (916)	845	3112
Sub-total	*540 (521)*	*10076 (7722)*	*3463*	*25115*	*449 (430)*	*2933 (2341)*	*3122*	*25238*
TOTAL	2296 (2521)	48661 (43183)	49499 (45600)	276640 (443834)	994 (1139)	14503 (12267)	22803 (25993)	127155 (287335)
Total conditions	67752 (66108)	587348 (494234)	2534594 (1280801)	10521934 (7268371)	61598 (57543)	321548 (264683)	2984975 (1632737)	12533289 (9271764)
% caused by alcohol	3.4 (3.8)	8.3 (8.7)	2.0 (3.5)	2.6 (6.1)	1.6 (2.0)	4.5(4.6)	0.76 (1.6)	1.01 (3.1)
Average number of years lost per death		21 (17)				15 (11)		

[a]mortality data, 1997, [b]morbidity data, 1996/97. [c]Wholly alcohol-caused conditions in bold font. [d]PYLL including deaths occurring between age 0 and age 69 years. [e]See note inset on pp.3. *Note: small numerical inconsistencies due to rounding errors*

Table 3.4a shows that in 1997 there were an estimated 3,290 lives lost due to high-risk alcohol consumption, accounting for 63,164 person-years of life lost before the age of 70 years. During 1996/97 a total of 72,302 hospital admissions and 403,795 hospital bed-days were caused by high-risk drinking. Overall, about 70% of all alcohol-caused deaths and hospitalisations occurred among males.

The most common conditions resulting in alcohol-caused death were alcoholic liver cirrhosis, alcohol dependence, road injuries, stroke and suicide. The greatest alcohol-caused cause of death for adult males was alcoholic liver cirrhosis, while females most commonly died from stroke. Alcohol-caused hospitalisations were most commonly the result of falls, alcohol-dependence, assaults and road injuries.

Overall, chronic conditions, e.g. alcoholic liver cirrhosis, alcohol dependence, contributed to the majority of alcohol-caused deaths (42% chronic, 28% acute, 22% stroke, 8% suicide). However, the greatest number of person-years of life lost were due to acute conditions, e.g. road injury, assault, drowning, (46% acute, 33% chronic, 6% stroke, 14% suicide).

There appears to have been a decline in the overall numbers of alcohol-caused deaths between 1992 (English *et al.,* 1995) and 1997. This reduction has been particularly marked for male road injury victims. However, male suicides increased by almost 10% between 1992 and 1997. Due to the youthful age of most male suicide victims, where the peak age of death is 20-29 years, estimates of PYLL were comparable to total life-years lost due to alcoholic liver cirrhosis. Despite the overall reduction in the number of alcohol-caused deaths, the average number of PYLL due to high-risk drinking has risen. Increases in PYLL were evident among most conditions, with road injury and aspiration being the major exceptions. Reasons for an increase in PYLL include the increasing average life expectancy for both males and females in the last decade and a possible increase in the frequency of alcohol-caused deaths in younger age groups.

During 1997, conditions wholly caused by alcohol contributed to about 37% of all alcohol-caused deaths, compared to 33% in 1992. A comparison of the number of deaths from wholly attributable conditions occurring in 1992 and 1997 indicated that while there was a reduction of 2% (21) among males, an increase of 15% (33) occurred

among females. The increase in wholly attributable deaths among females was largely due to death from alcohol dependence. Overall, this represented a small increase of about 1% in the number of wholly alcohol attributable deaths occurring between 1992 and 1997.

Alcohol-caused deaths contributed to approximately 2.5% of all deaths and 6.9% of all person-years of life lost occurring in 1997. In 1996/97, alcohol-caused hospitalisations contributed to 1.3% of all admissions and 1.75% of all hospital bed days. There was little overall change in the proportions of *all* deaths and PYLL which were caused by alcohol between 1992 and 1997. However, there did appear to be reductions in the proportions of all admissions and hospital bed-days due to high-risk alcohol consumption. As mentioned previously, hospital management practices may have an effect on admissions.

As noted earlier, the consequences of long term alcohol misuse tend to manifest among older persons in the form of chronic conditions (e.g. alcoholic liver cirrhosis, oesophageal cancer, hypertension). Overall, diseases of this type contribute to the greatest number of alcohol-caused deaths. However, since those who suffer from chronic alcohol-caused diseases tend to be older, they do not contribute to the largest numbers of PYLL. Instead, acute conditions, such as road fatalities and death by violence, account for the greatest proportions of PYLL. Acute forms of alcohol-caused injuries generally arise from bouts of intoxication or 'binge drinking' and occur predominantly among the younger age groups – males in particular.

The very different types of outcomes arising from long term misuse of alcohol versus bouts of intoxication, particularly in terms of premature life lost, has implications for the direction of future public policy and alcohol related health campaigns. There is no doubt that the bulk of the economic burden on society due to alcohol misuse arises from productivity losses due to premature death. Estimates of the cost of productivity losses also far outweigh any costs associated with health related medical and hospital treatments incurred from alcohol-caused disease and injury (Chikritzhs *et al.*, 1999; Collins & Lapsley, 1996).

4 CONCLUSIONS

This research has examined the prevalence and patterns of alcohol-caused deaths and hospitalisations across Australia using official health statistics. It represents an important update and development of previous work conducted in this area (English *et al.*, 1995). The main findings show that, in spite of a decline in per-capita alcohol consumption, the rates of alcohol-caused death show similar patterns to 1992 for both males and females. These deaths confer a considerable cost to society in both human and economic terms. Similarly, it would appear that the rate of alcohol-caused hospitalisation has been increasing over the period considered which also presents a considerable burden on the community's limited health resources.

The burden of alcohol-caused deaths is distributed unevenly across the population. Males are over-represented in mortality statistics compared to women, as are those living in non-metropolitan regions compared to metropolitan regions and Indigenous Australians compared to non-Indigenous Australians. This has implications for the allocation of resources for the prevention of alcohol-caused death.

It is also important to consider the different types of outcomes associated with two main types of drinking patterns, ie drinking to intoxication (acute conditions) versus long term alcohol misuse (chronic conditions), and their implications for public health policy and economic cost to the community. The largest proportion of all alcohol-caused deaths occur as a result of chronic conditions such as alcoholic liver cirrhosis and alcoholic dependence, however, the greatest proportion of prematurely lost years of life (PYLL) occur due to acute conditions such as road injury and assault. This discrepancy is a direct result of the youthful age distribution among people injured in acute alcohol-related events – those generally arising from drinking to intoxication. There is no doubt that the bulk of the economic burden on society due to alcohol misuse arises from productivity losses due to premature death. Estimates of the cost of productivity losses also far outweigh any costs associated with health related medical and hospital treatments incurred from alcohol-caused disease and injury (Chikritzhs *et al*. 1999; Collins & Lapsley 1996).

The geographic pattern of hospitalisation does not mirror that for alcohol-caused death and it is assumed this is most likely due to admission practices and bed availability as

well as demand. As other alcohol-caused harms are considered by the NAIP it will be of interest to consider the geographic overlap between measures. It may be that some locations with high levels of alcohol consumption and related disease and injury are inadequately resourced.

Currently, work is underway to update and fine tune estimates of relative risk and aetiologic fractions for some sentinel conditions, ie. road injury, stroke, falls and female breast cancer (personal communication, Bruno Ridolfo, AIHW). Because of these imminent changes, future efforts to quantify the burden of alcohol misuse on morbidity and mortality to the Australian community may reveal some major changes in the understanding of the relationship between alcohol and harm in terms of both trend and magnitude. For example, stroke is one of the largest causes of alcohol-caused death among women. However, the relationship between alcohol and stroke is complicated by the fact that the disease occurs in two main forms – ischaemic and haemorrhagic, both of which are associated with different pathological processes. There is evidence to suggest that while a harmful level of consumption is a strong risk factor for haemorrhagic stroke in particular, it also appears to afford a protective effect on the risk of suffering ischaemic stroke when consumed at low and moderate levels (Stampfer *et al.*, 1988; Thun *et al.*. 1997; Sacco *et al.*, 1999; Hillbom and Juvela, 1996). Current meta-analyses provide only one estimate of relative risk for stroke (English *et al.*, 1995), while new work will delineate between ischaemic and haemorrhagic stroke (personal communication, Bruno Ridolfo, AIHW). Since the vast majority of strokes are known to be ischaemic in nature - about 70% (Camargo 1996), it is possible that future estimates of the proportion of all strokes which are caused by alcohol may differ substantially to current estimates.

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APPENDICES

APPENDIX A

Table A1: Estimated national numbers and age-specific rates (per 10,000) of alcohol-caused deaths by age group and sex, 1997

	Males		Females	
Age group	**N**	**Age specific rate/10,000**	**N**	**Age specific rate/10,000**
0 to 4	11.37	0.17	2.93	0.05
5 to 9	6.22	0.09	2.38	0.04
10 to 14	9.14	0.14	3.43	0.05
15 to 19	91.43	1.38	19.31	0.31
20 to 24	152.99	2.19	37.43	0.55
25 to 29	139.48	1.92	31.48	0.43
30 to 34	104.04	1.46	33.76	0.47
35 to 39	121.75	1.65	35.31	0.48
40 to 44	152.06	2.21	57.99	0.84
45 to 49	186.23	2.87	59.35	0.93
50 to 54	212.57	3.81	76.20	1.42
55 to 59	187.17	4.33	48.90	1.16
60 to 64	217.48	6.04	56.14	1.55
65 to 69	238.36	7.10	86.53	2.46
70 to 74	218.62	7.79	168.20	5.13
75-79	123.60	6.50	20.02	0.78
80-84	70.08	6.44	88.62	4.96
85+	53.05	8.13	166.51	11.04

Table A2: Estimated national numbers and age-specific rates (per 10,000) of alcohol-caused hospitalisations by age group and sex, 1996/97

	Male		Female	
Age group	**N**	**Age specific rate/10,000**	**N**	**Age specific rate/10,000**
0 to 4	191	2.88	79	1.25
5 to 9	223	3.31	65	1.01
10 to 14	552	8.21	260	4.06
15 to 19	4134	62.27	1598	25.34
20 to 24	5476	78.52	1968	29.15
25 to 29	5157	70.81	2025	27.90
30 to 34	4592	64.61	1910	26.73
35 to 39	4728	64.10	2057	27.76
40 to 44	4598	66.76	2120	30.62
45 to 49	4658	71.72	2093	32.69
50 to 54	3880	69.59	2004	37.36
55 to 59	3171	73.28	1663	39.61
60 to 64	2696	74.90	1664	45.86
65 to 69	2015	60.02	924	26.32
70 to 74	1863	66.37	1257	38.33
75+	1564	42.97	1117	19.08

APPENDIX B

Table B1: Estimated, state-wide numbers of deaths due to wholly alcohol-caused conditions, males, 1990-1997

	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
1990	401	267	156	71	73	17	17	12
1991	336	245	161	69	73	23	11	18
1992	326	300	166	93	80	18	19	13
1993	327	164	137	80	72	23	16	10
1994	333	228	150	87	75	16	13	12
1995	349	196	161	89	87	25	27	12
1996	336	210	178	86	83	20	24	11
1997	324	227	182	82	88	24	24	8

Table B2: Estimated, state-wide numbers of deaths due to wholly alcohol-caused conditions, females, 1990-1997

	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
1990	110	61	39	23	27	5	7	4
1991	83	72	41	24	28	12	7	2
1992	98	45	40	13	24	7	7	
1993	95	50	36	21	29	4	5	3
1994	95	55	39	28	35	7	7	4
1995	109	44	43	22	24	8	10	3
1996	81	51	50	23	34	7	6	1
1997	97	52	41	18	25	5	13	3

APPENDIX C

Table C1: Estimated numbers of hospitalisations resulting from wholly alcohol-caused conditions by states/territory, males, 1993/94-1996/97

	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
1993/94	7010	3040	4611	1163	1406	277	142	66
1994/95	6282	2944	4550	1200	1574	258	151	87
1995/96	7404	2877	4452	1226	1571	331	156	76
1996/97	7366	3239	4240	1109	1529	309	170	83

Table C2: Estimated numbers of hospitalisations resulting from wholly alcohol-caused conditions by states/territory, females, 1993/94-1996/97

	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
1993/94	2398	1350	1636	379	543	70	52	36
1994/95	1980	1320	1800	466	564	98	52	32
1995/96	2754	1206	1688	478	668	118	48	38
1996/97	2686	1533	1926	483	669	183	61	39

APPENDIX D

Table D1: Number of adult male deaths resulting from wholly alcohol attributable conditions by state/territory, metropolitan/non-metropolitan, 1990-1997 (Age standardised rate in parentheses)

State	1990	1991	1992	1993	1994	1995	1996	1997
NSW								
Metro	250	225	187	198	189	195	180	184
	(1.90)	(1.68)	(1.38)	(1.44)	(1.35)	(1.37)	(1.23)	(1.23)
Non-metro	151	111	139	129	144	154	156	140
	(1.80)	(1.24)	(1.53)	(1.42)	(1.53)	(1.59)	(1.61)	(1.42)
VIC								
Metro	180	172	210	114	155	140	152	153
	(1.59)	(1.51)	(1.80)	(.97)	(1.30)	(1.16)	(1.22)	(1.21)
Non-metro	87	73	90	50	73	56	58	74
	(1.79)	(1.50)	(1.86)	(1.03)	(1.42)	(1.10)	(1.12)	(1.38)
QLD								
Metro	67	65	81	52	64	63	77	84
	(1.43)	(1.36)	(1.66)	(1.04)	(1.26)	(1.15)	(1.38)	(1.47)
Non-metro	89	96	85	85	86	98	101	98
	(1.53)	(1.59)	(1.34)	(1.32)	(1.28)	(1.41)	(1.42)	(1.33)
SA								
Metro	50	44	58	56	57	63	58	58
	(1.23)	(1.08)	(1.44)	(1.34)	(1.37)	(1.48)	(1.31)	(1.31)
Non-metro	21	25	35	24	30	26	28	24
	(1.34)	(1.59)	(2.20)	(1.57)	(1.83)	(1.58)	(1.70)	(1.42)
WA								
Metro	55	42	55	44	54	60	49	59
	(1.36)	(1.03)	(1.32)	(1.03)	(1.23)	(1.31)	(1.03)	(1.21)
Non-metro	18	31	25	28	21	27	34	29
	(1.12)	(1.99)	(1.49)	(1.69)	(1.24)	(1.62)	(1.92)	(1.61)
TAS								
Metro	9	10	8	6	7	9	8	12
	(1.32)	(1.46)	(1.09)	(.82)	(.97)	(1.20)	(1.08)	(1.57)
Non-metro	8	13	10	17	9	16	12	12
	(.80)	(1.21)	(.92)	(1.59)	(.84)	(1.44)	(1.09)	(1.07)
NT								
Metro	9	4	10	7	5	6	7	9
	(4.93)	(1.98)	(6.32)	(4.11)	(2.29)	(2.49)	(2.38)	(5.04)
Non-metro	8	7	9	9	8	21	17	15
	(2.29)	(4.94)	(2.87)	(3.49)	(2.11)	(6.47)	(5.17)	(4.10)
ACT								
Metro	12	18	13	10	11	11	10	8
	(1.28)	(2.09)	(1.43)	(.89)	(1.24)	(1.41)	(1.02)	(0.90)

Table D2: Number of adult female deaths resulting from wholly alcohol attributable conditions by state/territory, metropolitan/non-metropolitan, 1990-1997 (Age standardised rate in parentheses)

State	1990	1991	1992	1993	1994	1995	1996	1997
NSW								
Metro	67	54	59	51	60	53	49	62
	(0.48)	(0.37)	(0.41)	(0.35)	(0.41)	(0.35)	(0.32)	(0.40)
Non-metro	43	29	39	44	35	56	32	35
	(0.49)	(0.32)	(0.44)	(0.48)	(0.37)	(0.57)	(0.32)	(0.36)
VIC								
Metro	42	50	31	34	41	37	33	37
	(0.35)	(0.41)	(0.25)	(0.27)	(0.32)	(0.29)	(0.25)	(0.28)
Non-metro	19	22	14	16	14	7	18	15
	(0.37)	(0.43)	(0.28)	(0.31)	(0.28)	(0.13)	(0.33)	(0.28)
QLD								
Metro	16	18	17	16	16	19	22	18
	(0.34)	(0.36)	(0.32)	(0.30)	(0.30)	(0.34)	(0.37)	(0.29)
Non-metro	23	23	23	20	23	24	28	23
	(0.41)	(0.38)	(0.37)	(0.32)	(0.37)	(0.36)	(0.40)	(0.32)
SA								
Metro	17	19	11	17	22	15	17	13
	(0.40)	(0.42)	(0.24)	(0.37)	(0.49)	(0.33)	(0.36)	(0.28)
Non-metro	6	5	2	4	6	7	6	5
	(0.38)	(0.35)	(0.14)	(0.27)	(0.39)	(0.46)	(0.37)	(0.32)
WA								
Metro	20	19	18	16	24	16	20	14
	(0.49)	(0.45)	(0.41)	(0.35)	(0.52)	(0.33)	(0.40)	(0.28)
Non-metro	7	9	6	13	11	8	14	11
	(0.49)	(0.64)	(0.42)	(0.88)	(0.70)	(0.47)	(0.88)	(0.68)
TAS								
Metro	3	3	3	3	3	6	5	2
	(0.37)	(0.38)	(0.41)	(0.39)	(0.41)	(0.75)	(0.62)	(0.25)
Non-metro	2	9	4	1	4	2	2	3
	(0.18)	(0.84)	(0.36)	(0.08)	(0.36)	(0.17)	(0.17)	(0.25)
NT								
Metro	4	3	3	2	1	4	4	5
	(2.00)	(0.82)	(0.96)	(1.36)	(0.99)	(1.70)	(1.69)	(1.59)
Non-metro	3	4	4	3	6	6	2	8
	(4.01)	(1.71)	(1.87)	(2.24)	(1.52)	(3.46)	(0.79)	(3.63)
ACT								
Metro	4	2	0	3	4	3	1	3
	(0.39)	(0.20)	(0.00)	(0.27)	(0.50)	(0.40)	(0.12)	(0.27)