

2 Methods

2.1 Methods of quantification of drug caused morbidity and mortality

2.1.1 Aetiological fractions

An *aetiological fraction*—also known as an attributable proportion or attributable risk—is a form of indirect quantification of morbidity and mortality due to a specified risk factor. In this case the risk factor is the consumption of tobacco, alcohol or an illicit drug. Indirect methods involve the estimation of a probability measure of the likelihood of causation by the risk factor which is then applied to the total number of deaths, illnesses or injuries resulting from a specific cause. The distinguishing characteristic of indirect methods is that individual risk factor-caused cases are not identified. For example, if there is a probability of 0.22 that a case of low birthweight is caused by smoking, then the product of this probability (the aetiological fraction) and the total number of low-birthweight babies in a population gives an estimate of the number of low-birthweight cases attributable to smoking.

The aetiological fractions used in this report were developed using the methodology outlined by English et al. (1995). There are two major sources of aetiological fractions for drug-caused illnesses and injuries. First, the fractions can be estimated directly from a series of cases where each case can be identified as caused or not caused by the drug in question. For example, from a representative series of fire deaths subjected to review, an estimated 17% were caused by cigarette smoking. Thus, an aetiological fraction of 17% can be applied to all fire deaths in the population to estimate the number attributable to cigarette smoking.

The second, and more common, source of aetiological fractions is from studies of the comparative rates of death, illness or injury in groups of people exposed and not exposed, or exposed at varying levels, to the drug in question. English et al. present the following formulae for the fractions in two such cases. Each formula uses the ratio of the incidence rate of the condition among those exposed to the drug to the incidence rate of the condition among those not exposed, designated *RR*.

The first case applies where we have an estimate of the proportion of the total population exposed to the risk factor. In this case the formula for the aetiological fraction among those exposed to the risk factor is

$$F_r = \frac{(RR - 1)}{RR} \quad (1)$$

and among the total population is

$$F_a = \frac{p_e (RR - 1)}{p_e (RR - 1) + 1} \quad (2)$$

where p_e is the proportion of the total population exposed to the risk factor.

This can be extended to the situation where the factor has several categories. If, say, the factor has k categories, then the partial aetiological fraction for category i ($1 \leq i \leq k$) among those exposed to the risk factor is

$$F_{ri} = \frac{(RR_i - 1)}{RR_i} \quad (3)$$

and among the total population is

$$F_{ai} = \frac{p_i (RR_i - 1)}{\sum_{j=0}^k p_j (RR_j - 1) + 1} \quad (4)$$

where $i = 0$ is the baseline (non-exposed) category, p_i is the prevalence of the i th category of exposure in the total population and RR_i is the rate ratio for the i th category relative to the baseline category.

These fractions can be combined into a single fraction for all categories of exposure relative to the baseline category in the following formula:

$$F_a = \frac{\sum_{i=1}^k p_i (RR_i - 1)}{\sum_{i=0}^k p_i (RR_i - 1) + 1} \quad (5)$$

The second case is where we have an estimate of the prevalence of exposure to the factor among cases of the disease, rather than among the total population. In this case the aetiological fraction for the general population is

$$F_a = \frac{p_c (RR - 1)}{RR} \quad (6)$$

where p_c is the prevalence of exposure among cases of the disease.

In some cases, where the 'risk' factor has a protective effect against a specific disease, the aetiological fraction can take negative values. We followed Holman et al. (1990) in interpreting this as allowing an estimate of the number of prevented cases.

In some cases we calculated pooled relative risk estimates from a number of studies. In these cases we followed English et al. (1995) in using an estimate based on precision-based weighting. The formula for the pooled estimate of the relative risk from N studies using precision based weighting is as follows:

$$RR = \exp \left(\frac{\sum_{i=1}^N W_i \ln(RR_i)}{\sum_{i=1}^N W_i} \right) \quad (7)$$

where $W_i = 1/Var(\ln(RR))$.

A 95% confidence interval around the pooled estimate is

$$\left(\exp \left(\ln(RR) - 1.96 / \sum W_i \right), \exp \left(\ln(RR) + 1.96 / \sum W_i \right) \right) \quad (8)$$

A full discussion of pooling multiple study results is beyond the scope of this report. Such a discussion, along with details of estimates of $Var(\ln(RR))$, can be found in the report of English et al. (1995, pp. 25–38).

2.1.2 Measures of mortality

This report uses two measures of mortality: the number of people who die in Australia from a specific cause; and the potential years of life lost (PYLL) as a result of each cause. Both these measures are derived from the Institute's mortality database. The data are compiled on a calendar-year basis, the most recent year being 1998.

The number of deaths—the National Mortality Database

The National Mortality Database comprises data on all registered deaths in Australia. The data are collected by the State and Territory Registrars of Births, Deaths and Marriages, processed by the Australian Bureau of Statistics, and provided to the Institute by the Bureau. Registration of deaths is a legal requirement in Australia and is virtually complete; with the exception of deaths of foreign diplomatic personnel, all deaths that occur in Australia are within the scope of the Institute's mortality collection.

The mortality data held by the Institute for the period covered by this report are coded according to the World Health Organization's ninth revision of the International Classification of Diseases. The ICD-9 aims to derive a single cause of death based on strict rules for determining the underlying cause from the sequence of events leading to death. This single underlying cause of death is used as the basis for applying the aetiological fraction in this report.

Potential years of life lost

Potential years of life lost, or PYLL, is an alternative to a simple count of deaths as a measure of mortality. It provides a measure of the time lost because of premature mortality. It can be calculated in two ways: by choosing an arbitrary limit to life, in which case the PYLL is the difference between this limit and the actual age at death; or by equating the PYLL with a measure of average community life expectancy at the actual age of death. The PYLL presented in this report are calculated using the latter method.

The usual source of average life expectancy is a life table. It is possible to derive Australian PYLL estimates using a life table based on the actual population mortality experience in the year under study. But this leads to variation in the PYLL estimates over time and between different study populations because of differences in the specific life tables. For example, the death of a woman aged 30 from a drug-related cause in 1988 contributes around two years less to the drug-related mortality burden, measured in terms of PYLL, than if it had occurred in 1998 purely because of changes in life expectancy over that period.

An alternative approach is to derive the average life expectancy from a standard life table. This has the disadvantage that the PYLL estimates do not relate exactly to the population under study, but it has the advantage that each death in a specific age-sex group contributes the same amount to the measure of mortality irrespective of the year of death. The PYLL presented in this report are based on the life table used in the Australian Burden of Disease study to derive years of life lost (YLL) as a result of premature mortality (Mathers et al. 1999). This is a life table of projected cohort life expectancies for Australians alive in 1996. Unlike the usually quoted 'period' life expectancies (ABS 1999), which synthesise the currently observed mortality patterns across all age groups in the population, cohort life expectancies use projected trends in mortality rates to estimate the average life expectancies likely to be achieved by people currently alive.

The cohort life table gives estimates of life expectancy at birth as 85.69 years for women and 81.45 years for men. By comparison, the ABS life table representing the Australian mortality

experience for 1996 to 1998 gives a life expectancy at birth of 81.52 years for women and 75.86 years for men. Nevertheless, the specific choice of life table makes little difference to the results presented in this report in terms of comparisons between specific causes of death and between different risk factors.

Both English et al. (1995) and Holman et al. (1990) used a method of calculating PYLL due to a specific condition that adjusted the life expectancy for removal of that condition. We have followed the practice of the Australian Burden of Disease study by allocating the age-specific population average life expectancy to all deaths, regardless of the cause of death. This simplification allows easier comparison of PYLL between conditions and risk factors, and it allows the PYLL estimates to be added together across conditions. It does, however, mean that reduction of a risk factor cannot be taken as leading to a proportional reduction in PYLL. Those people saved from death due to the risk factor reduction would still remain at risk of death from other conditions, so they would be subject to a modified life expectancy that would be less than the average life expectancy used in the PYLL calculations.

The method used by English et al. allocated the PYLL to the age at which a person would have lived had they not died. We followed the Australian Burden of Disease Study in allocating the PYLL to the age at which death occurred. We also followed that study in applying a 3% time discount rate to years of life lost in the future to estimate the net present value of PYLL. This is standard practice in economic analysis, and, among other things, it avoids the tendency of PYLL calculations to over-emphasise deaths at young ages. The use of 3% a year as the discount rate follows the recommendation of the US Panel on Cost-Effectiveness in Health and Medicine (Gold et al. 1996). The discounted mean life expectancy at each age was calculated as

$$PYLL = \frac{(1 - e^{(-0.03L)})}{0.03} \quad (9)$$

where L is the corresponding undiscounted mean life expectancy.

2.1.3 Measures of morbidity

This report uses two measures of morbidity: the number of hospital separations attributable to a specific principal diagnosis; and the number of patient days attributable to a specific principal diagnosis. Both these measures are derived from the Institute's hospital morbidity database, which is compiled on a financial-year basis.

These two measures do not provide a complete picture of morbidity in the community because they do not cover morbidity where no medical care was sought or where medical care was provided outside the hospital system—by, for example, general practitioners. However, more complete national measures of cause-specific morbidity are not available in Australia at present.

Hospital separations—the National Hospital Morbidity Database

The National Hospital Morbidity Database is a collection of confidentialised records for admitted hospital patients provided to the Institute by the State and Territory health departments. Data on patients admitted in one year but separated (discharged, transferred or died) in another are included in the database for the year in which the separation occurred.

The database includes data from public acute hospitals and Department of Veterans' Affairs hospitals, public psychiatric hospitals, private acute and psychiatric hospitals, and private free-standing day hospital facilities. Exceptions in the public sector are public hospitals not

within the jurisdiction of a State or Territory health authority or the Department of Veterans' Affairs—for example, hospitals operated by the Department of Defence and hospitals located in offshore territories. In addition, in 1997–98, public hospital data were not available for a mothercraft hospital in the Australian Capital Territory, one small 'outpatient clinic' in Queensland, and for most separations from three small district public hospitals in Tasmania.

In the private sector, about 4,500 hospital separations were not included for New South Wales private hospitals, and separations were not available for two private free-standing day hospital facilities and one other private hospital in Tasmania, private free-standing day hospital facilities in the Australian Capital Territory, and the private hospital in the Northern Territory.

A person can have had multiple stays in hospital in one year, but it is not possible to identify such people on the database. Thus, a count of hospital separations will be an accurate guide to the number of episodes of hospital care in a year but will generally be an overestimate of the number of people treated in hospital in a year.

English et al. (1995) recommended that hospital separations for conditions relating to complications of pregnancy and birth be excluded if the birth occurred during the hospital stay. This is because the separation would probably have taken place even if the complication had been absent, so to include it would lead to an overestimate of the total attributable separations. We have followed that recommendation in this report.

2.2 Conditions included in this report

The conditions included in this report are those identified by English et al. (1995) as having a causal relationship with alcohol, tobacco or illicit drugs (Tables 2.1 to 2.3). To these we have added conditions identified by the National Health and Medical Research Council as related to environmental tobacco smoke (NHMRC 1997).

The mortality data are coded to the underlying cause of death, which is defined as the disease or injury that initiated the train of morbid events leading directly to death. These are coded according to version 9 of the International Classification of Diseases. Accidental and violent deaths are classified according to the external cause—that is, to the circumstances of the accident or violence that produced the fatal injury—rather than to the nature of the injury. These are coded using the ICD-9 external cause codes (denoted by the letter 'E' at the start of the code number).

Hospital separations and patient days are coded in most cases to the principal diagnosis, which is the diagnosis established after study to be chiefly responsible for occasioning the patient's episode of care in hospital. It does not include codes for external causes so in some cases—falls related to alcohol, for example—an external cause coding is used instead. The hospital data are coded using the clinical modification of the ICD-9 codes (ICD-9-CM), but the differences between ICD-9 and ICD-9-CM are small for the conditions included in this report and may be disregarded.

One consequence of this use of the principal diagnosis and the underlying cause of death is that the analysis takes no account of other conditions recorded on the death certificate or the hospital record. This is a limitation inherent in using the aetiological fractions derived by English et al. because the fractions were estimated on the basis of primary diagnosis and underlying cause of death.

In most cases the conditions included in the analyses for deaths and for hospital separations are coded in the same way. For some conditions, however, English et al. used an external

cause code when analysing mortality data but a principal diagnosis code for the corresponding condition when analysing data on hospital separations. We followed this practice for these cases, which are identified by footnotes in Tables 2.1 to 2.3. This sometimes leads to the conditions for each analysis not being directly comparable. For example, the data for deaths due to psychostimulant poisoning relate only to accidental poisoning, while the corresponding hospital separations also relate to deliberate poisoning. This also leads to some conditions being analysed differently for different risk factors. For example, suicide and self-inflicted injury are included in both the mortality and morbidity analyses for alcohol but only in the mortality analyses for illicit drugs. In the latter case, the self-inflicted injury is classified under the principal diagnosis according to the drug used. For example, attempted suicides using opiates are counted under opiate poisoning.

English et al. did not distinguish between types of hepatitis other than types A and B. Instead, they calculated a fraction for all non-A, non-B cases pooled together. Thus, although some forms of non-A, non-B hepatitis (such as type E) are not transmitted by injecting drug use, we followed their practice and pooled all non-A, non-B hepatitis into one group. The aetiological fraction for this condition identifies the proportion of the pooled group that is attributable to injecting drug use.

Table 2.1: Causes of death and principal diagnoses identified as alcohol-related conditions

Condition	ICD-9 code
Cancer	
Oropharyngeal cancer	141, 143–146, 148–149
Oesophageal cancer	150
Liver cancer	155
Laryngeal cancer	161
Female breast cancer	174
Alcoholism and alcoholic liver cirrhosis	
Alcoholic psychosis	291
Alcohol dependence/abuse	303, 305.0
Alcoholic liver cirrhosis	571.0–571.3
Road injuries	
	E810–E819
Other	
Epilepsy	345
Alcoholic poly-neuropathy	357.5
Hypertension	401–405
Ischaemic heart disease	410–414
Alcoholic cardiomyopathy	425.5
Supraventricular cardiac dysrhythmias	427.0, 427.2, 427.3
Heart failure	428–429
Stroke	430–438
Oesophageal varices	456.0–456.2
Gastro-oesophageal haemorrhage	530.7
Alcoholic gastritis	535.3
Unspecified liver cirrhosis	571.5–571.9
Cholelithiasis	574
Pancreatitis, acute and chronic	577.0, 577.1
Low birthweight	656.5, 764, 765
Psoriasis	696.1
Ethanol/methanol toxicity	980.0 ^(a) , 980.1 ^(a)
Alcoholic beverage poisoning	E860.0 ^(b)
Other ethanol and methanol poisoning	E860.1, E860.2 ^(b)
Fall injuries	E880–E888
Fire injuries	E890–E899
Drowning	E910
Aspiration	E911
Occupational and machine injuries	E919, E920
Suicide and self-inflicted injury	E950–E959
Assault	E960, E965, E966, E968, E969
Child abuse	E967

(a) Diagnosis code used only for calculating numbers of drug-caused hospital separations and patient days.

(b) External cause code used only for calculating numbers of drug-caused deaths and PYLL.

Source: English et al. (1995).

Table 2.2: Causes of death and principal diagnoses identified as tobacco-related conditions

Condition	ICD-9 code
Cancer	
Oropharyngeal cancer	141,143–146, 148–149
Oesophageal cancer	150
Stomach cancer	151
Anal cancer	154.2, 154.3
Pancreatic cancer	157
Laryngeal cancer	161
Lung cancer	162
Endometrial cancer	179, 182
Cervical cancer	180, 233.10
Vulvar cancer	184.4
Penile cancer	187.1–187.4
Bladder cancer	188
Renal parenchymal cancer	189.0
Renal pelvic cancer	189.1
Respiratory carcinoma in situ	231
Ischaemic heart disease	
Ischaemic heart disease	410–414
Chronic obstructive pulmonary disease	
Chronic obstructive pulmonary disease	490–492, 496
Other direct effect of smoking	
Tobacco abuse	305.1
Parkinson's disease	332
Pulmonary circulation disease	415.0, 416–417
Cardiac dysrhythmias ^(a)	427
Heart failure ^(a)	428–429
Stroke	430–438
Atherosclerosis	440–448
Pneumonia	480–487
Peptic ulcer	531–534
Crohn's disease	555
Ulcerative colitis	556
Ectopic pregnancy	633, 761.4
Spontaneous abortion	634, 761.8
Antepartum haemorrhage	640, 641, 762.0, 762.1
Hypertension in pregnancy	642, 760.0
Low birthweight	656.5, 764, 765
Premature rupture of membranes	658.1–658.2, 761.1

(continued)

Table 2.2 (continued): Causes of death and principal diagnoses identified as tobacco-related conditions

Condition	ICD-9 codes
SIDS (and smoking during pregnancy)	798.0
Fire injuries	E890–E899
Environmental tobacco smoke	
Lung cancer	162
Ischaemic heart disease	410–414
Asthma (under 15 years)	493
Lower respiratory illness (under 18 months)	464, 466, 480–486, 487 and 490
SIDS (and post natal smoking)	798.0

(a) The majority of heart failure and cardiac dysrhythmias are secondary to ischaemic heart disease.

Source: English et al. (1995).

Table 2.3: Causes of death and principal diagnoses identified as illicit drug-related conditions

Condition	ICD-9 code
Directly attributable to opiates	
Opiate dependence	304.0, 304.7
Opiate abuse	305.5
Opiate poisoning	965.00, 965.01, 965.02 ^(a)
Accidental opiate poisoning	E850.0, E850.1 ^(b)
Antepartum haemorrhage due to opiates	640, 641
Low birthweight due to opiates	764, 765, 656.5
Directly attributable to other illicit drugs	
Cannabis dependence	304.3
Cannabis abuse	305.2
Amphetamine dependence	304.4
Amphetamine abuse	305.7
Cocaine dependence	304.2
Cocaine abuse	305.6
Psychostimulant poisoning	969.7 ^(a)
Accidental poison by psychostimulants	E854.2 ^(b)
Hallucinogen dependence	304.5
Hallucinogen abuse	305.3
Hallucinogen poisoning	969.6 ^(a)
Other psychotropic drug poisoning	969.8, 969.9 ^(a)
Accidental poisoning by hallucinogens	E854.1 ^(b)
Anabolic steroid poisoning	962.1 ^(a)
Antepartum haemorrhage due to cocaine	640, 641
Low birthweight due to cocaine	764, 765, 656.5
Attributable to unclassifiable injecting drug use	
Hepatitis B	070.2, 070.3
Hepatitis non A, non B	070.4, 070.5
AIDS	279.1, 042–044
Infective endocarditis	421
Other related causes	
Drug psychoses	292
Maternal drug dependence	648.3
Newborn drug toxicity	760.7, 779.5
Road injuries	E810–E819
Suicide	E950–E959 ^(b)

(a) Diagnosis code used only for calculating numbers of drug-caused hospital separations and patient days.

(b) External cause code used only for calculating numbers of drug-caused deaths and PYLL.

Source: English et al. (1995).

2.3 Aetiological fractions selected for revision

The estimated values of the fractions for most conditions depend on both the prevalence of the risk factor and on the risk ratio associated with the specific condition. In all cases where data were available, the fractions have been revised to reflect the most recent estimation of risk factor prevalence. In addition, some fractions have been revised to reflect the effect of recent research results on our knowledge of the risk ratios.

English et al. (1995) used estimates of the current prevalence of tobacco smoking and alcohol consumption in their calculation of aetiological fractions for tobacco and alcohol. But many conditions have a long time lag between exposure to tobacco smoke or alcohol consumption and their associated ill-effects—in the case of cancers it may be many decades. So for these conditions estimates of current prevalence are not helpful in understanding the current associated disease burden. We followed the Australian Burden of Disease Study in using the method proposed by Peto et al. (1992) to adjust for this time lag for tobacco smoke. In the case of alcohol, however, there is no equivalent of the method used by Peto et al. to adjust for this time lag so we followed English et al. in using the current prevalence estimates for alcohol consumption in the calculation of the alcohol fractions. The prevalence of alcohol consumption, particularly of heavy drinking, has declined in recent decades, and it is thus likely that these methods underestimate the true aetiological fractions for some current health outcomes attributable to alcohol consumption.

Given the time and the resources available for this study, it was impossible to examine in detail examination all the conditions with which the three risk factors have been linked. Instead, we selected for detailed study those conditions that made the largest contribution to mortality and morbidity, as identified by English et al., and for which there was clear epidemiological evidence of a need to revise the risk ratios. These conditions are listed in Table 2.4. We used the risk ratios or case study estimates identified by English et al. or by the National Health and Medical Research Council environmental smoking study (NHMRC 1997) for the remaining fraction estimates.

Table 2.4: Conditions selected for detailed study and risk ratio revision

Cause/condition	ICD-9 code
Alcohol	
Female breast cancer	174
Stroke	430–438
Road injuries	E810–E819
Fall injuries	E880–E888
Tobacco	
Cervical cancer	180, 233.10
Peptic ulcer	531–534
Illicit drugs	
Road injuries	E810–E819

2.4 Literature search

We followed English et al. (1995) in searching for articles from the CD-ROM MEDLINE National Library of Medicine 1988–98 database supplied by Silver Platter. As with English et al., in all instances searches were restricted to articles published in English. The overwhelming majority of the relevant studies would be published in English, so few studies of relevance would have been excluded. Furthermore, the restriction to literature published in English increases the relevance of the studies to the Australian population. The search strategies were applied to conditions identified by English et al.

Much of the literature relating to illicit drugs in Australia is unlikely to be indexed on MEDLINE. It is more likely to be published in technical reports and monographs or in more specific CD-ROM collections. Our search strategy included the libraries of the National Drug and Alcohol Research Centre, the National Centre for Research into Prevention of Drug Addiction, and the Alcohol and Drugs Council of Australia. It also included the Victorian Anti-Cancer Council and the Centre for Behavioural Research in Cancer, the Australian Institute of Criminology in Canberra, the Australian Institute of Health and Welfare Injury Surveillance Unit, and the Australian Transport Safety Bureau for research into the relationship between alcohol consumption and road accidents.

The same methodology as that used for MEDLINE was also used to search specialist CD-ROM databases such as the Australian Medical Index (AMI), the National Library of Australia (1968–1998b), the Australian Public Affairs Information Service—Health (APAIS—Health), the National Library of Australia (1978–1998), the Aboriginal and Torres Strait Islander Health Bibliography (ATSIhealth), The School of Health Studies and Edith Cowan University (1988–1998), AusportMed, the National Sports Information Centre and the Australian Sports Commission (1989–1998), the Drug Database (DRUG), the Alcohol and Other Drugs Council of Australia (1974–1998), the Health and Society Database, the Australian Institute of Family Studies (1980–1998b), the Rural and Remote Health Database (RURAL), the Australian Rural Health Research Institute and Monash University (1966–1998), the Attorney-General's Information Service (AGIS), the Attorney-General's Department (1975–1998), the Australian Federal Police Digest (AFPD), the Australian Federal Police (1991–1998), the Australian Public Affairs Information Service (APAIS), the National Library of Australia (1978–1998a), the Australian Criminology Database (CINCH), the Australian Institute of Criminology (1968–1998), the Australian Family and Society Abstracts Database (Family), Australian Institute of Family Studies (1980–1998a), and HealthSTAR, the National Library of Medicine (1997–1998).

2.5 Prevalence of exposure data

2.5.1 Alcohol data

Classification of data on alcohol consumption

The criteria used to classify data on alcohol consumption were equivalent to those used by English et al. (1995), with the exception of the names used to categorise each level of consumption. Whereas English et al. refer to alcohol intake categorised according to the National Health and Medical Research Council's criteria (abstinence, low, hazardous, and

harmful), this report refers to these equivalent levels as abstinence, low, medium and high (Table 2.5).

Table 2.5: Alcohol intake levels used in this report

Intake level	Standard drinks per day (1 standard drink = 10 grams alcohol)		
	Males	Females	Persons
Abstinence	0.00–0.25	0.00–0.25	0.00–0.25
Low	0.26–4.00	0.26–2.00	0.26–3.00
Medium	4.01–6.00	2.01–4.00	3.01–5.00
High	6.01+	4.01+	5.01+

Following English et al., we included alcohol quantities up to one-quarter of a standard drink per day in the exposure category of ‘abstinence.’ This provided tolerance for the inclusion of studies with small amounts of baseline contamination (commonly less than one drink a week or less than one drink a month). Assignment of relative risk to low, medium or high levels of exposure varied according to whether subjects were males, females or a combined group of both sexes.

For the purpose of assigning equivalents of exposure, one alcohol drink a day was taken as equivalent to 10 grams of alcohol a day, 70 grams of alcohol a week, or 300 grams or 10 ounces a month (Table 2.6). In assigning results of published literature to one or more exposure categories, a median exposure level was estimated for each result in the literature, based on the exposure interval to which the estimate of relative risk related. These were then assigned to the abstinence, low, medium and high levels of exposure according to where the median exposure level fell. Binge drinkers and ex-drinkers were generally excluded.

Table 2.6: Approximate equivalents of alcohol consumption used in this report

Intake level	Standard drinks/day	grams/day	grams/week	grams/month	ounces/month
Males					
Abstinence	0.00–0.25	0.0–2.5	0.0–17.5	0.0–75	0.0–2.5
Low	0.26–4.00	2.6–40.0	17.6–280.0	76–1200	2.6–40.0
Medium	4.01–6.00	41.0–60.0	281.0–420.0	1201–1800	41.0–60.0
High	6.01+	61+	421+	1801+	61+
Females					
Abstinence	0.00–0.25	0.0–2.5	0.0–17.5	0.0–75	0.0–2.5
Low	0.26–2.00	2.6–20.0	17.6–140.0	76–600	2.6–20.0
Medium	2.01–4.00	21.0–40.0	141.0–280.0	601–1200	21.0–40.0
High	4.01+	41+	281+	1201+	41+

For some conditions—notably falls and motor vehicle injuries—the lack of published analytical studies necessitates the use of aetiological fractions reported from clinical or blood alcohol case series data. As did English et al., in these circumstances we assumed that all attributed cases were due to medium to high drinking levels. For road injuries, blood alcohol concentration was generally measured directly or estimated with the use of a breathalyser test. In this case concentrations of over 0.05–0.10 g/100 mL and over 0.10 g/100 mL were regarded as comparable to medium and high levels of alcohol intake. Thus aetiological

fractions derived from an exposure contrast between ‘illegal’ and ‘legal’ blood alcohol concentrations may be interpreted as the proportions of road injuries that might be avoidable if alcohol exposure were reduced to within the legal limit.

Data on the prevalence of alcohol consumption

English et al. (1995) estimated the prevalence of alcohol consumption from the 1989–90 National Health Survey conducted by the Australian Bureau of Statistics and the 1989 National Heart Foundation Risk Factor Prevalence Survey. Because of limitations in each of these data sources, they devised the following method of prevalence estimation based on a combination of the two sources:

- The sex- and age-specific prevalence measures of current drinkers (at any level) were based on the results of the Risk Factor Prevalence Survey.
- The sex- and age-specific prevalence measures at particular levels of drinkers’ intake were based on the results of the 1989–90 National Health Survey.

Table 2.7 shows the estimated prevalence of alcohol consumption for Australian males and females in 1989.

Table 2.7: Prevalence of alcohol consumption among Australians, by gender, 1989

Age	Males				Females			
	Abstinence	Low	Hazardous	Harmful	Abstinence	Low	Hazardous	Harmful
18–19	0.159	0.658	0.097	0.086	0.202	0.683	0.082	0.032
20–24	0.159	0.623	0.103	0.115	0.202	0.642	0.111	0.044
25–29	0.080	0.688	0.124	0.108	0.185	0.696	0.093	0.026
30–34	0.110	0.726	0.084	0.080	0.240	0.678	0.066	0.016
35–39	0.120	0.708	0.099	0.073	0.221	0.685	0.078	0.016
40–44	0.087	0.743	0.082	0.088	0.233	0.654	0.099	0.015
45–49	0.115	0.716	0.089	0.080	0.219	0.655	0.098	0.028
50–54	0.129	0.678	0.095	0.098	0.328	0.566	0.081	0.025
55–59	0.178	0.662	0.090	0.070	0.327	0.577	0.076	0.020
60–64	0.192	0.650	0.081	0.077	0.331	0.577	0.071	0.021
65–69	0.169	0.696	0.085	0.050	0.335	0.564	0.084	0.017
70–74	0.169	0.739	0.049	0.043	0.335	0.571	0.069	0.025
75–79	0.169	0.784	0.027	0.020	0.335	0.609	0.056	0.000
80+	0.169	0.806	0.013	0.012	0.335	0.632	0.030	0.004
18+	0.126	0.699	0.092	0.084	0.247	0.646	0.085	0.023

Source: Derived from the 1989–90 ABS National Health Survey and 1989 National Heart Foundation Risk Factor Prevalence Survey.

In updating the prevalence data using more recent sources of information, a number of sources were considered, including the 1995 ABS National Health Survey, the ABS 1997 Survey of Mental Health and Wellbeing, and the Department of Health and Aged Care’s 1998 National Drug Strategy Household Survey. Although the most dated, the 1995 National Health Survey was chosen to represent the alcohol prevalence data for updating the aetiological fractions. This was because of the more specific nature of the information collected (seven categories of alcoholic drinks) and the fact that adjustments were made for the alcohol content of a variety of brands within these categories—for example, low-alcohol

beer distinguished from full-strength beer (ABS 1995b). Furthermore, the National Health Survey had a sampling frame of 23,800 households, representing about 1 in 310 of the non-institutionalised population in Australia. The sample design ensured that within each State or Territory each person had a known and equal chance of selection. Overall, completed responses were obtained from 53,751 people (Donath 1999).

However, unlike the 1989–90 National Health Survey, the 1995 Survey asked about the quantity of alcohol consumed on up to three days of the week before the interview day, whereas the 1989–90 Survey used a seven-day retrospective diary. Furthermore, the 1995 Survey asked how many days in the previous week had alcohol been consumed. This was done so as to estimate weekly consumption among those who consumed alcohol on more than three days a week, by multiplying the number of days alcohol was consumed in the previous week, dividing that by three, and then multiplying by the total consumption across the three days on which it was assessed. This relies on the implicit assumption that average consumption for the three days where it was assessed was the same as on the days for which data were not obtained. However, as is evident from the 1989–90 Survey data, for those who consume alcohol on four or more days of the week there are large differences between consumption from Monday to Thursday and consumption on Fridays, Saturdays and Sundays (Donath 1999).

Since the 1995 Survey methodology directly estimates consumption for those consuming alcohol on fewer than four days, only the estimates for those consuming alcohol on four or more days appear to be problematic. This is important. The proportion of people who drink alcohol on four or more days is substantial: in 1995 it was estimated to be 26.3% for males and 11.7% for females (Donath 1999).

In order to obtain reliable estimates using the 1995 methodology, the day of the week of interview would have to be uniformly distributed for those who drank on four or more days of the week. But it appears this was not the case. With the 1995 Survey, the data show that, for people consuming alcohol in the previous week, far more interviews were conducted on Monday, Tuesday and Wednesday (26%, 24% and 22% respectively) than on weekends (4% Saturday and 1% Sunday) (Donath 1999).

Because of these difficulties, the 1995 Survey's data on weekly consumption were reweighted so as to give equal weight to each of the days of interview. Further, because there were so few interviews on Saturdays and Sundays, there were too few outcomes for the moderate and high levels of alcohol intake to allow this to be done by five-year age groups. Therefore, the average distribution of Saturday and Sunday for broader age groups (18–34, 35–64 and 65+) was used to provide the estimates to be applied to the five-year age groups.

As Donath did, we determined the prevalence of consumption for 18–24 year olds and used this to derive aetiological fractions for both 18–19 and 20–24 year olds. This overcame the small numbers that occurred in trying to estimate prevalence for 18–19 year olds, particularly females. The resulting 1995 Survey prevalence estimates for updating the aetiological fractions are described in Table 2.8.

Comparison of the 1995 and 1998 National Drug Strategy Household Survey estimates of alcohol consumption suggests that levels of alcohol intake remained relatively constant between 1995 and 1998. We took the prevalence estimates in Table 2.8 as applying to 1998.

Table 2.8: Prevalence of alcohol consumption among Australians, by gender, 1995

Age	Males				Females			
	Abstinence	Low	Hazardous	Harmful	Abstinence	Low	Hazardous	Harmful
18–19	0.153	0.697	0.067	0.083	0.225	0.618	0.130	0.027
20–24	0.153	0.697	0.067	0.083	0.225	0.618	0.130	0.027
25–29	0.135	0.717	0.075	0.073	0.258	0.624	0.097	0.021
30–34	0.162	0.680	0.088	0.070	0.222	0.668	0.091	0.020
35–39	0.197	0.675	0.078	0.050	0.363	0.502	0.112	0.023
40–44	0.186	0.660	0.097	0.057	0.346	0.540	0.094	0.021
45–49	0.186	0.649	0.108	0.057	0.392	0.485	0.092	0.031
50–54	0.149	0.707	0.081	0.063	0.386	0.461	0.114	0.038
55–59	0.193	0.0689	0.081	0.037	0.385	0.473	0.117	0.025
60–64	0.169	0.654	0.104	0.073	0.372	0.494	0.123	0.011
65–69	0.184	0.696	0.075	0.045	0.391	0.453	0.141	0.014
70–74	0.162	0.717	0.062	0.059	0.432	0.472	0.085	0.011
75–79	0.185	0.721	0.028	0.066	0.497	0.451	0.043	0.009
80+	0.204	0.714	0.053	0.030	0.373	0.568	0.043	0.016
18+	0.176	0.679	0.083	0.063	0.310	0.560	0.108	0.022

Source: AIHW analysis of 1995 ABS National Health Survey

Prevalence data on alcohol consumption during pregnancy

English et al. (1995) provided estimates of the prevalence in Australia of alcohol consumption during pregnancy. These were based on a 1993 Survey of 6,861 pregnant women in Tasmania, undertaken by the University of Tasmania's Department of Obstetrics and Gynaecology at the Queen Alexandria Hospital. Information on alcohol intake was provided by 5,417 of the 6,861 respondents (Table 2.9).

Table 2.9: Alcohol intake during pregnancy, Tasmania, 1993

Drinks	Prevalence
None	0.787
<3 per week	0.196
3–6 per week	0.015
2–3 per day	0.0009
4+ per day	0.0004

Source: English et al. (1995).

Thus, by apportioning the prevalence observed at two to three drinks a day equally between low and hazardous drinking, the total prevalence of low consumption during pregnancy was estimated at 0.212 and of hazardous or harmful consumption at 0.001.

We used results from the more recent 1998 National Drug Strategy Household Survey to update these estimates. There is no exact correspondence between the results collected by the Survey and our categorisation of intake as low, moderate or high, although the Survey's

results can be grouped approximately into these categories. The result is an estimated prevalence of low consumption during pregnancy of 0.293 and of hazardous or harmful consumption of 0.053.

2.5.2 Data on cigarette smoking

English et al. (1995) derived the prevalence of smoking in the adult Australian population aged 18 years or more from the 1989–90 National Health Survey. They determined prevalences for never, former and current cigarette smoking. The prevalence of current cigarette smoking was described in terms of three categories according to the number of cigarettes smoked each day (one to 14, 15 to 24, and 25 or more). ‘Current smoking’ was defined as smoking at the time of interview and ‘former smoking’ as smoking at any time prior to interview.

This approach, which described as current smokers those smoking a minimum of one cigarette a day, is consistent with the studies that have uncovered the harmful effects of smoking, whereby regular or current smoking is quantified in terms of a minimum of one cigarette a day (Doll 1998). The 1995 National Health Survey defined a regular smoker as someone smoking a minimum of one cigarette a day (ABS 1995b). A current smoker who smoked less than one cigarette a day was defined as an occasional smoker. For the purpose of quantifying smoking status, however, only regular smokers (one or more a day) were counted as smokers; occasional smokers were classified as ‘never smoked’. The ex-smokers were those who indicated smoking at some time but who were not occasional or regular smokers at the time of interview.

Data on cigarette smoking prevalence

The 1995 National Health Survey was not considered as a replacement source for updated prevalence data because unlike the 1989–90 Survey, it did not assess the number of cigarettes smoked. Two other recent sources of prevalence data on smoking are the 1995 Australian survey by the Victorian Anti-Cancer Council (Hill et al. 1998—see Tables 2.10 and 2.11) and the more recent 1998 National Drug Strategy Household Survey by the Department of Health and Aged Care.

The Victorian Anti-Cancer Council Survey results are described below. It should be noted that smoking prevalence is calculated only to age 70 or more years because age was restricted to this level in the original data collection. This contrasts with the work of English et al. (1995) who had determined smoking prevalence by five-year age groups and to age 80 and over. Furthermore, for the purpose of analysis the small number of pipe and cigar users were excluded from the estimates derived from the Victorian Anti-Cancer Council Survey. As just described above, only regular smokers (one or more a day) were counted as smokers; occasional smokers were classified as having never smoked.

While the National Drug Household Survey was the more recent and had the ability to be analysed more completely by five-year age groups, the Victorian Anti-Cancer Council Survey’s estimates of prevalence by the Centre for Behavioural Research in Cancer were adopted for the revision of the aetiological fractions. This was because the Anti-Cancer Council Survey data contained cigarette consumption data that matched the work of English et al.

Table 2.10: Smoking prevalence among Australian males, 1995

Age	Never smoked	Ex-smoker	Current smokers cigarettes per day			
			All smokers	1–14	15–24	25+
16–17	75.5	4.3	20.2	9.6	5.3	5.3
18–19	58.5	6.2	35.4	26.2	9.2	0.0
20–24	56.2	12.4	31.3	16.6	9.2	5.5
25–29	52.2	18.3	29.5	16.3	7.2	6.0
30–34	46.7	23.0	30.2	10.3	6.5	13.4
35–39	47.1	27.8	25.1	6.2	7.2	11.7
40–44	49.3	29.3	21.4	6.0	5.4	10.0
45–49	45.2	33.2	21.6	4.6	5.8	11.2
50–54	40.0	34.9	25.1	10.2	6.2	8.7
55–59	37.1	39.4	23.4	7.4	5.7	10.3
60–64	42.4	45.8	11.9	4.0	2.8	5.1
65–69	31.8	56.3	12.0	4.2	4.7	3.1
70+	37.3	54.5	8.1	3.6	1.2	3.3
Total 16+	45.9	32.1	22.0	8.5	5.6	7.9

Source: Hill et al. (1998).

Table 2.11: Smoking prevalence among Australian females, 1995

Age	Never smoked	Ex-smoker	Current smokers cigarettes per day			
			All smokers	1–14	15–24	25+
16–17	70.3	9.5	20.3	13.5	5.4	1.4
18–19	47.6	14.3	38.1	25.0	9.5	3.6
20–24	53.7	15.3	31.0	14.0	9.1	7.9
25–29	51.6	16.9	31.5	16.9	8.6	6.0
30–34	45.7	23.7	30.6	13.9	8.2	8.5
35–39	52.1	24.8	23.1	10.0	7.5	5.6
40–44	52.0	23.2	24.7	8.1	8.1	8.5
45–49	62.3	24.1	13.6	3.6	3.6	6.4
50–54	55.5	23.5	21.0	7.0	7.5	6.5
55–59	59.4	24.0	16.6	6.3	5.7	4.6
60–64	67.8	18.0	14.1	5.4	6.3	2.4
65–69	63.3	24.3	12.4	6.5	0.0	5.9
70+	67.3	26.0	6.8	3.5	1.2	2.1
Total 16+	56.9	21.7	21.4	9.5	6.2	5.7

Source: Hill et al. (1998).

Comparison of the 1995 and 1998 National Drug Strategy Household Survey estimates of the prevalence of tobacco consumption suggests that levels of tobacco use remained relatively

constant over the period. We took the prevalence estimates in Tables 2.10 and 2.11 as applying to 1998.

Prevalence data on cigarette smoking during pregnancy

English et al. (1995) provided estimates of the prevalence in Australia of cigarette smoking during pregnancy. These were based on a 1993 survey of 6,861 pregnant women in Tasmania, undertaken by the University of Tasmania’s Department of Obstetrics and Gynaecology at the Queen Alexandria Hospital. Information on cigarette smoking was provided by 5,428 of the 6,861 respondents (Table 2.12).

Table 2.12: Cigarette smoking during pregnancy, Tasmania, 1993

Smoking status		Current smokers cigarettes: per day		
Non-smoker	Current smoker	1–9	10–20	21+
0.71	0.29	0.13	0.12	0.04

Source: English et al. (1995).

We used results from the 1998 National Drug Strategy Household Survey to update these estimates. The estimated proportion of women who were either occasional or regular smokers during pregnancy in 1998 was 0.279.

Cigarette smoking and exposure to environmental tobacco smoke among infants and children aged less than 16 years

Revised estimates of exposure to environmental tobacco smoke were provided by the Anti-Cancer Council of Victoria’s Centre for Behavioural Research in Cancer; they were based on unpublished data from a 1997 survey conducted only in Victoria.

For households with children aged less than 2 years it was estimated that, in 1997, 24.4% of such households would have had a female smoker (assumed to be the mother). Of the households with female smokers, 51.6% were households in which there was no restriction or ban on smoking indoors. Overall, this results in an estimate of 12.6% of households containing a child aged less than 2 years having a female smoker who was not restricted to smoking outdoors.

Similarly, for children aged less than 16 years and for whom there was a female smoker (assumed to be the mother) of 10 or more cigarettes a day in the household, it was estimated that the prevalence of exposure was 21.3%. Again, of such households, 25.3% were households in which there was no restriction or ban placed on smoking indoors. Overall, this results in an estimate of 5.4% of households containing a child aged less than 16 years having a female smoker who was not restricted to smoking outdoors.

Cigarette smoking and exposure to environmental tobacco smoke among non-smoking spouses

The NHMRC’s report on the health effects of passive smoking (NHMRC 1997) used the proportion of non-smokers with a spouse who was a current smoker as a measure of exposure to environmental tobacco smoke. That report derived the prevalence measure from the 1989–90 ABS National Health Survey. We calculated the corresponding estimates from the 1995 National Health Survey. Details of these estimates are presented in Section 4.2.3.