Estimating the burden of alcohol abuse in South Africa in 2000

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Acronyms and Abbreviations

AAF	Alcohol attributable fraction
AIDS	Acquired Immune Deficiency Syndrome
AFR-E	Afr-E region of Sub-Saharan Africa
ASSA2002	Actuarial Society of South Africa 2002, model
BAC	Blood alcohol level
BOD	Burden of Disease
CAGE	Cutting down, Annoyance by criticism, Guilty feeling and Eye-openers
CFR	Case fatality rate
CRA	Comparative Risk Assessment
DALYs	Disability adjusted life years
DHS	Demographic and Health Survey
DISMOD	Disease model
FAS	Fetal alcohol syndrome
ICD	International Classification of Disease
IHD	Ischaemic heart disease
LBW	Low birth weight
MRC	Medical Research Council
NIMSS	National Injury Mortality Surveillance Study
PAF	Population attributable fraction
RTI	Road traffic injuries
RR	Relative risk
SA BOD list	South African Burden of Disease list
SADHS	South African Demographic and Health Survey
SA NBD	South African National Burden of Disease Study
WHO	World Health Organization
YLDs	Years lived with disability
YLLs	Years of life lost due to premature mortality

Executive Summary

This study set out to estimate the burden of disease attributable to alcohol by sex and specific age groups in South Africa in 2000 as part of the Comparative Risk Assessment for South Africa. The analysis follows the World Health Organisation Comparative Risk Assessment methodology. Population-attributable fractions (PAFs) calculated from modelled prevalence estimates and relative risks based on the global review were applied to the revised South African National Burden of Disease study for 2000. The alcohol-attributable fractions for injuries were directly determined from blood alcohol concentration (BAC >0.05 g/100 ml) at the time of injury. Monte Carlo simulation modelling techniques were used to quantify uncertainty in the estimates. Deaths and disability-adjusted life years (DALYs) from IHD, stroke, hypertensive heart disease, diabetes, certain cancers, liver cirrhosis, epilepsy, alcohol use disorder, depression and intentional and unintentional injuries as well as burden from foetal alcohol syndrome (FAS) and low birthweight.

Alcohol harm accounted for an estimated 7.1% (95% uncertainty interval: 6.6%-7.5%) of all deaths and 7.0% (95% uncertainty interval: 6.6%-7.4%) of total DALYS in 2000. Injuries and cardiovascular incidents ranked first and second in terms of attributable deaths. Top rankings for overall alcohol-attributable burden were interpersonal violence (39.0%), neuropsychiatric conditions (18.4%) and road traffic injuries (14.3%). Interpersonal violence accounted for 42.8% of the injury DALYs attributed to alcohol in males and 25.9% in females. In terms of alcohol-attributable disability, alcohol use disorders ranked first (44.6%), interpersonal violence second (23.2%), and FAS third (18.1%).

Despite the fact that many South Africans do not drink, alcohol abuse results in a considerable burden of disease in SA. Particular attention needs to be given to preventing and reducing the burden of alcohol-related homicide and violence, alcohol-related road traffic accidents, alcohol use disorders, and FAS. Multilevel interventions are required to target high-risk drinkers, in addition to creating awareness in the general population of the problems associated with alcohol abuse. Focus should now shift from legislation and regulation to making resources available for implementing intervention strategies. These should include a coherent liquor outlet policy, increasing random breath analysis of drivers, brief interventions, and other forms of treatment for high-risk and hazardous drinkers, as well as training and accreditation of treatment and prevention programmes.

Changing the pattern of drinking in South Africa is essential if the alcohol-related burden is to be reduced. This will require a co-ordinated national intervention strategy. An adequate information base should underpin the implementation of a national alcohol strategy and enable monitoring and evaluation.

1. Background

The health and social outcomes that result from the use of alcohol are complex. This is particularly so in South Africa with its colonial and apartheid history. Traditionally, the indigenous peoples of South Africa - the Bantu and the Khoikhoi - consumed intoxicating drinks derived from the fermentation of plants, fruits and grains, with alcohol consumption playing an important role in social and ritual gatherings. Wine was used by whites in South Africa from the time of the first governor of the Cape, Jan van Riebeeck in 1652. Sailors, visiting the Cape, used wine and malt beer, both western brews, to prevent scurvy on the long sea voyages. Parry and Bennetts (1998) note that malt was first brewed in 1657 and wine was first produced in 1659 in the Cape. The growth of the wine and beer industry meant that the production of alcohol was not dependent on maize production or the availability of fruit. From the seventeenth century, European settlers at the Cape also used alcohol in part payment for labour and it became more easily accessible among indigenous populations, so leading to misuse. Over time Western brews were introduced into the interior of the country, by Afrikaners moving away from British rule at Cape, as well as by prospectors, entrepreneurs and soldiers (*ibid*.).

In terms of the development of alcohol policies, one draconian law, implemented towards the end of the nineteenth century by the British colonial power, that had annexed the Cape from the Dutch, prohibited drinking in non-white population groups. The rationale for this law was to prevent 'social decay and disorder' in their black subjects (Parry and Bennetts, 1998). In 1909 the Native Beer Act was passed, this stipulated that African beer could only be consumed legally within municipal beer halls in Durban. This Act was implemented throughout South African towns and cities (*ibid*).

Whereas some scholars viewed the development of alcohol policy in South Africa as an *ad hoc* response to alcohol-related social problems, Charles van Onselen (1982) documented alcohol regulation as serving the interests of the mining industry, that is, white capital. He highlighted the "complex relationship between alcohol and the emergence of a modern urban-industrial system, based on mining and the exploitation of migrant labour." In the 1890's, unskilled workers were initially encouraged to consume alcohol and the mine owners profited from the sale of alcohol to their workers. However, after 1896, when the deep mines on the Witwatersrand went into production, the mine owners needed a sober labour force and the sale of liquor to blacks was restricted (Ambler and Crush, 1992; Mager, 2004).

Viticulture in South Africa has also had an impact on drinking behaviours, particularly in the Western and Northern Cape. Farm workers were effectively controlled through the regular supply of crude wine as part of their wages. Similarities in the circumstances of Coloured farm workers at the Cape and African mine workers on the Witwatersrand has contributed to the culture of heavy drinking that currently exists in South Africa (Mager, 2004).

Under the apartheid government which ended in 1994 and preceding governments, legislation controlled where Africans and coloureds could buy and consume liquor, how much they could buy, who they could drink with, who produced and procured it, as well as the quality of the alcohol available to them. Mager (2004) highlighted that a prime example of the iniquitous use of alcohol legislation to affect social control, including control of the leisure of blacks, was the lifting of the prohibition of selling European liquor to Africans in the same year that the South African 1962 Sorghum and Beer Act stipulated state control of the brewing of grain beer for sale in African townships. Brady and Rendall-Mkosi (2005) reported that the profits from these beer monopolies in turn funded racial and residential segregation during apartheid. In addition, although Africans were used to a nutritious, home-made sorghum drink, under apartheid the mass produced grain beer was depleted of its vitamin and nutritional contents as sorghum was replaced by cheaper maize products (Mager, 2004).

Beer halls and 'shebeens' nurtured vibrant subcultures which have persisted in impoverished environments (Ambler and Crush, 1992). For some the alcohol trade, (the preparation and sale thereof), became a means of economic survival. The illegal liquor trade serves as a means whereby, mainly older women, support their extended families through small-scale shebeens (Brady and Rendall-Mkosi, 2004).

During the apartheid era, the brewing and drinking of alcohol in illegal 'shebeens', (liquor outlets), in the black townships became a form of resistance against oppressive laws and apartheid (Brady and Rendall-Mkosi, 2004). People could be sociable and discuss politics, at the same time, drunkenness blotted out stress and feelings of alienation (Mager, 2004). In the 1976 student uprising in South Africa, beer halls and bottle stores were destroyed as symbols of exploitation and oppression (*ibid*.). The students also perceived liquor as a cause of their parents' political inertia and attacked liquor purveyors and consumers. Today, ironically, the past struggle against apartheid is used to justify excessive use of alcohol – "they have the freedom to do as they please" (Brady and Rendall-Mkosi, 2004).

Currently, the formal part of the South African liquor industry comprises 23 0000 licensed outlets, with about 180 000 informal liquor outlets across the country, mostly shebeens. According to (2005(a)), it is expected that most provinces will liberalise the restrictions on the retail selling of alcohol in order to draw the unregulated outlets into the regulated market and in so doing implement a coherent liquor outlet policy.

Alcohol use and abuse, has clearly been of a mercurial nature in the socio-political and economic spheres in South Africa (Brady and Rendall-Mkosi, 2004). Similarly, alcohol as a risk factor for disease has both positive and negative aspects. Small amounts of alcohol, a depressant drug, can make one feel relaxed and provides a feeling of well-being. Moderate alcohol consumption does not harm most

people, whereas regular heavy drinking does cause health problems over time (Whelan and Gijsbers, 2000). Unregulated homebrews or concoctions can be particularly damaging to health as they may have poisonous additives, such as battery acid or methylated spirits.

Medical literature reviewed by Rehm *et al*. (2004) suggests that on the whole, the impact of alcohol consumption on chronic diseases and injuries is negative. An example of a harmful effect of alcohol consumption on chronic diseases is the increased risk of high blood pressure. According to Walker and colleagues (2005), the prime target for the toxic effects of alcohol is the liver and chronic alcohol abuse can result in alcoholic cirrhosis that predisposes persons to infections. However, the review by Rhem *et al.* (2004) highlighted that there are beneficial relationships between alcohol and IHD and cerebrovascular disease for certain combinations of average volume of consumption and patterns of drinking. For example, light to moderate drinking in a regular pattern for older persons can have a beneficial effect on IHD by reducing blood clot formation and reducing plaque deposits in arteries. There is also some evidence that alcohol consumption offers some protection against gallstones and evidence from cohort studies shows that moderate alcohol consumption, offers protection for type II diabetes, perhaps through the effects of alcohol on insulin sensitivity (Rimm *et al.,* 1995).

Rhem *et al.* (2004) found that there is increasing evidence that the volume of alcohol consumed as well as the patterns of drinking are relevant to health. In terms of detrimental patterns of drinking, binge drinking, (defined as occasional bouts of heavy drinking), is considered the worst scenario. The cardio-protective effect is determined by the volume and pattern of drinking and has the most relevance to countries with established market economies. The effects of alcohol misuse are exacerbated by poor nutritional status and may lead to infectious diseases in the short-term and degenerative diseases in the longer- term.

The toxic and beneficial effects describe the biochemical effects of alcohol on bodily functions and not the intermediate impacts of intoxication and dependence. Intoxication mediates mainly for acute outcomes such as intentional and unintentional injuries - both fatal and non-fatal. Even small amounts of alcohol slow thought processing, slow down reaction time and generally impair co-ordination and alertness, increasing the risk of all types of injures. Alcohol dependence is a disorder in itself but also impacts on both chronic and acute physiological and social consequences.

The South African population structure is skewed towards young people who tend to drink heavily at the weekend which results in increased mortality and morbidity from accidents and crime and violence. In South Africa, 46% of the cases of mortality due to non-natural causes for which blood alcohol levels were obtained, had blood alcohol concentrations (BACs), greater than or equal to 0.05g/100ml, the legal limit for driving (Matzopoulos *et al.*,2002). A study carried out between 1999 and 2001 of patients treated in trauma units in three South African cities found that across the sites and for each respective

year of the survey between 17% and 67% of patients had BACS greater than or equal to 0.05g/100ml (Plüddemann *et al.*, 2004). The pharmacological effects of alcohol are likely to increase the likelihood of aggressive behaviour (Room *et al.*, 2005). People commit crimes while under the influence of alcohol (Parry and Dewing, 2006); and heavy drinking by adults often leads to disrupted family life with domestic violence and child neglect (Brady and Rendall-Mkosi, 2004). Alcohol use is also associated with unsafe sexual practices and the increased risk of contracting HIV (Morojele *et al*., 2006). This coupled with the high prevalence estimate of 12 % for HIV/AIDS among South Africans (Dorrington *et al*., 2004) is cause for concern. According to a 2003 study, almost one in five HIV patients studied at a large infectious disease clinic in Cape Town, met criteria for an alcohol use disorder (Olly *et al.,* 2003); these patients were also more likely to have symptomatic HIV infection.

Foetal Alcohol Syndrome (FAS) was recognised as a distinct birth defect in 1973 (Jones *et al.*, 1973) and has been identified as an important public health problem in certain regions and among specific population groups in South Africa. This is particularly the case for pregnant women in poorer communities in the Western Cape where it was considerably higher than the percentages found in other surveys (42.8% versus 25% in the USA (May *et al.,* 2000). These rates are 18 to 141 times greater than in the United States. The study of FAS in a South African community in the Western Cape Province by May *et al.,* (2000) demonstrates that the "historical presence of the wine industry in the Western Cape and the drinking patterns that have developed have produced a high FAS rate." The culture of heavy drinking by male partners has also undermined the cultural taboo of women drinking during child-bearing years (Viljoen, 2005). Other factors associated with FAS, identified by a review of various studies by May *et al*. (2000) included a greater risk of FAS among women characterised by advanced maternal age, high gravidity and parity, low socioeconomic status and severe drinking patterns, particularly heavy episodic use. In the literature on alcohol abuse, heavy drinking women are often found to be co-habitors with alcoholic males, having alcohol abusing parents, initiating drinking at an early age and taking other drugs (May *et al.,* 2000). These factors demonstrate that FAS is a complex social problem.

Alcohol causes considerable disease burden and in 2000, 3.2% (1.8 million) of global deaths and 4% (58.3 million) of global Disability Adjusted Life Years, (DALYs), have been attributed to alcohol exposure (Rhem *et al.,* 2004). The comparable global figures in 1990 were 1.5% of global deaths and 3.5% of global DALYs. In 2000, the DALY burden for AFR-E was estimated to be 0.8% for females and 3.5% for males, accounting for 6.1 million deaths (Rhem *et al*., 2003; Rhem *et al.,* 2004).

The WHO AFR-E sub-region comprises 20 countries. This high mortality, developing sub-region which includes South Africa was rated as having the seventh highest consumption level of all 14 WHO regions, with an estimated 7.1 litres of absolute alcohol consumed per adult per year. Given the fairly

high level of abstainers in the AFR-E region, this translates to a rate of 16.6 litres per drinker, which according to Rhem *et al.,* (2003) is the highest rate in the world.

Alcohol abuse is not conducive to economic development and contributes to a cycle of poverty. Room *et al.* (2003) reported that levels of alcohol consumption have increased in many developing countries. The global burden of alcohol in 2000 in terms of death and disability was estimated by Rhem *et al.,* (2004) at between 1.6% (for high mortality developing sub-regions) and 9.2% (for developed sub-regions) of total disability adjusted life years lost. These figures have been interpreted to infer that as countries develop, their burden from alcohol abuse will increase.

All in all, a very bleak picture of alcohol related health and social problems emerges for South Africa. The AFR-E figures for burden attributed to alcohol are likely to be too low to accurately reflect this burden in South Africa. The aim of the study is therefore to make quantitative estimates of the alcohol-attributable disease burden by sex and specific age groups in South Africa in 2000. The findings of the study (Schneider *et al.,* 2007) are reported alongside 16 other risk factors that formed the South African Comparative Risk Assessment (Norman *et al*., 2007). This methodological note provides fuller details about the study and highlights additional findings.

2. Methods

2.1 Comparative risk assessment and health outcomes for alcohol

The methods developed by Ezzati *et al.* (2002) for the WHO Comparative Risk Assessment, (CRA) study (WHO, 2002) and applied to alcohol by Rhem *et al.* (2004) have been used in this study. Broadly the method is to estimate the amount of disease or injury burden attributable to exposure to the risk factor alcohol, by comparing the current risk factor distribution to a counterfactual distribution. For alcohol, this counterfactual distribution is the theoretical minimum distribution, an exposure distribution that will result in the lowest possible risk in the population.

Although the intake of alcohol has been related to more than 60 health outcomes (English *et al.,* 1995, Gutjhar *et al.,* 2001, Ridolfo and Stevenson, 2001), the SA study is restricted to health outcomes identified from meta-analyses in the global review by Rhem *et al.* (2004) as well as FAS and low birthweight (LBW). Pancreatitis, cholelithiasis, spontaneous abortion and psoriasis are not listed separately in the SA National Burden of Disease (SANBD) study by Bradshaw *et al.* (2003) and revised by Norman *et al.* (2007), and have been left out of the analysis. The health outcomes related to alcohol with ICD-10 codes (WHO, 1988) are listed in Table 1.

Table 1: Alcohol related health outcomes

Health outcomes	ICD-10 codes
Cancers (neoplasms)	
Mouth/oropharynx	C06,C10
Oesophagus	C15
Liver	C22
Larynx	C32
Breast	D05
Cardiovascular diseases	
Hypertensive diseases	I10-I13
Ischaemic heart disease	I20-I25
Ischaemic stroke (cerebral infarction)	I63
Haemorrhagic stroke (intra-cerebral haemorrhage)	I61
Other chronic diseases	
Diabetes (non-insulin dependent)	E11
Cirrhosis of liver	K70, K71,K74,K76
Effects of prenatal alcohol exposure	
Foetal alcohol syndrome	Q86.0
Low birth weight	P07
Neuropsychiatric conditions	
Depression (unipolar major depression)	F32
Epilepsy	G40
Alcohol dependence	Z72
Acute adverse effects	
Intentional injuries	X60-X84, Y87
Unintentional injuries	V01-V99

The selected health outcomes include four groups of conditions attributable to alcohol:

- Chronic conditions and LBW, where alcohol may be a detrimental (e.g. cancer) or beneficial (e.g. type II diabetes) contributing cause. The burden attributable to alcohol consumption in the population was estimated by comparing the current observed level of alcohol consumption to a counterfactual of no consumption and the relative risk (RR) of disease occurrence. In the case of IHD, two dimensions of alcohol consumption are defined as exposure variables: average volume of alcohol consumption, and pattern of drinking;
- Acute conditions, such as intentional and unintentional injuries, where alcohol is a contributing cause was assessed through categorical attribution;
- Unipolar depression, where a review of global data by Rhem *et al.* (2004) revealed an association with alcohol dependence that was used to predict the alcohol-attributable fraction (AAF) from the prevalence of alcohol dependence by sub-region,
- Those which are 100% alcohol attributable, such as alcohol use disorders and FAS.

2.2 Prevalence of exposure to alcohol

Two dimensions of alcohol consumption are defined as exposure variables, namely, the average volume of alcohol consumption and the pattern of drinking. A hypothetical scenario that provides a reference for hypothetical risk reduction, should take both dimensions into account. For the global review, analyses involving both volume of alcohol consumed and drinking patterns were included for the health outcomes, IHD and injuries, as these were the two main ICD categories for which there are sufficient evidence of a causal link to drinking patterns.

The 1998 South African Demographic and Health Survey (SADHS) reported that 45% of men and 17% of women 15 years and older reported that they currently consume alcohol (Department of Health, Medical Research Council and Macro International, 2001). Although this is relatively low, the particular segment of the population that drink; as well as the pattern of drinking is particularly worrying, given the epidemiological profile and social problems in South Africa. One third of the current drinkers in SADHS reported risky drinking over weekends. (Risky drinking is defined as five or more drinks per day for men and three or more drinks per day for women).

Prevalence of alcohol consumption based on the average volume of alcohol consumption was not available from the 1998 SADHS as the alcohol consumption questions determines current alcohol use i.e. use in the past 30 days to assess weekend and weekday consumption. The response options for these questions are specified categories that did not correspond to levels of drinking categories based on the number of drinks in the global risk assessment by Rhem *et al.* (2004). While it would be possible to obtain an average number of drinks from the mid-point of the category for the approximate number of drinks consumed, convert this to alcohol consumed and then allocate to the specified categories, this would not be satisfactory, as the distribution may be asymmetrical across the category. Another source for the prevalence of population alcohol consumption categories was the 2001 World Health Survey (WHS) that was conducted in South Africa (WHO, 2003). It is difficult to compare the results from these surveys as they do not have the same response categories as each other (or as the CRA study). Both surveys observed very high levels of abstinence, particularly among women. However, it is considered that these surveys are likely to understate the extent of alcohol consumption as people often do not respond truthfully to the sensitive issue of alcohol consumption.

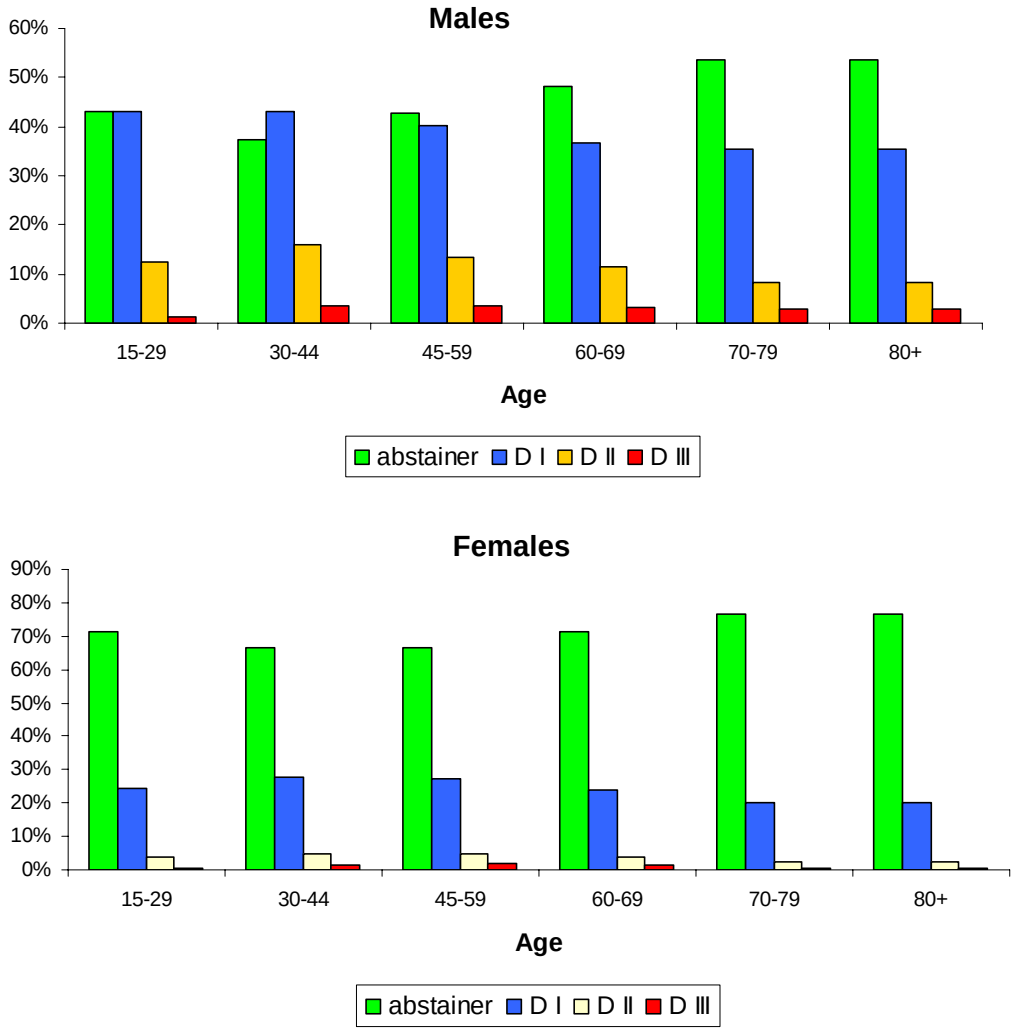
The production figures for South Africa reported in the Alcohol Beverage Review (2004) result in a per capita consumption figure of 6.98 litres of alcohol and is of the same order of magnitude of 7.8 for South Africa from the recent World Health Statistics Report (WHO, 2005). (Both the latter figures are for round about the year 2000 and are for recorded consumption only). It is not clear whether the industry figure over-estimates or under-estimates the amount of alcohol consumed. The production figures do not reflect imported alcohol. However they do include the whole South African Customs Union.

Rhem *et al.* (2004) estimated that the adult per capita consumption in South Africa was 10.21 for after 1998 (in 2000) in litres of pure alcohol. The figure is 12.41 litres of alcohol if the recorded consumption is adjusted to include estimates of unrecorded consumption, such as home brews. These figures are relatively lower than most developed countries (Room *et al.*, 2002). As many people do not drink alcohol at all, the amount consumed per drinker is about 20 litres of absolute alcohol consumed per year, among the highest in the world, for a country.

It was not possible to reconcile the alcohol consumption figures reported by industry and implied by the prevalence observed in either of the national population based surveys. Production figures based on the excise collected for the Southern African Customs Union were converted into litres of alcohol by type and then into the number of drinks per year. The prevalence figures on drinking from each population based survey were converted into the number of drinks per day and compared with the estimate based on production. The AFR-E sub-region consumption prevalence data from the global CRA study by Rhem *et al.* (2004) matched the production figures for SA in 2000, far better than either the World Health Survey 2001 or the SADHS 1998. Distributional information on drinkers for the AFR-E sub-

region, was obtained form the 1998 SADHS and the age-specific prevalence of drinking was calculated on the assumption that the average per capita consumption and the proportion of male and female abstainers were correct. It was therefore decided to use the prevalence of alcohol exposure levels by age and sex from the AFR-E sub-region.

The drinking categories DI, DII and DII from the CRA study are based on the Australian study (English *et al*., 1995). The categories comprise consumption in grams of pure alcohol per day and are shown in Figures 2.1 and Table 2. Men and women have different category cut points, as alcohol affects men and women differently due to body size.



Definitions of categories of risk factor levels
Level 1: Abstinence abstainer: 0-<0.25g/day Males, 0-<0.25g/day females
Level 2: (Dl) drinking category I: Males >0.25–<40 g/day; females >0.25–<20 g/day
Level 3: (Dll) drinking category II: Males 40–<60 g/day; females 20 - <40g/day
Level 4: (Dlll) drinking category III: Males≥60 g/day; Females ≥40 g/day

Figure 2.1: Alcohol consumption in males and females by age, South Africa 2000
Source: Prevalence for AFR-E region modelled by Rehm *et al*. 2004.

Table 2: Estimated prevalence of alcohol exposure levels by age and sex, South Africa, 2000

Sex	Average volume of consumption category	Age groups (yrs)					
		15-29	30-44	45-59	60-69	70-79	≥80
Males	Abstinence	0.43	0.38	0.43	0.48	0.54	0.54
	D I	0.43	0.43	0.40	0.37	0.35	0.35
	D II	0.13	0.16	0.14	0.12	0.08	0.08
	D III	0.01	0.03	0.03	0.03	0.03	0.03
Females	Abstinence	0.72	0.66	0.66	0.72	0.78	0.78
	D I	0.24	0.28	0.27	0.24	0.20	0.20
	D II	0.04	0.05	0.05	0.24	0.03	0.03
	D III	0.01	0.01	0.02	0.01	0.01	0.01

Source: Rehm *et al*. (2004) prevalence for AFR-E region.
Definitions of categories of risk factor levels:
Abstinence: Males 0-<0.25 g/d, females, 0-<0.25 g/d ;
Drinking category D l: Males >0.25-<40 g/d, females >0.25-<20 g/d;
Drinking category D II: Males 40–<60 g/d, females 20-<40 g/d;
Drinking category D III: Males ≥60 g/d, females ≥40 g/d.

In order to estimate the extent of FAS, it is necessary to know the prevalence of alcohol consumption among pregnant women. There is limited data on the prevalence of alcohol consumption during pregnancy. Only 13 out of 191 pregnant women (7%) interviewed in the 1998 SADHS acknowledged current drinking. This figure was considered unreliable due to the small number of pregnant women in the sample, and also probably an under-estimate due to the particularly sensitive nature of the question.

Data from three underprivileged areas in the Western Cape suggests little awareness of the health risks of alcohol as 23.7% of the sample of 636 pregnant women attending 17 antenatal clinics reported alcohol intake sufficient to place unborn children at risk (Coxford and Viljoen, 1999). We therefore assumed the same prevalence of drinking as in non-pregnant women, (ie.16.8%) weighted by the estimated number of births in women of child-bearing age obtained from the ASSA2002 model. The respective weights were 0.75 and 0.25 for the child-bearing age groups 15-29 and 30-49 years.

2.3 Population attributable fraction

Population-attributable fractions (PAFs) by age and sex and cause were calculated in MS Excel using the formula:

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

where p_i is the prevalence of exposure level i, RR_i is the RR of disease in exposure level i and k is the total number of exposure (Walter, 1995). As the exposure variable has several categories, the formula

above is a multi-level extension of the usual attributable fraction formula. Estimates of the RRs were obtained from the meta-analyses reported in the global review by Rhem, Room, Montiero, *et al.* (2004) and other studies. These are presented in Table 3. It can be seen that the RR for Type 2 diabetes mellitus (males and females), haemorrhagic stroke (females) and ischaemic stroke (females) are less than 1 indicating that alcohol consumption has beneficial effects for these conditions.

Table 3: Relative risks (RRs) and 95% confidence intervals for alcohol-related disease for different drinking categories) D I to D III relative to abstainers

Health outcome	Abstainer	RR category D I	RR category D II	RR category D III
Cancer mouth/pharynx[a]	1	1.45 (1.32 – 1.60)	1.85 (1.49 – 2.30)	5.39 (4.67 – 6.22)
Cancer oesophagus[a]	1	1.80 (1.63-1.99)	2.37 (2.03 – 2.76)	4.26 (3.70 – 4.90)
Cancer liver[a]	1	1.45 (1.09 – 1.94)	3.03 (1.33 – 6.92)	3.6 (2.05 – 6.32)
Cancer larynx[a]	1	1.83 (1.51 – 2.22)	3.90 (2.13 – 7.13)	4.93 (3.41 – 7.15)
Cancer breast[b] (females) <45 years	1	1.15 (1.04 – 1.28)	1.41 (1.20 – 1.67)	1.46 (0.99 – 2.14)
Cancer breast[b] (females) 45+ years	1	1.14 (1.05 – 1.24)	1.38 (1.24 – 1.53)	1.62 (1.24 – 2.13)
Type 2 diabetes mellitus[c] (males)	1	1.00 (0.98 – 1.01)	0.57 (0.28 – 1.01)	0.73 (0.55 – 1.06)
Type 2 diabetes mellitus[c] (females)	1	0.92 (0.80 -1.08)	0.87 (0.78 – 1.03)	1.13 (0.97 – 1.22)
Epilepsy[c] (males)	1	1.23 (0.99 – 1.54)	7.52 (5.93 – 9.55)	6.83 (5.41 – 8.65)
Epilepsy[c] (females)	1	1.34 (0.99 – 1.79)	7.22 (5.70 – 9.16)	7.52 (5.93 – 9.55)
Hypertension[d]	1	1.4 (1.3 – 1.5)	2.0 (1.8 – 2.3)	4.1 (3.1 – 5.9)
Ischaemic heart disease*	1	0.82 (0.80 -0.83)	0.84 (0.80 – 0.88)	0.88 (0.84 – 0.92)
Ischaemic stroke[b] (males)	1	0.94 (0.78 – 1.13)	1.33 (1.07 – 1.66)	1.65 (0.95 – 2.86)
Ischaemic stroke[b] (females)	1	0.52 (0.42 – 0.65)	0.64 (0.44 – 0.95)	1.06 (0.36 – 3.12)
Haemorrhagic stroke[b] (males)	1	1.27 (0.83 – 1.94)	2.19 (1.47 – 3.28)	2.38 (1.18 – 4.77)
Haemorrhagic strokes[b] (females)	1	0.59 (0.38 – 0.92)	0.65 (0.36 – 1.19)	7.98 (3.25 – 19.6)
Cirrhosis[c]	1	1.26 (1.25 – 1.26)	9.54 (9.31 – 9.77)	13.0 (12.68 – 13.32)
Low birthweight[e]	1	1	1.40 (1.19 – 1.67)	1.40 (1.19 – 1.67)

T2DM = Type 2 diabetes mellitus. For T2DM (males and females), haemorrhagic stroke (females) and ischaemic stroke (females) have beneficial effects related to alcohol consumption.

Source: Unless otherwise stated Gutjahr *et al.* (2001) and Ridolfo and Stevenson (2001) as reported by Rehm *et al*. (2004).

* RRs from Corrao *et al*. (2000) were not used in analysis; instead the AAFs for IHD for AFR-E predicted from multilevel analysis were used.

Definition of categories of risk factor levels:

Abstinence: Males 0-<0.25 g/d, females, 0-<0.25 g/d ;
Drinking category D l: Males >0.25-<40 g/d, females >0.25-<20 g/d;
Drinking category D II: Males 40–<60 g/d, females 20-<40 g/d;
Drinking category D III: Males ≥60 g/d, females ≥40 g/d.

[a] English *et al.* (1995).
[b] Ridolfo and Stevenson (2001).
[c] Gutjahr *et al.* (2001) CIs derived from English *et al.* (1995).
[d] Corrao *et al.* (1999).
[e] Rehm *et al*. (2004).

2.3.1 IHD

Based on the overall drinking pattern in SA, only the harmful effects for IHD were considered in the case of IHD. AFR-E estimates of the attributable fraction incorporating the effect of the alcohol consumption from Corraro *et al.* (2000) are included in Table 5 for completeness but were not used. The standard method of calculating PAFS using the appropriate RRs and prevalence and a counterfactual distribution was not used for IHD as there is a lack of data on the relationship between exposure including patterns of drinking and IHD. If one were to use the relative risks from individual-level studies, these may overestimate the cardio-protective effects due to over-representation in the cohort of people with more regular drinking styles.

Rhem *et al.* (2003) developed a multi-level model to determine the AAF based on per capita consumption and drinking pattern. In terms of patterns of drinking, countries were rated on a four point scale with "4" being the most harmful. Practices that were particularly harmful included: not drinking with meals, drinking in public places, drinking daily or nearly daily, drinking to intoxication, festive drinking and high unusual quantities of alcohol per occasion. These occasions include community events such as funerals and may include communal drinking (passing around a common container).
Beneficial effects are expected for countries with a consumption pattern of 1. In countries with a detrimental pattern 3, alcohol showed a detrimental impact on IHD for males only (Rehm *et al.,* 2003). For females the impact on IHD was zero (i.e. no marked impact of alcohol.) The patterns most detrimental occur in four sub-regions, including AFR-E and South Africa is categorized as having a type 3 pattern.

IHD mortality, per capita consumption and the appropriate drinking pattern value was used to obtain the predicted AAFs for IHD for each country (Rhem *et al.,* 2003). As in the global study, the effects are halved to adjust for potential confounding.

2.3.2 Mental health

Alcohol dependence, by definition, is fully attributed to alcohol, (AAF =1). In the case of depression related to alcohol use, it is only that fraction of depression for which the onset of alcohol problems precede the depression which can be attributed to alcohol. It is necessary to estimate the AFF based on the prevalence of alcohol dependence. The CAGE questionnaire, which was included in the 1998 SADHS, can serve as a screening instrument for possible alcohol dependence. (The questions focus on Cutting down, Annoyance by criticism, Guilty feeling, and Eye-openers. The acronym "CAGE" helps the physician to recall the questions). Even when a stringent cut-off (an affirmative response to 3 questions instead of the usual of 2) on the CAGE questions was used with the 1998 SADHS data, the prevalence of problematic alcohol use was too high to be plausible.

Although the co-morbidity of alcohol problems and mental health has been established, the direction of causal relationships is often complex. For example, it is known that depressed people often use alcohol to deal with already established depression. Indirect quantitative estimates of the proportion of the depressive disorders attributable to alcohol can be derived from the high correlation between alcohol dependence and the proportion of the depressive disorders with preceding alcohol use-disorders, using the country specific alcohol dependence rate (Rhem *et al.,* 2004).

The AAF for major depression in AFR-E modelled from AFR-E prevalence of alcohol dependence in the global study by Rhem *et al.* (2004) was used due to the lack of reliable local estimates of alcohol dependence. The prevalence of alcohol dependence for AFR-E is 2.89% for males and 0.31% for females and the AAF for major depression is 3.56 for males and 0.37 for females in AFR-E - in people aged ≥15 years the figure is 0.04. The prevalence of risky weekday drinking in the 1998 SADHS was similar to the prevalence of alcohol dependence in AFR-E, supporting the decision to use these modelled estimates for South Africa.

2.3.3 Intentional and unintentional injuries

The AAFs for acute consequences such as injuries are usually directly determined from the blood alcohol concentration (BAC) at the time of the injury. For example, road accidents are attributed to alcohol according to whether the driver responsible for the accident tested positive for alcohol and to what degree.

For intentional and unintentional injuries we used 2001 data from the National Injury Mortality Surveillance System (NIMSS) (Matzopolous *et al.,* 2002). National AAFs for injury mortality were based on the percentage of fatal injuries positive for blood alcohol concentration (BAC) ≥ 0.05 g/100 ml using NIMSS data by age, sex and injury cause obtained from those mortuaries with academic forensic support (personal communication H. Donson). Although the risk of accidents increases with higher BAC levels, and the NIMSS provides different levels of percentage blood alcohol content (BAC), and in this analysis only one level of percentage BAC is used, namely, ≥0.05 g/per 100ml.

Alcohol has a differential affect on fatal and non-fatal injury outcomes. In general, more severe outcomes are related to alcohol than less severe outcomes. Consequently, the AAFs for mortality should be higher than the AAFs for morbidity. Unfortunately, most research to determine AAFs for injury did not explicitly separate mortality and morbidity. Ridolfo and Stevenson (2001) explicitly separated the AAFs for motor vehicle accidents. In the case of males they determined AAFs of 0.32 for deaths and 0.24 for hospitalizations. Based on their work and that of Cherpitel (1994, 1996), the ratio of AAF for morbidity from motor vehicle accidents was estimated in the global study as two thirds of the AAF for

mortality (Rhem *et al.*, 2004). The ratio for other kinds of injury is lower, and to be conservative, these ratios were set at 0.44 for both men and women.

Injury morbidity AAFs were calculated as the percentage of non-fatal injuries positive for BAC ≥ 0.05 g/100 ml using 1999-2001 data from the three-city study of Plüddemann *et al.* (2004). The morbidity to mortality relationship observed in each of the three cities (Cape Town, Port Elizabeth and Durban) were averaged to obtain a morbidity to mortality ratio of 0.61 for interpersonal violence and 0.42 for road traffic injuries (RTIs), and applied to the national AAF for injury mortality to derive national injury morbidity AAFs. For all other injury categories we used a ratio of 0.44 (two-thirds of the RTI ratio or the product of 0.67 and 0.42).

In the case of other injuries (excluding violence and transport) NIMMS data for the Western Cape was available. In this case the ratio was approximately 0.5 (the same as transport for Cape Town). The same method used by Rehm *et al.* (2004) was used for the South African study and the figure used was 0.18, (=0.67x 0.42), that is, two thirds of the ratio for motor vehicle accidents.

The AAFs used in the South African study are shown in Table 4.

Table 4: Estimated alcohol attributable fractions, South Africa 2000

Males	**0-4**	**5-14**	**15-29**	**30-44**	**45-59**	**60-69**	**70-79**	**80+**
Cancer mouth/ pharynx				0.32	0.31	0.29	0.26	0.26
Cancer oesophagus				0.40	0.38	0.36	0.33	0.33
Cancer liver				0.38	0.35	0.33	0.29	0.29
Cancer larynx				0.49	0.46	0.44	0.39	0.39
Cancer female breast				0.00	0.00	0.00	0.00	0.00
Diabetes				-0.09	-0.08	-0.07	-0.05	-0.05
Epilepsy			0.50	0.57	0.54	0.51	0.44	0.44
Hypertension				0.30	0.29	0.27	0.24	0.24
Ischaemic heart disease*			0.07	0.08	0.08	0.07	0.07	0.07
Stroke – harm				0.18	0.15	0.13	0.10	0.09
Stroke YLDs				0.11	0.09	0.08	0.06	0.06
Stroke – benefit				-0.01	-0.01	-0.01	-0.01	-0.01
Stroke benefit YLDs				-0.02	-0.02	-0.02	-0.02	-0.02
Cirrhosis liver				0.66	0.63	0.60	0.54	0.54
Alcohol use disorders	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Depression**			0.04	0.04	0.04	0.04	0.04	0.04
LBW	0.01							
FAS	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
IHD			*-0.11*	*-0.12*	*-0.11*	*-0.09*	*-0.08*	*-0.08*
Females	**0-4**	**5-14**	**15-29**	**30-44**	**45-59**	**60-69**	**70-79**	**80+**
Ca mouth/ pharynx				0.18	0.19	0.16	0.12	0.12
Ca oesophagus				0.24	0.25	0.22	0.18	0.18
Ca liver				0.20	0.21	0.17	0.14	0.14
Ca larynx				0.29	0.30	0.26	0.21	0.21
Ca female breast				0.06	0.06	0.05	0.04	0.04
Diabetes				-0.03	-0.03	-0.02	-0.02	-0.02
Epilepsy				0.31	0.33	0.28	0.21	0.21
Hypertension				0.14	0.15	0.12	0.10	0.10
Ischaemic heart disease			0.00	0.00	0.00	0.00	0.00	0.00
Stroke – harm				0.05	0.06	0.04	0.02	0.02
Stroke YLDs				0.02	0.02	0.01	0.00	0.00
Stroke – benefit				-0.15	-0.15	-0.13	-0.11	-0.11
Stroke benefit YLDs				-0.17	-0.16	-0.14	-0.12	-0.12
Cirrhosis				0.38	0.41	0.34	0.26	0.26
Alcohol use disorders	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Depression			0.00	0.00	0.00	0.00	0.00	0.00
LBW	0.003							
FAS	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
IHD				*-0.06*	*-0.06*	*-0.05*	*-0.04*	*-0.04*

Source: *Rehm *et al.* (2004) predicted by multilevel analysis, **Rehm *et al.* (2004).

2.4 Attributable burden

The estimated PAFs were applied to the number of deaths, years of life lost due to premature mortality (YLLs), years of life lived with disability (YLDs) and disability-adjusted life years (DALYs) for each selected outcome from the revised SANBD study for 2000 (Norman, Bradshaw, Schneider *et al.,* 2006). The calculations for stroke and FAS, however, were not straight-forward. The calculation of the attributable burden was complicated by the fact that haemorrhagic and ischaemic stroke are affected differently by alcohol but the SA NBD does not include the stroke sub-types as end-points.

2.4.1 Stroke

Haemorrhagic stroke is closely related to blood pressure levels, which are adversely affected by alcohol, while moderate to low alcohol consumption affords some protection from ischaemic stroke. The Ridolfo and Stevenson (2001) meta-analysis used in the WHO CRA study indicates an increased risk of haemorrhagic stroke in males even at low consumption levels. For women, there is a strong protective effect when less than 40g/day of alcohol is consumed. Ischaemic stroke effects are similar to those for IHD, i.e. low to moderate alcohol consumption can afford some protection. The protective effect is more pronounced in females. However, the SA burden of disease endpoint is 'total stroke' and not stroke subtypes.

Estimates of the subtypes for stroke were estimated. Total stroke deaths and DALYs were adjusted by the age-specific proportions of ischaemic and haemorrhagic fatal and non-fatal strokes for the AFR-E region, using the method of Lawes *et al*. (2004) and the Clinical Trials research Unit (2002). A study conducted in Pretoria found the case fatality rate (CFR) to be 22% at one month for ischaemic stroke and 58% for haemorrhagic stroke (Rosman, 1986) which was similar to estimated CFRs for stroke subtypes for the AFR-E region.

In the global Comparative Risk Assessment Risk (CRA) analyses, mean cholesterol levels were used to estimate the proportion of strokes due to haemorrhagic and ischemic stroke. The level of cholesterol is important in determining the overall ratio of cerebrovascular ischaemic to cerebrovascualr haemorrhagic. The Clinical Trials Research Unit of Auckland University estimated the age specific, stroke subtype proportions for fatal and non-fatal events using data from four 'gold standard' incidence studies (Clinincal Trials Research Unit, 2002). As the overall percentage of haemorrhagic stroke was 20% in the AFR-E region, the age specific proportions were adjusted by 1.33 (20/15). From the 'gold standard' incidence studies included in a recent review, the case fatality rate for AFR-E is 20%. Hence, scenario for AFR-E is as follows: 60% for ischaemic stroke and 20% for haemorrhagic stroke and a case fatality rate, (CFR), of 20%. A study by Rosman in Pretoria (South Africa), (1986), found the case fatality to be 22% at one month for ischaemic stroke and 58% for haemorrhagic stroke, similar to the

estimated case fatality rates for the stroke subtypes for the AFR-E region. The AFR-E percentages were then converted to proportions of ischaemic and haemorrhagic stroke within the fatal and non-fatal groups.

2.4.2 FAS

The diagnosis of FAS is applied to a child who has growth retardation with central nervous system anomalies and characteristic facial dysmorphology (Viljoen, 2005). There are no biological markers to assist with the diagnosis of FAS and diagnosis may be very inaccurate at birth; FAS is diagnosed with more confidence between the ages of 3 years and 10 years. There are three major case-definition categories: growth retardation, facial dysmorphic features and head circumference. Other clinical features of FAS include central nervous system (CNS) dysfunction, such as, decreased intelligence and hyperactivity. These characteristics of FAS are not always universally present and depend on the timing and dose of alcohol exposure, maternal liver function and maternal nutrition and many other risk factors. In our estimate of the burden associated with FAS, we explicitly exclude alcohol–related birth defects (ARBD) and alcohol-related neuro-developmental disorder (ARND) as we do not have the empirical data to estimate the burden related to these conditions. ARBD and ARND are also the result of children exposed to maternal alcohol use but do not fulfill the full criteria for a diagnosis of FAS.

The disease impact of FAS is measured in terms of morbidity as the South African cause of death data do not have any deaths ascribed to FAS. The calculation of YLDs requires the incidence of FAS, the life expectancy and disability weights. There are no national incidence data. Studies of FAS in specific communities have results ranging from an incidence of 11.8 to 103 per 1 000 births as seen in Table 5 (Prof Viljoen personal communication). Some of these rates are extraordinarily high. However, the studies have tended to focus on communities where FAS is known to be a problem.

Table 5: FAS incidence rates from recent South African studies

Province	Characteristic of study population	Year	FAS incidence rate
Western Cape	1 small town community Population: 35 364 urban + 986 rural	1997	40.5 – 46.4/1000
Western Cape	2 small town community Population: 35 364 urban + 9861 rural	1999	75.0/1000
Western Cape	1 small town community Population: 35 364 urban + 9861 rural	2001	Estimated 87/1000
Gauteng	A These are high risk areas of JHB B C D	2001 Total	22.7/1000 11.8/1000 31.1/1000 41.7/1000 26.5/1000
Northern Cape	Town A (Population: 27 000) Town B (Population: 55 322)	2002 2003	103/1000 estimated 68/1000

Source: Prof Viljoen personal communication.

The incidence rates ranged from 11.8 per 1000 to an extreme of 103 per 1000. We have used an estimated national incidence rate of FAS has 14 per 1 000 births. This figure is based on an incidence of 11.8 per 1000 births occurring in 92% of births in 2000 in SA and an incidence of 40 per 1000 in the 8% of births in the coloured population. Given that national surveys show about 70% of South African women do not drink alcohol at all, this seems to be a plausible estimate.

For the calculation of YLDs, the age at onset is 0.0 years. The duration is 58.6 for males and 66.5 for females, based on the life expectancy obtained from 1990 South African pre-AIDS figures, and used in the SA NBD study. (Even though the life expectancy of children diagnosed with FAS is lower than that of the general population, (RR= 2), no adjustment for this decreased life expectancy was incorporated as no deaths are ascribed to FAS for this study). The disability weights have been calculated using the Dutch weights (Stoutard *et al.,* 1997) for different levels of mental disorder, as shown in Table 6.

The mean IQ of children with FAS, of 77.5 and standard deviation of 13.4 was obtained from a study in a community in the Western Cape (May *et al.,* 2000). By applying the normal distribution to this data, the proportions of children with FAS in each of the categories of mental retardation could be estimated and used to derive a weighted disability weight for the children with FAS. Based on this data, an overall disability weight of 0.125 was derived.

Table 6: The Dutch disability weights

Level of mental disorder	Disability weight
Mental retardation (IQ = 70-84)	0.09
Mild mental handicap IQ = 50-69)	0.29
Moderate mental handicap (IQ =35-49)	0.43
Severe mental handicap (IQ =20-34)	0.82
Extreme mental handicap (IQ< 20)	0.76

Source: Stouthard *et al.,* 1997.

Based on an incidence of 14 per 1000, a mean IQ of 77.5 and a SD of 13.4 and no adjustment to the life expectancy the total DALYs for the year 2000 are 62 466; 100% of theses DALYS are attributed to alcohol. It should be noted that the FAS attributable DALYs under-estimates the full impact of FAS, in that the behavioural and learning disabilities resulting from FAS also have high social consequences that are not included. These are not easily quantifiable in terms of DALYs.

2.4.3 Uncertainty analysis

Monte Carlo simulation modeling techniques were used to present uncertainty ranges around point estimates reflecting all the main sources of variability in the calculations. The @RISK software version 4.5 for Excel was used, which allows multiple recalculations of a spreadsheet, each time choosing a value from distributions defined for input variables. For prevalence of average volume of consumption categories, the estimated uncertainty ranges around AFR-E point estimates from the global CRA study were used (Rhem *et al.,* 2004). For the RR input variables a normal distribution was specified, with the natural logarithm of the RR estimates as the entered means of the distribution and the standard errors of these estimates. The 95% confidence intervals were calculated or obtained from the published 95% confidence intervals (CIs) for the RRs for alcohol-related disease for different drinking categories (see Table III). For each of the output variables (namely attributable burden as a percentage of total burden in SA 2000), 95% uncertainty intervals were calculated bounded by the 2.5^{th} and 97.5^{th} percentiles of the 2000 iteration values generated.

3. Results

Just under 37 000 deaths were attributable to alcohol in 2000, with considerable variation across sex and age groups (Figure 3.1). For each female death attributable to alcohol there were just over 4 male deaths, mostly as a result of the large number of fatal injuries in young adult men. Figure 3.1 also indicates the beneficial effects of alcohol consumption in terms of prevention of deaths from stroke and diabetes among older men and women (shown below the axis). These are particularly noticeable for stroke in older women. When the deaths that are prevented are taken into account, the total mortality loss attributed to alcohol is just over 33 699 deaths.

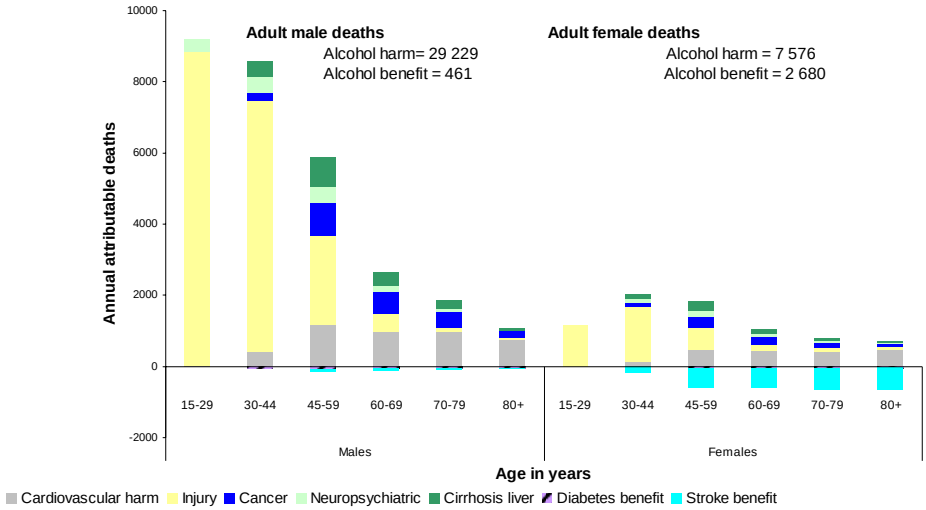


Figure 3.1: Annual alcohol-attributable adult deaths by age and sex (including beneficial effects), SA 2000

Including the disability related to alcohol abuse, and excluding the beneficial effects, more than 1.1 million DALYs were attributable to alcohol in 2000. Figure 3.2 depicts the alcohol-attributable DALYs by cause, and injuries accounted for 63.1% of the burden. Interpersonal violence accounted for the largest proportion of the injury burden, i.e. 39.0%, with 42.8% and 25.9% of the alcohol-attributable DALYs in males and females respectively.

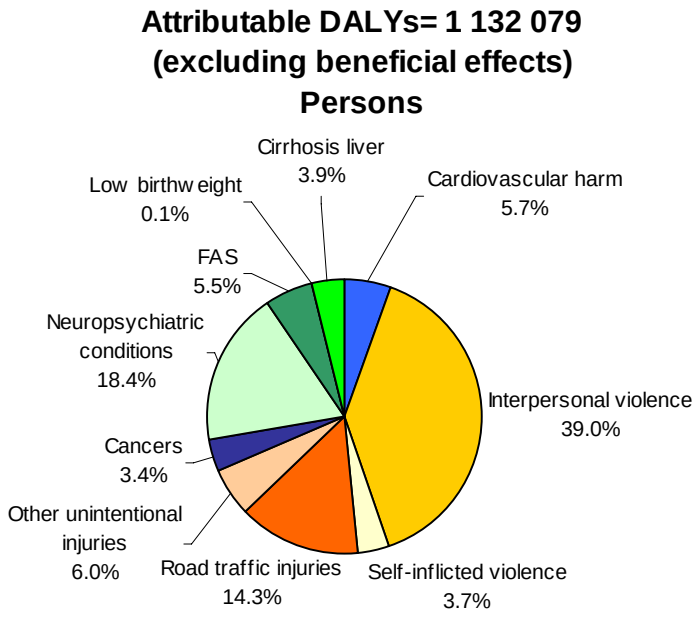


Figure 3.2: Alcohol-attributable DALYs for persons (excluding beneficial effects), SA 2000

Figure 3.3 shows the injury burden attributable to alcohol which is substantially higher for men than for women. Violence accounts for 69.5% of the DALYs attributed to alcohol in males and 58.3% in females. The violence part of these figures are disaggregated the by age groups, in order to highlight the large burden of violence in young adults. Violence accounts for 58.1% of the alcohol attributable DALYs for men between the ages of 15 and 44, and 48.8% is the figure for women between the ages of 15 and 44. Figure 3.3 also reflects the large RTI component of injury.

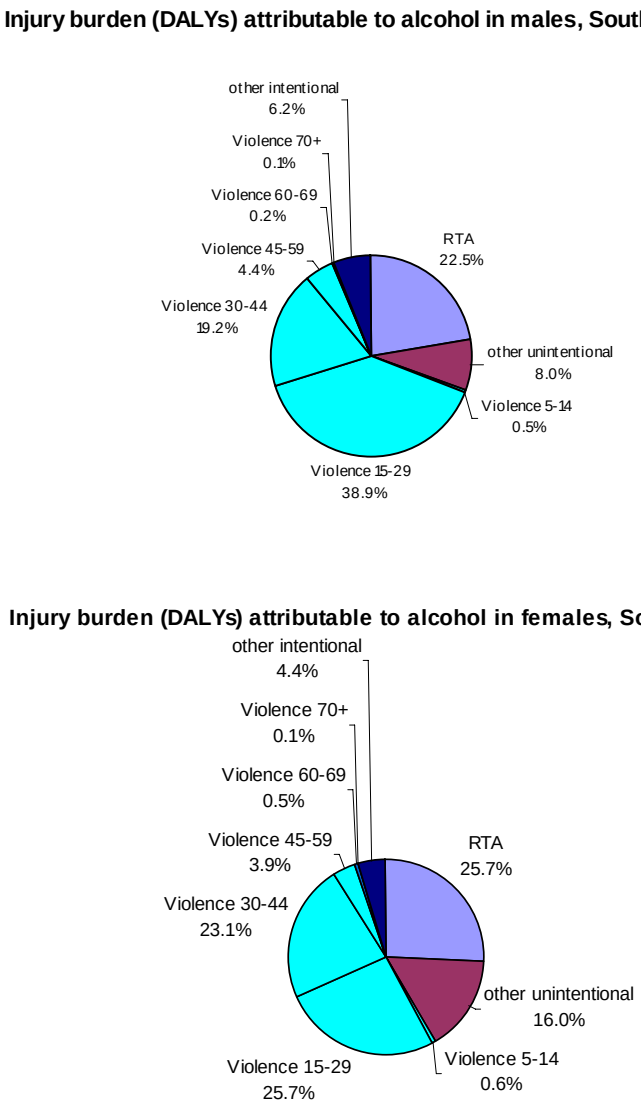


Figure 3.3: Alcohol-attributable injury DALYs for males and females, SA 2000

Alcohol accounted for 7.1% of all deaths (95% uncertainty interval: 6.6%-7.5%) and 7.0% of all DALYs (95% uncertainty interval: 6.6%-7.4%) for SA in 2000 (Table 7). The alcohol-attributable-burden is particularly marked for men, accounting for 10.4% of DALYs (95% uncertainty interval: 9.6% -11.1%). In the case of women, alcohol accounted for 3.3% of total DALYs (95% uncertainty interval: 3.1%-3.5%). From Table IV it can be seen that homicide and violence (39.0%), alcohol dependence or use disorders (14.7%) and RTIs (14.3%) are the top three rankings in terms of alcohol-attributable DALYs for persons. FAS is ranked fourth and accounts for 5.5% (62 466) of alcohol-attibutable DALYs (this despite no deaths attributed to FAS for this study). For YLLs the top rankings are homicide and violence (45.9%), RTIs (19.6%) and suicides (5.4%). However, in terms of alcohol-attributable disability (YLDs), alcohol use disorders ranks first (44.6%), homicide and violence second (23.2%) and FAS third (18.1%). These are followed by epilepsy and RTIs, accounting for 3.5% and 2.3% of alcohol-attributable YLDs respectively.

Table 7: Burden attributable to alcohol in males, females and persons, SA 2000

	Males					Females					Persons				
	PAFs	**Deaths**	**YLLs**	**YLDs**	**DALYs**	**PAFs**	**Deaths**	**YLLs**	**YLDs**	**DALYs**	**PAFs**	**Deaths**	**YLLs**	**YLDs**	**DALYs**
Cancer mouth/pharynx	28.5%	283	3353	166	3 519	16.4%	63	755	30	785	25.2%	345	4 108	197	4 304
Cancer oesophagus	37.2%	1 289	14 743	228	14 971	23.0%	437	5 194	70	5 264	32.1%	1 726	19 937	298	20 235
Cancer liver	30.3%	519	6 474	72	6 546	17.0%	161	1 829	24	1 852	25.8%	680	8 303	95	8 398
Cancer larynx	42.9%	271	2 888	0	2 888	28.0%	30	375	0	375	40.4%	301	3 263	0	3 263
Female breast cancer	0.0%	0	0	0	0	5.6%	165	2 160	165	2 326	5.6%	165	2 160	165	2 326
Type II diabetes mellitus beneficial	-6.4%	-301	-3 518	-954	-4 471	-2.3%	-189	-1 949	-521	-2 470	-3.9%	-491	-5 467	-1 475	-6 942
Epilepsy	53.7%	968	21 591	7 688	29 279	22.2%	208	3 446	4 492	7 938	41.2%	1 176	25 037	12 180	37 217
Hypertensive disease	26.0%	1 340	13 219	377	13 596	12.4%	1 302	11 231	255	11 486	17.3%	2 642	24 450	631	25 081
Ischaemic heart disease	7.6%	1 292	11 958	768	12 726	0.0%	0	0	0	0	4.4%	1 292	11 958	768	12 726
Stroke harmful	12.3%	1 635	17 422	1 331	18 753	3.8%	631	7 234	343	7 577	7.5%	2 266	24 656	1 674	26 330
Stroke beneficial	-1.1%	-160	-1 409	-284	-1 694	-13.0%	-2 491	-22 487	-3 308	-25 795	-7.9%	-2 650	-23 896	-3 593	-27 489
Cirrhosis liver	54.6%	1 932	28 209	4 836	33 046	31.3%	651	9 358	1 433	10 791	46.1%	2 582	37 567	6 269	43 836
Alcohol use disorders/dependence	100.0%	550	9 489	106 973	116 462	100.0%	210	3 563	46 536	50 099	100.0%	760	13 052	153 509	166 561
Depression	3.6%	0	0	3 591	3 591	0.4%	0	0	678	678	1.5%	0	0	4 269	4 269
Low birthweight	0.3%	19	637	47	685	0.3%	16	543	41	584	0.3%	36	1 181	88	1 269
Foetal alcohol syndrome	100.0%	0	0	31 181	31 181	100.0%	0	0	31 285	31 285	100.0%	0	0	62 466	62 466
Road traffic injuries	49.9%	4 935	123 834	6 779	130 613	29.2%	1 231	30 485	1 252	31 737	43.9%	6 166	154 319	8 031	162 350
Poisonings	31.3%	67	1 814	0	1 814	24.4%	47	1 034	0	1 034	28.4%	114	2 848	0	2 848
Falls	21.3%	185	3 576	1 287	4 863	7.7%	19	282	1 375	1 657	14.7%	204	3 858	2 662	6 520
Fires	51.0%	980	24 391	4 076	28 468	46.9%	668	14 534	2 073	16 606	49.4%	1 648	38 925	6 149	45 074
Drownings	56.8%	231	6 149	0	6 149	21.3%	21	467	0	467	50.8%	252	6 615	0	6 615
Other unintentional injuries	5.9%	70	1 857	4 895	6 752	0.0%	0	0	0	0	4.8%	70	1 857	4 895	6 752
Suicides	36.3%	1 430	36 790	7	36 797	18.5%	244	5 429	9	5 438	32.3%	1 674	42 218	16	42 235
Homicide and violence	47.3%	11 253	322 492	53 113	375 604	31.1%	1 488	38 946	26 855	65 800	43.9%	12 741	361 437	79 967	441 405
Total incl beneficial effects		**28 787**	**645 958**	**226 178**	**872 136**		**4 912**	**112 428**	**113 085**	**225 513**		**33 699**	**758 386**	**339 263**	**1 097 649**
95% uncertainty interval lower upper		26 370 30 706	586 296 701 650	217 514 234 189	804 513 935 290		3 983 6 287	100 564 128 303	106 925 116 811	209 003 242 672		31 090 36 212	696 654 817 558	328 635 347 786	1 026 986 1 164 342
% total burden (incl beneficial effects)		**10.5%**	**11.2%**	**8.4%**	**10.3%**		**2.0%**	**2.3%**	**4.0%**	**2.9%**		**6.5%**	**7.1%**	**6.2%**	**6.8%**
95% uncertainty interval lower upper		9.6% 11.2%	10.2% 12.2%	8.0% 8.7%	9.5% 11.0%		1.6% 2.5%	2.0% 2.6%	3.8% 4.2%	2.7% 3.1%		6.0% 6.9%	6.5% 7.7%	6.0% 6. 3%	6.3% 7,2%
Total excl beneficial effects		**29 248**	**650 885**	**227 416**	**878 301**		**7 592**	**136 864**	**116 914**	**253 778**		**36 840**	**787 749**	**344 331**	**1 132 079**
95% uncertainty interval lower upper		26 923 31 134	591 943 707 043	219 142 235 376	811 511 942 384		6 968 8 516	127 107 149 041	111 027 120 236	239 277 268 131		34 499 38 925	728 402 846 395	333 509 352 766	1 062 852 1 197 765
% total burden (excl beneficial effects)		**10.7%**	**11.3%**	**8.4%**	**10.4%**		**3.1%**	**2.8%**	**4.2%**	**3.3%**		**7.1%**	**7.4%**	**6.2%**	**7.0%**
95% uncertainty interval lower upper		9.8% 11.4%	10.3% 12.3%	8.1% 8.7%	9.6% 11.1%		2.8% 3.4%	2.6% 3.0%	4.0% 4.3%	3.1% 3.5%		6.6% 7.5%	6.8% 7.9%	6.0% 6.4%	6.6% 7.4%

PAFs = population attributable fractions; YLLs = years of life lost; YLDS = years lived with disability; DALYs = disability adjusted life years

4. Discussion

Alcohol abuse results in a considerable health burden in SA. Despite assuming that about 50% of men and 70% of women do not drink any alcohol, alcohol accounts for 7.0% of all DALYs in SA. Alcohol harm ranked third in terms of percentages of total DALYs for the 17 risk factors included in the SA CRA study. If the beneficial effects of alcohol are included, then alcohol accounts for 6.5% of total deaths and 6.8% of total DALYs. While the ranking against other risk factors remains the same for the percentage total DALYs, alcohol ranks sixth when the beneficial effects are included and not fourth in terms of percentage total deaths.

In 2000, 3.2% (1.8 million) of global deaths and 4.0% (58.3 million) of global DALYs were attributed to alcohol exposure. The DALY burden for high-mortality developing subregions (including AFR-E) was estimated to be 1.6% of total DALYs - compared to 9.2% for developed regions. The extent of the SA burden is more similar to the experience in developed countries than to that in high-mortality developing regions. This is largely accounted for by the high alcohol-related injury burden in SA. The WHO global CRA study estimated that 28% of the unintentional and 12% of the intentional injury burden was attributable to alcohol (Rehm *et al*. (2004). In SA these figures are 20.2% for unintentional and 40.9% for intentional injuries.

There is a need for local epidemiological data on the contribution of alcohol to poor health outcomes. In particular, data are needed on the association between alcohol consumption and increased risk of HIV/AIDS which was not quantified in this study due to a lack of data. A critical assumption in this analysis has been the use of AFR-E sub-region consumption prevalence data from the global CRA study, (Rehm *et al*. (2004), rather than available prevalence data for SA. This was done because the prevalence data did not match production figures as a result of the high prevalence of reported abstinence. Given the high estimated burden, it is clear that there is an urgent need to improve the population based data to reliably monitor the use of alcohol. Furthermore, as a large part of the estimated burden of alcohol abuse results from injury which emphasises the need to ensure good quality alcohol-related data are collected in the mortuary surveillance system. However, the narrow uncertainty band for these estimates does suggest that the results of our study are robust.

Many negative effects related to alcohol, including social and economic consequences, are not captured in this analysis. Room *et al.* (2003) have shown that the costs of these negative consequences exceed direct health costs, highlighting the need for a public health response to this risk factor. A review of policy relevant strategies to deal with alcohol harm reveals that to date, alcohol interventions have been fragmented across different departments and levels of government, and are poorly distributed (Parry, 2005a). There is no single strategy for reducing the social, economic and health burden associated with alcohol misuse. According to Parry *et al.* (2003), multi-level interventions are required to foster the responsible development of the alcohol industry on the one hand and simultaneously reduce the burden

imposed by alcohol on the other. Taking the high burden of alcohol-related problems, insufficient revenue to cover social costs associated with alcohol misuse, and the relatively low real price of alcohol into consideration, it is recommended that a moderate real increase in excise taxes on all alcoholic beverages be levied. There is compelling evidence that young drinkers are especially responsive to price (Grossman *et al.,* 1995) and that taxes contain moderate and heavy drinking and control the level of alcohol-related problems in developing countries (Edwards *et al.,* 1994).

Parry and Bennetts (1998) identified a number of individual and population-based strategies to address alcohol misuse in SA. This list incorporates most of the WHO-recommended short-term alcohol intervention strategies found to be effective in a review by Barbor *et al*. (2003) and an assessment of the feasibility of their implementation in SA (Table 8). Strategies having proven effectiveness include regulating physical availability of alcohol, drinking/driving counter-measures, and brief interventions for hazardous drinkers. (A brief intervention for alcohol problems involves a structured motivational interviewing technique aimed at enhancing motivation to change (Barbor and Higgins-Biddle, 2001; Kaner *et al.,* 2007).

According to Parry (2005b) strategies which also need to be considered within the SA context include workplace interventions, broad-based community development initiatives, and specific interventions aimed at drunken pedestrians. There should also be specific programmes directed at pregnant women and drunk drivers (Parry *et al.,* 2005). Various product restrictions should also be implemented, such as restricting the size of beer containers and stopping 'papsakke' (wine in plastic bags). There should also be increased restrictions on alcohol marketing and increased alcohol counter-advertising.

It is of critical importance to exercise constraint when consuming alcohol. Certain groups, such as pregnant women or machine operators, should abstain from alcohol use, and motor vehicle drivers should avoid consuming alcohol. Van Heerden and Parry (2001) have called for sensible drinking and the SA Department of Health's Food-Based Dietary Guidelines (2004) recommends sensible drinking or 'low-risk drinking' as: "for those who drink - no more than four units of alcohol per day for men and two per day for women, with at least two alcohol-free days per week". Public health experts question the appropriateness of these so-called weekly 'low-risk' maximums for sensible alcohol consumption (Parry, 2002). New Canadian guidelines stipulate fourteen standard drinks per week for men and nine for women, with a maximum of two drinks per day (Bondy *et al.,*1999).

Table 8: Relevance to SA of strategies indicated by Barbor, *et al*. (2003) as having proven effectiveness

Specific strategy	Effectiveness	Cost to implement	Target group	Application in SA
Regulating physical activity				
Changes in minimum purchasing age	+++	Low	B	Not feasible at present; rather enforce existing limits.
Government monopoly on retail sales	+++	Low	A	Not feasible to reintroduce this.
Restrictions on hours/days of sale	++	Low	A	Only feasible if enforced.
Outlet density restrictions	++	Low	A	Need to regulate the market first.
Alcohol taxation				
Increase excise taxes on alcohol	+++	Low	A	Government is moving in the right direction.
Drinking/driving countermeasures				
Sobriety check-points	++	Moderate	A	Should consider increasing random breath testing.
Lowered BAC limits	+++	Low	A	Current efforts should focus on enforcing existing limits.
Administrative licence suspension	++	Moderate	C	Useful, given the over-burdened courts in SA.
Graduated licensing for novice drivers	++	Low	B	Implementation would be very feasible in SA.
Brief interventions				
Brief interventions for hazardous drivers	++	Low	B	Good option, but primary practitioners need training.

Source: Parry, 2005(b)

A - general population;
B - high-risk drinkers or groups considered to be vulnerable to the effect of alcohol;
C - persons already manifesting harmful drinking and alcohol dependence.
++ = moderate; +++ = high

5. Conclusion and recommendations

Despite the fact that many South Africans do not drink, alcohol abuse results in a considerable burden of disease in SA. The National Liquor Act of 2003 (Republic of SA, 2004) aims to promote a sustainable liquor industry, and encourages responsible drinking to reduce the social and economic costs of alcohol abuse. Focus should now shift from legislation and regulation to making resources available for implementing intervention strategies. These should include a coherent liquor outlet policy, increasing random breath analysis of drivers, brief interventions, and other forms of treatment for high-risk and hazardous drinkers, as well as training and accreditation of treatment and prevention programmes. Changing the pattern of drinking in South Africa is essential if the alcohol-related burden is to be reduced.

A co-ordinated national intervention strategy - ideally a National Plan with provincial components that include civil society - is required especially given the linkages of alcohol to other national priorities such as crime and violence, RTIs and HIV/AIDS. An adequate information base should underpin the implementation of a national alcohol strategy and enable monitoring and evaluation.

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