The burden of death, disease and disability due to alcohol in New Zealand

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ALCOHOL ADVISORY COUNCIL OF NEW ZEALAND
Kaunihera Whakatupato Waipiro o Aotearoa

PO Box 5023
Wellington
New Zealand

www.alac.org.nz and www.waipiro.org.nz

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SUMMARY

This study estimated the burden of death, disease and disability attributable to alcohol consumption in New Zealand. We used the comparative risk assessment (CRA) methodology that was developed by the World Health Organisation (WHO) for measuring the impact of important risk factors on health at a regional and global level. We applied the CRA approach at a country level, and for Mäori and non-Mäori separately where this has been possible. The best estimates of alcohol consumption in the population were combined with best estimates of alcohol-disease relationships from the international epidemiological literature, to calculate the proportion of each alcohol-related condition that was attributable to alcohol. Where reliable local data have been identified these have been incorporated into the analysis, in the place of estimates generated from the international literature or WHO models. In two separate analyses we have estimated mortality in 2000, and disability-adjusted life years in 2002.

In the analysis of mortality, we estimated that 3.9% of all deaths in New Zealand in 2000 were attributable to alcohol consumption (approximately 1040 deaths), and that approximately 980 deaths were prevented by alcohol, resulting in a net loss of about 60 lives. Since many of the alcohol-attributable deaths occurred before middle age and the deaths prevented by alcohol were almost entirely amongst older people, the balance of years of life lost (YLL) and gained due to alcohol consumption was much less favorable. There were 17,200 years of life estimated to be lost, but only 5,300 years of life were estimated to have been gained; a net loss of almost 12,000 years of life due to alcohol.

The burden of mortality from alcohol use was not evenly spread in the population. The rate of alcohol-attributable YLLs in men was four to five times the rate in women, largely due to high alcohol-related mortality in men in the 15-44 year age group. These differences between men and women were seen in both the Mäori and non-Mäori populations. However, both Mäori men and Mäori women had higher mortality and YLL rates than non-Mäori of the same age. Overall, Mäori had 4 times the alcohol-related mortality of non-Mäori, and more than double the rate of years of life lost due to alcohol.

Injury was a major contributor to alcohol-related mortality, being responsible for 51% of deaths (532 deaths) and 72% of years of life lost (12,434 YLLs). Most alcohol-related deaths before middle age were due to injury. Cancers accounted for a further 24% of alcohol-related deaths and 14% of YLLs, with the remainder being due to other chronic diseases.

Most of the positive effects of alcohol consumption were seen in prevention of ischaemic heart disease deaths in older people who had a pattern of drinking characterised by frequent low volume intake (78% of all deaths prevented). Reduction in deaths due to stroke, diabetes and complications of cholelithiasis made up the remainder.

In a separate analysis incorporating estimates of morbidity as well as mortality, the loss of 33,500 disability-adjusted life years (DALYs) was attributed to alcohol for the New Zealand population in 2002. This comprised 7.4% of all DALYs lost in the population (10% of all DALYs in men and 4% in women). The largest single cause of DALYs lost was alcohol use disorders, responsible for 49%. The benefits of alcohol resulted in a gain of approximately 7,500 DALYs, and these were evenly split between men and women. Overall, there was a net loss of 26,000 DALYs attributable to alcohol, with 76% lost by men.

These figures underestimate the adverse impacts of alcohol consumption on health, as they exclude many mental health outcomes, and important social consequences. While elimination of alcohol consumption is not realistic, and may not be desirable, evidence-based policy measures could minimize alcohol-related mortality, morbidity and disability while keeping the alcohol-related health benefits.

Five major messages have emerged from this analysis:

- There are no health benefits of drinking alcohol before middle age
- The pattern of drinking is very important in determining the health effects of alcohol consumption
- Injury is responsible for half of all alcohol-attributable deaths and almost three-quarters of the years of life lost due to alcohol
- There is a large burden of disability due to alcohol use disorders that is not reflected in mortality figures
- The health burden of alcohol falls inequitably on Mäori.

1. INTRODUCTION

1.1 Objective

The object of this report was to estimate:

- Deaths attributable to alcohol, for Mäori, non-Mäori and the total population of New Zealand in 2000*.
- The burden of disease and disability, including non-fatal conditions, that was attributable to alcohol, for the total population of New Zealand in 2002†.

1.2 Motivation

It has long been recognised that alcohol drinking influences the health of populations. Although alcohol has clear and major detrimental effects on health through its contribution to many diseases and injuries, there has also been evidence of significant health benefits.[4 5]

Previous quantitative studies of the overall health impact of alcohol drinking on the New Zealand population[6 7] have attempted the complicated task of estimating the net effect of the impact of alcohol on many aspects of health. The comparative risk assessment (CRA) methodology developed for the World Health Report 2002[1 2] has provided an opportunity to update and refine these estimates.

1.3 Overview

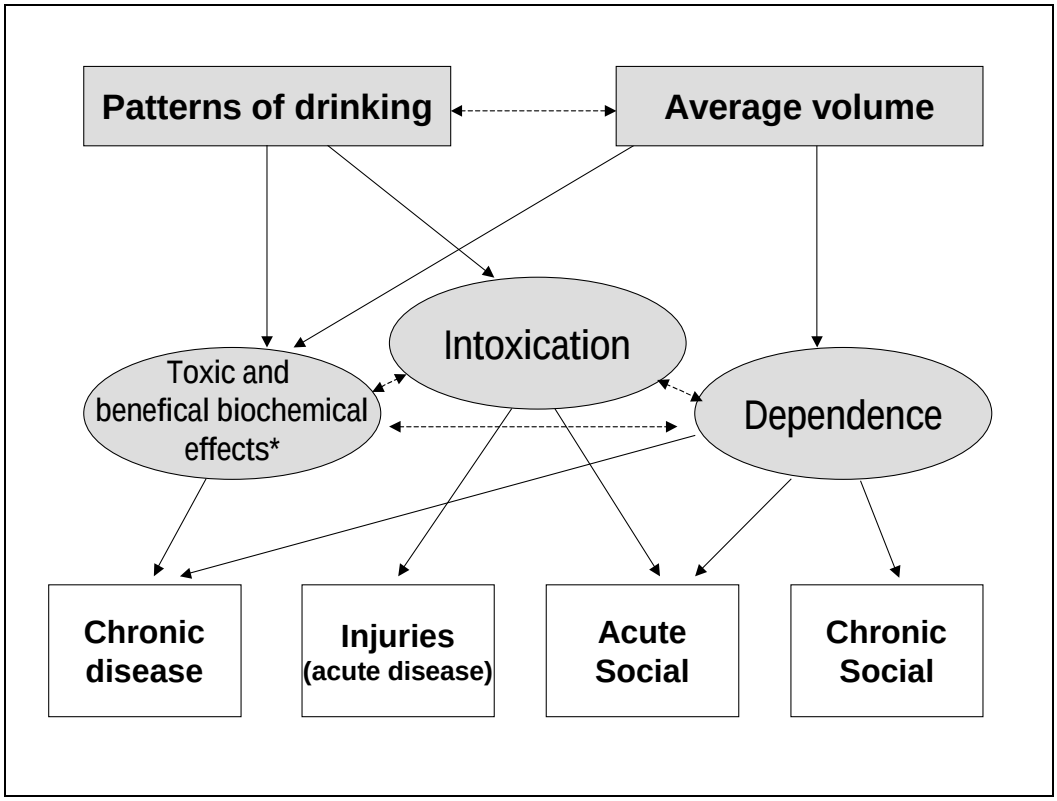
Alcohol as a risk factor for disease and injury

The relationship between alcohol consumption and health is complex. Biological and social effects of alcohol use result from three main intermediaries or pathways: intoxication, dependence and direct biochemical effects. These effects relate to both the average volume of alcohol consumed and the pattern of drinking. A simple model of the relationships between alcohol and health outcomes is shown in Figure 1.

Social outcomes of alcohol consumption, such as family problems, public disorder or workplace problems, are not included in this report unless they are included in ICD-10 (International Classification of Diseases Version 10), although it is recognised that they contribute to health in a broad sense.

* the most recent available NZHIS mortality data
† the most recent available WHO projections of DALY burden

Figure 1: Conceptual model of alcohol consumption, intermediate mechanisms, and long-term consequences



* independent of intoxication or dependence

Adapted from: Rehm et al. 2003[5]

Direct biochemical effects include both harmful and beneficial effects that are not the result of intoxication or dependence. Examples are chronic pancreatic and liver damage on one hand, and improvements in blood lipid and coagulation profiles on the other.

Intoxication is a powerful mediator of acute adverse outcomes, and can also contribute to some chronic diseases. The effects on risk of injury are evident even at moderate levels of consumption, when there may be little subjective experience of intoxication.

Alcohol dependence, a disorder in itself, mandates regular alcohol consumption. It thus mediates the impact of alcohol on all classes of health outcome.

Average volume of drinking, or total alcohol consumption in a population, has been used to measure relationships between alcohol and disease. However, average consumption is not a good predictor of intoxication and consequent injury, or of health benefits derived from small frequent doses of alcohol, such as reduction in ischaemic heart disease. Such effects are better predicted by including measures of pattern of drinking. “Pattern of drinking” refers to the way in which most alcohol is consumed, such as in irregular heavy drinking occasions, or binge drinking, compared with light to moderate drinking on a daily basis. These data are now increasingly collected in population research.

Comparative risk assessment (CRA)

This methodology was developed by the World Health Organisation (WHO) as a systematic approach to measuring the burden of disease attributable to a range of important global risk factors.[1 2 8 9] The use of a comparable approach to estimation has allowed the health burden due to different risk factors to be compared and ranked for the first time on a global and regional scale. CRA aims to combine best estimates of the risk factor distribution in the population, with best estimates of risk factor-disease relationships from the international epidemiological literature to measure the impact of each major risk factor. In the World Health Report 2002, CRA was used to estimate global and regional burden of disease due to a range of risk factors, including alcohol, but no country level estimates were calculated.

This report employs the CRA approach at a country level, and for Mäori and non-Mäori separately where this has been possible. Where reliable local data have been identified these have been incorporated into the analysis, in the place of estimates generated from the international literature or WHO models.

The WHO CRA for alcohol drew heavily on existing reports on the quantification of drug-caused mortality and morbidity in Australia[10-12] and Canada,[13 14] as well as reviewing new epidemiological evidence about the association of alcohol with health outcomes.[15] (See Rehm et al.[3] for an overview) New methods were developed for incorporating the effect of pattern of drinking for some conditions, and for modeling estimates where reliable data on the individual level were lacking.[3]

Mäori

This report has endeavoured to take an approach consistent with the Treaty of Waitangi. That is, the mortality analyses of alcohol attributable burden of disease for Mäori and non-Mäori have been conducted separately. In terms of the CRA country-level framework, Mäori, non-Mäori, and New Zealand have been treated as separate “countries”. In the second analysis, combining fatal and nonfatal health outcomes, separate analyses have not been possible as the estimates of disability-adjusted life years lost for each condition have been supplied by the WHO Global Burden of Disease Study and did not include separate estimates for Mäori and non-Mäori.

Mäori and non-Mäori have different alcohol consumption patterns on average. Non-Mäori are more likely to be alcohol drinkers and drink more often, but drink less on a typical drinking occasion, when compared with Mäori. The differences are such that average alcohol consumption per day among Mäori and non-Mäori is similar,[16] but the impacts on health are likely to differ substantially.

There is little direct epidemiological evidence concerning the effects of alcohol on health in Mäori, and uncertainty about the generalisation of findings from other populations. Further research needs to be undertaken to address these issues.

2. DATA AND METHODS

2.1 Terminology and definitions

Age groups: All of the analyses in this report categorise the population into eight age groups: 0-4 years, 5-14, 15-29, 30-44, 45-59, 60-69, 70-79, 80+. These age categorisations were chosen to be consistent with the groups used by WHO.

Ethnicity classification (Mäori, non-Mäori): Where possible, analyses have been conducted for Mäori and non-Mäori separately as well as for the total New Zealand (NZ) adult population. Denominator populations used in the calculation of rates are those derived for the appropriate year by Statistics New Zealand from Census data. They rely on self-identification of ethnicity, and a hierarchy of ethnic groups when multiple ethnic group affiliations are identified, to establish membership of the Mäori ethnic group. Thus, any person identifying with Mäori ethnic group is classified here as Mäori, and all others as non-Mäori.

Numerator data are from the New Zealand Health Information Service (NZHIS) mortality database for 2000, which also uses self-identification of ethnicity but is more prone to misclassification due to the way data are collected. This is has been shown to result in underestimation, rather than overestimation, of Mäori mortality.[17] [18] No other ethnic groups are differentiated in these analyses.

Drinking categories (abstainer, I, II, III): The categories of average alcohol consumption used through out this report are consistent with those used in the WHO comparative risk assessment for alcohol, based on the work of English et al.[10] The cut points for the categories differ between men and women, to reflect the different biological effects of alcohol on men and women on average. Alcohol consumption data are converted to equivalents in grams of pure alcohol per day and the categories are:

Abstainer		
Drinking category I:	Women 0.01-19.99	Men 0.01-39.99
Drinking category II:	Women 20-39.99	Men 40-59.99
Drinking category III:	Women 40+	Men 60+

Patterns of drinking (1,2,3,4): As part of the WHO CRA for alcohol a method was developed to incorporate pattern of drinking, as well as average volume of alcohol consumption, into the assessment of ischaemic heart disease and injuries. We have used that system to categorise populations (Mäori, non-Mäori and total NZ) into one of four patterns of drinking. Higher scores indicate more health risks for a given volume of alcohol consumption.[3] Details are given in section 2.2 and Appendix A.

Years of life lost (YLLs): YLLs are a measure of premature mortality. They measure deaths in units of time (life years) rather than events (mortality). There are a number of

alternative approaches to calculating years of life lost, and the approach used in this report is outlined below in section 2.4.

Years of life lost due to disability (YLDs): YLDs are calculated from the number of years lived with a health condition multiplied by the disability weight previously estimated for that condition (in the range 0-1). YLDs are used to describe the impact of alcohol on morbidity, and contribute towards DALY estimates.

Disability adjusted life years (DALYs): DALYs are a summary measure of population health. One DALY can be thought of as one lost year of "healthy" life, and the burden of disease in DALYs as a measurement of the gap between current health of the population and an ideal situation where everyone lives to old age in full health. DALYs for a specific health condition in a population are calculated as the sum of the years of life lost due to premature mortality (YLLs) and the years lost due to disability (YLD) from the specified condition.

Alcohol attributable fraction (AAF): the AAF is the proportion of disease in a population that would be prevented if there was no alcohol consumed. This is also known as the population attributable fraction (PAF) or population attributable risk (PAR) due to alcohol. The AAF varies for different health conditions, and subgroups of the population. An AAF can be negative if alcohol reduces risk or is protective for the defined group and condition.

2.2 Prevalence and patterns of exposure to alcohol

2.2.1 Measurement of exposure to alcohol

The impact of alcohol consumption on the health of a population is primarily dependent upon how often, how much and over what period of time alcohol is consumed. Other factors may also impact on certain disease categories, such as whether or not is it is drunk with food (influencing how fast the alcohol is absorbed into the bloodstream and thus influencing injury and some cardiovascular conditions),[19] or whether or not it is consumed in conjunction with operating machinery or vehicles (influencing injury based on the amount consumed in the situation).[20]

While the relationship of alcohol to some conditions, such as injuries, is determined by the volume consumed on a particular occasion, for other conditions, such as cancers and liver cirrhosis, the period of time over which tissues are exposed to high levels of alcohol is most important. Cardiovascular conditions may be affected by both volume consumed and regularity of drinking; with some intermediate factors being long ranging (e.g. lipid profile) and some short acting (e.g. platelets).

Ideally, surveys undertaken to measure alcohol consumption in a population would measure all these aspects of drinking patterns. They would survey a representative

sample of the population, selected through random or other probability sampling techniques, and include an age range that covers all users of alcohol. The methodology used to conduct the survey would be culturally appropriate and questions framed in such a way that bias was minimised, avoiding 'loaded' questions or judgmental language.

The effects of patterns of drinking on health have only recently been explored (for more detail see [21 22]). In New Zealand, most studies that have measured alcohol consumption have investigated alcohol in its relation to a particular health condition or behaviour, and have included only selected consumption measures. Thus, the Heart and Health Study conducted in Auckland in 1992/93, which examined long-term use of alcohol in order to describe its relationship with coronary heart disease, measured 'usual' drinking but not binge drinking.[23 24] The Sleep Survey investigated problem sleeping, and measured frequency of drinking and numbers of drinks on a typical occasion (personal communication, R. Harris, Wellington). No single study has included all aspects of alcohol consumption relevant to risk relating to alcohol consumption.

The most comprehensive surveys of recent drinking habits in New Zealand have been the National Alcohol Survey 2000 and Te Ao Waipiro 2000 (a Mäori National Alcohol Survey) conducted by the Alcohol and Public Health Research Unit (APHRU) in Auckland, now the Centre for Social Health Outcomes Research and Evaluation (SHORE).[25 26] These two studies included a large number of both Mäori and non-Mäori respondents and had response rates of 72% and 74% respectively.

The methods of the two SHORE surveys are detailed elsewhere.[25 26] Briefly, for each, a computerised questionnaire and database of residential land-line telephone numbers was established. Telephone numbers were randomly selected from within geographic strata, and dialed automatically. For the Mäori survey, three random digit dialing (RDD) samples, which differed only in geographic coverage and age range of respondents were used. In addition, a database of published telephone numbers for households where there were any persons identified on the electoral rolls as having Mäori ancestry was sampled and this latter source made up 75% of the survey. Within each household, a further random selection was then made for each individual within the household to determine if they were to be interviewed. Responses were entered into the database during the interview. In both surveys, audits were undertaken to ensure the interviews occurred correctly.

2.2.2 Average daily consumption

We calculated average daily consumption by using the 2000 National Alcohol and Te Ao Waipiro surveys for New Zealanders aged between 15 and 65 years, weighted to represent consumption for the whole adult NZ population. The estimated total consumption accounted for was found to be 98% of the total alcohol reported by Statistics New Zealand as available for consumption nationally.[25 26] For determining alcohol-related mortality and morbidity in this study for those older than 65 years and for assessing the health effects of drinking in pregnancy it was necessary to estimate exposure based on findings from other surveys (see later).

We used these data to classify each participant into one of four levels of average daily consumption of alcohol, corresponding to those used by the WHO Global Burden of Disease Study (based originally on the English et al. 1995, series of meta-analyses[10]). Measures are in average grams of pure alcohol per day.

Abstainer

Drinking category I:	Women 0.01-19.99	Men 0-39.99
Drinking category II:	Women 20-39.99	Men 40-59.99
Drinking category III:	Women 40+	Men 60+

By classifying people into categories rather than simply as drinkers or non-drinkers, it is possible to more accurately assess the risks (and benefits) of drinking according to the amount consumed.[19]

2.2.3 Pattern of drinking

Average daily drinking volumes do not necessarily capture heavy drinking episodes or the environmental factors that put people at risk. The working group on the Comparative Risk Assessment within the WHO Global Burden of Disease 2000 project developed a scoring system that classifies pattern of drinking for populations, using evidence about average harmful and beneficial effects of alcohol consumption. In this system, higher scores indicate more health risks for a given volume of alcohol consumption.[3]

Under the WHO classification, pattern values are assigned:
0–3 points is classified as Pattern 1
4–6 points as Pattern 2
7–9 points as Pattern 3
10–17 points as Pattern 4

Alcohol consumption in New Zealand has previously been classified as Pattern 2.[3] Information in the National Alcohol and Te Ao Waipiro surveys was used to classify pattern of drinking for non-Mäori and Mäori populations separately.[25 26] The scoring system and scores for non-Mäori and Mäori are shown in Table 1. Non-Mäori were

allocated to pattern 2 (score=5) and Mäori to a Pattern 3 (score=8). There is some uncertainty around these scores because not all indicators are reported explicitly, but small changes would not alter the pattern categorisation. Mäori and non-Mäori patterns differ by a single item of the scoring system: usual quantity per drinking session.

Table 1. Classification of pattern of drinking in Mäori and non-Mäori using WHO criteria

Component	Scored item	Possible score	Non-Mäori	Mäori
Heavy drinking occasions (maximum 11 points)				
Daily drinking	Males: less than 20% drink daily	1	1	1
	Females: less than 10% drink daily	1	1	1
Frequency of getting drunk	Males: • most drinkers usually get drunk when drinking • most drinkers often get drunk	2 1	0	0
	Females: most drinkers usually or often get drunk	1	0	0
Usual quantity per drinking session	Males: • more than 60% typically consume 4+ drinks per session • between 40% and 60% consume 4+ drinks per session	2 1	1	2
	Females: • more than 50% consume 4+ drinks per session • between 35% and 50% consume 4+ drinks per session	2 1	0	2
Fiesta drinking	Males: fiesta drinking commonly occurs	1	0	0
	Females: fiesta drinking commonly occurs	1	0	0
Drinking with meals (maximum 4 points)				
Males	• rarely or never drink with meals • sometimes drink with meals	2 1	1	1
Females	• rarely or never drink with meals • sometimes drink with meals	2 1	1	1
Drinking with in public places (maximum 2 points)				
Drinking in public	Males: common and everyday	1	0	0
	Females: common and everyday	1	0	0
Total score (possible range 0 –17 points)			**5**	**8**

Based on the 2000 National Alcohol and Te Ao Waipiro surveys[25 26]

2.2.4 Extension to the over 65 age groups

The National Alcohol and Te Ao Waipiro surveys did not include people over 65 years of age. However, an important part of the impact of alcohol was expected to occur in those aged over 65 years because of the association of alcohol with ischaemic heart disease. Therefore prevalence estimates were made using other New Zealand studies.

Data were available from five studies.[23 27-30] These studies used questionnaires that were less detailed than National Alcohol and Te Ao Waipiro surveys, and only some of the studies were based on population samples.[16]

All five studies showed declining prevalence of drinking with increasing age. However, for people under 65 years these studies reported considerably lower proportions of drinkers in high exposure categories than the more recent National Alcohol and Te Ao Waipiro surveys. This may reflect recent changes in consumption following liberalisation in the laws relating to retail sales of alcohol in addition to the different methods of measurement.

New Zealand population percentages in each exposure category, for each sex and age group over 65 were estimated in the following manner. The proportions of participants in the 60-65 year age group from the combined 5 studies in each of the four exposure groups was calculated, then compared to those obtained from this age group in the National Alcohol and Te Ao Waipiro surveys. The resulting set of ratios was used to adjust the data from the five studies for the over 65 year olds, pooled by sex, age group and Mäori/non-Mäori status. The adjusted results are shown in Table 2, and were used in the analysis.

Table 2. Estimated alcohol consumption, for Mäori, non-Mäori and Total population (%), by sex and age group*

	Exposure level	Age group 15-29	30-44	45-59	60-69	70-79	80+
Males							
Mäori	Abs.	11.7	19.0	24.1	20.4	29.7	47.7
	I	52.8	58.6	59.5	67.8	62.3	48.7
	II	10.4	8.7	6.4	6.0	4.1	1.9
	III	25.2	13.8	10.0	5.8	3.9	1.7
Non-Mäori	Abs.	12.2	11.9	11.3	15.8	24.4	40.0
	I	59.8	66.9	72.7	73.5	68.6	56.7
	II	8.6	9.2	8.5	7.1	4.7	2.3
	II	19.3	12.0	7.5	3.5	2.3	1.1
Total	Abs.	11.9	12.3	11.9	17.8	26.6	42.2
	I	58.3	66.4	71.3	70.0	65.3	53.9
	II	9.7	9.1	8.7	8.2	5.5	2.6
	III	20.1	12.3	8.1	4.0	2.7	1.2
Females							
Mäori	Abs.	17.0	17.3	16.1	22.9	39.9	46.8
	I	57.1	65.8	67.4	68.3	55.1	49.4
	II	12.4	11.4	11.3	7.2	4.1	3.1
	III	13.5	5.4	5.2	1.6	0.9	0.7
Non-Mäori	Abs.	13.4	16.9	31.1	39.8	54.6	61.6
	I	59.2	68.8	59.5	53.6	41.6	35.6
	II	11.4	6.7	7.2	4.4	2.6	1.9
	III	16.0	7.6	2.3	2.2	1.3	0.9
Total	Abs.	16.2	17.5	17.6	25.8	41.9	48.4
	I	57.2	65.9	66.4	66.3	53.6	48.1
	II	12.6	11.0	11.0	6.5	3.7	2.9
	III	14.0	5.6	5.0	1.4	0.8	0.6

where exposure levels are defined as:

Abs:	Abstainer		
I:	Women 0-19.99	Men 0-39.99	grams of alcohol per day
II:	Women 20-39.99	Men 40-59.99	
III:	Women 40+	Men 60+	

* based on the 2000 National Alcohol and Te Ao Waipiro surveys (15-65 years), and adjusted 5 studies dataset (65-80+ years). (See 2.2.4 above)

2.2.5 Alcohol exposure for pregnant women

For perinatal and maternal conditions, data are required on alcohol consumption during pregnancy, but few surveys have specifically measured consumption in this period, especially during early pregnancy when the risks to the fetus are greatest. Given recent changes in consumption in New Zealand that are evident in the National Alcohol and Te Ao Waipiro survey data, particularly in the youngest age groups, we used the most recent New Zealand data, from a study of pregnant women in Wellington surveyed in 1999.[31] This study reported that at about 24 weeks gestation 19.7% of Mäori and 26.2% of non-Mäori had drunk alcohol over the previous 7 days.

Recent research suggests that drinking pattern, rather than average number of drinks per week, is likely to be the most significant factor affecting adverse pregnancy outcomes. Some investigators have found that the risk of defects is highest when women concentrate their weekly drinking by having five drinks or more in one day, while maintaining a weekly consumption of seven drinks.[32-34] Nevertheless, due to individual differences in sensitivity to alcohol, and the likelihood that at certain developmental time points the fetus is more sensitive to the effects of alcohol, it cannot be assumed that drinking fewer than five drinks per day is a safe threshold.

For this project, the prevalence of drinking in pregnancy from the Wellington survey (19.7% in Mäori and 26.2% in non-Mäori) was apportioned over the three exposure categories according to the distribution of levels of drinking amongst women drinkers in the wider population, by age group. Adjusted results are shown in Table 3. In this survey, responders were more likely to be married, non-smokers, with tertiary education, not receiving a benefit, and slightly older, than non-responders and there have been reports that women of higher socio-economic status are more likely to continue drinking during pregnancy.[35] However, two unpublished New Zealand studies suggest that these data may underestimate the proportion of women who are at-risk drinkers during pregnancy. In a report for ALAC in 2001, a survey of midwives estimated 7% of the pregnant population were regular drinkers and about 13% were drinking more than a glass a day or were regular binge drinkers.[36] The "Nutrition during Pregnancy" report to the Ministry of Health in 1999 estimated that 10% of pregnant women drank to intoxicating levels .[37]Another survey is currently being conducted by SHORE (2004), and will provide better estimates for future use.

Table 3. Estimated alcohol consumption, for Mäori, non-Mäori and total pregnant women in New Zealand (%)

	Exposure level	Age group 15-29	Age group 30-44	Total 15 - 44
Mäori	Abs.	80.3	80.3	80.3
	I	13.5	16.3	14.2
	II	2.6	1.6	2.3
	III	3.6	1.8	3.1
Non-Mäori	Abs.	73.8	73.8	73.8
	I	18.0	20.9	19.5
	II	3.9	3.6	3.8
	III	4.3	1.7	2.9
Total	Abs.	74.1	74.1	73.9
	I	17.7	20.7	19.1
	II	3.9	3.5	3.7
	III	4.3	1.7	3.1

where exposure levels are defined as:

Abs: Abstainer
I: Women 0.01-19.99
II: Women 20-39.99
III: Women 40+ grams of alcohol per day

Based on a survey by McLeod in Wellington, 1999 [31]

2.3 Alcohol-related conditions

2.3.1 Conditions included in the study

The selection of the conditions considered in this report to be wholly or partially attributable to alcohol is based on established epidemiological relationships, and so is inherently conservative. Conditions that have been considered by the CRA group to meet a basic set of criteria establishing alcohol as a contributing cause are summarized in Table 4. This assessment process included the review of a series of meta-analyses, [10 13-15] consideration of new research, and the documentation of biological evidence (for details see Rehm et al.[5]).

The conditions related to alcohol can be categorized into three groups:

- wholly alcohol-attributable conditions, with an alcohol-attributable fraction (AAF) of 100%
- chronic conditions where alcohol is a contributing cause (detrimental or beneficial)
- acute conditions where alcohol is a contributing cause

Table 4. Conditions where alcohol is considered a contributing cause

Always caused by alcohol (alcohol attributable fraction AAF = 1)
Alcohol abuse
Alcoholic psychoses
Alcohol dependence syndrome
Alcoholic fatty liver
Alcoholic hepatitis
Alcoholic cirrhosis of the liver
Alcoholic liver disease, other
Alcoholic hepatic failure
Alcoholic polyneuropathy
Alcohol cardiomyopathy
Alcoholic gastritis
Chronic pancreatitis (alcohol induced)
Fetal alcohol syndrome (dysmorphic)
Poisoning by exposure to alcohol
Detrimental impact of alcohol with AAF < 1
Low birth weight (including prematurity and intrauterine growth retardation)
Pulmonary and other respiratory tuberculosis
Pneumonia and influenza
Malignant neoplasm of lip, oral cavity and pharynx
Malignant neoplasm of oesophagus
Malignant neoplasm of liver and intrahepatic bile ducts
Malignant neoplasm of larynx
Breast cancer
Diabetes
Hypertension
Ischaemic heart disease
Cerebrovascular disease
Diseases of oesophagus, stomach and duodenum
Oesophageal varices
Fibrosis and cirrhosis of the liver, without mention of alcohol
Acute pancreatitis
Chronic pancreatitis (not alcohol induced)
Ischaemic heart disease
Cardiac arrhythmias
Unipolar depression
Epilepsy
Psoriasis
Conditions where alcohol is potentially beneficial
Diabetes
Ischaemic heart disease
Cerebrovascular disease (stroke)
Cholelithiasis
Acute conditions: injuries and poisonings (with AAF < 1)
Road traffic injuries
Water transport accidents
Air transport accidents
Accidental falls
Injuries caused by fire and flames
Accidental drowning and submersion
Suicide and self inflicted injury
Homicide and injury purposely inflicted by other person
Other injuries, including work-related injuries
Excessive cold
Poisoning

For the purposes of estimating the burden of death and disease in New Zealand due to the effect of alcohol on these conditions, we have included as many of these conditions as possible (Table 5).

There are two main reasons for conditions being omitted or combined into broader categories. First there is a lack of detailed epidemiological data from which to estimate risk for some conditions, and we have been restricted to conditions or groups of conditions where reliable estimates of the alcohol-attributable fraction can be derived. Secondly, when dealing with routinely collected data, misclassification is reduced by combining categories. We are confident that this simplification will not significantly affect the estimates as most of the alcohol-related burden is in a few disease categories.[38]

Consistent with the methods used for the WHO CRA for Alcohol report,[3] the analysis of outcomes in people under 15 years of age was restricted to the secondary effects of drinking by another person, for example, children injured in a car crash attributable to alcohol, or the effects of alcohol on birth weight. This excludes the consequences of drinking in under 15 year olds, mainly because there is little epidemiological evidence about prevalence or risk of alcohol consumption in this group.

Table 5. Classification of alcohol-related conditions included in this report

ICD-10 3 digit codes	Conditions
Cancers	
C00-14	Mouth and oropharyngeal cancers
C15	Oesophagus cancer
C22	Liver cancer
C32	Laryngeal cancer
C50	Breast cancer
Diabetes	
E10-14	Diabetes mellitus
Neuro-psychiatric disorders	
F10	Alcohol use disorders
F32-33	Unipolar depressive disorders
G40-41	Epilepsy
Cardiovascular disorders	
I10-13	Hypertensive heart disease
I20-25	Ischaemic heart disease
I47-48	Cardiac arrhythmias
I85	Oesophageal varices
I60-69	Stroke, ischaemic or haemorrhagic
Digestive disorders	
K80	Cholelithiasis
K85	Pancreatitis
K70	Alcoholic liver cirrhosis
K74	Unspecified liver cirrhosis
Conditions arising during pregnancy	
O03	Spontaneous abortion
P05-07	Low birth weight
Q86	Fetal alcohol syndrome
Injuries	
V02-04, V09, V12-14, V19-79, V86-89, Y32, Y85	Road traffic injuries
X45, X65, Y15	Alcohol poisoning
X40-44, X46-49, Y10-14, Y16-18	Other poisonings
W00-19, Y30-31	Falls
W65-74, Y21	Drownings
V01, V05-06, V10, V11, V15-18, V80-85, V90-99, W20-64, W75-99, X00-39, X50-59, Y19-Y20, Y22-29, Y86	Other unintentional injuries
X60-64, X66-84,	Self-inflicted injuries
X85-Y09	Violence
Y35, Y87	Other intentional injuries

2.3.2 Assessing population burden of alcohol-related conditions

Two complementary approaches to assessing the burden of these conditions in the New Zealand population have been taken.

Mortality

The first analysis is restricted to fatal outcomes only, with number of deaths and years of life lost (YLL) as measures of burden. Mortality for each of the conditions affected by alcohol has been estimated using routinely collected mortality data from the New Zealand

Health Information Service (NZHIS) mortality database. The number of deaths by 5 year age group, sex and Mäori/non-Mäori status, for each included condition by ICD-10 code (Table 5) were obtained for the year 2000, the most recent year for which data were available.

Disability adjusted life years (DALYs)

Non-fatal outcomes are also important in assessing the burden of alcohol-related conditions in the population. The relative importance of fatal and nonfatal outcomes to the overall health burden varies between conditions, and so the contribution of different conditions to the overall impact of alcohol on health is not reflected in the analysis of deaths alone.

Our second analysis summarises the impact of alcohol using disability-adjusted life years (DALYs), a measure which integrates fatal (measured by YLLs) and nonfatal outcomes (measured by years of life lived with disability (YLD) for each condition. Ideally, estimation of DALYs would utilise local data on disease incidence (rather than mortality) for each condition, for Mäori and non-Mäori separately. It would also require health state valuations for each condition that are appropriate to New Zealanders, and for Mäori and non-Mäori separately. As epidemiological evidence for disease incidence is limited and there are no New Zealand-specific health state evaluations, we have calculated DALY estimates only for the total New Zealand population. We have done so using the DALY burden for each condition in New Zealand in 2002, as estimated by the World Health Organisation. These estimates form part of the Global Burden of Disease (GBD) 2000 Study and were not available for Mäori and non-Mäori separately. The methods used in estimating the DALY burden for each GBD category of conditions is described in detail in the GBD2000 report.[39] Briefly, in these data, mortality estimates are based on analysis of latest available national information on levels of mortality and cause distributions. YLD estimates are based on the GBD2000 analyses of incidence, prevalence, duration and severity of conditions for the relevant epidemiological subregion, together with national and subnational level information available to WHO. These DALY estimates represent the best estimates of WHO, based on the evidence available in mid-2002.

2.3.3 Estimating alcohol-disease relationships and alcohol-attributable fractions (AAF)

A few conditions are, by definition, wholly attributable to alcohol. For all other conditions with an established relationship to alcohol use, the proportion of the burden that is attributed to alcohol must be established from the available epidemiological evidence.

As indicated above, both average volume of alcohol consumption and episodes of intoxication have been shown to influence alcohol-related burden of disease. Therefore the way in which the AAF for each condition is estimated depends on whether the condition is due to acute or chronic use of alcohol and whether the pattern of drinking has been established as an important determinant of the incidence of the condition. In all

cases the estimation of risk is made by comparison of drinkers (at various levels) with abstainers. That is, zero is the theoretical minimum exposure to alcohol.

The effect of pattern of drinking may be underestimated in many conditions, as pattern information has not been routinely collected in epidemiological studies. Conditions where the pattern of consumption has been demonstrated to have an effect independently of average volume of alcohol are ischaemic heart disease, unintentional injuries and intentional injuries.

In this report, the conditions that are affected by episodes of intoxication have alcohol-attributable fractions that are very different for Mäori (drinking pattern 3) and non-Mäori (drinking pattern 2).

Many of the estimates of relative risk and alcohol attributable fractions used in our analyses have been collated or developed by the WHO expert working group on alcohol for the global comparative risk assessment.[1 2] The details of the original epidemiological evidence for the risk relationships, and of the modeling techniques used to complement patchy empirical data have been published by the WHO.[3] The conditions for which New Zealand data have been used to modify these estimates are described in the text.

Harmful effects of alcohol on chronic conditions

For a number of chronic conditions where alcohol is a contributing cause, risk of disease increases with average volume of alcohol consumption, and we have no evidence that the pattern of drinking affects risk. For these conditions we have used the risk estimates derived from meta-analyses summarized in the WHO report, [3] in combination with prevalence data on levels of alcohol consumption in Mäori and non-Mäori (described in Section 2.2 above) to estimate alcohol-attributable fractions according to the following formula:

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

where p_i is the prevalence of exposure of level i, and RR_i is the risk at level i relative to no exposure. The AAFs for all non-zero levels of exposure (F) is obtained by adding the F_i. [40]

This group of conditions included cancers (of the mouth and pharynx, larynx, oesophagus, liver and breast), hypertensive disease, epilepsy, cardiac arrhythmias, oesophageal varices, pancreatitis, and low birth weight.

In the case of low birth weight the prevalence of drinking in pregnant women rather than the general population was used to calculate the attributable fraction.

Mental health

Co-morbidity of alcohol problems and other mental health conditions is well recognised. However, establishing the direction of causal relationships is difficult in this area, and there may be several causal pathways co-existing. Depression was included in the WHO study as it was considered there was sufficient evidence for a causal relationship. However, AAFs had never been estimated and there were no relative risk estimates for the contribution of alcohol to depression. Quantitative estimates of the proportion of depressive disorders attributable to alcohol were derived from the high correlation observed between the prevalence of alcohol dependence in a population and the proportion of depressive disorders with preceding alcohol-use disorders, in settings where such data are available.[3] In New Zealand, until the Survey of Mental Health and Wellbeing is completed, the only available data on general population prevalence of alcohol dependence comes from the Christchurch Psychiatric Epidemiology Study conducted in 1986.[41] The prevalence of alcohol abuse and/or dependence was 14.1% in men and 2.6% in women, with some further variation by age group.

Table 6. Six-month prevalence of alcohol abuse and/or dependence in Christchurch 1986

Age group	**18-24 years**	**25-44 years**	**45-64 years**	**Total**
Men	20.5%	13.0%	11.3%	14.1%
Women	4.4%	2.8%	1.2%	2.6%

In a further paper from the same study[42] the authors report that alcohol abuse alone and alcohol dependence (with or without abuse) were almost equally common, suggesting the prevalence of alcohol dependence was approximately 50% of the figures in Table 6.

These proportions do not reflect changes in drinking patterns over the last 17 years, or differences between Mäori and non-Mäori drinking, and so are likely to be conservative. The AAFs for major depression have been estimated from these data using the WHO CRA method, in the absence of anything more complete or up to date, and are included in Table 7.

Potentially beneficial effects of alcohol on chronic conditions

Type II diabetes

There is growing evidence that low to moderate alcohol consumption reduces the risk of Type II diabetes, and relative risks have been estimated by Gutjahr et al.,[15] based on review of the international literature. The risk curve for diabetes is J-shaped with maximal benefit at moderate levels of average alcohol consumption. The importance of drinking pattern on this relationship has not yet been established. Evidence from a recent New Zealand study suggests a similar J-shaped relationship starting at lower levels of alcohol

consumption in men.[43] However, we have not employed these estimates in these analyses since the small number of heavy drinkers in the study sample makes estimation only possible for the lower drinking categories. Instead we have used the WHO estimates, recognizing they may underestimate any benefit. The New Zealand data demonstrate that the relationship is similar for Mäori and non-Mäori, and so the same estimates of relative risk have been used for both groups.

Stroke

Stroke includes ischaemic and haemorrhagic subtypes. Ischaemic stroke accounts for approximately 80% of stroke mortality in New Zealand and has similar risk factors as ischaemic heart disease. The risk of haemorrhagic stroke is more strongly related to blood pressure, which is adversely influenced by alcohol consumption. Ischaemic and haemorrhagic stroke are thus affected differently by alcohol, with low to moderate consumption conferring some protective effect in ischaemic stroke, and this effect being more pronounced in women than men. The risk of all types of stroke is increased at high levels of alcohol consumption.[44] For haemorrhagic stroke, the meta-analysis conducted by Ridolfo and Stevenson[12] that was used in the WHO CRA suggests an increase in risk in males even at low levels of consumption but a strong protective effect for women in drinking less than 40 g/day of alcohol. However, a recent meta-analysis[45] and cohort study[46] have not shown such a difference between men and women. Moreover, the marked protective effect in women with low and moderate drinking, and the very high risk (RR=8) associated with heavy drinking in women described in the Ridolfo and Stevenson meta-analysis were not confirmed in the more recent reports.

Thus, the relationship between alcohol and subtypes of stroke appears complex, and the evidence is still somewhat conflicting.[45 47] It seems likely that pattern of drinking, in addition to average volume, will affect the risk of ischaemic stroke, but no reliable evidence for this is yet available. We have therefore taken the same approach for Mäori as for non-Mäori.

As well as the uncertainty about alcohol-related risk and stroke subtype, the subtype of stroke is commonly misclassified or unclassifiable in the collection of mortality and morbidity data. Consequently, in this analysis we have used the relative risk estimates for the effect of different levels alcohol consumption on total stroke incidence from the recent meta-analysis of Reynolds et al.,[45] and applied these to the combined mortality data for ischaemic, haemorrhagic and unclassified stroke.

Ischaemic heart disease

Ischaemic heart disease (IHD), or coronary heart disease, is one of the leading causes of death in New Zealand, as in the rest of the world. Mäori are disproportionately affected by

this burden and it is the main reason for the increasing difference in life expectancy between Mäori and non-Mäori.[7 48]

The relationship reported between alcohol and IHD is complex and epidemiological evidence is evolving. No systematic differences have been confirmed between the effects of different alcohol-containing beverages.[49] The beneficial effects of alcohol on IHD are maximal at low average volumes of drinking and although some studies have shown a benefit with all levels of drinking, a recent meta-analysis[50]demonstrates a J-shaped curve, with increased risk of IHD with heavy drinking, relative to abstainers. The beneficial effect is less for women than men and less for fatal heart attacks than non-fatal. The maximum protective effect was estimated by Corrao at 20g alcohol per day. Interestingly, the best-designed studies in this meta-analysis showed the smallest benefit, suggesting than many studies have over estimated the protection due to alcohol because of confounding. Inconsistency between the findings of individual studies may be partly due to the effect of pattern of drinking on IHD, [3] but there is some concern that much of the evidence comes from studies conducted in low risk groups.

For these reasons, the WHO alcohol CRA group developed a multilevel model for the impact of average volume and pattern of drinking on IHD mortality, using data from all over the world. Pattern of drinking was described by one of four categories (Appendix A), and details of the modeling are given in the WHO report. [3] This analysis found a beneficial effect on IHD mortality for countries with the most beneficial pattern of drinking (pattern 1), little impact for countries with pattern 2 drinking, and increasingly detrimental effects for pattern 3 (men only) and 4 (men and women).

A population-based case-control study of IHD in non-Mäori was carried out in Auckland in 1992. Analyses of the contribution of alcohol to IHD have recently been published [24] and provide local estimates of the protective effect of drinking. This effect was considerably stronger than the estimates used in the CRA for pattern 2 drinkers. However, there were few category III drinkers in this study especially among women, making the estimates of risk in this category very imprecise. Given this limitation and the evidence from the meta-analysis of Corrao that better quality studies showed a smaller protective effect, we have used the more conservative estimates from the CRA for these calculations.

We have used the same relative risk estimates for calculating the AAF for both Mäori and non-Mäori, although the WHO classification of pattern of drinking is different for the two ethnic groups, and the WHO approach would attribute no preventive effect to alcohol consumption in Mäori. Recent literature supports the importance of pattern of drinking in determining the benefits for alcohol for prevention of heart disease (see summaries by McKee and Britton,[51]Puddey et al.,[52]and Rehm[19]). However, the difference in pattern of drinking category for Mäori and non-Mäori was based on the criterion of “usual quantity per drinking session” alone, and without considering the effect of different drinking patterns of age-sex subgroups in the population. We considered that there was important

uncertainty about whether this would result in a significant difference in the relationship of alcohol to IHD in Mäori and non-Mäori, although it is clearly important for acute effects of alcohol such as injury. In the US, where African Americans and white Americans are also mainly distinguished by usual quantity per drinking session, the relationship between average alcohol intake and coronary disease has been reported for both groups from the same cohort, representative of the general population. While a significant protective effect was found for white Americans,[53] no protective effect was found for African Americans.[54] However, small numbers of events limited the precision of the risk estimates for African Americans, and did not rule out a modest protective effect. Modeling of the effect of alcohol on coronary disease events in the whole cohort demonstrated significant effect modification by ethnicity in moderate drinking categories. There are no similar data for Mäori.

An investigation of the relationship of frequency and volume of alcohol with some IHD risk factors in Mäori and non-Mäori has been conducted recently (personal communication, Dr Dale Bramley). These analyses demonstrated that the associations of average frequency and average volume of alcohol drinking with HDL cholesterol and the total cholesterol:HDL ratio were very similar in Mäori and non-Mäori, suggesting that there was no important effect of differences in pattern of drinking However, there were insufficient data to determine whether this was true for the other CVD risk factors studied (systolic and diastolic blood pressure, serum glucose) and there were no markers of the effects of alcohol on blood clotting. These data, derived from pooling five studies,[23 27-30] were not representative of the Mäori and non-Mäori populations overall and probably over-represented low risk drinkers.

Thus, without case-control or cohort data from a representative sample of the Mäori population, it is not clear what the impact of alcohol on IHD is in Mäori. It probably lies somewhere between the protective effect described for non-Mäori and no effect. The analysis for this report has assumed a protective effect for Mäori the same as for non-Mäori, but we have assessed the impact of this assumption by recalculating alcohol-related health burden excluding any beneficial effect on IHD for Mäori (see Section 3.5).

Cholelithiasis

A strong inverse relationship between alcohol consumption and gallstones has been substantiated in a series of large studies. Complications of cholelithiasis are responsible for around 30 deaths and significant morbidity each year in New Zealand. We have used the estimates of relative risk for this condition derived from the international literature by WHO.[3]

Table 7 summarises the relative risk estimates for the major chronic disease categories discussed above.

Table 7: Relative risk for major chronic disease categories by sex and average drinking category

Disease	ICD-10 (3-digit)	Males			Females		
		Drinking category			Drinking category		
		I	II	III	I	II	III
Cancers							
Mouth and oropharynx cancers	C00–C14	1.45	1.85	5.39	1.45	1.85	5.39
Oesophagus cancer	C15	1.80	2.38	4.36	1.80	2.38	4.36
Liver cancer	C22	1.45	3.03	3.60	1.45	3.03	3.60
Laryngeal Cancer	C32	1.83	3.9	4.93	1.83	3.9	4.93
Female breast cancer							
<45 years of age	C50	NA	NA	NA	1.15	1.41	1.46
≥45 years of age		NA	NA	NA	1.14	1.38	1.62
Diabetes mellitus	E10–E14	1.00	0.57	0.73	0.92	0.87	1.13
Neuro-psychiatric conditions							
Alcohol-use disorders	F10	AAF 100%	AAF 100%	AAF 100%	AAF 100%	AAF 100%	AAF 100%
Unipolar major depression	F32–F33	AAFs were estimated indirectly based on prevalence of alcohol dependence					
Epilepsy	G40–G41	1.23	7.52	6.83	1.34	7.22	7.52
Cardiovascular diseases							
Hypertensive disease	I10–I13	1.40	2.00	4.10	1.40	2.00	2.00
Ischaemic heart disease	I20–I25	0.82	0.83	1.00	0.82	0.83	1.12
Cardiac arrhythmias	I46-49	1.51	2.23	2.23	1.51	2.23	2.23
Cerebrovascular disease (stroke)	I60–I69	0.91	1.03	1.76	0.72	0.82	2.01
Oesophageal varices	I85	1.26	9.54	9.54	1.26	9.54	9.54
Digestive diseases							
Alcoholic cirrhosis of the liver	K70	1.30	9.50	13.00	1.30	9.50	13.00
Cholelithiasis	K80	0.82	0.68	0.50	0.82	0.68	0.50
Pancreatitis	K85	1.30	1.80	3.20	1.30	1.80	3.20
Conditions arising during pregnancy							
Spontaneous abortion	O03	NA	NA	NA	1.20	1.76	1.76
Low birth weight	P05–P07	1.00	1.40	1.40	1.00	1.40	1.40
Fetal alcohol syndrome	Q86	AAF 100%	AAF 100%	AAF 100%	AAF 100%	AAF 100%	AAF 100%

NA Not applicable

Drinking exposure categories:

I: Women >0-<20g; Men >0-<40g /day

II: Women 20-<40g; Men 40-<60g /day

III: Women >40g; Men >60g /day

(Adapted from Rehm et al.[3])

Acute adverse effects of alcohol: Injuries

The acute effects of alcohol are related to intoxication, and less often to poisoning. As a result, there is a marked effect of pattern of drinking on the alcohol attributable fractions (AAFs) for all causes of acute harm from drinking. The difference in pattern of drinking between Mäori and non-Mäori shown in Table 1 translates into a higher risk of acute consequences of drinking in Mäori than non-Mäori, and the AAFs for injury reflect this.

Alcohol use has been associated with an increase in risk of injury in a wide variety of settings, including all kinds of road users (car occupants, motorcyclists, cyclists, pedestrians), falls, fires, drowning, sports injuries, work-related injuries, as well as self-inflicted injuries and violence.[4 5 55] Even moderate doses of alcohol have cognitive and psychomotor effects such as increased reaction time, impaired cognitive processing, and reduced co-ordination and vigilance that are relevant to the risk of injury.[56]

Unintentional injuries

Studies relating average volume of drinking to risk of injury have found that risk of injury is positively related to average consumption of alcohol, and that increased risk starts at relatively low volumes of intake.[57] However, as one would expect, injury risk is determined largely by level of blood alcohol at the time of exposure to risk, and so is strongly related to specific patterns of drinking. Frequency of heavy drinking and intoxication are both associated with injury in general [58] as well as with death due to injury.[59] In studies of the relationship between drinking pattern and risk of injury from driving, the highest risk was found for individuals who consume relatively large amounts on some occasions, and whose highest amounts are markedly greater than the average amount per occasion, after adjusting for other drinking pattern variables and characteristics of the drinker ("binge drinking").[60-63]

Road traffic injury is the largest cause of unintentional injury death and disability, and is also the best studied. Case-control studies comparing blood alcohol concentration (BAC) levels of drivers in crashes with control drivers not involved in crashes have consistently found increasing risk with increasing levels of BAC. All levels of BAC are associated with an increased risk of crashes relative to a BAC of zero, but the risk curve gets steeper at high BACs. [64]

Although the effects of alcohol on ability to drive may be similar, alcohol attributable fractions for traffic related injuries (and also injuries in other settings) vary by time and place. This is not only because drinking patterns differ but also due to the varying prevalence of other risk factors. Thus, the generalisation of AAFs for injuries is less certain than for the risk relationships between alcohol and chronic diseases.

In New Zealand, AAFs for traffic injury can be calculated directly from the Auckland Car Crash Injury Study (ACCIS), a population-based case control study of risk factors for

crashes resulting in hospitalisation or death of a car occupant.[65] This study, carried out in 1998-9, although limited in precision by its size, has been used to calculate age, sex and ethnicity-specific AAFs for a general population of drivers.

Deriving AAFs for injury in New Zealand

As injuries are influenced by both average volume of alcohol consumption and intoxication, the WHO CRA group modeled this relationship in the same way as for IHD. A multilevel analysis was conducted that included average volume of alcohol and pattern of drinking. They found no difference in injury risk between pattern 1 and pattern 2 drinking, but pattern 3 had a significantly higher injury risk for both sexes that was more marked in males. Using this model, AAFs for New Zealand were derived from Australian AAFs, since these reflected the most up-to-date information.[12] Australian AAFs were adjusted for differences in per capita alcohol consumption but not for differences in pattern as overall New Zealand has the same drinking pattern as Australia (pattern 2).

To estimate the AAFs for injury in Mäori, the effect of the different pattern of drinking needed to be incorporated. The CRA model found that a scaling factor of 2.1 was required to adjust for the difference between pattern 2 drinking and pattern 3 drinking; that is, the proportion of injury attributable to alcohol in Mäori is treated as twice that of non-Mäori. However, it was not possible to adjust for differences in socioeconomic status or age structure in the model as would be done when considering two different countries. Using local data from ACCIS, we can directly measure AAFs for car crash injury in Mäori, non-Mäori and the total population (Appendix B). This demonstrates that scaling by a factor of 1.5 from the total population AAFs is more realistic. Therefore, this has been used to adjust the AAFs for injury from pattern 2 drinkers (non-Mäori) to pattern 3 drinkers (Mäori) in the New Zealand context. In the case of children aged under 15 years who are injured as the result of alcohol impaired driving, the overall AAF from the ACCIS study is 22%. However due to small numbers, no subgroup analysis is possible. We have applied these estimates to both boys and girls and scaled non-Mäori AAFs up by 1.5 for Mäori.

Another important aspect of the relationship of alcohol to injury is the difference in the contribution of alcohol to fatal and non-fatal outcomes. The CRA group has estimated that the AAFs for non-fatal traffic injury are two-thirds that of fatal injury, and for other types of (non-traffic) injury the proportion is 44%. In ACCIS, the overall AAF and the estimates for men and women separately were very close to the CRA estimates. In ACCIS, 27% of injury crashes were attributable to alcohol compared with 29% in CRA, 33% vs 36% for men, and 18% vs 15% for women (Appendix B). However, the ACCIS AAFs were predominantly based on non-fatal injury crashes, with no change in the estimates when fatal crashes were excluded, whereas the CRA model is for fatal crashes and would estimate nonfatal crashes at just 66% of these AAFs.[3]

Although the ACCIS data do not allow a precise estimation of the AAF for fatal crashes separately, there is no evidence in this study that it is higher than for non-fatal crashes. One possible explanation for this is that the ACCIS study considered all levels of alcohol as potentially contributing to risk and to burden of injury, and found a significant contribution to the total burden from very low levels of blood alcohol concentration (<50mg/100ml).[65] In many previous analyses, drivers with blood alcohol levels below the legal limit (rather than zero) have been used as the reference group when calculating risk relationships.[10 12 14] On the basis of the ACCIS findings being recent and of New Zealand origin, we have used the AAFs from the ACCIS study in the road traffic accident analyses in this report.

Other injury estimates

For acute consequences of alcohol consumption other than road traffic crashes, estimates of relative risk at different levels of alcohol are rare. Therefore, we have used CRA estimates of AAFs derived from review of the literature. These are almost entirely based on case series. Estimates from Australian data have been adjusted for the difference in per capita consumption between New Zealand and Australia, while assuming the same pattern of drinking (Pattern 2).

We have used these AAFs for pattern 2 drinking for non-Mäori, and scaled them by 1.5 for Mäori, as discussed above. We have applied the CRA scaling factor of 0.44 to the AAF for non-fatal injuries compared with fatal injuries, in the absence of any local data.

2.3.4 AAFs for alcohol-related conditions

The alcohol attributable fractions given in Table 8 for Mäori and Table 9 for non-Mäori represent the proportion of each condition that would be prevented if everyone had the same risk as abstainers, that is if no one drank alcohol. In some cases these attributable fractions are negative (e.g. ischaemic heart disease), which means that abstaining from alcohol does not confer the lowest risk for these conditions.

Table 8: AAFs for all alcohol-related conditions : Mäori population by age group and sex

	Mäori Male								Mäori Female							
	0-4	**5-14**	**15-29**	**30-44**	**45-59**	**60-69**	**70-79**	**80+**	**0-4**	**5-14**	**15-29**	**30-44**	**45-59**	**60-69**	**70-79**	**80+**
Mouth and oropharynx cancers			58.8%	48.5%	43.3%	37.9%	32.7%	23.7%			51.6%	41.2%	30.0%	27.3%	20.9%	17.8%
Laryngeal cancer			63.3%	56.1%	51.8%	49.1%	44.1%	34.5%			59.2%	51.6%	44.2%	39.7%	32.0%	27.9%
Oesophagus cancer			58.5%	51.2%	47.4%	45.0%	40.7%	32.1%			53.9%	47.3%	39.4%	36.1%	29.1%	25.5%
Liver cancer			52.4%	44.4%	39.7%	36.6%	31.7%	23.2%			47.8%	39.2%	32.1%	27.9%	21.4%	18.2%
Breast cancer											17.3%	14.2%	11.1%	9.5%	7.1%	5.9%
Diabetes mellitus			-12.7%	-8.0%	-5.8%	-4.3%	-2.9%	-1.3%			-4.3%	-5.7%	-5.7%	-4.8%	-3.6%	-3.1%
Alcohol use disorders			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Unipolar depressive disorders			17.4%	13.0%	11.3%	6.2%	3.1%	1.6%			3.7%	2.8%	1.2%	0.7%	0.4%	0.2%
Epilepsy			69.4%	60.0%	53.3%	46.9%	38.8%	25.1%			64.5%	51.0%	42.4%	35.0%	25.2%	20.7%
Hypertensive heart disease			52.3%	42.8%	38.0%	33.8%	29.1%	21.1%			45.9%	36.7%	27.5%	24.6%	18.8%	16.0%
Ischaemic heart disease			-12.7%	-13.7%	-13.4%	-15.2%	-13.5%	-10.0%			-14.4%	-15.6%	-13.5%	-11.6%	-8.6%	-7.2%
Cardiac arrhythmias			41.4%	36.5%	33.6%	32.9%	29.4%	22.6%			39.0%	34.5%	29.5%	26.2%	20.6%	17.8%
Stroke (including all subtypes)			12.8%	5.2%	2.4%	-1.5%	-2.6%	-3.1%			6.7%	-0.2%	-3.5%	-3.1%	-2.8%	-2.5%
Oesophageal varices			76.0%	67.4%	60.9%	54.1%	45.7%	30.3%			71.4%	58.4%	49.0%	41.3%	30.3%	25.1%
Alcoholic liver cirrhosis			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Cholelithiasis			-36.0%	-27.2%	-23.4%	-22.5%	-18.6%	-12.6%			-30.7%	-24.6%	-18.1%	-15.3%	-10.8%	-8.9%
Pancreatitis			44.3%	35.4%	31.1%	27.5%	23.4%	16.6%			38.3%	30.0%	22.2%	19.6%	14.8%	12.5%
Low birth weight	2.1%								2.1%							
Fetal alcohol syndrome	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Alcohol poisoning	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Non-alcohol poisoning			51.9%	28.6%	28.6%	28.6%	14.3%	14.3%			39.9%	26.0%	26.0%	26.0%	12.1%	12.1%
Road traffic injuries	32.9%	32.9%	60.4%	58.7%	20.6%	13.7%	13.7%	13.7%	32.9%	32.9%	36.8%	34.6%	10.8%	3.1%	3.1%	3.2%
Falls			39.4%	39.4%	39.4%	30.4%	21.5%	21.5%			24.3%	24.3%	24.3%	15.6%	6.9%	6.9%
Drownings			46.5%	55.5%	55.5%	44.8%	44.8%	44.8%			43.3%	52.0%	52.0%	41.6%	41.6%	41.6%
Other unintentional injuries	28.6%	28.6%	51.9%	51.9%	43.0%	43.0%	43.0%	43.0%	8.7%	8.7%	39.9%	39.9%	32.9%	32.9%	32.9%	32.9%
Self-inflicted injuries			17.9%	17.9%	13.1%	13.1%	6.0%	6.0%			11.6%	11.6%	9.2%	9.2%	5.8%	5.8%
Violence	25.1%	25.1%	48.3%	48.3%	48.3%	48.3%	48.3%	48.3%	24.3%	24.3%	46.8%	46.8%	46.8%	46.8%	46.8%	46.8%
Other intentional injuries			23.9%	23.9%	23.9%	23.9%	11.9%	11.9%			23.1%	23.1%	23.1%	23.1%	11.6%	11.6%

Table 9: **AAFs for all alcohol-related conditions : Non-Mäori population by age group and sex**

	Non-Mäori Men								Non-Mäori Women							
	0-4	**5-14**	**15-29**	**30-44**	**45-59**	**60-69**	**70-79**	**80+**	**0-4**	**5-14**	**15-29**	**30-44**	**45-59**	**60-69**	**70-79**	**80+**
Mouth and oropharynx cancers			54.4%	47.6%	42.1%	35.3%	31.0%	24.3%			48.9%	38.7%	38.5%	30.5%	24.3%	21.8%
Laryngeal cancer			60.1%	56.4%	53.4%	48.9%	44.4%	36.6%			57.7%	52.2%	52.1%	45.6%	37.9%	34.5%
Oesophagus cancer			55.5%	51.6%	48.7%	44.6%	40.9%	34.2%			52.0%	46.4%	46.5%	41.1%	34.5%	31.6%
Liver cancer			48.6%	44.4%	41.0%	36.2%	31.7%	24.8%			46.2%	40.1%	40.0%	33.1%	26.1%	23.3%
Breast cancer											16.6%	14.6%	14.5%	11.7%	8.9%	7.9%
Diabetes mellitus			-9.8%	-7.7%	-6.0%	-4.2%	-2.7%	-1.3%			-4.6%	-6.4%	-6.6%	-6.6%	-5.1%	-4.5%
Alcohol use disorders			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Unipolar depressive disorders			17.4%	13.0%	11.3%	6.2%	3.1%	1.6%			3.7%	2.8%	1.2%	0.7%	0.4%	0.2%
Epilepsy			64.6%	59.2%	53.7%	45.7%	37.6%	25.4%			64.9%	56.3%	55.9%	43.9%	33.3%	29.0%
Hypertensive heart disease			48.1%	42.3%	37.8%	32.2%	28.2%	22.0%			32.8%	30.2%	30.3%	26.5%	21.3%	19.1%
Ischaemic heart disease			-13.9%	-15.7%	-17.0%	-16.9%	-15.1%	-11.8%			-12.1%	-15.1%	-15.5%	-15.4%	-11.7%	-10.3%
Cardiac arrhythmias			39.4%	37.6%	36.2%	33.6%	30.4%	24.8%			37.9%	35.2%	35.3%	31.3%	25.5%	23.0%
Stroke (including all subtypes)			8.7%	3.3%	-0.6%	-3.9%	-4.5%	-4.4%			-4.8%	-17.7%	-18.6%	-23.2%	-18.0%	-15.9%
Oesophageal varices			71.8%	66.5%	60.9%	52.4%	43.8%	30.2%			70.3%	61.7%	61.2%	48.1%	36.2%	31.3%
Alcoholic liver cirrhosis			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%			100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Cholelithiasis			-32.3%	-28.7%	-26.6%	-23.1%	-19.6%	-14.4%			-28.4%	-24.3%	-24.5%	-20.1%	-14.6%	-12.6%
Pancreatitis			40.3%	35.0%	31.1%	26.2%	22.7%	17.5%			36.2%	29.0%	28.9%	22.9%	17.9%	15.8%
Low birth weight	2.6%								2.6%							
Fetal alcohol syndrome	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Alcohol poisoning	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Non-alcohol poisoning			34.6%	19.1%	19.1%	19.1%	9.5%	9.5%			26.6%	17.3%	17.3%	17.3%	8.1%	8.1%
Road traffic injuries	21.9%	21.9%	40.3%	39.1%	13.8%	9.1%	9.1%	9.1%	21.9%	21.9%	24.6%	23.1%	7.2%	2.1%	2.1%	2.1%
Falls			26.3%	26.3%	26.3%	20.3%	14.3%	14.3%			16.2%	16.2%	16.2%	10.4%	4.6%	4.6%
Drownings			31.0%	37.0%	37.0%	29.8%	29.8%	29.8%			28.9%	34.7%	34.7%	27.7%	27.7%	27.7%
Other unintentional injuries	19.1%	19.1%	34.6%	34.6%	28.6%	28.6%	28.6%	28.6%	5.8%	5.8%	26.6%	26.6%	21.9%	21.9%	21.9%	21.9%
Self-inflicted injuries			17.9%	17.9%	13.1%	13.1%	6.0%	6.0%			11.6%	11.6%	9.2%	9.2%	5.8%	5.8%
Violence	16.7%	16.7%	32.2%	32.2%	32.2%	32.2%	32.2%	32.2%	16.2%	16.2%	31.2%	31.2%	31.2%	31.2%	31.2%	31.2%
Other intentional injuries			23.9%	23.9%	23.9%	23.9%	11.9%	11.9%			23.1%	23.1%	23.1%	23.1%	11.6%	11.6%

2.4 Calculation of alcohol attributable burden of disease and injury: mortality, YLLs and DALYs

Alcohol attributable mortality has been calculated by multiplying the mortality from each alcohol-related condition in 2000 by the alcohol attributable fraction for that condition (Tables 8 and 9), for each age, sex and ethnicity subgroup.

Years of life lost (YLL) are a measure of the burden of premature mortality. YLLs have been derived from our mortality data by the 'remaining life expectancy method'. Rather than assessing the gap between age at death and a "cut-off" age such as 65 years, this method measures the difference between age at death and life expectancy remaining at that age.

There are a number of limitations in using the population's own current life table to determine life expectancy remaining,[66] not the least of which is how to deal with differences between Mäori and non-Mäori life expectancy. Therefore, a model life table is used to define the standard for comparison. In this analysis, the Coale and Demeny model life table West level 26 has been used, which provides a life expectancy at birth of 82.5 years for women and 80 years for men. This standard was chosen as it was used in the GBD Study.[67] It provides consistent life expectancies for Mäori and non-Mäori at any given age.

To reflect society's preference for present rather than future benefits, health losses that occur in the future are discounted relative to those in the present. A discount rate of 3 percent per annum has been used as recommended by WHO. [67]

Alcohol attributable DALYs have been calculated by multiplying the DALY count for each alcohol-related condition in New Zealand in 2002 (provided by WHO) by the alcohol attributable fraction for that condition (Tables 8 and 9), for each age and sex subgroup. DALY counts are age-weighted and discounted at 3 per cent per annum.

Mortality, YLL, and DALY rates have been derived from the counts, using 2000 and 2002 mid-year population estimates provided by Statistics New Zealand. Rates were age standardised by the direct method using the WHO World population as the standard population.[68]

The calculations presented in this report were facilitated by the use of country-level burden of disease templates developed and provided by WHO.

3. ALCOHOL-ATTRIBUTABLE MORTALITY

3.1 Overview

We estimated that approximately 1037 deaths in New Zealand in 2000 were attributable to alcohol consumption; representing 3.9% of all deaths. Alcohol consumption was also estimated to prevent 981 deaths in the same year, resulting in a net loss of 56 lives.

The mortality burden was not evenly distributed by sex or ethnicity (Table 10). In non-Mäori women, deaths prevented by alcohol consumption outweighed deaths caused, but in all men and in Mäori women more deaths were caused than prevented. The standardised alcohol-related death rate for men was considerably higher than for women in both Mäori and non-Mäori. The alcohol-related death rate for Mäori overall was 4.2 times the rate for non-Mäori, when standardisation to the WHO world population had eliminated the effect of differences in the age structure of the two populations. More lives were lost due to alcohol as well as fewer deaths prevented by alcohol in Mäori compared with non-Mäori, relative to the size of their populations.

Table 10: Mortality attributable to alcohol, by ethnicity and sex, 2000

	Ethnicity	Deaths caused	% of all deaths	Deaths prevented	Net deaths (count)	Net deaths (rate)*
Males	Mäori	161	11.3	47	114	37.8
	Non-Mäori	557	4.5	476	81	9.7
	Total	718	5.2	523	195	13.6
Females	Mäori	45	3.9	26	19	1.9
	Non-Mäori	273	2.3	431	-158	-0.8
	Total	319	2.5	457	-139	-0.1
Total	Mäori	206	8.0	73	133	19.0
	Non-Mäori	831	3.4	907	-77	4.5
	Total	1037	3.9	981	56	6.7

* rate per 100,000 age-standardised to WHO world population

3.2 Causes of alcohol-attributable deaths

The overall figures for alcohol-related deaths are the balance of deaths caused and deaths prevented by the effect of alcohol on different fatal conditions. As Table 11 shows, more than half of the alcohol-related mortality was due to injuries (including intentional and unintentional injuries).

Table 11: Proportions of alcohol-attributable mortality due to cancer, other chronic disease and injuries

	Men	Women	% of all deaths attributable to alcohol (n=1037)
Cancers	135	113	23.9%
Other chronic diseases	147	108	24.6%
Injuries	435	97	51.3%

The numbers of deaths estimated to be prevented by alcohol consumption in New Zealand are shown in Table 12. Almost all were due to alcohol drinking reducing the risk of ischaemic heart disease and stroke.

Table 12: Deaths prevented by alcohol consumption, by cause of death

	Men	Women	% of all deaths prevented by alcohol (n=981)
Ischaemic heart disease	466	299	78.0%
Stroke	40	136	18.0%
Diabetes	13	19	3.3%
Cholelithiasis	3	3	<1%

There was a strong effect of pattern of drinking on the major cause of lives lost (injury). Over and above the effect of average volume of alcohol consumed, the difference in pattern of drinking between pattern 2 and pattern 3, as described above, increased the detrimental effect on injuries. There was also a lesser preventive effect of alcohol on ischaemic heart disease in Mäori due to the smaller proportion the Mäori population in the oldest age groups and the smaller proportion of people who drank any alcohol. These differences are seen in the summary of mortality by cause of death for Mäori and non-Mäori in Table 13.

Table 13: Mortality attributable to alcohol by major disease and injury categories, 2000

	Mäori			Non-Mäori			Total		
	Men	Women	All	Men	Women	All	Men	Women	All
Mouth and oropharynx cancers	4	1	5	30	8	37	34	9	43
Laryngeal cancer	1	0	1	12	1	13	14	1	15
Oesophagus cancer	8	1	9	44	19	64	53	20	73
Liver cancer	10	1	11	25	12	37	35	13	48
Breast cancer		6	6		64	64		70	70
Diabetes mellitus	-4	-3	-8	-9	-16	-25	-13	-19	-32
Alcohol use disorders	3	0	3	14	4	18	17	4	21
Unipolar depression	0	0	0	0	0	0	0	0	0
Epilepsy	1	1	2	17	5	22	18	6	24
Hypertensive heart disease	5	3	8	18	25	44	23	28	51
Ischaemic heart disease	-42	-20	-62	-424	-279	-703	-466	-299	-765
Cardiac arrhythmias	2	1	3	21	31	52	23	32	70
Stroke (all subtypes)	0	-2	2	-40	-134	-174	-40	-136	-176
Oesophageal varices	0	0	0	1	1	2	1	1	2
Alcoholic liver cirrhosis	4	4	8	57	29	86	61	33	94
Cholelithiasis	0	-1	-1	-3	-2	-5	-3	-3	-5
Pancreatitis	0	0	1	3	3	6	3	3	6
Low birth weight	0	0	0	0	0	1	1	1	1
Fetal alcohol syndrome	0	0	0	0	0	0	0	0	0
Alcohol poisoning	3	1	4	4	1	5	7	2	9
Non-alcohol poisoning	1	0	2	5	3	8	6	3	9
Road traffic injuries	37	11	48	75	17	92	112	29	140
Falls	5	1	6	18	7	25	23	8	31
Drownings	4	1	4	11	2	13	14	3	17
Other unintentional injuries	53	8	60	147	29	176	200	36	236
Self-inflicted injuries	12	1	13	47	7	54	58	9	67
Violence	6	3	9	5	4	10	12	7	19
Other intentional injuries	0	0	0	0	0	0	0	0	0
All causes	**114**	**19**	**133**	**81**	**-158**	**-77**	**195**	**-138**	**56**

3.3 Alcohol-attributable deaths by age group

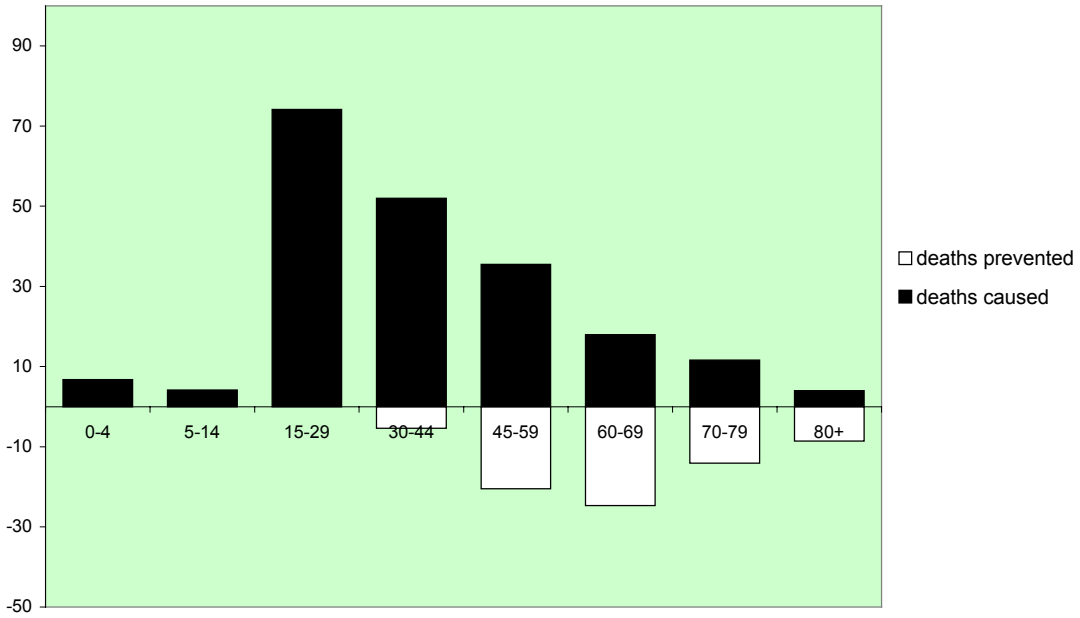
The predominance of injury as a cause of death in children and young adults, and of ischaemic heart disease and stroke in older adults, means that the balance of risks and benefits of alcohol consumption varied with age. Table 14 shows overall mortality due to alcohol by age group. Figures 2 and 3 show the balance between detrimental and preventive effects of alcohol for Mäori and non-Mäori, at different ages.

Table 14: Net mortality attributable to alcohol by ethnicity and age group, 2000

		0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+
Males	Mäori	5	2	63	37	11	-3	-1	-1
	Non-Mäori	4	3	115	115	35	-5	-79	-107
	Total	9	5	178	152	46	-8	-80	-108
Females	Mäori	2	2	11	9	4	-4	-1	-4
	Non-Mäori	1	3	23	24	38	-2	-54	-193
	Total	3	5	34	33	42	-6	-55	-197
Total	Mäori	7	4	74	46	15	-6	-2	-5
	Non-Mäori	5	6	138	139	73	-7	-133	-300
	Total	12	10	212	186	88	-13	-135	-305

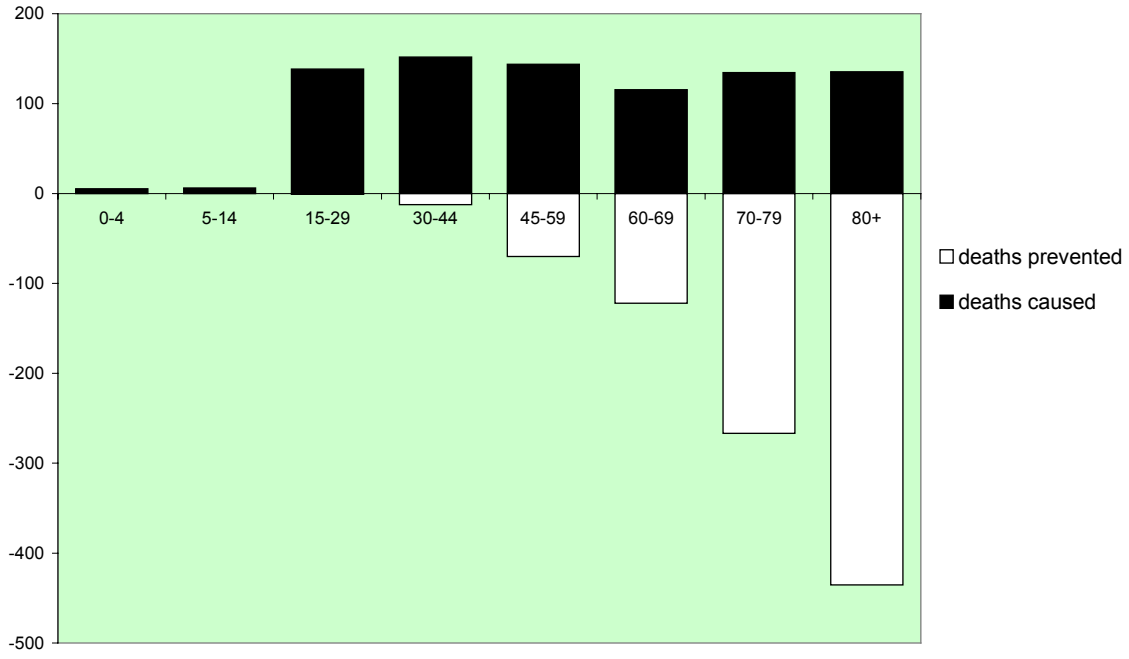
Figures 2 and 3 show graphically the impact of pattern of drinking on the balance of health risks and benefits from alcohol consumption. Although average volumes of alcohol consumed were approximately the same in Mäori and non-Mäori, Figure 2 (Mäori) represents a pattern of drinking with more abstainers but higher individual consumption in those that drank; with more of the alcohol being consumed in heavy drinking sessions. This results in a very marked impact on young people. Small proportions of the population in older age groups means the benefits were small.

Figure 2: Deaths caused and prevented by alcohol consumption, 2000 Mäori, by age group



In the non-Mäori population a substantial number of lives continue to be lost at older ages, again reflecting the greater proportion of the non-Mäori population in older age groups than of the Mäori population.

Figure 3: Deaths caused and prevented by alcohol consumption, 2000 Non-Mäori, by age group



Further details of Mäori and non-Mäori mortality by sex, age group and cause of death are given in Table C1 and Table C2 in Appendix C.

3.4 Years of life lost due to alcohol

Estimating years of life lost (YLL) due to alcohol-attributable disease or injury incorporates the impact of deaths at different ages. Table 15 summarises the net effects of alcohol on YLL for the 2000 year. As with mortality, the burden is not evenly spread in the population, but higher in men than women, higher in Mäori than non-Mäori. The standardised rate of YLL attributable to alcohol is 4-5 times higher in men than in women, in both Mäori and non-Mäori. Amongst women, Mäori have twice the rate of non-Mäori and amongst men Mäori have two and a half times the rate of non-Mäori.

Table 15: Net years of life lost (YLL) attributable to alcohol by ethnicity and sex, 2000

	Ethnicity	Years of life lost (count)	Years of life lost (rate) *
Males	Mäori	3143	1100
	Non-Mäori	6533	442
	Total	9676	548
Females	Mäori	769	240
	Non-Mäori	1468	112
	Total	2237	136
Total	Mäori	3912	656
	Non-Mäori	8001	276
	Total	11913	339

* rate per 100,000 age-standardised to WHO world population

Table 16 shows the proportions of alcohol-attributable YLLs by condition. Injury is the leading cause of alcohol-related YLLs (72%). As many injury deaths occur in younger age groups, injury is responsible for an even larger proportion of alcohol-attributable YLLs than was estimated for deaths (51%).

Table 16: Proportions of alcohol-attributable years of life lost by condition

	Men	Women	% of all alcohol attributable YLLs (n=17 207)
Cancers	1189	1186	13.8%
Other chronic diseases	1509	827	13.6%
Injuries	10234	2200	72.3%

Years of life gained by alcohol consumption are summarised in Table 17 by condition. Over 80% of the life years gained were from reduction in IHD. These benefits are mostly in older age groups.

Table 17: Years of life gained by alcohol consumption, by condition

	Men	Women	% of all years of life gained due to alcohol
Ischaemic heart disease	3033	1298	81.8%
Stroke	142	538	12.8%
Diabetes	123	129	4.8%
Cholelithiasis	11	17	<1%

The balance of gains and losses due to alcohol in different age groups as measured by YLLs is shown in Figures 4, 5 and 6, for Mäori and non-Mäori.

Figure 4: YLL caused and prevented by alcohol consumption, by age group, 2000: Mäori

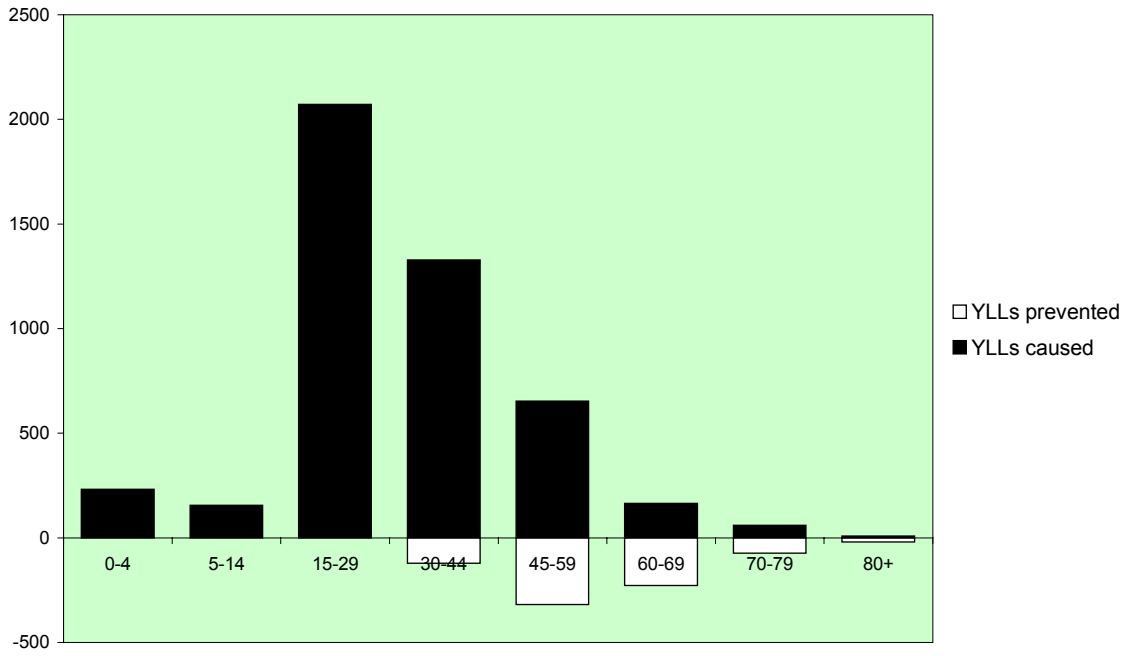


Figure 5: YLL caused and prevented by alcohol consumption, by age group, 2000: Non-Māori

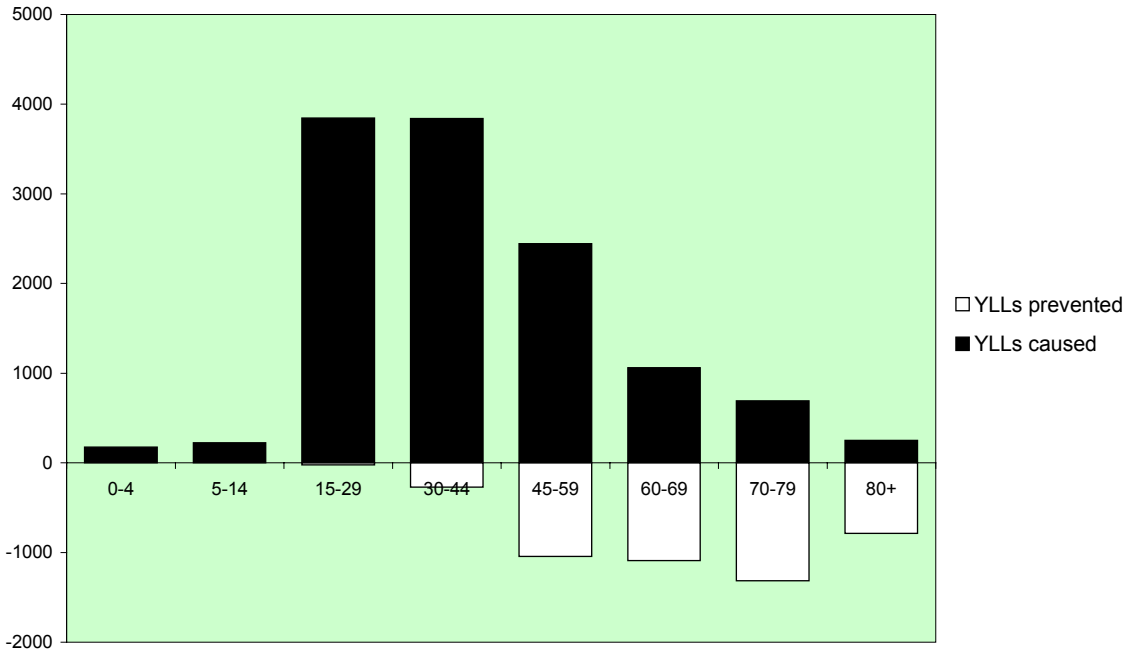
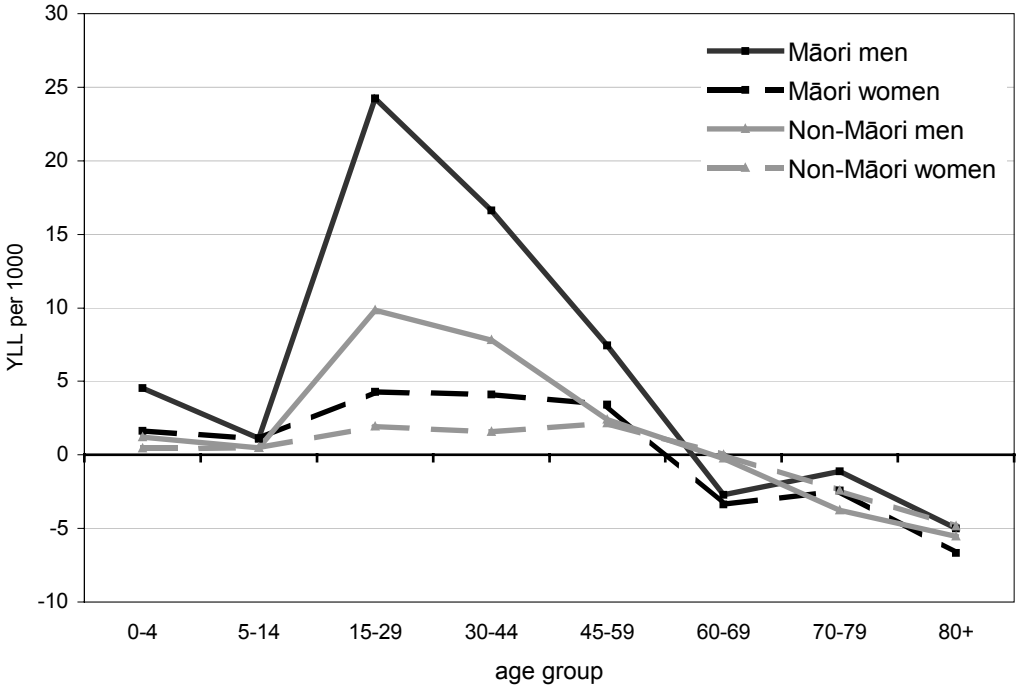


Figure 6: Age-specific rates of net years of life lost due to alcohol, by ethnicity and gender, 2000



3.5 Alcohol and ischaemic heart disease in Mäori : A sensitivity analysis

The effect of alcohol consumption on ischaemic heart disease in Mäori in New Zealand is less well understood than in populations where it has been directly studied. The analyses in this report have assumed Mäori accrue the same benefit in the prevention of ischaemic heart disease from drinking the same average volumes of alcohol as non-Mäori, even though the pattern of drinking is different. As discussed in Section 2.3.3, this is the best-case scenario.

This section demonstrates the effect of changing this assumption about IHD benefit on the estimates of alcohol-related mortality and years of life lost. The measures of burden have been recalculated assuming that there is no effect on IHD rates in Mäori from drinking alcohol, because of a less beneficial pattern of drinking. This is consistent with the approach of the WHO comparative risk assessment group.

Table 18 shows the results for alcohol-attributable mortality assuming no prevention of IHD by alcohol in Mäori. Compared with the previous analysis (figures in parentheses) there are 42 fewer deaths prevented by alcohol in Mäori men and 20 fewer in Mäori women. Total net deaths for the whole population attributable to alcohol rise from 56 to 118. Due to small numbers of the Mäori population in the age groups most affected by IHD, these death counts translate into large increases in alcohol-related death rates. The age-standardised rate for Mäori men increases from 38 to 72 per 100,000 and for Mäori women it increases from under 2 to almost 16 per 100,000.

Table 18: Mortality attributable to alcohol, by ethnicity and sex, 2000, assuming no IHD benefit in Mäori. Previous estimates shown in parentheses.

	Ethnicity	Deaths caused	% of all deaths	Deaths prevented	Net deaths (count)	Net deaths (rate)*
Males	Mäori	161	11.3	5 *(47)*	156 *(114)*	72.0 *(37.8)*
	Non-Mäori	557	4.5	476	81	9.7
	Total	718	5.2	481 *(523)*	237 *(195)*	15.6 *(13.6)*
Females	Mäori	45	3.9	6 *(26)*	39 *(19)*	15.7 *(1.85)*
	Non-Mäori	273	2.3	431	-158	-0.8
	Total	319	2.5	437 *(457)*	-119 *(-139)*	0.6 *(-0.1)*
Total	Mäori	206	8.0	11 *(73)*	195 *(133)*	42.1 *(19.0)*
	Non-Mäori	831	3.4	907	-77	4.5
	Total	1037	3.9	918 *(981)*	118 *(56)*	8.1 *(6.7)*

* rate per 100,000 age-standardised to WHO world population

The decrease in the estimated deaths prevented by alcohol consumption translates into 450 extra years of life lost in Mäori men, and 200 in Mäori women compared with previous estimates (Table 19).

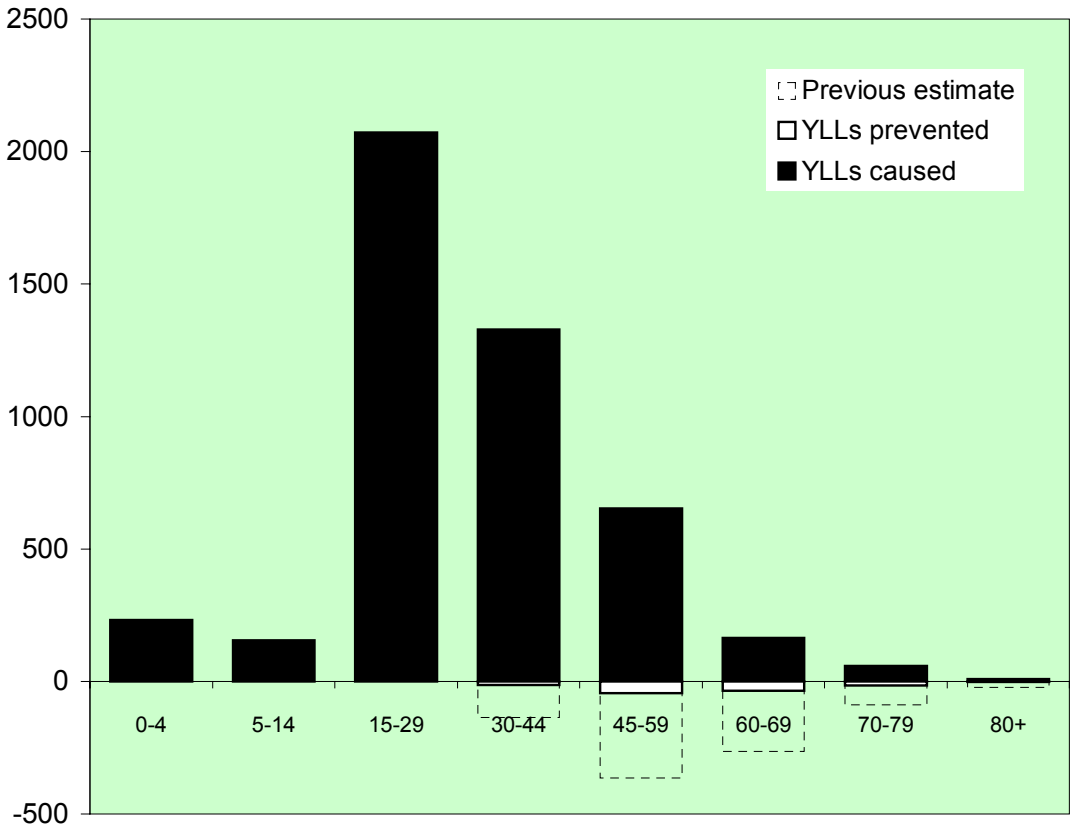
Table 19: Net years of life lost (YLL) attributable to alcohol by ethnicity and sex, 2000, assuming no IHD benefit in Mäori. Previous estimates shown in parentheses.

	Ethnicity	Years of life lost (count)		Years of life lost (rate) *	
Males	Mäori	3593	*(3143)*	1377	*(1100)*
	Non-Mäori	6533		442	
	Total	10126	*(9676)*	599	*(548)*
Females	Mäori	969	*(769)*	354	*(240)*
	Non-Mäori	1468		112	
	Total	2437	*(2237)*	140	*(136)*
Total	Mäori	4562	*(3912)*	848	*(656)*
	Non-Mäori	8001		276	
	Total	12563	*(11913)*	354	*(339)*

* rate per 100,000 age-standardised to WHO world population

Figure 7 shows graphically the balance of gains and losses due to alcohol consumption in terms of years of life lost or gained, by age group in Mäori, and the comparison with the previous estimates.

Figure 7:YLL caused and prevented by alcohol consumption, by age group, 2000, assuming no IHD benefit in Mäori: Mäori population



Under the assumption of no beneficial effect of alcohol on IHD for Mäori, there are virtually no health benefits for Mäori from drinking alcohol at any age. Overall death counts, rates and YLLs for the total NZ population (Tables 18 and 19) tend to obscure this difference between Mäori and non-Mäori.

4. DISABILITY ADJUSTED LIFE YEARS (DALYS) LOST DUE TO ALCOHOL

4.1 Overview

As described in the methods section above, DALYs lost due to alcohol consumption in New Zealand were calculated for the year 2002, using a variation of the approach used for calculation of mortality.

We used estimates from the WHO Global Burden of Disease project of the number of DALYs lost or gained in New Zealand during 2002 for each GBD disease category. To these we applied the same attributable fractions used in the mortality analysis to estimate the number and rate of DALYs lost due to alcohol consumption.

There were two limitations of this approach additional to those in the mortality analysis. The first was that we were unable to calculate DALY burdens for Mäori and non-Mäori separately and the whole population was included as pattern 2 drinkers; the second was that a few of the conditions in the mortality analysis did not correspond to GBD categories and were omitted. In particular, cardiac arrhythmias and oesophageal varices were not included, and fetal alcohol syndrome (FAS), which did not appear in the mortality analysis as there were no deaths attributed to FAS in 2000, could not be included due to lack of DALY data. However, the DALY analysis did allow the burden of morbidity due to alcohol to be described in addition to the burden of mortality and years of life lost.

Table 20 below shows the estimated DALYs lost due to alcohol in New Zealand in 2002. The burden in men was three times that in women, accounting for 76% of all alcohol-attributable DALYs lost.

Table 20: Alcohol attributable DALYs; total NZ population 2002

	DALYs lost	% of all DALYs lost	DALYs gained	Net DALYs lost	DALY rate*
Males	23,540	10.4%	3,910	19,630	1,075
Females	10,003	4.4%	3,662	6,341	386
Total	33,543	7.4%	7,572	25,971	726

* rate per 100,000 age-standardised to WHO world population

4.2 Alcohol attributable DALYs lost by cause

Table 21 below shows the alcohol-attributable DALY burden by cause of DALYs lost. The very large contribution of alcohol use disorders to the overall burden is clearly seen, with approximately 49% of alcohol-related DALYs. The importance of alcohol use disorders was not clearly seen in the preceding analyses, as deaths in those affected are usually coded according to the more proximal cause e.g. liver cirrhosis, and much disability from

alcohol use disorders does not result in death. Table 22 shows the estimates of DALYs gained that can be attributed to alcohol consumption, by cause.

Table 21: Alcohol attributable DALYs lost, by major disease and injury categories, 2002

	Men	Women	Total	Percentage of all alcohol-attributable DALYs lost
Mouth and oropharynx cancers	267	110	376	1.1%
Oesophagus cancer	345	127	473	1.4%
Liver cancer	284	92	375	1.1%
Breast cancer		1123	1123	3.3%
Alcohol use disorders	11688	4821	16509	49.2%
Unipolar depressive disorders	1762	500	2261	6.7%
Epilepsy	808	527	1335	4.0%
Hypertensive heart disease	187	146	333	1.0%
Cirrhosis of the liver	635	249	884	2.6%
Low birth weight	33	30	63	0.2%
Poisonings	68	32	101	0.3%
Road traffic injuries	3019	843	3862	11.5%
Falls	609	155	764	2.3%
Fires	115	44	159	0.5%
Drownings	360	39	399	1.2%
Other unintentional injuries	1597	734	2331	6.9%
Self-inflicted injuries	1452	331	1783	5.3%
Violence	304	100	404	1.2%
Other intentional injuries	7	0	7	<0.1%
Total	**23540**	**10003**	**33543**	**100%**

Table 22: Alcohol attributable DALYs gained, by major disease and injury categories, 2002

	Men	Women	Total	Percentage of all alcohol-attributable DALYs gained
Diabetes mellitus	394	320	714	9.4%
Ischaemic heart disease	3324	1547	4870	64.3%
Stroke (all subtypes)	192	1795	1989	26.3%
Total	**3910**	**3662**	**7572**	**100%**

4.3 Alcohol attributable DALYs lost by age group

Table 23 shows once again the huge health burden due to alcohol use by young people in New Zealand, and the estimated benefits for those over 60 years of age.

Table 23: Net alcohol attributable DALYs lost by age group, 2002

	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+
Men	210	468	9112	8047	2539	-30	-497	-219
Women	115	267	4181	2745	803	-555	-597	-618
Total	325	735	13293	10792	3342	-585	-1094	-837

Disability adjusted life years are the sum of life years lost and years lived with disability due to the health condition. Figures 8-11 show the components of the net DALYs calculated as lost due to alcohol in New Zealand in 2002. They illustrate how the number of deaths alone (Figure 8) is a poor indication of the burden due to alcohol, as injury and other disorders in young people are associated with high numbers of years of life lost (Figure 9), and high numbers of years lived with disability for every death (Figure 10), whereas for each death prevented at older ages (Figure 8) the number of YLLs and YLDs saved is small (Figures 9 and 10).

Figure 8: Net number of deaths caused or prevented by alcohol consumption, 2002

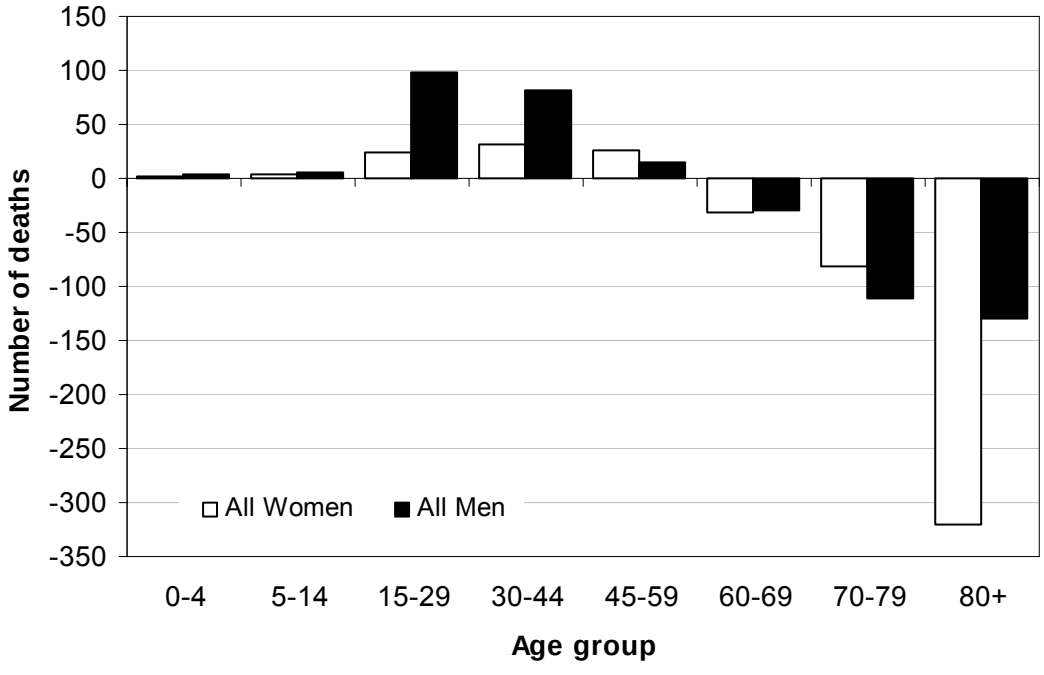


Figure 9: Net number of YLLs caused or prevented by alcohol consumption, 2002

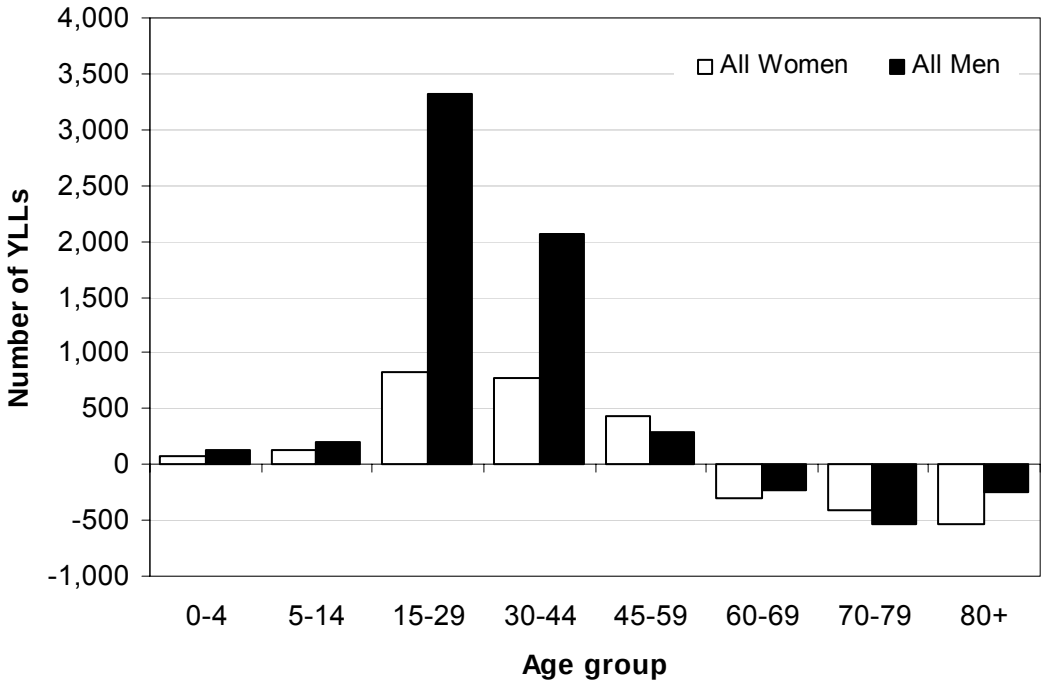


Figure 10: Net number of years lived with disability (YLDs) caused or prevented by alcohol consumption, 2002

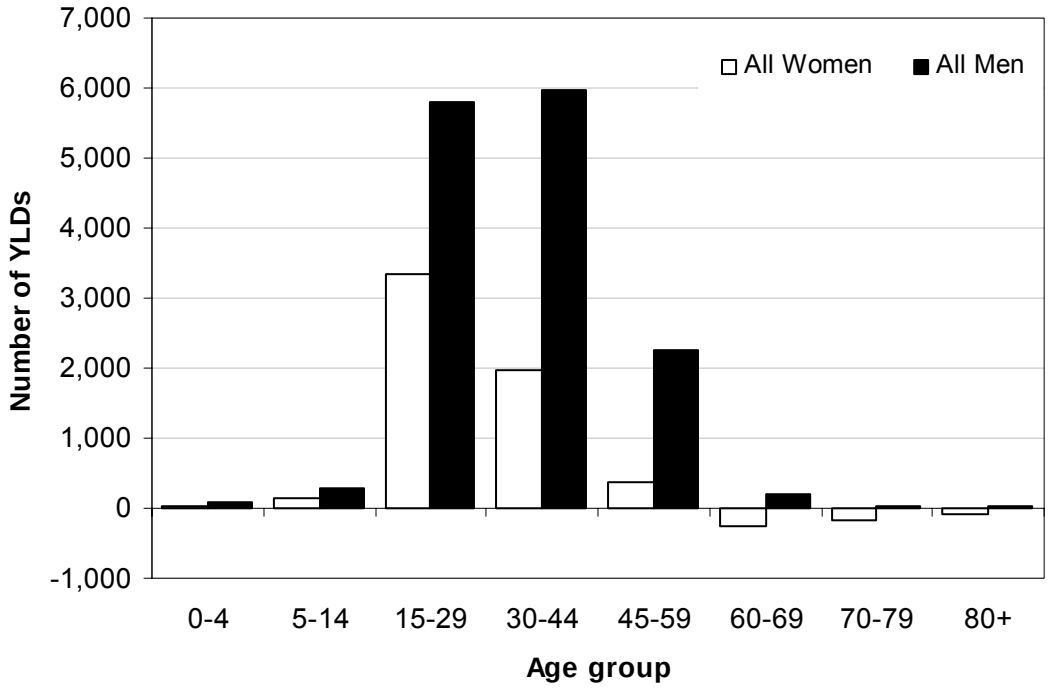
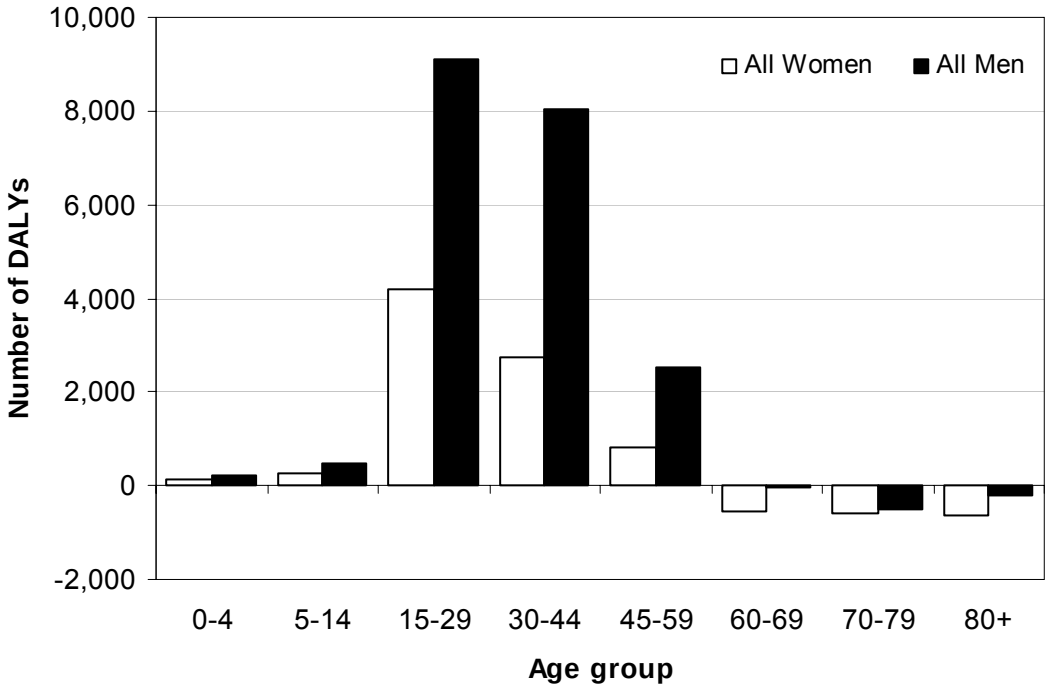


Figure 11: Net number of DALYs caused or prevented by alcohol consumption, 2002



5. DISCUSSION

5.1 Summary of findings

In this analysis we have estimated that about 1040 deaths in New Zealand in 2000 were attributable to alcohol consumption, and that 980 deaths were prevented by alcohol, resulting in a net loss of approximately 60 lives. Since many of the alcohol-attributable deaths occurred before middle age and the deaths prevented were almost entirely amongst older people, the balance of years of life lost and gained due to alcohol consumption was much less favorable. While 17,200 years of life were estimated to be lost, only 5,300 years of life were estimated to have been gained; a net loss of nearly 12,000 years of life due to alcohol.

The burden of mortality from alcohol use was not evenly spread in the population. The rate of alcohol-attributable YLLs in men was four to five times the rate in women, largely due to high alcohol-related mortality in men in the 15-44 year age group. These differences between men and women were seen in both the Mäori and non-Mäori populations. However, both Mäori men and Mäori women had much higher mortality and YLL rates than non-Mäori of the same age.

Injury was a major contributor to alcohol-related mortality, being responsible for 51% of deaths (532 deaths) and 72% of years of life lost (12,434 YLLs). Most alcohol-related deaths before middle age were due to injury. Cancers accounted for a further 24% of alcohol-related deaths and 14% of YLLs, with the remainder being due to other chronic diseases.

Most of the positive effects of alcohol consumption were seen in prevention of ischaemic heart disease deaths in older people who had a pattern of drinking characterised by frequent low volume intake (78% of all deaths prevented). Reduction in deaths due to stroke, diabetes and complications of cholelithiasis made up the remainder.

In a separate analysis incorporating estimates of morbidity as well as mortality, the loss of 33,500 disability-adjusted life years was attributed to alcohol for the New Zealand population in 2002. This comprised 7.4% of all DALYs lost in the population (10% of all DALYs in men and 4% in women). The largest single cause of DALYs lost was alcohol use disorders, responsible for 49%. The benefits of alcohol resulted in approximately 7,500 DALYs gained, and these were evenly split between men and women. Overall, there was a net loss of 26,000 DALYs attributable to alcohol, with 76% affecting men.

5.2 Limitations of the study

This report has attempted to bring together the most up-to-date methodology and estimates of alcohol-disease relationships with the most reliable available data on prevalence and pattern of drinking in New Zealand. However, there are a number of important limitations of this study that should be considered when interpreting or using the results reported here.

Uncertainty:

The relationship of alcohol to population health has several dimensions that make the estimation of burden more complex than for other major risk factors. We need to account for protective effects as well as harmful effects, the impact of pattern of drinking as well as volume, and acute as well as long-term consequences. A degree of uncertainty exists in all of the estimates used in calculating the health effects of alcohol. We have not attempted to quantify this uncertainty in our analyses but acknowledge that it is considerable, and arises from

- problems with exposure measurement,
- problems in determination of risk relationships,
- problems with outcome assessment, especially non-fatal outcomes.

Mäori:

The evidence base for estimating the burden of alcohol for Mäori is very small, with little specific information on risk relationships and non-fatal outcomes for Mäori. The extrapolation from available data sources to Mäori may be less appropriate than for non-Mäori.

Scope:

Social outcomes of alcohol consumption have not been included in the analysis unless they have resulted in a condition that is included in the ICD-10 coding system. It is acknowledged that there are a range of adverse social outcomes, and potentially some beneficial ones, that result from alcohol consumption and that these have an impact on health. Estimates from other countries indicate that the costs of social consequences of alcohol exceed the cost of direct health consequences.[69]

The impact of alcohol on mental health has been included in this analysis only in a limited way, through estimating the attributable fraction for unipolar depression. Previous studies of this type have not been able to include any mental health outcomes due to lack of reliable quantification of causal relationships and inadequate information on the prevalence of mental health conditions. Depression has been included on the strength of evidence of a causal relationship and its importance due to relatively high prevalence in the population, but the estimates used are subject to considerable uncertainty.

Disability:

Most of the analyses are based on deaths from alcohol-attributable conditions, and so exclude the contribution of disability to the health burden. The DALY analysis gives some insight into the contribution of disability, but is limited to a smaller range of conditions, and cannot provide separate Mäori and non-Mäori information as separate DALY information was not available. The DALY analysis assumes the same drinking pattern for the whole NZ population (Pattern 2), and the DALY weights have been generalized from non-New Zealand populations.

Exposure measurement:

The methodology does not take into account the lag time between exposure to alcohol and the development of each condition. Indeed for some conditions this may be variable or unknown. While current exposure distributions are appropriate for acute conditions, such as injuries, they may not be reasonable estimates of exposure over the long period preceding death from cancer. Current exposure is used here as a proxy for exposure at the time appropriate to causation of each condition.

Pattern of drinking:

The categorisation of pattern of drinking into one of only four categories (two of which are extreme) is very simplistic. We have applied this categorisation to Mäori and non-Mäori populations separately in our analysis based on average behaviour, but pattern of drinking is likely to vary by other characteristics, such as age group, geographical location and socio-economic status within these populations. This is particularly so for the most detrimental pattern of drinking, irregular heavy drinking occasions. One of the consequences of the assignment of Pattern 3 to Mäori and Pattern 2 to non-Mäori is marked differences in the attributable fractions, and therefore attributable burden, of pattern-sensitive conditions (injury and ischaemic heart disease). Local data on car crash injury confirmed an increased AAF for injury in Mäori, but uncertainty that the differences in pattern of drinking between Mäori and non-Mäori would substantially reduce the benefits of alcohol consumption for ischaemic heart disease meant the same AAFs were applied to both groups.

Risk relationships:

Some of the exposure-disease relationships used in the analysis are still being modified by continuing research. In particular, the degree of benefit in ischaemic heart disease, stroke and diabetes is still the subject of disagreement, and some of this may be the result of pattern as well as volume of drinking being influential. Methods to account for the effect of pattern of drinking are still being developed, and have only been considered for

IHD and injury in this report. There is little New Zealand research quantifying any of these relationships, particularly for Mäori, and most available estimates come from low risk populations. In conditions where pattern of drinking is important, locally derived risk estimates would provide a more reliable basis for estimation of burden.

Elderly people:

Uncertainty in the estimates of burden may be even greater in elderly people who are not usually included in major epidemiological studies, and may have an increased sensitivity to some of alcohol's effects. Accurate data on the volume and pattern of alcohol consumption in the elderly are scarce, and often extrapolated from younger drinkers or based on small numbers. These problems are even more pronounced amongst Mäori, where the elderly population is small and the appropriateness of extrapolating from other sources is unknown. Finally, the cause of death in older people is more likely to be misclassified than in younger people.

5.3 Comparison with previous analyses

Three previous New Zealand reports have quantified alcohol-related mortality in New Zealand, and one has previously estimated DALYs lost due to alcohol.

The first estimate of the proportion of NZ deaths caused or prevented by alcohol drinking was published by Scragg in 1995.[6] This analysis used the methods of Holman et al.[11] and found that alcohol consumption in 1987 resulted in a net reduction of 416 (1.5%) deaths but a net loss of 9525 person-years of life. The major differences in approach between this and the current analysis were that exposure to alcohol was dichotomised (drinkers/non-drinkers) by Scragg, broader age groups were used and years of life lost were calculated from NZ life tables rather than standard international ones. The use of 1987 NZ life expectancy (71.1 for men and 77.1 for women at birth), rather than life expectancy at birth of 80 years for men and 82.5 for women used by the GBD and the current study, will have resulted in substantially fewer years of life lost for each death. In addition, many risk relationships have been better characterised in the interim, and Mäori and non-Mäori have been considered separately, with the adjustment for pattern of drinking in injury and IHD.

In 1999 the Ministry of Health published estimates of alcohol attributable mortality and YLLs that were also lower than our current estimates in *Our Health, Our Future,* a population health monitoring report.[66] They reported a net reduction in deaths as a result of alcohol consumption (-59 deaths overall) and 3367 net attributable YLLs in 1996. The methods used were similar to those of this study, but exposure data came from a different source (New Zealand Health Survey). The exposure data used in the current study are both more comprehensive and more recent. Also, in the current study some risk estimates have been updated, and adjustment for pattern of drinking in injury and IHD has resulted in a reduction in estimated benefits and increase in estimated harmful consequences.

The Burden of Disease and Injury in New Zealand was published by the Ministry of Health in 2001,[70] providing DALY estimates for major groups of conditions for the same year (1996), as the mortality study described above. Population attributable risk estimates for alcohol from the mortality study were applied to DALY counts for alcohol-related conditions, in order to rank alcohol against other major risk factors. For men, alcohol was considered to be responsible for 5% loss and 3% gain of DALYs, with a net loss of 2%. The net DALYs lost for women were less than 1%.

A further publication from the Ministry of Health Public Health Intelligence group in 2004[71] reported on the causal structure of mortality in New Zealand in 1997. A number of major risk factors were compared with respect to impact on mortality. Alcohol was estimated to be responsible for 800 deaths in this year, approximately 3% of all deaths, which is 30% lower than the estimates from our analysis for the year 2000.

5.4 Public health implications

Alcohol is responsible for a considerable burden of ill-health. While elimination of alcohol consumption is not realistic, and may not be desirable, an evidence-based approach to promoting safer drinking has the potential to reduce both acute and chronic consequences of alcohol.

The focus on exposure to proximal risk factors for health conditions (such as alcohol consumption) produces health information devoid of context. The public health response to such information must be informed by an understanding of the social structural and cultural determinants of these risk factors.

Five major messages have emerged from this analysis:

- There are no health benefits of drinking alcohol before middle age

Most of the benefits of alcohol consumption accrue in the elderly, and so benefits that appear large in terms of mortality are less impressive when expressed as years of life lost or DALYs. The benefits are associated with regular low volume intake, and risks associated with heavy drinking persist into old age.

- The pattern of drinking is very important in determining the health effects of alcohol consumption

It is increasing clear that for drinkers consuming the same average volume of alcohol, pattern of drinking has a major influence on both benefits and harms. Moving towards patterns of drinking that are safer in terms of physical health outcomes is also likely to reduce the unmeasured social consequences of alcohol consumption.

- Injury is responsible for half of all alcohol-attributable deaths and almost three-quarters of the years of life lost due to alcohol

Changes in the pattern as well as the context of much alcohol consumption will be needed to substantially reduce the burden of injury due to alcohol. Even amongst low volume regular drinkers, there are increased risks of injury associated with alcohol.

- There is a huge burden of disability due to alcohol use disorders that is not reflected in mortality figures

Our perception of the burden of alcohol is highly influenced by which measures are used to quantify it. While acknowledging the limitations of the DALY metric, this

analysis has highlighted the very substantial morbidity from alcohol abuse and dependence in the community.

- The health burden of alcohol falls inequitably on Mäori.

The combination of more harmful drinking patterns and a smaller proportion of the population in the older age groups where benefits accrue, means that the Mäori population is more adversely affected by alcohol than non-Mäori population. Almost all health benefits from alcohol consumption are in non-Mäori, and using measures of the health effects of alcohol for the combined NZ population obscures these disparities.

5.5 Recommendations for alcohol policy

Alcohol use has net negative effects on burden of disease, and the size of these effects is large enough to call for public health interventions. Moreover, the benefits of drinking on cardiovascular events such as CHD and stroke are based on specific patterns of drinking, which are associated with small risk for other disease endpoints, so that there theoretically should be a way to only reduce the burden of alcohol use while keeping the benefits. Empirical research has shown, however, that this difficult to achieve as drinking behaviour in a society is socially and culturally determined, and behaviour of a particular group of people cannot be changed without effects on other groups.[72]

The following suggestions are thus made to minimise the overall alcohol-related burden of disease, based on the specific epidemiological situation in New Zealand:

- Prevention of alcohol-related injury should be a major focus of preventive efforts, given that more than 70% of the overall burden occurs in this category. Road traffic injuries are one large category where reductions have been shown empirically to result from controls on BAC with, for example, random breath testing, or by lowering the limit of legal BAC.[73] There are a number of other evidenced-based specific measures for drink driving which should also be considered for implementation in New Zealand, such as zero tolerance for new drivers. However, one should not lose sight of the fact that that injuries other than traffic injuries make an even bigger contribution to the burden of alcohol-related injury. Specific prevention policies should be implemented here as well, e.g. in the workplace, coupled with adequate enforcement efforts. This will not be possible in all areas, e.g. in the prevention of household falls. In this area, information and education have been the predominant measures, but empirical research does not show much demonstrable and sustained effectiveness of such policies.[73] [74] One possible secondary prevention strategy would be brief interventions in emergency rooms, thus applying a measure of proven effectiveness for reducing alcohol-related problems and harm to a new setting.[75]

- Secondly, measures should be taken to reduce the disproportionate burden of alcohol for the Mäori. Based on the epidemiology, such measures would specifically address heavy drinking occasions. While there are a multitude of evidenced-based measures for reducing alcohol-related harm in general, not much research has been conducted on changing drinking patterns. Again, brief interventions have been empirically shown to change drinking patterns[73 75] and should be considered in programmes tailored towards problem drinkers in the Mäori population. Other measures would require specific knowledge of where the alcohol-related burden occurs, and planning access restrictions or harm reduction measures specifically in these places (e.g. bar programs).

- Finally, the overall drinking culture in New Zealand should be taken into account. Alcoholic beverage prices have been dropping over the past decades relative to income in most established market economies. Thus, taxation may be a suitable means in New Zealand to reduce alcohol related problems.[73] Advertising and sponsorship also influence drinking culture and bans of such behaviours have been shown to reduce alcohol consumption and alcohol-related harm.[76]

In summary, there are a number of policies that have been shown to be effective in reducing alcohol-related harm and, if properly implemented, could markedly reduce the size of the alcohol-related health burden in New Zealand.

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Disclaimer

Any findings, conclusions or opinions are those of the authors and are not to be attributed to ALAC.

APPENDIX A: "PATTERN OF DRINKING" VARIABLES AND THEIR RELATIVE WEIGHTS

HEAVY DRINKING OCCASIONS

(Maximum of 11 points for this component)

Daily drinking

Less than 20% daily drinking for males: 1 point
Less than 10% daily drinking for females: 1 point

Frequency of getting drunk

Most male drinkers usually get drunk when they are drinking: 2 points
Most males drinkers often get drunk: 1 point
Most female drinkers usually or often get drunk: 1 point

Usual quantity per drinking session

Males: more than 60% typically consume four or more drinks per session: 2 points
Males: between 40% and 60% consume four or more drinks per session: 1 point
Females: more than 50% consume four or more drinks per session: 2 points
Females: between 35% and 50% consume four or more drinks per session: 1 point

Fiesta binge drinking

Males: fiesta drinking commonly occurs: 1 point
Females: fiesta drinking commonly occurs: 1 point

Drinking with meals

(Maximum of 4 points for this component)
Males: rarely or never with meals: 2 points
Males: sometimes with meals: 1 point
Females: rarely or never with meals: 2 points
Females: sometimes with meals: 1 point

Drinking in public places

(Maximum of 2 points for this component)
Males: common and everyday: 1 point
Females: common and everyday: 1 point

Scoring

(possible range: 0–17 points)
Scoring by summation of individual questions: range 0–17
10–17 points: assign a pattern value of 4
7–9 points: assign a pattern value of 3
4–6 points: assign a pattern value of 2
0–3 points: assign a pattern value of 1

APPENDIX B: ALCOHOL ATTRIBUTABLE FRACTIONS FOR HOSPITALISATION OR DEATH FROM CAR CRASH

Data from Auckland Car Crash Injury Study

	Drivers with BAC 3-50 mg/100ml				Drivers with BAC >50 mg/100ml					
	prevalence (%)	RR	AF	PAF(%)	prevalence (%)	RR	AF	PAF(%)	**AAF**	**Mäori AAF**
Total cases	7.2	3.8	0.74	5.33	23.2	19.3	0.95	22.04	**27.4**	
Mäori	9.1	3.8	0.74	6.73	35.5	19.3	0.95	33.73	**40.5**	
NonMäori	6.7	3.8	0.74	4.96	19.8	19.3	0.95	18.81	**23.8**	
Men	7.2	4.3	0.76	5.47	28.7	27.9	0.96	27.55	**33.0**	
Women	7.1	5.1	0.80	5.68	12.7	18.1	0.94	11.94	**17.6**	
15-29 men	9.6	6.0	0.83	7.97	34.0	19.3	0.95	32.30	**40.3**	**60.4**
30-44 men	6.4	3.8	0.74	4.74	36.2	19.3	0.95	34.39	**39.1**	**58.7**
45-59 men	1.9	3.8	0.74	1.41	13.0	19.3	0.95	12.35	**13.8**	**20.6**
60-69 men	5.4	3.8	0.74	4.00	5.4	19.3	0.95	5.13	**9.1**	**13.7**
70+ men	5.4	3.8	0.74	4.00	5.4	19.3	0.95	5.13	**9.1**	**13.7**
15-29 women	7.9	3.8	0.74	5.85	19.7	19.3	0.95	18.72	**24.6**	**36.8**
30-44 women	7.4	3.8	0.74	5.48	18.5	19.3	0.95	17.58	**23.1**	**34.6**
45-59 women	9.7	3.8	0.74	7.18	0.0	19.3	0.95	0.00	**7.2**	**10.8**
60-69 women	2.8	3.8	0.74	2.07	0.0	19.3	0.95	0.00	**2.1**	**3.1**
70+ women	2.8	3.8	0.74	2.07	0.0	19.3	0.95	0.00	**2.1**	**3.1**
fatal to driver	5.8	3.8	0.74	4.29	26.5	19.3	0.95	25.18	**29.5**	**44.2**
nonfatal driver injury	7.5	3.8	0.74	5.55	24.9	19.3	0.95	23.66	**29.2**	**43.8**
secondary injury	6.6	3.8	0.74	4.88	17.9	19.3	0.95	17.01	**21.9**	**32.8**

2 oldest age-sex groups have been combined due to small numbers

APPENDIX C: ALCOHOL-ATTRIBUTABLE MORTALITY: BY ETHNICITY, SEX, AGE-GROUP, AND CAUSE, 2000

TABLE C1 : MÄORI

	Mäori men								Mäori women								Totals		
	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	Men	Women	All
Mouth and oropharynx cancers	0	0	0	1	0	2	1	0	0	0	0	1	0	0	0	0	4	1	5
Laryngeal cancer	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	1
Oesophagus cancer	0	0	0	0	3	2	2	0	0	0	0	0	0	1	0	0	8	1	9
Liver cancer	0	0	0	1	4	4	1	0	0	0	0	0	1	0	0	0	10	1	11
Breast cancer	0	0	0	0	0	0	0	0	0	0	0	1	2	1	0	0	0	6	6
Diabetes mellitus	0	0	0	0	-1	-2	-1	0	0	0	0	0	-1	-1	-1	0	-4	-3	-8
Alcohol use disorders	0	0	0	1	1	0	1	0	0	0	0	0	0	0	0	0	3	0	3
Unipolar depressive disorders	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Epilepsy	0	0	0	1	1	0	0	0	0	0	1	0	0	0	0	0	1	1	2
Hypertensive heart disease	0	0	0	0	1	1	1	1	0	0	0	0	1	0	0	0	5	3	8
Ischaemic heart disease	0	0	0	-4	-12	-15	-8	-3	0	0	0	-1	-5	-6	-4	-4	-42	-20	-62
Cardiac arrhythmias	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	2	1	3
Ischaemic stroke	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-1	-1
Haemorrhagic stroke	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-1	-1
Oesophageal varices	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Alcoholic liver cirrhosis	0	0	0	0	2	2	0	0	0	0	0	1	1	1	1	0	4	4	8
Cholelithiasis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-1	-1
Pancreatitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Low birth weight	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Fetal alcohol syndrome	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Alcohol poisoning	0	0	3	0	0	0	0	0	0	0	0	0	1	0	0	0	3	1	4
Non-alcohol poisoning	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	2
Road traffic injuries	2	1	19	11	2	1	0	0	1	2	4	3	1	0	0	0	37	11	48
Falls	0	0	2	2	1	0	0	0	0	0	0	0	0	0	0	0	5	1	6
Drownings	0	0	0	2	1	0	0	0	0	0	0	1	0	0	0	0	4	1	4
Other unintentional injuries	2	1	28	15	6	0	0	1	0	0	4	2	1	0	0	0	53	8	62
Self-inflicted injuries	0	0	7	4	1	0	0	0	0	0	1	0	0	0	0	0	12	1	13
Violence	1	0	3	2	0	0	0	0	0	0	1	1	0	0	0	0	6	3	9
Other intentional injuries	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total deaths caused	5	2	63	41	25	14	8	3	2	2	11	11	11	4	3	1	161	45	206
Total deaths prevented	0	0	0	-4	-14	-17	-9	-4	0	0	0	-2	-7	-8	-5	-5	-47	-26	-73
Net deaths : all causes	5	2	63	37	11	-3	-1	-1	2	2	11	9	4	-4	-1	-4	114	19	133

TABLE C2 : NON-MÄORI

	Non-Mäori men								Non-Mäori women								Totals		
	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	Men	Women	All
Mouth and oropharynx cancers	0	0	0	1	8	10	9	3	0	0	0	0	1	1	3	2	30	8	37
Laryngeal cancer	0	0	0	0	3	4	4	1	0	0	0	0	1	0	0	0	12	1	13
Oesophagus cancer	0	0	0	2	4	12	17	9	0	0	0	0	1	2	6	10	44	19	64
Liver cancer	0	0	1	1	5	5	9	3	0	0	0	0	2	4	3	3	25	12	37
Breast cancer	0	0	0	0	0	0	0	0	0	0	0	9	22	11	12	10	0	64	64
Diabetes mellitus	0	0	0	0	-2	-2	-3	-2	0	0	0	0	-1	-3	-5	-7	-9	-16	-25
Alcohol use disorders	0	0	0	4	3	4	3	0	0	0	0	0	1	1	1	1	14	4	18
Unipolar depressive disorders	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Epilepsy	0	0	2	9	3	1	2	0	0	0	1	1	1	1	1	0	17	5	22
Hypertensive heart disease	0	0	0	1	1	3	8	6	0	0	0	0	2	2	6	15	18	25	44
Ischaemic heart disease	0	0	0	-8	-49	-80	-150	-137	0	0	0	-2	-12	-24	-68	-174	-424	-279	-703
Cardiac arrhythmias	0	0	0	0	0	1	4	15	0	0	0	0	1	1	5	26	21	31	52
Ischaemic stroke	0	0	0	0	0	-2	-8	-13	0	0	0	-1	-3	-3	-15	-52	-22	-74	-96
Haemorrhagic stroke	0	0	0	0	0	-2	-6	-10	0	0	0	-1	-4	-5	-11	-39	-18	-60	-78
Oesophageal varices	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	0	1	1	2
Alcoholic liver cirrhosis	0	0	0	4	13	20	17	3	0	0	0	1	13	7	6	2	57	29	86
Cholelithiasis	0	0	0	0	0	0	-1	-1	0	0	0	0	0	0	-1	-1	-3	-2	-5
Pancreatitis	0	0	0	0	1	1	1	0	0	0	0	0	0	0	1	2	3	3	6
Low birth weight	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Fetal alcohol syndrome	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Alcohol poisoning	0	0	1	1	0	2	0	0	0	0	0	1	0	0	0	0	4	1	5
Non-alcohol poisoning	0	0	3	1	0	0	0	0	0	0	2	1	0	0	0	0	5	3	8
Road traffic injuries	1	2	34	29	6	2	2	1	0	2	8	5	1	0	0	0	75	17	92
Falls	0	0	2	3	1	1	4	8	0	0	0	0	0	0	1	5	18	7	25
Drownings	0	0	3	5	1	1	0	0	0	0	0	0	2	0	0	0	11	2	13
Other unintentional injuries	2	1	48	45	27	12	9	5	0	0	9	7	6	3	0	3	149	29	178
Self-inflicted injuries	0	0	18	16	8	3	1	1	0	0	2	3	1	1	0	0	47	7	54
Violence	0	0	2	2	1	0	0	0	0	0	1	1	1	0	0	1	5	4	10
Other intentional injuries	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total deaths caused	4	3	115	124	86	81	89	56	1	3	23	28	58	34	46	80	557	273	831
Total deaths prevented	0	0	-1	-8	-51	-86	-167	-163	0	0	0	-4	-19	-36	-100	-272	-476	-431	-907
Net deaths : all causes	**4**	**3**	**115**	**115**	**35**	**-5**	**-79**	**-107**	**1**	**3**	**23**	**24**	**38**	**-2**	**-54**	**-193**	**81**	**-158**	**-77**