The quantification of drug-caused mortality and morbidity in Australia, 1998

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Summary

The aetiological fraction methodology and the associated fraction estimates enable estimation of the proportion of cases of an illness or injury that can be attributed to a risk factor. This report presents aetiological fraction estimates attributing deaths and hospital separations resulting from a range of specific illnesses or injuries to tobacco, alcohol and illicit drugs. The fractions represent a revision of the fractions originally presented by Holman et al. (1990) and later revised by English et al. (1995). Also presented here are estimates of 1998 mortality and 1997–98 hospital separations attributable to alcohol, tobacco and illicit drugs based on the revised fractions.

Deaths attributable to alcohol, tobacco and illicit drugs

In 1998 an estimated 19,019 people died in Australia as a result of tobacco smoking. A further 1,023 deaths can be attributed to illicit drugs. For tobacco smoking, the majority of deaths (14,799) occurred at ages 65 and over. However, because of the time lag between exposure to tobacco smoke and the onset of many diseases, particularly cancer and chronic obstructive pulmonary disease, many of these deaths represent the result of tobacco smoking at a much earlier age. The majority of deaths attributed to illicit drugs (649) occurred between the ages of 15 and 34 years.

The effect of alcohol consumption on illness and injury is more complex. In 1998 an estimated 3,271 people died as a consequence of hazardous and harmful levels of alcohol consumption. In addition to the harmful effects, however, when consumed at moderate levels alcohol appears to be associated with a decrease in heart disease and stroke. The number of people in Australia who drink at moderate levels far outweighs the number who drink at hazardous or harmful levels, so this apparent protective effect is greater for the overall population than the harmful effect for deaths, though not for potential years of life lost. Thus the estimated net reduction in deaths associated with alcohol consumption in 1998 was 2,371 but the estimated net potential years of life lost due to alcohol consumption in 1998 was 21,147.

The reason that alcohol appears to be associated with a net decrease in deaths but a net increase in potential years of life lost is because the decrease applies to illnesses which occur at older ages while the harmful effects apply across all ages. Deaths at younger ages contribute more potential years of life lost than deaths at older ages. Thus in 1998 the net effect of alcohol consumption at ages below 65 years was to cause an estimated 2065 deaths, leading to 47,887 potential years of life lost, while the net effect at ages 65 years and over was associated with a decrease of 4,436 deaths or 26,739 potential years of life lost.

Hospital separations attributable to tobacco, alcohol and illicit drugs

In 1997–98, 142,525 hospital separations in Australia were attributable to tobacco smoking and 14,471 to illicit drugs. For tobacco, the majority of separations (74,379) occurred at ages 65 and over; for illicit drugs the majority of separations (10,876) occurred at ages 15 to 34.

In 1997–98 an estimated 71,422 separations could be attributed to harmful and hazardous levels of alcohol consumption. However, once the estimate is adjusted for the decrease in heart disease and stroke associated with moderate alcohol consumption, the net overall number of separations was 43,033.

Revision of the aetiological fractions

Aetiological fractions depend on the prevalence of a risk factor and the associated relative risk of a particular illness or injury. The fractions presented here have been revised where possible—from the earlier reports of Holman et al. (1990) and English et al. (1995)—to incorporate the most recent estimates of the prevalence of use of tobacco, alcohol and illicit drugs. In addition, the relative risk estimates for some conditions have been revised to incorporate the results of recent research. The following conditions were selected for this detailed study and risk-ratio revision:

- in relation to alcohol—breast cancer, stroke, road injuries and fall injuries;
- in relation to tobacco—cervical cancer and peptic ulcer;
- in relation to illicit drugs—road injuries.

The relative risk of breast cancer associated with alcohol consumption was examined because recent research suggests that the risk varies with age. However, the analyses presented in this report failed to show a statistically significant difference in the risk for older women compared with younger women, and the overall risk-ratio estimate incorporating the results of recent research was similar to that derived by English et al.

The relative risk of stroke associated with alcohol consumption was examined because recent research suggests that the risk differs between ischaemic and haemorrhagic stroke. The analyses presented in this report support this, so different fractions were estimated for the two different types of stroke.

The relative risk of road injuries associated with alcohol consumption was examined using only Australian data—rather than a combination of Australian and international data, as used by English et al. Separate fractions were derived for motor vehicle drivers or motorcycle riders and for pedestrians, and these in turn were derived separately for hospital separations and deaths.

The relative risk of fall injuries associated with alcohol consumption was examined because of evidence that the risk varies with age. The analyses presented in this report support this, so separate fractions were derived for people aged 65 years and over and for people aged less than 65.

The relative risk of cervical cancer associated with tobacco smoking was examined because of recent research results on the causes of this cancer. Similarly, the relative risk of peptic ulcer associated with tobacco smoking was examined because of recent research results on the causes of peptic ulcer.

English et al. did not derive a fraction road injuries associated with illicit drug use because of the lack of suitable Australian data. More recent research has, however, provided such data so an estimate of this aetiological fraction is presented here.

In addition to using recent prevalence data and relative risk estimates, the aetiological fraction methodology for tobacco was revised in two ways. The first was to adjust for the time lag between tobacco exposure and the onset of related illnesses. English et al. used an estimate of current smoking prevalence in their calculation of aetiological fractions for tobacco. But for, many conditions there is a long time lag between exposure to tobacco smoke and the associated ill-effects—in the case of cancer it may be many decades. So for these conditions estimates of the current prevalence of smoking are not helpful in understanding the current associated disease burden.

We followed the Australian Burden of Disease Study (Mathers et al. 1999) in using the method proposed by Peto et al. (1992) to adjust for this time lag. Peto et al. proposed using an artificial compound prevalence measure of tobacco exposure, derived from a comparison between lung cancer rates in the country of interest and lung cancer rates among non-smokers observed in a large long-term follow-up study in the United States. This method was used here to determine tobacco exposure for cancer and chronic obstructive pulmonary disease. The mean time between tobacco exposure and the other illnesses and injuries discussed in this report is considerably shorter than that for cancer and chronic obstructive pulmonary disease, so estimates of current tobacco exposure were used for these other conditions.

The second modification to the methodology for tobacco involved the inclusion of estimates of passive exposure to tobacco smoke and its health effects in pregnancy and childhood. These estimates were based on data in the National Health and Medical Research Council's report on passive smoking (NHMRC 1997).

As with tobacco, current exposure to alcohol does not reflect the relevant exposure for some current outcomes, such as cirrhosis and cancers. There is, however, no equivalent of the method used by Peto et al. to adjust for this time lag. This report followed English et al. in using current prevalence estimates for alcohol consumption in calculating the aetiological fractions. The prevalence of alcohol consumption, particularly of heavy drinking, has declined in recent decades, so it is likely that these methods underestimate the true aetiological fractions of some current health outcomes attributable to alcohol consumption.

The final modification to the methodology of English et al. was to estimate the full attributable effect of alcohol consumption, including the apparent benefits of moderate consumption. English et al. calculated aetiological fractions for hazardous and harmful alcohol consumption (as defined by the NHMRC) relative to low alcohol consumption. These differed from the earlier estimates derived by Holman et al., which were calculated with abstention as the reference category. Using low alcohol consumption as the reference category, English et al. sought to reflect more accurately the idea that unsafe drinking—as opposed to low alcohol consumption, which may be protective—is the cause for concern.

Even at low levels of consumption, however, alcohol raises the risk of some conditions. Further, the approach taken by English et al. does not allow for the quantification of conditions prevented as a result of the beneficial effects of low levels of alcohol consumption. This report followed the earlier approach of Holman et al. and derived fractions to reflect both the risks and benefits of alcohol at all levels of consumption relative to abstaining from alcohol. Hence the estimates of alcohol-related deaths and hospital separations represent the net effect of both the alcohol-related harm and the alcohol-related benefit. The only exception to this is the fraction for the effect of alcohol on road traffic accidents: although there is some evidence that low levels of alcohol consumption raise the

risk of road traffic accidents at some ages, we followed English et al. in deriving the aetiological fraction with the legal level of alcohol consumption in drivers as the reference level.

Public health efforts in Australia are directed towards reducing unsafe alcohol consumption, rather than alcohol consumption per se. Therefore, although the primary purpose of this report is to estimate the total effect of alcohol consumption, it also presents, as Appendix A, a separate calculation using the approach taken by English et al. These data represent the extra effect of alcohol consumption for the 'unsafe' drinker compared with the 'responsible' drinker (English et al. 1995, p. 58), where unsafe and responsible consumption are defined by the NHMRC guidelines for responsible drinking (NHMRC 1992).

Preface

This study was partially funded by the Commonwealth Department of Health and Aged Care as part of the Memorandum of Understanding between it and the Australian Institute of Health and Welfare.

The work was carried out at the Australian Institute of Health and Welfare by Bruno Ridolfo and Chris Stevenson under the supervision of Dr Colin Mathers, who provided overall guidance for the project and valuable comments on early drafts of the report.

Bruno Ridolfo was responsible for the literature reviews, revision of the relative risk estimates, and estimation of the aetiological fractions for alcohol and breast cancer, stroke, road injuries and fall injuries; tobacco and cervical cancer and peptic ulcer disease; and illicit drugs and road injuries. He also prepared the population prevalence data for alcohol and tobacco consumption and contributed substantially to the fraction estimates for environmental tobacco smoke.

Chris Stevenson was responsible for preparing prevalence estimates for environmental tobacco smoke among adults and for completing the estimation of the associated aetiological fractions. He was also responsible for all the remaining aetiological fraction estimates, for preparing the estimates of 1998 mortality and 1997–98 hospital separations presented in Chapters 6 and 7 and for overall preparation of the report.

Dr Theo Vos, from the Victorian Department of Human Services, and Professor D'Arcy Holman, from the Department of Public Health in the University of Western Australia, provided valuable comments on an earlier draft of Chapters 1 to 5. In particular, Professor Holman suggested the inclusion of the partial fractions for hazardous and harmful alcohol consumption that are presented in Appendix A.

1 Introduction

In 1987 the Department of Community Services and Health (now the Department of Health and Aged Care) commissioned a study to determine methods for quantifying drug caused morbidity and mortality in Australia. The study was undertaken by Dr D'Arcy Holman (then director of the Epidemiology Branch of the Health Department of Western Australia) and Professor Bruce Armstrong (then Professor of Epidemiology and Cancer Research and Director of the National Health and Medical Research Council's Research Unit in Epidemiology and Preventive Medicine at the University of Western Australia). The primary objective of the project was to estimate the number of drug-caused deaths, hospital separations, patient days and years of life lost.

The resultant report was released by the Department of Community Services and Health in 1990 in conjunction with a departmental document that included modifications to the methods and time-series estimates of drug-caused deaths in Australia (Holman et al. 1990). The report dealt with harmful (and protective) effects attributed to three main drug groupings: alcohol, tobacco, and illicit drugs. It presented the proportion of cases attributed to the drug (called the *aetiological fraction*) for a comprehensive list of illnesses and causes of death or injury.

In view of the considerable research knowledge that accumulated after the publication of the Holman et al. report, the Department of Human Services and Health (as it was then known) commissioned a second study to revise and update the original methods and to apply the updated methods to more recent data. This study was undertaken by Dr Dallas English (Senior Lecturer in the Department of Public Health in the University of Western Australia) and Professor D'Arcy Holman (by this time Professor in the Department of Public Health in the University of Western Australia). Their report presented fully revised aetiological fractions for tobacco, alcohol and illicit drugs, along with estimates of the attributable hospital separations and patient days, deaths and potential years of life lost in Australia in 1992 (English et al. 1995).

In 1997 the Department of Health and Family Services (as it was then known) commissioned a further revision of the aetiological fractions methodology as part of the Memorandum of Understanding between the Department and the Institute. This report presents the results of the revision, along with estimates for 1998 (for mortality) and 1997–98 (for hospital separations). As with the previous two studies, this report uses the ninth revision of the World Health Organization's International Classification of Diseases (ICD-9) to classify causes of death, illness and injury.

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

$$F_r = (RR-1)/RR \qquad (1)$$

and among the total population is

$$F_a = p_e(RR-1)/\left(p_e(RR-1)+1\right) \qquad (2)$$

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 ($1 \le i \le k$) among those exposed to the risk factor is

$$F_{ri} = \left(RR_i - 1\right) \Big/ RR_i \tag{3}$$

and among the total population is

$$F_{ai} = p_i\left(RR_i - 1\right) \Big/ \sum_{j=0}^{k} p_j\left(RR_j - 1\right) + 1 \tag{4}$$

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

$$F_a = \sum_{i=1}^{k} p_i\left(RR_i - 1\right) \Big/ \sum_{i=0}^{k} p_i\left(RR_i - 1\right) + 1 \tag{5}$$

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

$$F_a = p_c\left(RR - 1\right) \Big/ RR \tag{6}$$

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:

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

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

A 95% confidence interval around the pooled estimate is

$$\left(\exp\left(\ln\left(RR\right) - 1.96 \Big/ \sum W_i \right), \exp\left(\ln\left(RR\right) + 1.96 \Big/ \sum W_i \right) \right) \tag{8}$$

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

$$PYLL = \left(1 - e^{(-0.03L)}\right) \Big/ 0.03 \qquad (9)$$

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

	Standard drinks per day (1 standard drink = 10 grams alcohol)		
Intake level	**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 then 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

	Males				Females			
Age	**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 occured 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

	Males				Females			
Age	**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 of 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

			Current smokers cigarettes per day			
Age	Never smoked	Ex-smoker	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

			Current smokers cigarettes per day			
Age	Never smoked	Ex-smoker	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.

3 Alcohol

3.1 Introduction

English et al. (1995) calculated aetiological fractions for hazardous and harmful alcohol consumption (as defined by the National Health and Medical Research Council) relative to low alcohol consumption. This was a departure from the earlier estimates derived by Holman et al. (1990), which were calculated with abstention as the reference category. With low alcohol consumption as the reference category, English et al. sought to reflect more accurately the idea that unsafe drinking—as opposed to low alcohol consumption, which may be protective—is the cause for concern.

However, even at low levels of consumption, alcohol increases the risk of some conditions. Further, the approach taken by English et al. does not allow for the quantification of conditions apparently prevented as a result of low levels of alcohol consumption. We followed the approach of the earlier study by Holman et al. and derived fractions to reflect both the risks and the benefits of alcohol at all levels of consumption relative to abstaining from alcohol. Hence our estimates of alcohol-related deaths and hospital separations represent the net effect of both the alcohol-related harm and the alcohol-related benefit. The only exception to this is the fraction for the effect of alcohol on road traffic accidents: although there is some evidence that low levels of alcohol consumption increase the risk of road traffic accidents at some ages, we followed English et al. in deriving the aetiological fraction with the legal blood alcohol concentration as the reference level.

Public health efforts in Australia are directed at reducing unsafe alcohol consumption, rather than alcohol consumption per se. Therefore, although the primary purpose of this report is to estimate the total effect of alcohol consumption, it also presents, in Appendix A, a separate calculation using the approach taken by English et al. These data represent the extra effect of alcohol consumption for the 'unsafe' drinker compared with the 'responsible' drinker (English et al. 1995, p. 58), where unsafe and responsible consumption are defined by the NHMRC guidelines for responsible drinking (NHMRC 1992).

As with tobacco, current exposure to alcohol does not reflect the relevant exposure for some current outcomes, such as cirrhosis and cancers. There is, however, no equivalent of the method used by Peto et al. (1992) to adjust for this time lag. We followed English et al. in using current prevalence estimates for alcohol consumption in calculating the aetiological fractions for alcohol. The prevalence of alcohol consumption, particularly heavy drinking, has declined in recent decades, so it is likely that these methods underestimate the true aetiological fractions of some current health outcomes attributable to alcohol consumption.

3.2 Revised aetiological fractions for alcohol

3.2.1 Alcohol and breast cancer among females

Drinking alcohol increases the risk of breast cancer, and it appears the mechanism may be a result of increased levels of oestradiol in the circulation (Davis et al. 1997). Reichman (1993)

demonstrated an increase in both total oestrogen levels and the amount of bioavailable oestrogens in association with alcohol consumption (30 g/day) among pre-menopausal women aged 21–40 years. This relationship between alcohol intake and increased levels of oestradiol in the circulation was confirmed more recently by Muti et al. (1998).

Ginsberg et al. (1996) demonstrated the effect that alcohol ingestion (0.7 g/kg) for a number of consecutive days can have on circulating levels of oestradiol among post-menopausal women receiving oral oestrogen as part of hormone replacement therapy when compared with women not on hormone replacement therapy. Alcohol did not significantly raise oestradiol levels among post-menopausal women not on hormone replacement therapy, but acute alcohol ingestion resulted in sustained and significant elevation in circulating oestradiol, to levels 300% higher than the level targeted for post-menopausal women on hormone replacement therapy (Ginsburg et al. 1996; Ginsburg et al. 1995a; Ginsburg et al. 1995b). While the alcohol dose (0.7 g/kg) used in this study was quite high, making the results less definitive, the study does demonstrate that in certain circumstances alcohol may have extreme effects on oestrogen levels and identifies a potential mechanism for increased breast cancer risk among post-menopausal women (Davis et al. 1997).

The epidemiological data on alcohol and breast cancer suggest a dose–response relationship that is very modest (Longnecker 1995b). While there are three meta-analyses (Longnecker 1994; Longnecker et al. 1988; Roth et al. 1994) that support a weak dose–response relationship, a causal role for alcohol was at the time still thought debatable (Longnecker 1995a; Longnecker 1995c). The association reported in the most recent meta-analysis (38 studies) by Longnecker (1994), however, was statistically significant, albeit very modest. The weighted average dose–response curve found in the 38 studies shows that, while results varied markedly between studies, on average for each alcoholic drink consumed daily the risk increased by 10% (Davis et al. 1997; Longnecker 1995b). A later study by Longnecker (1995a), which assessed risk based on cumulative (lifetime) alcohol intake, found that there was an almost 40% increase in women who averaged one drink a day and a 70% increase among those averaging two drinks a day (Davis et al. 1997).

Longnecker (1995a; 1995b) estimated that even if alcohol were causal—because in general women do not drink much and because the effect on risk, if any, is subtle—only around 4% of all breast cancers among women would be attributable to alcohol consumption (Davis et al. 1997). This is consistent with the work of English et al. (1995), who found that some 3% of breast cancer among females in Australia is caused by medium (≅ >2 ≤4 drinks a day) and high (≅ >4 drinks a day) levels of alcohol consumption.

A more recent Italian study (Mezzetti et al. 1998) has, however, attributed 10.7% of female breast cancer to alcohol intake of >20 g/day (95% CI: 4.4–17.0). Furthermore, the corresponding figure for pre-menopausal women (21.1%; 95% CI: 10.9–31.4) was four times that for post-menopausal women (5.4%; 95% CI: –2.5–3.4). This contrasts with the finding of English et al. that the test for heterogeneity in pooled relative risk estimates between pre-menopausal women (<45 years) and post-menopausal women (≥45 years) was consistent with no difference between the two groups. English et al. combined the two groups for subsequent analysis, and pooled relative risks were determined for use in calculating the aetiological fraction.

Epidemiological evidence for reviewing the aetiological fraction for alcohol and female breast cancer

Ferraroni et al. (1998) reported on 19 studies that examined heterogeneity due to age or menopausal status in the association of alcohol intake and the risk of female breast cancer.

Eight of the studies (Ewertz 1991; Kato et al. 1989; Katsouyanni et al. 1994; La Vecchia et al. 1989; Meara et al. 1989; Schatzkin et al. 1989; Sneyd et al. 1991; Willett et al. 1987) are reported to show no substantial heterogeneity. However, five studies (Friedenreich et al. 1993; Levi et al. 1996; Rohan & McMichael 1988; Schatzkin et al. 1987; van't Veer et al. 1989) reported an association that was stronger at a younger age or in pre-menopausal women. On the other hand, one study was indicative of a trend in risk only among peri-menopausal women (Chu et al. 1989), and four case-control studies (Ferraroni et al. 1991; Longnecker et al. 1995a; Martin Moreno et al. 1993; Richardson et al. 1989) and one prospective investigation (Hiatt et al. 1988) showed a stronger association among women aged more than 50 years or who were post-menopausal.

Recent results reported by Mezzetti et al. (1998) and Ferraroni et al. (1998) have found the elevation in risk for female breast cancer among alcohol drinkers to be significant among pre-menopausal women, rather than post-menopausal women. Mezzetti et al. found that the proportion of breast cancer cases attributable to alcohol for pre-menopausal females (21.1%; 95% CI: 10.9–31.4) was four times that for post-menopausal women (5.4%; 95% CI: –2.5–13.4).

Studies used to revise the aetiological alcohol and female breast cancer

The studies used to revise the aetiological alcohol and female breast cancer are listed in Appendix B. The literature search identified 52 papers for detailed review; this yielded 17 studies with data suitable for inclusion in the review of the fractions. Only 16 of the 17 studies were, however, included in the calculations: the results reported by Mezzetti et al. (1998) appeared to be based on the same data used by Ferraroni et al. (1998) and were therefore excluded (Appendix B, Tables B.3 and B.4). The 16 studies were combined with the studies reviewed by English et al. (1995) (Appendix B, Tables B.1 and B.2) and used to recalculate the aetiological fraction.

Revised pooled relative-risk estimates for alcohol exposure and female breast cancer

While English et al. (1995) found that the test for heterogeneity between studies of the association between alcohol and female breast cancer was consistent with no difference for the effect of menopausal status, this was inconsistent with the more recent literature (Ferraroni et al. 1998; Mezzetti et al. 1998). Therefore, those studies reviewed by English et al. and the studies from our review were classified into studies that examined women of all ages, of pre-menopausal age (<45 years) and post-menopausal age (≥45 years) and separate risk ratios calculated for each group. The risk ratio estimates for women aged under 45 years were not statistically significantly different from those for women aged 45 and over, so the aetiological fractions were based on the combined risk ratio. Table 3.1 shows the revised risk-ratio estimates.

Revised aetiological fractions for alcohol and female beast cancer

Aetiological fractions for alcohol and female breast cancer were calculated using the formulae and the prevalence estimates based on the ABS National Health Survey, as shown in Chapter 2, and the revised pooled estimates of relative risk shown in Table 3.1. Table 3.2 shows the results.

Based on the sum of the partial aetiological fractions for ages 18 and over, the overall female aetiological fraction for breast cancer caused by low, medium and high drinking levels was

estimated to be 0.121. Thus, around 12% of female breast cancer for ages 18 years and over may be attributable to low, medium and high levels of alcohol intake.

Table 3.1: Revised pooled estimates of relative risk for alcohol exposure and female breast cancer

		Low		Medium		High	
Sex	**Age**	**RR**	**95% CI**	**RR**	**95% CI**	**RR**	**95% CI**
Female	All	1.14	1.09–1.20	1.41	1.32–1.50	1.59	1.43–1.78
	Under 45 years	1.15	1.04–1.28	1.41	1.2–1.67	1.46	0.99–2.14
	45 years and over	1.14	1.05–1.24	1.38	1.24–1.53	1.62	1.24–2.13

Source: AIHW analysis of studies listed in Appendix B, Tables B.1 and B.3.

Table 3.2: Revised aetiological fractions for alcohol exposure and breast cancer

	Level of exposure		
Age	**Low**	**Medium**	**High**
Exposed population			
All ages	0.12	0.29	0.37
General population			
18–19	0.075	0.046	0.014
20–24	0.075	0.046	0.014
25–29	0.077	0.035	0.011
30–34	0.082	0.033	0.010
35–39	0.062	0.041	0.012
40–44	0.067	0.034	0.011
45–49	0.060	0.033	0.016
50–54	0.057	0.041	0.020
55–59	0.059	0.043	0.013
60–64	0.061	0.045	0.006
65–69	0.056	0.051	0.007
70–74	0.060	0.032	0.006
75–79	0.058	0.016	0.005
80+	0.072	0.016	0.008
Total (18+)	**0.069**	**0.039**	**0.012**

Source: AIHW analysis of revised relative risk estimates in Table 3.1 and prevalence data from Chapter 2.

3.2.2 Alcohol and stroke

In the past 30 years many studies have linked both habitual and acute heavy drinking to an increased risk of stroke (Camargo 1996). The role of smaller amounts of alcohol is more complex (Camargo 1989; Camargo 1996; Sacco et al. 1999).

When limited to observational investigation, epidemiological research on alcohol consumption presents important methodological problems, given that alcohol consumption

is a complex and varying phenomenon that is difficult to measure (Camargo 1996). Epidemiological research on stroke is also complicated by methodological difficulties, particularly concerning stroke identification and classification (Camargo 1996). Silent or clinically undetected strokes may contribute to imprecise ascertainment of stroke incidence, which will tend to weaken any statistical association with true risk factors.

Epidemiological studies of alcohol and stroke should take account of potential confounders. If alcohol causes increased stroke risk because of alcohol-induced hypertension alone, then controlling for blood pressure would eliminate the association between alcohol and stroke. The confounded association may be of greater interest than the adjusted association (Camargo 1996).

Additional problems can arise if stroke is regarded as a single pathological entity. As a minimum, ischaemic (thrombo-embolic) strokes should be differentiated from haemorrhagic strokes (intracerebral haemorrhage and subarachnoid haemorrhage). While some risk factors, for example age and hypertension, are common to all types of stroke, other factors may have distinctive associations. Therefore, if a factor is strongly associated with one type of stroke and weakly (or inversely) associated with another, failure to differentiate between stroke types would tend to obscure real associations (Camargo 1996). Ischaemic stroke accounts for 70–80% of all strokes.

Studies in the last 10 years have confirmed that alcohol consumption has a distinctively different association with haemorrhagic stroke as opposed to ischaemic stroke (Camargo 1996). In the main, they have shown an increased risk of haemorrhagic stroke associated with increasing alcohol consumption in a dose-dependent fashion (Sacco et al. 1999), so that even moderate levels of drinking increase the risk of haemorrhagic stroke (Camargo 1996). On the other hand, Sacco et al. confirmed the findings of a number of earlier case-control studies that moderate alcohol consumption (up to two drinks a day in the past year) relative to the absence of any alcohol consumption was significantly protective for fatal or first non-fatal ischaemic stroke. They found an odds ratio (OR) of 0.51 with a 95% confidence interval of 0.39 to 0.67. This protective effect was evident for consumption of up to five drinks a day (OR=0.58; 95% CI: 0.35–0.94).

Kiechl et al. (1994) examined the dose-dependent promotion or deceleration of carotid atherosclerosis by alcohol. Their findings supported the likelihood of a U-shaped association with ischaemic stroke. This study of Italian men aged 40–79 years used logistic regression to examine the potential relationship between alcohol and carotid atherosclerosis. Alcohol consumption was quantified in terms of grams a day and classified into four categories: no current use; ≤50 g/day; 51–99 g/day; and ≥100 g/day). (English et al. considered one alcohol drink a day as equivalent to 10 grams of alcohol a day.)

With alcohol consumption treated as a continuous variable, logistic regression showed that the age-adjusted overall effect of drinking was moderate disease promotion (slope coefficient β=+0.0067, df=1, $p<0.01$). However, classification of alcohol consumption into four equally spaced groups (50 g/day each) was strongly suggestive of a U-shaped trend. The quadratic (non-linear) model was confirmed as having the best fit.

Atherosclerotic risk in men who consumed ≥ 100 g/day was more than twice that of abstainers, but light drinkers fared better than abstainers (OR=0.44; p=0.01). Furthermore, when the analysis was restricted to the alcohol estimates obtained with diet records (as opposed to recall for questionnaire responses) this further strengthened the U-shaped trend (≤ 50 g/day: OR=0.38; ≥100 g/day: OR=3.67). The use of past alcohol consumption, while continuing to reflect the U-shaped association with carotid atherosclerosis, did so to a lesser degree. For females, the relationship between low amounts of alcohol (≤50 g/day; n=112)

and the lowered risk for carotid atherosclerosis was similar to that observed for males (OR: 0.47; p=0.01).

In examining the association between alcohol consumption and carotid atherosclerosis Kiechl et al. (1994) adjusted for behavioural variables such as social class and physical activity and for body mass index. The adjustment did not improve the fit of the regression model and yielded similar risk estimates. Furthermore, adjustment for smoking and restriction of the analysis to non-smokers (n=310) did not result in major changes to the adjusted odds ratios.

Given that an ex-drinker might have stopped drinking because of health problems, Kiechl et al. also took account of the potential effect of the inclusion of past drinkers in the reference group (current non-drinkers). This was done by excluding previous drinkers and reclassifying ex-drinkers as light, moderate or severe based on self-reported amounts of previous alcohol consumption. While both of these tended to lessen the beneficial effects of low amounts of alcohol compared with no alcohol (OR= 0.52 and 0.51, compared with 0.44), the residual relationship remained statistically significant.

Carnago (1996) noted that, overall, the reduction in relative risk of ischaemic stroke associated with low alcohol consumption (up to two drinks a day in the past year), as opposed to no alcohol consumption, outweighed the increased relative risk for haemorrhagic stroke. This resulted in reduced total stroke relative risk for low alcohol consumption as opposed to no alcohol consumption.

However, Carnago also reported studies showing this result may be influenced by factors associated with race. Studies that differentiated between white and black populations did not support effect modification by race. But studies of people of Japanese origin found the reduction in the risk of ischaemic stroke was attenuated to the extent that there was an absence of reduced relative risk due to low alcohol consumption for all stroke once the increased risk for haemorrhagic stroke was taken into account. This suggests that the effect of alcohol on stroke may be different for people of Asian origin. In Australia, 1996 census data show that some 4.8% of the Australian population had Asia as a place of birth (ABS 1997).

Epidemiological evidence for reviewing the aetiological fraction for alcohol and stroke

The literature suggests that, as well as updating the current fractions with references for 1994 to 1998, the following matters should also be taken into account where possible.

Distinguishing between studies reporting on ischaemic, haemorrhagic and all stroke

Since the alcohol – ischaemic stroke relationship has been shown to be U- or J-shaped, low or medium drinking will confer a health benefit because of a reduction in the incidence of ischaemic stroke. While there will be an increased incidence of haemorrhagic stroke with an equivalent increase in the level of alcohol consumption, this should not translate into an increase in total stroke incidence since, as noted, ischaemic stroke accounts for 70–80% of all strokes combined (Camargo 1996). This relationship therefore requires that ischaemic and haemorrhagic stroke be examined separately.

Inclusion of the protective effect of low alcohol intake on stroke incidence and death in any quantification of the overall effect of alcohol and stroke

Sacco et al. (1999) confirmed the finding of a number of earlier case-control studies, conducted predominantly among white subjects, that low alcohol consumption (up to two drinks a day in the past year) relative to no alcohol consumption was significantly protective for fatal or first non-fatal ischaemic stroke (OR=0.51; 95% CI: 0.39–0.67). The unadjusted odds ratio shows this protective effect to be evident for consumption of up to five drinks a day (OR=0.58; 95% CI: 0.35–0.94).

Among those with low alcohol consumption, continued consumption at this level (particularly among the elderly) reduces the risk of ischaemic stroke (Sacco et al. 1999), confirming that the J-shaped association with alcohol is protective for the onset of fatal or first non-fatal ischaemic stroke. Given that the prevalence of exposure to alcohol at this level in the community is 60% to 80% among males in all age groups and 55% to 70% among females (English et al. 1995), this effect would be expected to be substantial. Furthermore, this is also important because ischaemic stroke accounts for 70–80% of all strokes.

Jamrozik et al. (1994) cited an example of this effect. In this Perth study, heavy consumption of alcohol (≥61 g/day) was relatively rare, so it was implicated in at most one in nine strokes, despite its higher relative risk (unadjusted odds ratio=2.51; 95% CI: 1.33–4.74). However, between 2% and 30% of additional strokes appear to have been avoided by almost half the study group consuming one or two alcoholic drinks daily.

If possible, distinguishing between studies reporting on incidence as opposed to death and deriving fractions pertinent to each

The incidence of stroke is largely determined by the distribution of risk factors (including alcohol) within the population . But case fatality is more likely to be strongly related to the type of stroke, the severity of the stroke, and the availability of diagnostic procedures and treatment (Grobbee et al. 1996). Studies that have stroke incidence as an outcome far outnumber those with stroke death as an outcome (English et al. 1995).

One recent study (He et al. 1995) conducted in the Chinese population sought to differentiate the association between alcohol consumption and the relative risks for stroke incidence and stroke mortality. In this instance, the increase in relative risk for alcohol consumption and stroke incidence was slightly higher than that for stroke death for both the unadjusted (RR: 1.38; 1.29–1.48 compared with RR: 1.25; 1.15–1.35) and adjusted (RR: 1.36; 1.19–1.56 compared with RR: 1.17; 1.16–1.19) estimates. This suggests that it may be preferable to have separate risk estimates for incidence and death, but the limited availability of studies that have examined incidence and death separately makes this problematic.

Studies used to revise the aetiological fraction for alcohol and stroke

The studies used to revise the aetiological fraction for alcohol and stroke are listed in Appendix B. The literature search identified 40 papers for detailed review; this yielded 14 studies with usable data (Appendix B, Table B.5). These were combined with the studies identified by English et al. (1995—see Appendix B, Table B.6) to calculate the risk-ratio estimates. While English et al. found the test for heterogeneity between studies of ischaemic and haemorrhagic stroke was consistent with no difference in the effect of alcohol, this was inconsistent with the literature review just outlined. Therefore, only studies that differentiated between these types of stroke were used in the calculations.

The test of heterogeneity between the sexes was consistent with different effects among men and women, so only studies reporting sex-specific relative risk estimates were used for the derivation of pooled estimates. We also followed English et al. in excluding studies of Asian origin. Appendix B, Table B.7 lists the individual risk ratio estimates used in calculating the pooled risk. In instances where relative risks or odds ratios with 95% confidence intervals were not reported for a particular study, these were calculated from data presented within the paper (Hillbom et al. 1995; Juvela et al. 1995).

Revised pooled estimates of relative risk for alcohol exposure and stroke

As with English et al. (1995), our revised pooled estimates of relative risk are based on the 'fixed-effects' assumption. While this is not valid in all instances (for example, where the test for heterogeneity is significant) the alternative random effects estimate is subject to limitations and disadvantages, the main one being that more weight is given to smaller studies than occurs under the 'fixed effects' assumption. The revised relative risk estimates are listed in Tables 3.3 and 3.4.

Compared with abstainers, the pooled relative risk of ischaemic stroke for males among low-level drinkers was 0.94 (95% CI: 0.78–1.13), among medium level-drinkers 1.33 (95% CI: 1.07–1.66) and among high-level drinkers 1.65 (95% CI: 0.95–2.86). Similarly, compared with abstainers, the pooled relative risk of ischaemic stroke for females among low-level drinkers was 0.52 (95% CI: 0.42–0.65), among medium-level drinkers 0.64 (95% CI: 0.44–0.95) and among high–level drinkers 1.06 (95% CI: 0.36–3.12).

Compared with abstainers the pooled relative risk of haemorrhagic stroke among male low level drinkers was 1.27 (95% CI: 0.83–1.94), among male medium level drinkers 2.19 (95% CI: 1.47–3.28) and among male high level drinkers 2.38 (95% CI: 1.18–4.77). Similarly, compared with abstainers, the pooled relative risk of haemorrhagic stroke among female low level drinkers was 0.59 (95% CI: 0.38–0.92), among female medium level drinkers 0.65 (95% CI: 0.36–1.19) and among female high level drinkers 7.98 (95% CI: 3.25–19.6).

Table 3.3: Revised pooled estimates of relative risk for alcohol exposure and ischaemic stroke

		Level of alcohol exposure					
		Low		Medium		High	
Sex	Age	RR	95% CI	RR	95% CI	RR	95% CI
Male	All	0.94	0.78–1.13	1.33	1.07–1.66	1.65	0.95–2.86
Female	All	0.52	0.42–0.65	0.64	0.44–0.95	1.06	0.36–3.12

Source: AIHW analysis of studies listed in Appendix B, Table B.7.

Table 3.4: Revised pooled estimates of relative risk for alcohol exposure and haemorrhagic stroke

		Level of alcohol exposure					
		Low		Medium		High	
Sex	Age	RR	95% CI	RR	95% CI	RR	95% CI
Male	All	1.27	0.83–1.94	2.19	1.47–3.28	2.38	1.18–4.77
Female	All	0.59	0.38–0.92	0.65	0.36–1.19	7.98	3.25–19.60

Source: AIHW analysis of studies listed in Appendix B, Table B.7.

Revised aetiological fractions for alcohol and stroke

The overall male aetiological fraction for ischaemic stroke caused by low, medium and high levels of alcohol consumption was estimated to be 0.027 (Table 3.5). Thus, for males, 2.7% of ischaemic stroke may be attributable to medium and high levels of alcohol intake after adjusting for the protective effect due to low-level alcohol intake. The overall female aetiological fraction for ischaemic stroke caused by low, medium and high drinking levels was estimated to be –0.441. Thus, relative to abstainers, an overall protective effect for ischaemic stroke was attributable to low and medium levels of alcohol intake among females.

Table 3.5: Revised aetiological fractions for alcohol exposure and ischaemic stroke

	Male			Female		
	Level of exposure			Level of exposure		
Age	Low	Medium	High	Low	Medium	High
Exposed population						
All ages	–0.06	0.25	0.39	–0.91	–0.55	0.06
General population						
18–19	–0.040	0.021	0.052	–0.451	–0.071	0.002
20–24	–0.040	0.021	0.052	–0.451	–0.071	0.002
25–29	–0.042	0.024	0.046	–0.449	–0.052	0.002
30–34	–0.039	0.028	0.044	–0.495	–0.051	0.002
35–39	–0.040	0.025	0.032	–0.334	–0.056	0.002
40–44	–0.038	0.031	0.036	–0.366	–0.048	0.002
45–49	–0.038	0.034	0.036	–0.316	–0.045	0.003
50–54	–0.041	0.026	0.040	–0.299	–0.055	0.003
55–59	–0.041	0.027	0.024	–0.310	–0.058	0.002
60–64	–0.038	0.033	0.046	–0.330	–0.062	0.001
65–69	–0.041	0.024	0.029	–0.297	–0.070	0.001
70–74	–0.042	0.020	0.038	–0.304	–0.041	0.001
75–79	–0.043	0.009	0.043	–0.282	–0.020	0.001
80+	–0.043	0.017	0.020	–0.383	–0.022	0.001
Total (18+)	**–0.040**	**0.027**	**0.040**	**–0.387**	**–0.056**	**0.002**

Source: AIHW analysis of revised relative risk estimates in Table 3.3 and prevalence data in Chapter 2.

The overall male aetiological fraction for haemorrhagic stroke was estimated to be 0.269 (Table 3.6). Thus, for males, 27% of haemorrhagic stroke may be attributable to low, medium and high levels of alcohol intake. The overall female aetiological fraction for haemorrhagic stroke was estimated to be –0.124. Thus, relative to abstainers, an overall protective effect for haemorrhagic stroke was attributable to low and medium levels of alcohol intake among females: this outweighed the harmful effect of alcohol on the risk of haemorrhagic stroke at high intake levels.

Table 3.6: Revised aetiological fractions for alcohol exposure and haemorrhagic stroke

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Medium**	**High**	**Low**	**Medium**	**High**
Exposed population						
All ages	0.21	0.54	0.58	–0.69	–0.53	0.87
General population						
18–19	0.136	0.058	0.083	–0.285	–0.051	0.212
20–24	0.136	0.058	0.083	–0.285	–0.051	0.212
25–29	0.140	0.065	0.073	–0.300	–0.040	0.169
30–34	0.133	0.076	0.070	–0.329	–0.038	0.166
35–39	0.136	0.069	0.051	–0.225	–0.043	0.175
40–44	0.130	0.084	0.058	–0.249	–0.037	0.161
45–49	0.127	0.093	0.057	–0.202	–0.033	0.220
50–54	0.139	0.070	0.063	–0.182	–0.038	0.257
55–59	0.140	0.072	0.038	–0.206	–0.044	0.185
60–64	0.126	0.088	0.072	–0.245	–0.052	0.089
65–69	0.140	0.066	0.047	–0.215	–0.057	0.113
70–74	0.144	0.055	0.060	–0.227	–0.035	0.088
75–79	0.148	0.025	0.069	–0.215	–0.017	0.072
80+	0.149	0.048	0.032	–0.271	–0.017	0.127
Total (18+)	**0.134**	**0.072**	**0.064**	**–0.258**	**–0.042**	**0.176**

Source: AIHW analysis of revised relative risk estimates in Table 3.4 and prevalence data in Chapter 2.

3.2.3 Alcohol and road injuries

Alcohol is the main cause of deaths on Australian roads: it is implicated in about one-third of all motorist deaths (Federal Office of Road Safety (FORS) 1996). However, alcohol has an even greater involvement in pedestrian fatalities: it is implicated in some 45% of fatalities among adult and youth pedestrians (FORS 1996).

Crashes involving adult and youth pedestrians tend to have greater alcohol involvement than other crashes (FORS 1996). In 1992, in cases where the blood alcohol concentration (BAC) of the parties involved was known, intoxication (BAC >0.05 g/100 mL) was implicated in 47% of deaths among adult and youth pedestrians, in 43% of single-vehicle crashes, and in 27% of multiple-vehicle crashes.

Furthermore, the blood alcohol concentration tends to be more extreme in crashes involving pedestrians (FORS 1996). In 1992 BACs averaged 0.217g/100 mL among intoxicated pedestrian victims, 0.181 g/100 mL among intoxicated motorists in fatal single-vehicle crashes, and 0.164 g/100 mL among intoxicated motorists in fatal multiple-vehicle crashes.

Epidemiological evidence for reviewing the aetiological fraction for alcohol and motor vehicle accidents

English et al. (1995) used information from case-control studies of relative risk and from BAC case series to derive the aetiological fraction for alcohol and motor vehicle accidents. They combined two studies that examined the relationship of motor vehicle deaths (Lloyd 1992) and injuries (McLean et al. 1980) to BAC, using exposure gradations (>0–0.05 g/100 mL, >0.05–0.10 g/100 mL and >0.10 g/100 mL) and the pooled relative risks to determine the aetiological fraction.

Although BAC data for police-reported hospitalisations associated with motor vehicle and motorcycle accidents are incomplete for Australia in 1996 (55% unknown), the New South Wales data are relatively complete (14% unknown). They suggest a less prominent role for alcohol in hospitalisations compared with accidents with a fatal outcome. If the records with unknown BAC are excluded, only 9% of hospitalisations recorded a BAC >0.05 g/100 mL. The corresponding figure with the unknowns included was 8%. This compares with a BAC >0.05 g/100 mL for 22% (unknowns included) or 24% (unknowns excluded) of fatally injured motor vehicle drivers and motorcycle riders in the same year (FORS 1997). Based on this evidence, the relative risk estimates from Lloyd's (1992) study of deaths caused by motor vehicle accidents were used to revise the aetiological fraction for motor vehicle driver and motorcycle rider deaths due to alcohol. The relative risk estimates from McLean's (1980) study of serious crashes provided estimates for the aetiological fraction for motor vehicle driver and motorcycle rider hospitalisations due to alcohol.

The FORS data on pedestrian hospitalisations as a result of accidents is less complete than the data on motor vehicle driver and motorcycle rider hospitalisations as a result of accidents. However, a South Australian case study also demonstrates the pattern of a somewhat higher BAC for pedestrian fatalities when compared with pedestrian hospitalisations (Holubowycz 1995). A BAC ≥0.10 g/100 mL was evident among 50% of male and 38% of female pedestrian fatalities for ages 16 and older. This compares with 39% of male and 30% of female pedestrian hospital admissions for ages 16 years and older. Since there are no relative risk estimates for the contribution of BAC ≥0.10 g/100 mL to either pedestrian fatalities or pedestrian hospitalisation, unpublished prevalence data for 1992 to 1996 provided by FORS and the Holubowycz prevalence data are the basis for determining these fractions.

English et al. used blood alcohol case series publications to calculate a pooled estimate, weighted by study size, of the aetiological fraction for road injuries caused by alcohol. They calculated separate fractions for males and females using combined Australian and international data and based on the criteria of (1) BAC >0.10 g/100 mL and (2) the use of case series for all road injuries (the majority being drivers), car drivers or motorcycle drivers. Specific case series of pedestrians, passengers or pedal cyclists were excluded. We modified this by using Australian case series data only (provided by FORS) and, based on the evidence just described, derived separate fractions for vehicle driver and motorcycle rider accidents and pedestrian accidents.

Revised blood alcohol case series for deaths due to motor vehicle accidents in Australia, 1996

Fatalities and hospitalisations with BACs of >0.05–<0.10 g/100 mL and ≥0.10 g/100 mL were obtained from FORS for 1996. The data were used to derive the prevalence of BAC >0.05–0.10 gms/100 mL and ≥0.10 g/100 mL among fatally injured motor vehicle drivers and motorcycle riders (Table 3.7) and pedestrians (Table 3.8) in Australia .

Table 3.7: Blood alcohol prevalence for motor vehicle driver and motorcycle rider accident deaths, 1996

	Males		Females		Persons	
	Blood alcohol concentration		Blood alcohol concentration		Blood alcohol concentration	
Age	>0.05–<0.10	≥0.10	>0.05–<0.10	≥0.10	>0.05–<0.10	≥0.10
16–19	0.03	0.28	0.08	0.08	0.04	0.23
20–29	0.04	0.37	0.02	0.16	0.04	0.33
30–49	0.03	0.38	0.02	0.19	0.03	0.35
50+	0.01	0.09	0.00	0.00	0.01	0.06
16 +	0.03	0.31	0.02	0.11	0.03	0.27

Source: Federal Office of Road Safety.

Table 3.8: Blood alcohol prevalence for pedestrian accident deaths, 1996

	Males		Females	
	Blood alcohol concentration		Blood alcohol concentration	
Age	>0.05–<0.10	≥0.10	>0.05–<0.10	≥0.10
16–19	0.06	0.69	0.00	0.50
20–29	0.03	0.58	0.11	0.11
30–49	0.02	0.51	0.00	0.42
50+	0.03	0.16	0.00	0.06
16+	0.03	0.40	0.01	0.17

Source: Federal Office of Road Safety.

Revised blood alcohol case series for hospitalisations due to motor vehicle accidents in Australia, 1994 to 1996

The aetiological fractions for hospitalisations are based on FORS case series data on hospitalisation due to motor vehicle accidents for 1994 to 1996. The scope of the hospitalisation crash data provided by police services differs between various Australian jurisdictions as a result of differing crash reporting requirements and practices. Not all reportable road traffic accidents come to police attention (in particular, those involving cyclists and motorcyclists) and classification by police at an accident scene (as to whether or not hospital admission occurs) is uncertain. Some people sent to hospital may simply be treated in accident and emergency departments before being sent home (FORS 1998).

Although Australian data are reported in Tables 3.9 and 3.10, it is important to note the incomplete nature of data on blood alcohol concentration across all age groups. New South Wales has the lowest level of missing values for road accident victims admitted to hospital;

Western Australia and Queensland have the highest levels (O'Conner & Trembath 1995). New South Wales data dominate the Australian data.

Table 3.9: Blood alcohol concentration among motor vehicle drivers and motorcycle riders hospitalised as a result of accidents, 1994 to 1996

	Males		Females		Persons	
	Blood alcohol concentration		Blood alcohol concentration		Blood alcohol concentration	
Age	>0.05–<0.10	≥0.10	>0.05–<0.10	≥0.10	>0.05–<0.10	≥0.10
16–19	0.07	0.17	0.03	0.08	0.05	0.15
20–29	0.06	0.31	0.03	0.14	0.05	0.26
30–49	0.04	0.23	0.02	0.12	0.03	0.19
50+	0.03	0.08	0.01	0.02	0.02	0.06
16 +	0.05	0.23	0.02	0.10	0.05	0.08

Source: Federal Office of Road Safety.

Table 3.10: Blood alcohol concentration among pedestrians hospitalised as a result of accidents, 1994 to 1996

	Males		Females	
	Blood alcohol concentration		Blood alcohol concentration	
Age	>0.05–<0.10	>=0.10	>0.05–<0.10	>=0.10
16–19	0.07	0.35	0.07	0.16
20–29	0.07	0.45	0.05	0.19
30–49	0.03	0.46	0.03	0.21
50+	0.08	0.23	0.00	0.03
16 +	0.06	0.37	0.01	0.06

Source: Federal Office of Road Safety.

Revised relative effect estimates for alcohol exposure and death due to motor vehicle and motorcycle accidents

The estimates of relative risk are based on data reported by Lloyd (1992) for alcohol and fatal road accidents. The age range and BAC for females were aggregated so as to provide sufficient data in the relevant cells.

These relative risk values are presented using abstainers as the base (BAC=0—Table 3.11). However, we followed English et al. (1995) and based the aetiological fractions on the risk relative to the lowest exposure category. These rescaled relative risk ratios are presented in Table 3.12.

Table 3.11: Risk estimates for alcohol exposure and motor vehicle driver and motorcycle rider deaths as a result of accident

		Blood alcohol concentration					
		>0–0.05		>0.05–0.10		>0.10	
Sex	Age	RR	95% CI	RR	95% CI	RR	95% CI
Male	<21	2.01	1.12–3.60	10.38	5.31–20.29	67.35	35.40–128.11
	21–29	1.14	0.58–2.23	4.96	2.59– 9.49	113.88	73.24–177.07
	30–50	1.33	0.66–2.69	5.74	2.92–11.27	142.43	89.15–227.56
	>50	1.44	0.61–3.37	3.75	1.38–10.19	45.15	22.58–90.27
	All ages	1.45	1.04–2.04	5.86	4.18–8.23	96.82	75.03–124.94
		Blood alcohol concentration					
		>0.01–0.10			>0.10		
		RR	95% CI	RR		95% CI	
Female	<30	1.78	0.72–4.39	72.59		34.83–151.29	
	30+	2.22	0.99–4.96	52.36		18.86–145.37	
	All ages	2.01	1.10–3.66	65.17		36.19–117.38	

Source: Lloyd 1992.

Table 3.12: Rescaled risk estimates for alcohol exposure and motor vehicle driver and motorcycle rider accident deaths

		Blood alcohol concentration		
		>0–0.05	>0.05–0.10	>0.10
Sex	Age	RR	RR	RR
Male	<21	1.00	5.16	33.50
	21–29	1.00	4.35	99.89
	30–50	1.00	4.31	107.09
	>50	1.00	2.60	31.35
	All ages	1.00	4.03	66.59
		Blood alcohol concentration		
		>0.01–0.10		>0.10
		RR		RR
Female	<30	1.00		40.78
	30+	1.00		23.58
	All ages	1.00		32.45

Source: AIHW analysis of data in Table 3.11.

The test of heterogeneity shows that these relative risk values do not vary significantly by age, so we based the fractions on the relative risk for all ages.

Revised aetiological fractions for alcohol and motor vehicle and motorcycle accident deaths

The aetiological fractions were based on the prevalence data in Tables 3.7 and 3.8. The fraction for motor vehicle driver and motorcycle rider deaths also used the all-ages relative risk values presented in Table 3.12. The age-specific prevalences from Table 3.7 were applied at ages over 15 years. However, motor vehicle accident deaths at ages under 15 years presumably involved passengers rather than drivers, so we followed Holman et al. (1990) and applied the all-ages fraction to these age groups (Table 3.13).

There are no equivalent relative risk estimates for pedestrians, so the fraction was taken to be the prevalence value from Table 3.8.

Table 3.13: Revised aetiological fractions for alcohol exposure and motor vehicle driver and motorcycle rider deaths

	Males		Females
	Blood alcohol concentration		Blood alcohol concentration
Age	**>0.05–<0.10**	**≥0.10**	**≥0.10**
Exposed			
All ages	0.752	0.985	0.969
General population			
<15	0.023	0.305	0.107
15–19	0.023	0.276	0.078
20–24	0.030	0.364	0.155
25–29	0.030	0.364	0.155
30–34	0.023	0.374	0.184
35–39	0.023	0.374	0.184
40–44	0.023	0.374	0.184
45–49	0.023	0.374	0.184
50–54	0.008	0.089	0.000
55–59	0.008	0.089	0.000
60–64	0.008	0.089	0.000
65–69	0.008	0.089	0.000
70–74	0.008	0.089	0.000
75–79	0.008	0.089	0.000
80+	0.008	0.089	0.000
All ages	**0.023**	**0.305**	**0.107**

Source: AIHW analyses of data in Tables 3.7 and 3.12.

The overall aetiological fraction for driver and rider road accident deaths caused by driving or riding with a BAC >0.05 g/100 mL was estimated as 0.328 among males and 0.107 among females. For those aged 20–29 years of age, the overall aetiological fraction was estimated as 0.395 among males and 0.155 among females. Among 30–50 year olds the overall aetiological fraction was estimated as 0.397 among males and 0.184 among females and for people aged 50 years and over it was 0.096 among males and nil among females. These numbers differ slightly from the sums of the corresponding numbers in Table 3.13 because of rounding.

English et al. (1995) used a blood alcohol criterion of ≥0.10 g/100 mL to identify the members of a road injuries case series that could be attributed to alcohol. We applied this criterion to the FORS 1996 case series of pedestrian deaths, so that the prevalence data for ≥0.10 g/100 mL from Table 3.8 were used to estimate the aetiological fraction for pedestrian road injuries caused by alcohol among males and females (Table 3.14).

Table 3.14: Revised aetiological fractions for alcohol exposure and pedestrian deaths

	Males	Females
	Blood alcohol concentration	Blood alcohol concentration
Age	>=0.10	>=0.10
General population		
16–19	0.69	0.50
20–29	0.58	0.11
30–49	0.51	0.42
50+	0.16	0.06
16+	0.40	0.17

Source: AIHW analysis of data from Table 3.8.

For people aged 16–19 years, the aetiological fraction for pedestrian road accident deaths caused by driving with a BAC >0.10 g/100 mL was estimated as 0.69 for males and 0.50 for females. For those aged 20–29 years, the aetiological fraction was estimated as 0.58 for males and 0.11 for females. Among 30–50 year olds the aetiological fraction was estimated as 0.51 for males and 0.42 for females, and for people aged 50 years or more it was 0.16 for males and 0.06 for females.

Revised aetiological fractions for alcohol and motor vehicle and motorcycle accident hospitalisations

The revised fractions for motor vehicle and motorcycle accident hospitalisations were based on the FORS data in Tables 3.9 and 3.10. Relative risk estimates for drivers and riders were derived from the study by McLean et al. (1980). As with the fractions for deaths, the fractions for pedestrian hospitalisations were taken directly from the prevalence estimates in Table 3.10 using a blood alcohol criterion of ≥0.10 g/100 mL.

Table 3.15: Risk estimates for alcohol exposure and motor vehicle and motorcycle accident hospitalisations: males and females, all ages

Blood alcohol concentration	RR	95% Confidence interval
0.01–0.03	0.69	0.32–1.47
0.04–0.06	1.83	0.87–3.85
0.07–0.09	3.20	1.20–8.48
0.10 and over	12.94	6.60–25.36

Source: Derived from McLean et al. (1980).

For the purposes of calculating the aetiological fractions for driver and rider accident hospitalisations, the relative risks of 3.13 (95% CI: 1.20–8.48) and 12.94 (95% CI: 6.60–25.36) were used for BAC >0.05–0.10 and >0.10 g/100 mL respectively.

Table 3.16: Revised aetiological fractions for alcohol exposure and driver and motorcycle rider accident hospitalisations

	Males		Females	
	Blood alcohol concentration		Blood alcohol concentration	
Age	**>0.05–<0.10**	**≥0.10**	**>0.05–<0.10**	**≥0.10**
Exposed				
All ages	0.688	0.923	0.688	0.923
General population				
<15	0.034	0.212	0.014	0.092
15–19	0.048	0.157	0.021	0.074
20–24	0.041	0.286	0.021	0.129
25–29	0.041	0.286	0.021	0.129
30–34	0.028	0.212	0.014	0.111
35–39	0.028	0.212	0.014	0.111
40–44	0.028	0.212	0.014	0.111
45–49	0.028	0.212	0.014	0.111
50–54	0.021	0.074	0.007	0.018
55–59	0.021	0.074	0.007	0.018
60–64	0.021	0.074	0.007	0.018
65–69	0.021	0.074	0.007	0.018
70–74	0.021	0.074	0.007	0.018
75–79	0.021	0.074	0.007	0.018
80+	0.021	0.074	0.007	0.018
All ages	**0.034**	**0.212**	**0.014**	**0.092**

Source: AIHW analyses of data in Tables 3.9 and 3.15.

For people aged 15–19 years, the aetiological fraction for driver and rider road accident hospitalisations caused by driving with a BAC >0.05 g/100 mL was estimated as 0.205 for males and 0.094 for females. For those aged 20–29 years, the aetiological fraction was estimated as 0.327 for males and 0.150 for females. Among 30–49 year olds, the aetiological fraction was estimated as 0.240 for males and 0.124 for females, and for people aged 50 years or more it was 0.094 for males and 0.025 for females. The overall fraction for all ages was estimated at 0.247 for males and 0.106 for females (Table 3.16). As with driver and rider accident deaths, this all-ages fraction was applied to deaths at ages below 15. These numbers differ slightly from the sums of the corresponding numbers in Table 3.16 because of rounding.

For people aged 16–19 years, the aetiological fraction for pedestrian hospitalisation caused by walking with a BAC >0.10 g/100 mL was estimated as 0.35 for males and 0.16 for females. For those aged 20–29 years, the aetiological fraction was estimated as 0.45 for males and 0.19 for females. Among the 30–49 year olds, the aetiological fraction was estimated as 0.46 for males and 0.21 for females, and for people aged 50 years or more it was 0.23 for males and 0.03 for females (Table 3.17).

Table 3.17: Revised aetiological fractions for alcohol exposure and pedestrian hospitalisations

	Males	Females
	Blood alcohol concentration	Blood alcohol concentration
Age	>=0.10	>=0.10
General population		
16–19	0.35	0.16
20–29	0.45	0.19
30–49	0.46	0.21
50+	0.23	0.03
16+	0.37	0.06

Source: AIHW analysis of data in Table 3.10.

3.2.4 Alcohol and fall injuries

Falls are a leading cause of injury hospitalisation across the majority of age groups. In New South Wales from 1989–90 to 1995–96 falls were the leading cause of injury hospitalisation in every age group except 15–29 years. Among 5–9 year olds, the main type of fall resulting in hospitalisation is falling from play equipment (28.0%) (Public Health Division 1997). Falls among this age group account, however, for very few deaths.

While alcohol does not generally play an aetiological role in fall morbidity and mortality in people aged less than 15 years, its contribution to fall injury among those aged 18–64 years has been described as substantial (Mosenthal et al. 1995). The occurrence of falls increases with age. Among older women living in the community, the proportion having one or more falls in a year increases from 35.0% for 65–79 year olds to 45.0% for 80–89 year olds and 55.0% for those older than 90 years (NHMRC 1993). Coupled with this greater likelihood of falling is an increased likelihood of injury and fractures, resulting in disproportionately high levels of morbidity and mortality attributable to fall injury among the elderly.

In the elderly, fall aetiology is multifactorial. It involves the combination of intrinsic factors (age-related physiological decline or disease) and extrinsic factors (environmental hazards or activity-related risk) in conjunction with drugs (pharmaceutical therapy or alcohol interacting with concurrently administered drugs or on the central nervous system directly) (NHMRC 1993).

English et al. (1995) calculated an aetiological fraction based on three case series for falls caused by alcohol. The aetiological fraction estimate was 0.34, which was applied to both males and females across all age groups. One series included deaths among people aged 15 years or more (Centers for Disease Control 1984), the second included deaths at all ages (Rutledge & Messick 1992), and the third examined the prevalence of acute intoxication (>0.1 g/100 mL) among trauma patients aged 18 years or more (Rivara et al. 1993).

The main study to contribute to the pooled estimate—and the only study to examine incidence—was that by Rivara et al. While the proportion of intoxication among the 398 falls was 0.38, the distribution of intoxication by age group suggests that these falls must have been skewed towards the young (Rivara et al. 1993, p. 909). Overall, the proportion of intoxication for the two oldest age groups in the study (55–64 years and greater than 64 years) was 0.28 and 0.13 respectively. Furthermore, only 200 of the 2,657 study participants

were older than 65 years (57% were younger than 35 years) and the study was overwhelmingly male (77%).

Rutledge and Messick (1992) examined 6,662 deaths among males and females. Of these, 142 were fall related. Of the fall-related deaths, 41 (29%) were classified as intoxicated (>0.1 g/100 mL). Again, from the distribution of intoxication by age (Rutledge & Messick 1992, p. 738), these fall deaths must have been skewed towards the young. The prevalence of intoxication beyond age 65–70 years was minimal, irrespective of the cause of death. From the data presented, it was not possible to differentiate the proportion of males and females in the study.

The study carried out by the Center for Disease Control (1984) reported on 3,293 deaths resulting from intentional and unintentional injuries in Erie County, New York, from 1973 to 1983. These deaths were for people aged 15 years or more who died within eight hours of injury. Intoxication was defined as BAC > 0.1 g/100 mL. Fifty-two deaths were due to falls; 21% of these involved intoxication. From the data presented, it was not possible to differentiate either the proportion of males and females or the age distribution among the 52 fall deaths.

Epidemiological evidence for reviewing the aetiological fraction for alcohol and falls

The differential distribution of harmful/hazardous alcohol intake across age groups

The 1989–90 Australian Bureau of Statistics National Health Survey (ABS 1994) showed that, while 13.9% of 18–24 year olds reported a level of alcohol intake classified as medium or high risk, this decreased to 5.5% for respondents aged 65 years or more. For high-risk consumption the figures were 6.0% and 1.6% respectively. Similar findings were reported for the 1995 National Health Survey, where the prevalence of high-risk alcohol consumption was 4.0% for 15–24 year olds, 1.8% for 65–74 year olds and 1.4% for people aged 75 years or more (ABS 1995a).

Results from the 1997 National Survey of Mental Health and Well-being show the proportions of respondents identified with an alcohol abuse disorder within the 12 months preceeding the survey as 10.6% for those aged 18–34 years and 1.9% for those aged 55 years or more. Similarly, for those with alcohol as a substance abuse disorder, while 13.5% of 18–34 year olds used alcohol in the preceding 12 months, only 3.1% of those aged 55 or more acknowledged alcohol use (Hall et al. 1998).

Of the studies examined by English et al. (1995), Rivara et al. (1993) reported the overall prevalence of acute intoxication (BAC ≥ 0.1 g/100 mL) among trauma patients at its highest among those age 25–34 years (43.2%) and declining to its lowest among those aged 65 years or more (12.6%).

Review of more recent literature identifies a number of studies (Cumming & Klineberg 1994; Cummings et al. 1995; Johnell et al. 1995; Nguyen et al. 1996; O'Neill et al. 1996; Sheahan et al. 1995) that found no association between alcohol consumption and the risk of osteoporotic fractures (mainly hip or distal forearm) after falls among people aged 45 years or more. In some instances, however, the studies were restricted to women (Cummings et al. 1995; Johnell et al. 1995; O'Neill et al. 1996) and none of these studies differentiated between falls occurring within an institutional or a community setting. These features are associated with the exposure of interest (alcohol). Nelson et al. (1992), whose study was restricted to

community-dwelling individuals aged 65 years or more, reported no association between fall injuries and self-reported average weekly alcohol use.

While English et al. derived one aetiological fraction for all age groups, our aetiological fraction has been revised by developing separate fractions for people aged less than 65 years and for people aged 65 years or more, based on the studies reviewed. This is consistent with the epidemiological evidence of a differential distribution across age groups of harmful and hazardous alcohol intake, as described.

The differential distribution between the sexes of both alcohol abuse and acute alcohol intoxication

Both the 1989–90 and 1995 National Health Survey results identified differences in the prevalence of high-risk alcohol consumption between the sexes. The prevalence of high-risk consumption for men was 7.1% for 1989–90 and 8.3% for 1995; the corresponding figures for women were 1.6% and 2.2%. This disparity was apparent across all age groups examined. Of the studies reported by English et al., Rivara et al. identified the disparity in the occurrence of overall acute intoxication in the presence of trauma among both males (39.9%) and females (22.4%).

Again, while English et al. derived one aetiological fraction for both sexes, our aetiological fraction has been revised by developing fractions specific to both males and females, based on the studies reviewed. This is consistent with the epidemiological evidence of a differential distribution of both alcohol abuse and acute intoxication between the sexes.

Falls occurring in individuals aged 65 years or more and living in institutions

The distribution of causes of falls will differ for frail high-risk individuals living in institutions such as residential aged care facilities when compared with that for falls among community-living individuals, where acute alcohol intake is more likely to be a cause of instability.

A number of studies of falls among nursing home residents or the hospitalised elderly have examined the contribution of medications, rather than alcohol, to the falls (Cumming 1996; Rubenstein et al. 1994; Salgado et al. 1994; Thapa et al. 1995; Yip & Cumming 1994). In the residential aged care environment a reduction in psychotropic drug use is likely to be a higher priority than a reduction in acute alcohol use.

Unpublished data from the Institute's National Injury Surveillance Unit show that, for 1996–97, almost one-quarter (12,081 or 24.2%) of the 49,867 falls and one-third (216,817 or 38.5%) of the 562,904 patient days attributable to falls among people aged 65 years or more had their place of occurrence as a residential institution. As might be expected, females accounted for over two-thirds of the 12,081 falls resulting in hospital admissions (8,499 or 70.3%) and two-thirds (145,084 or 66.9%) of the 216,817 patient days resulting from these admissions.

For people aged 65–69 years, the mean length of stay for institutional falls (30.7 days) was four times that for non-institutional falls (7.6 days). Furthermore, the unpublished data from the Institute's National Injury Surveillance Unit show that, while less than 10% of the 6,417 falls in this age group were institutional (549 or 8.5%), these falls accounted for over a quarter (16,616 or 27.0%) of fall injury patient days for this age group. Among people aged 85 years or more, the mean length of stay for institutional falls (15.4 days), while higher, was close to that for non-institutional falls (13.6 days). However, this is likely to reflect the higher occurrence and earlier onset of death among very elderly institutionalised people who are hospitalised for fall injury. Almost one-third (5,551 or 28.4%) of the 19,561 falls in people

aged 85 years or more occurred in an institution, and a slightly higher proportion (85,637 or 31.0%) of all patient days (276,604 days) were similarly due to institutional falls for this age group.

The fractions derived in this section relate to the effect of acute intoxication on falls. They were derived primarily using studies that did not differentiate on the basis of residence. Hence they can be applied to deaths and hospital separations data collected without reference to a person's usual residence. However, given the primacy of drug use, rather than alcohol, in the aetiology of falls in nursing homes and hospitals, we recommend that the fractions not be applied to data focusing on falls in institutions.

Although we acknowledge that chronic alcohol abuse results in individuals requiring institutional care and that disability arising from chronic alcohol abuse will contribute to falls, the effect of chronic alcohol abuse on falls is beyond the scope of this analysis.

Separate aetiological fractions for reporting on incidence or prevalence as opposed to death

Alcohol is more likely to be present among fall fatalities than among non-fatal injuries arising from falls (Hingson & Howland 1993); the severity of the fall injury is also likely to be related to blood alcohol concentration (Smith & Kraus 1988). As a result, two separate fractions—one for fall-related injuries and one for fall-related deaths—would seem desirable.

Studies used to revise the aetiological fraction for alcohol and falls

The studies used to revise the aetiological fraction for alcohol and falls are listed in Appendix B. The literature search for studies on which to base a revised aetiological fraction for alcohol and falls was not restricted to blood alcohol concentration as the criterion for intoxication. English et al. (1995) examined only studies that had BAC as a measure of exposure, so the literature for both the 1985 to 1993 and 1994 to 1998 periods was reviewed to locate papers that examined falls and exposure to alcohol.

Ninety papers that met the search criteria were found. Of these, 44 were reviewed in detail and 15 were chosen as presenting results in a suitable way for use in re-analysis of the aetiological fraction for alcohol and falls (not including the three papers used by English et al.). A range of measures of exposure was allowed for, and where possible data were derived from within each paper so as to determine exposure by age and sex (Appendix B, Table B.8).

Revised aetiological fractions for alcohol and falls

Using the data available (Appendix B, Table B.8) and the epidemiological criteria just discussed, aetiological fractions for falls attributable to alcohol were produced by weighting the contribution of each study by its size. The aetiological fractions were determined separately for males and females aged less than 65 years and 65 years or more (Table 3.18).

Overall, among people aged less than 65 years, 22% of male falls and 14% of female falls were attributable to alcohol; this compares with 12% of male and 4% of female falls among people aged 65 years or more.

Table 3.18: Revised aetiological fractions for alcohol and falls

	Hazardous/harmful alcohol consumption	
Age	**Males**	**Females**
General population		
< 65 years	0.22	0.14
≥ 65 years	0.12	0.04

Source: AIHW analysis of data presented in sources listed in Appendix B, Table B.8.

Differentiating between exposure resulting in death, as opposed to hospitalisation, resulted in few studies remaining available for the derivation of aetiological fractions. Hartshorne et al. (1997) provide prevalence estimates for the contribution of alcohol to falls resulting in death that are consistent with the assumption that alcohol would be more prevalent as a cause of more serious falls that might result in death as opposed to injury. While restricted to 19 males and four females, these results— and those of Rutledge & Messick (1992) and the Centers for Disease Control (1984)—do suggest the need for separate fractions for falls leading to mortality, when the results of more studies become available. Given the limited data having mortality as the sole outcome, the best current estimates of studies pertaining to falls and fatal outcomes remain those derived above.

3.2.5 Aetiological fractions for alcohol updated with recent prevalence data

Where possible, the aetiological fractions were revised to incorporate updated estimates of the prevalence of alcohol consumption. The conditions discussed in sections 3.2.1 to 3.2.4 were also based on revised risk-ratio estimates. Table 3.19 lists the conditions for which the aetiological fractions were revised to incorporate updated estimates of the prevalence of alcohol consumption but that were based on the risk-ratio estimates derived by English et al. (1995). Table 3.20 lists the conditins for which the aetiological fractions were not revised. Table 3.21 lists the values of the revised aetiological fractions. The remainder of this section presents a discussion of those fractions whose revision raises difficulties in addition to the simple application of the updated prevalence estimates to the risk-ratio estimates of English et al.

Ischaemic heart disease

English et al. found a significant body of evidence to support the attribution of a protective effect against ischaemic heart disease to moderate alcohol intake. They found that the protective effect is fully realised within low drinking levels and that no additional benefit is gained with increasing intake. Further, they found that the small apparent increase in risk of ischaemic heart disease with high drinking levels relative to low drinking levels was very weak and not well substantiated with corroborating evidence. They concluded that there was inadequate evidence that the marginal exposure between low and hazardous or harmful alcohol intake is either a cause of or protective against ischaemic heart disease. Since this marginal risk was the focus of their study, they did not calculate an aetiological fraction for alcohol and ischaemic heart disease.

Our study focuses on both the harms and the benefits of alcohol consumption relative to abstaining from alcohol, so we calculated an aetiological fraction using the unscaled risk ratios derived by English et al. and the updated prevalence data.

Heart failure

The ICD-9 classifications 428 (heart failure) and 429 (ill-defined descriptions and complications of heart disease) are non-specific categories that do not identify the underlying pathology. We followed English et al. in assigning deaths and hospital separations in these categories to specific heart disease codes according to the proportional distribution of cases in each of the specific codes. Hence the aetiological fraction is effectively a weighted average of those applying to each specific heart disease condition.

Since ischaemic heart disease was the predominant cause of heart failure, it was the condition with the largest contribution to the weighted average. English et al. excluded ischaemic heart disease from their calculations, so they also excluded heart failure. However, because we included ischaemic heart disease, we also calculated a fraction for heart failure.

Unspecified liver cirrhosis

While ICD-9 provides a classification for cirrhosis of the liver caused by alcohol, it is possible that not all cases of alcoholic liver cirrhosis are recorded as such on death certificates or in morbidity records. English et al. derived relative risk estimates for all liver cirrhosis and alcohol. These were 1.26 for low alcohol consumption and 9.54 for hazardous or harmful levels of consumption. They applied their prevalence estimates to derive overall fractions for liver cirrhosis. If the proportion of total cirrhosis cases assigned to ICD-9 codes for alcoholic liver cirrhosis is less than the corresponding sex-specific aetiological fraction, then the fraction to be applied to unspecified liver cirrhosis is calculated so as to make up the difference. Specifically, the fraction F for unspecified liver cirrhosis is calculated as

$$F = \left[F_a(a+b) - a\right] \Big/ b$$

where

F_a = the overall aetiological fraction for liver cirrhosis and alcohol

a = the number of liver cirrhosis cases assigned to alcoholic liver cirrhosis

b = the number of liver cirrhosis cases assigned to unspecified liver cirrhosis.

The numerator of this formula is an estimate of the total liver cirrhosis cases attributable to alcohol, using the fraction F_a, less the number actually coded to alcoholic liver cirrhosis (a). This is divided by the number of liver cirrhosis cases assigned to unspecified liver cirrhosis (b). The result is an estimate of the proportion of unspecified liver cirrhosis cases that should have been attributed to alcohol.

Low birthweight

English et al. derived risk ratios for low birthweight and alcohol and found a small increase in risk due to hazardous and harmful consumption in pregnancy relative to low consumption. They found an aetiological fraction of 0.0004, which they omitted from their calculations because of its small size. However, when the risk of any alcohol consumption is assessed relative to abstaining, we find an increase in risk resulting from high levels of consumption that is almost exactly balanced by a decrease in risk resulting from low levels of consumption. We included this fraction in our calculations, despite the fact that its overall value is close to zero, to enable estimation of the cases associated with high-level drinking and the benefits associated with low-level drinking.

Table 3.19: Conditions where aetiological fractions were based on the English et al. risk-ratio estimates but revised to incorporate updated prevalence estimates

Condition	Source of prevalence data
Oropharyngeal cancer	AIHW analysis of 1995 ABS National Health Survey
Oesophageal cancer	AIHW analysis of 1995 ABS National Health Survey
Liver cancer	AIHW analysis of 1995 ABS National Health Survey
Laryngeal cancer	AIHW analysis of 1995 ABS National Health Survey
Hypertension	AIHW analysis of 1995 ABS National Health Survey
Ischaemic heart disease	AIHW analysis of 1995 ABS National Health Survey
Supraventricular cardiac dysrhythmias	AIHW analysis of 1995 ABS National Health Survey
Heart failure	AIHW analysis of 1995 ABS National Health Survey
Unspecified liver cirrhosis	AIHW analysis of 1995 ABS National Health Survey
Cholelithiasis	AIHW analysis of 1995 ABS National Health Survey
Low birthweight	1998 National Drug Strategy Household Survey
Psoriasis	AIHW analysis of 1995 ABS National Health Survey
Suicide and self-inflicted injury	AIHW analysis of 1995 ABS National Health Survey

3.3 Aetiological fractions for alcohol left unrevised

The aetiological fractions for alcohol that were not revised are listed in Table 3.30. A number of the conditions have an aetiological fraction of one. They are conditions—for example, alcoholic psychosis—that are defined by association with alcohol. The remainder are conditions for which no more recent data could be found on which to base a revision, so they have been left at the values derived by English et al. (1995). They are discussed in the following paragraphs.

3.3.1 Epilepsy

English et al. recommended the use of an aetiological fraction for epilepsy of 0.15. This estimate was derived from four clinical case series and was recommended for use until further epidemiological evidence accumulates.

3.3.2 Oesophageal varices

Apart from some rare conditions such as portal or hepatic vein occlusion due to thrombosis or portal lymphadenopathy, virtually all oesophageal varices are a result of liver cirrhosis. On this basis, English et al. recommended applying the overall liver cirrhosis fraction to oesophageal varices.

3.3.3 Gastro-oesophageal haemorrhage

English et al. based their fraction for gastro-oesophageal haemorrhage on a case series of 38 patients in Belfast. The estimate was 0.47 and, in the absence of better or more recent data, we used the same value.

3.3.4 Pancreatitis, acute and chronic

English et al. did not identify any epidemiological studies that examined the relative risk of acute pancreatitis in association with alcohol, and none of the corresponding studies they identified for chronic pancreatitis were in a form suitable for inclusion in a meta-analysis. Instead, they derived separate fractions for acute pancreatitis and for chronic pancreatitis from case series—five for acute pancreatitis and another five for chronic pancereatitis. Their estimates were 0.24 for acute pancreatitis and 0.84 for chronic pancreatitis and, in the absence of better or more recent data, we used the same values.

3.3.5 Fire injuries

English et al. identified only one epidemiological study in which the risk of fire injuries in association with alcohol intake was examined, but the results were not presented in a form suitable for their report. Instead, they used five blood alcohol case series to derive the fraction. Their estimate was 0.44 and, in the absence of better or more recent data, we used the same value.

3.3.6 Drowning

English et al. did not identify any epidemiological studies that examined the relative risk of drowning in association with alcohol. Instead, they used six blood alcohol case series to derive the fraction. Their estimate was 0.34 and, in the absence of better or more recent data, we used the same value.

3.3.7 Aspiration

English et al. were advised by staff of the Australian Bureau of Statistics that use of the aspiration code in adults is virtually confined to cases of aspiration of vomitus in alcoholics. Hence they assigned the fraction a value of one at ages 15 and over. We used the same value.

3.3.8 Occupational and machine injuries

English et al. did not identify any epidemiological studies that examined the relative risk of occupational and machine injuries in association with alcohol. Instead, they used two blood alcohol case series to derive the fraction. Their estimate was 0.07 and, in the absence of better or more recent data, we used the same value.

3.3.9 Assault

English et al. did not identify any epidemiological studies that examined the relative risk of assault in association with alcohol. Instead, they used five clinical case series to derive the fraction. Their estimate was 0.47 and, in the absence of better or more recent data, we used the same value.

3.3.10 Child abuse

English et al. did not identify any epidemiological studies that examined the relative risk of child abuse in association with alcohol. Instead, they used eight clinical case series to derive

the fraction. Their estimate was 0.16 and, in the absence of better or more recent data, we used the same value.

Table 3.20: Aetiological fractions not revised

Condition	Reason for not revising fraction	Fraction value
Alcoholic psychosis	Fraction = 1 by definition	1.00
Alcohol dependence/abuse	Fraction = 1 by definition	1.00
Alcoholic liver cirrhosis	Fraction = 1 by definition	1.00
Epilepsy	Insufficient information on which to base a revision	0.15
Alcoholic poly neuropathy	Fraction = 1 by definition	1.00
Alcoholic cardiomyopathy	Fraction = 1 by definition	1.00
Oesophageal varices	Use fraction for unspecified liver cirrhosis	Males 0.59, females 0.56
Gastro-oesophageal haemorrhage	Insufficient information on which to base a revision	0.47
Alcoholic gastritis	Fraction = 1 by definition	1.00
Pancreatitis, acute and chronic	Insufficient information on which to base a revision	Acute 0.24, chronic 0.84
Ethanol/methanol toxicity	Fraction = 1 by definition	1.00
Alcoholic beverage poisoning	Fraction = 1 by definition	1.00
Other ethanol and methanol poisoning	Fraction = 1 by definition	1.00
Fire injuries	Insufficient information on which to base a revision	0.44
Drowning	Insufficient information on which to base a revision	0.34
Aspiration	Fraction assigned the value of 1	1.00
Occupational and machine injuries	Insufficient information on which to base a revision	0.07
Assault	Insufficient information on which to base a revision	0.47
Child abuse	Insufficient information on which to base a revision	0.16

Table 3.21: Revised values for fractions based on the English et al. risk-ratio estimates and updated prevalence data

1. Fractions directly updated with population prevalence data

Oropharyngeal cancer (ICD-9 codes 141, 143–146, 148–149)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.310	0.459	0.814	0.310	0.459	0.814
General population						
18–19	0.181	0.033	0.210	0.185	0.073	0.079
20–24	0.181	0.033	0.210	0.185	0.073	0.079
25–29	0.189	0.038	0.188	0.193	0.057	0.062
30–34	0.181	0.044	0.182	0.205	0.053	0.059
35–39	0.191	0.042	0.138	0.159	0.067	0.071
40–44	0.182	0.050	0.155	0.172	0.056	0.064
45–49	0.179	0.056	0.154	0.152	0.054	0.095
50–54	0.191	0.042	0.166	0.141	0.066	0.114
55–59	0.201	0.045	0.105	0.150	0.070	0.077
60–64	0.173	0.052	0.188	0.162	0.076	0.034
65–69	0.199	0.040	0.126	0.147	0.087	0.044
70–74	0.197	0.032	0.158	0.159	0.054	0.036
75–79	0.198	0.014	0.177	0.159	0.029	0.030
80+	0.214	0.030	0.088	0.188	0.027	0.050
Total (18+)	**0.185**	**0.042**	**0.168**	**0.175**	**0.063**	**0.068**

Oesophageal cancer (ICD-9 code 150)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.444	0.578	0.765	0.444	0.578	0.765
General population						
18–19	0.290	0.048	0.141	0.281	0.101	0.050
20–24	0.290	0.048	0.141	0.281	0.101	0.050
25–29	0.300	0.054	0.124	0.294	0.078	0.040
30–34	0.288	0.064	0.120	0.310	0.072	0.037
35–39	0.298	0.059	0.090	0.246	0.094	0.046
40–44	0.286	0.072	0.101	0.265	0.079	0.041
45–49	0.280	0.080	0.101	0.240	0.078	0.063
50–54	0.300	0.059	0.109	0.224	0.095	0.076
55–59	0.309	0.062	0.067	0.233	0.099	0.050
60–64	0.275	0.075	0.125	0.247	0.106	0.022
65–69	0.308	0.057	0.082	0.226	0.121	0.028
70–74	0.310	0.046	0.104	0.247	0.076	0.023
75–79	0.315	0.021	0.118	0.249	0.041	0.020
80+	0.328	0.041	0.056	0.291	0.038	0.033
Total (18+)	**0.292**	**0.061**	**0.110**	**0.268**	**0.088**	**0.044**

Liver cancer (ICD-9 code 155)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.310	0.670	0.722	0.310	0.670	0.722
General population						
18–19	0.188	0.082	0.130	0.172	0.164	0.044
20–24	0.188	0.082	0.130	0.172	0.164	0.044
25–29	0.194	0.092	0.114	0.183	0.128	0.035
30–34	0.184	0.107	0.109	0.196	0.120	0.033
35–39	0.191	0.100	0.082	0.149	0.150	0.040
40–44	0.181	0.119	0.091	0.163	0.128	0.036
45–49	0.176	0.132	0.090	0.147	0.125	0.054
50–54	0.193	0.100	0.099	0.135	0.150	0.065
55–59	0.197	0.105	0.061	0.140	0.157	0.043
60–64	0.174	0.125	0.112	0.148	0.167	0.018
65–69	0.198	0.096	0.074	0.134	0.188	0.024
70–74	0.201	0.079	0.096	0.150	0.122	0.020
75–79	0.209	0.037	0.111	0.155	0.066	0.018
80+	0.213	0.071	0.052	0.185	0.063	0.029
Total (18+)	**0.187**	**0.102**	**0.100**	**0.165**	**0.143**	**0.038**

Laryngeal cancer (ICD-9 code 161)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.454	0.744	0.797	0.454	0.744	0.797
General population						
18–19	0.276	0.093	0.155	0.257	0.189	0.053
20–24	0.276	0.093	0.155	0.257	0.189	0.053
25–29	0.283	0.104	0.137	0.276	0.149	0.043
30–34	0.270	0.122	0.131	0.292	0.139	0.041
35–39	0.282	0.114	0.099	0.227	0.177	0.049
40–44	0.267	0.136	0.110	0.249	0.151	0.045
45–49	0.259	0.151	0.109	0.225	0.148	0.068
50–54	0.283	0.114	0.119	0.205	0.177	0.081
55–59	0.293	0.121	0.074	0.214	0.186	0.053
60–64	0.255	0.142	0.135	0.227	0.198	0.023
65–69	0.293	0.110	0.090	0.204	0.223	0.030
70–74	0.297	0.090	0.115	0.233	0.147	0.025
75–79	0.309	0.042	0.134	0.244	0.081	0.023
80+	0.318	0.082	0.064	0.285	0.075	0.037
Total (18+)	**0.275**	**0.117**	**0.121**	**0.249**	**0.167**	**0.047**

Hypertension (ICD-9 codes 410–405)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.020	0.301	0.512	–0.176	0.213	0.441
General population						
18–19	0.012	0.025	0.077	–0.096	0.036	0.022
20–24	0.012	0.025	0.077	–0.096	0.036	0.022
25–29	0.013	0.029	0.068	–0.099	0.028	0.017
30–34	0.012	0.034	0.065	–0.107	0.026	0.017
35–39	0.012	0.031	0.048	–0.077	0.031	0.019
40–44	0.012	0.037	0.054	–0.084	0.026	0.017
45–49	0.012	0.041	0.054	–0.075	0.025	0.025
50–54	0.013	0.031	0.059	–0.070	0.031	0.030
55–59	0.013	0.032	0.036	–0.072	0.032	0.020
60–64	0.012	0.039	0.068	–0.077	0.034	0.009
65–69	0.013	0.029	0.043	–0.069	0.039	0.011
70–74	0.013	0.024	0.056	–0.074	0.024	0.009
75–79	0.013	0.011	0.063	–0.071	0.012	0.007
80+	0.013	0.021	0.030	–0.091	0.012	0.013
Total (18+)	**0.012**	**0.032**	**0.059**	**–0.087**	**0.030**	**0.018**

Ischaemic heart disease (ICD-9 codes 410–414)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	–0.220	–0.190	–0.136	–0.220	–0.190	–0.136
General population						
18–19	–0.147	–0.013	–0.012	–0.129	–0.024	–0.004
20–24	–0.147	–0.013	–0.012	–0.129	–0.024	–0.004
25–29	–0.152	–0.014	–0.010	–0.129	–0.018	–0.003
30–34	–0.143	–0.016	–0.010	–0.139	–0.017	–0.003
35–39	–0.141	–0.015	–0.007	–0.102	–0.020	–0.003
40–44	–0.138	–0.018	–0.008	–0.110	–0.017	–0.003
45–49	–0.136	–0.020	–0.008	–0.098	–0.016	–0.004
50–54	–0.149	–0.015	–0.009	–0.093	–0.020	–0.005
55–59	–0.145	–0.015	–0.005	–0.095	–0.021	–0.003
60–64	–0.137	–0.019	–0.010	–0.100	–0.022	–0.001
65–69	–0.146	–0.014	–0.006	–0.091	–0.025	–0.002
70–74	–0.151	–0.012	–0.008	–0.094	–0.015	–0.001
75–79	–0.151	–0.005	–0.009	–0.089	–0.008	–0.001
80+	–0.149	–0.010	–0.004	–0.115	–0.008	–0.002
Total (18+)	**–0.143**	**–0.015**	**–0.009**	**–0.115**	**–0.020**	**–0.003**

Supraventricular cardiac dysrhythmias (ICD-9 codes 427.0, 427.2, 427.3)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.338	0.552	0.552	0.338	0.552	0.552
General population						
18–19	–0.147	–0.013	–0.012	–0.129	–0.024	–0.004
20–24	–0.147	–0.013	–0.012	–0.129	–0.024	–0.004
25–29	–0.152	–0.014	–0.010	–0.129	–0.018	–0.003
30–34	–0.143	–0.016	–0.010	–0.139	–0.017	–0.003
35–39	–0.141	–0.015	–0.007	–0.102	–0.020	–0.003
40–44	–0.138	–0.018	–0.008	–0.110	–0.017	–0.003
45–49	–0.136	–0.020	–0.008	–0.098	–0.016	–0.004
50–54	–0.149	–0.015	–0.009	–0.093	–0.020	–0.005
55–59	–0.145	–0.015	–0.005	–0.095	–0.021	–0.003
60–64	–0.137	–0.019	–0.010	–0.100	–0.022	–0.001
65–69	–0.146	–0.014	–0.006	–0.091	–0.025	–0.002
70–74	–0.151	–0.012	–0.008	–0.094	–0.015	–0.001
75–79	–0.151	–0.005	–0.009	–0.089	–0.008	–0.001
80+	–0.149	–0.010	–0.004	–0.115	–0.008	–0.002
Total (18+)	**–0.143**	**–0.015**	**–0.009**	**–0.115**	**–0.020**	**–0.003**

Cholelithiasis (ICD-9 code 574)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	–0.220	–0.471	–1.000	–0.220	–0.471	–1.000
General population						
18–19	–0.155	–0.026	–0.051	–0.133	–0.050	–0.016
20–24	–0.155	–0.026	–0.051	–0.133	–0.050	–0.016
25–29	–0.159	–0.030	–0.045	–0.133	–0.037	–0.012
30–34	–0.150	–0.035	–0.043	–0.143	–0.035	–0.012
35–39	–0.147	–0.030	–0.030	–0.105	–0.042	–0.013
40–44	–0.145	–0.038	–0.035	–0.113	–0.035	–0.012
45–49	–0.142	–0.042	–0.035	–0.101	–0.034	–0.018
50–54	–0.156	–0.032	–0.038	–0.096	–0.042	–0.022
55–59	–0.149	–0.031	–0.022	–0.098	–0.043	–0.014
60–64	–0.145	–0.041	–0.045	–0.103	–0.046	–0.006
65–69	–0.151	–0.029	–0.027	–0.094	–0.052	–0.008
70–74	–0.157	–0.024	–0.036	–0.096	–0.031	–0.006
75–79	–0.157	–0.011	–0.040	–0.090	–0.015	–0.005
80+	–0.153	–0.020	–0.018	–0.117	–0.016	–0.009
Total (18+)	**–0.149**	**–0.032**	**–0.038**	**–0.118**	**–0.040**	**–0.013**

Psoriasis (ICD-9 code 696.1)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.367	0.375	0.545	0.367	0.375	0.545
General population						
18–19	0.262	0.026	0.065	0.244	0.053	0.022
20–24	0.262	0.026	0.065	0.244	0.053	0.022
25–29	0.268	0.029	0.057	0.251	0.040	0.017
30–34	0.258	0.035	0.055	0.264	0.037	0.016
35–39	0.261	0.031	0.040	0.210	0.048	0.020
40–44	0.254	0.038	0.046	0.225	0.040	0.018
45–49	0.249	0.043	0.046	0.205	0.040	0.027
50–54	0.267	0.032	0.049	0.194	0.049	0.033
55–59	0.268	0.033	0.030	0.199	0.051	0.022
60–64	0.248	0.041	0.057	0.209	0.054	0.009
65–69	0.269	0.030	0.036	0.193	0.062	0.012
70–74	0.273	0.024	0.046	0.205	0.038	0.010
75–79	0.276	0.011	0.052	0.202	0.020	0.008
80+	0.279	0.021	0.024	0.240	0.019	0.014
Total (18+)	**0.259**	**0.033**	**0.050**	**0.229**	**0.046**	**0.019**

Suicide and self–inflicted injury (ICD-9 codes E950–E959)

	Male			Female		
	Level of exposure			Level of exposure		
Age	**Low**	**Hazardous**	**Harmful**	**Low**	**Hazardous**	**Harmful**
Exposed population						
All ages	0.286	0.569	0.603	0.286	0.569	0.603
General population						
18–19	0.187	0.059	0.084	0.169	0.118	0.028
20–24	0.187	0.059	0.084	0.169	0.118	0.028
25–29	0.192	0.066	0.074	0.177	0.091	0.022
30–34	0.182	0.078	0.071	0.188	0.085	0.021
35–39	0.186	0.071	0.052	0.145	0.107	0.025
40–44	0.179	0.086	0.059	0.158	0.090	0.023
45–49	0.174	0.096	0.059	0.142	0.089	0.035
50–54	0.190	0.072	0.064	0.132	0.108	0.042
55–59	0.192	0.074	0.039	0.137	0.112	0.027
60–64	0.173	0.091	0.073	0.144	0.118	0.012
65–69	0.193	0.068	0.048	0.130	0.134	0.015
70–74	0.197	0.056	0.061	0.143	0.085	0.012
75–79	0.202	0.026	0.071	0.144	0.045	0.011
80+	0.204	0.050	0.033	0.174	0.043	0.018
Total (18+)	**0.184**	**0.074**	**0.065**	**0.160**	**0.101**	**0.024**

2. Other updated fractions

Heart failure: deaths (ICD-9 code 428–429)

	Male			Female		
	Year			Year		
Age	**1996**	**1997**	**1998**	**1996**	**1997**	**1998**
General population						
18–19	–0.034	–0.016	–0.016	0.005	0.000	0.000
20–24	–0.049	–0.043	0.007	–0.026	–0.026	–0.045
25–29	–0.087	–0.006	–0.049	–0.056	–0.032	0.004
30–34	–0.068	–0.029	–0.081	–0.075	–0.071	0.028
35–39	–0.069	–0.103	–0.132	–0.081	–0.089	–0.043
40–44	–0.103	–0.132	–0.112	–0.049	–0.029	–0.082
45–49	–0.101	–0.096	–0.115	–0.070	–0.081	–0.072
50–54	–0.119	–0.126	–0.119	–0.060	–0.066	–0.084
55–59	–0.126	–0.121	–0.124	–0.088	–0.087	–0.075
60–64	–0.131	–0.126	–0.130	–0.101	–0.102	–0.102
65–69	–0.137	–0.136	–0.136	–0.094	–0.094	–0.083
70–74	–0.143	–0.140	–0.139	–0.088	–0.088	–0.086
75–79	–0.138	–0.139	–0.139	–0.079	–0.077	–0.077
80+	–0.133	–0.135	–0.135	–0.101	–0.101	–0.099
Total (18+)	**–0.133**	**–0.135**	**–0.135**	**–0.100**	**–0.101**	**–0.099**

Note: Fractions for heart failure deaths are derived as a weighted average of the fractions for other specified heart conditions. The weights are derived from the number of deaths coded to each specific condition. Hence the fractions vary by year.

Heart failure: separations (ICD-9 code 428–429)

	Male			Female		
	Year			Year		
Age	**1996**	**1997**	**1998**	**1996**	**1997**	**1998**
General population						
18–19	0.048	0.075	0.061	0.053	0.057	–0.038
20–24	0.075	0.072	0.075	0.048	0.046	–0.038
25–29	0.048	0.059	0.055	0.022	0.018	–0.054
30–34	0.019	0.022	0.018	0.010	0.004	–0.064
35–39	–0.051	–0.052	–0.036	–0.004	0.000	–0.028
40–44	–0.083	–0.078	–0.077	–0.027	–0.031	–0.041
45–49	–0.098	–0.094	–0.089	–0.038	–0.035	–0.024
50–54	–0.111	–0.108	–0.102	–0.044	–0.049	–0.008
55–59	–0.107	–0.103	–0.098	–0.050	–0.046	–0.020
60–64	–0.101	–0.098	–0.096	–0.055	–0.051	–0.034
65–69	–0.101	–0.096	–0.092	–0.050	–0.045	–0.019
70–74	–0.097	–0.094	–0.092	–0.043	–0.038	–0.041
75–79	–0.084	–0.083	–0.078	–0.035	–0.033	–0.052
80+	–0.081	–0.077	–0.070	–0.051	–0.048	–0.065
Total (18+)	**–0.081**	**–0.077**	**–0.070**	**–0.046**	**–0.044**	**–0.065**

Note: Fractions for heart failure separations are derived as a weighted average of the fractions for other specified heart conditions. The weights are derived from the number of separations coded to each specific condition. Hence the fractions vary by year.

Unspecified liver cirrhosis: deaths (ICD-9 code 571.5–571.9)

	Male			Female		
	Year			Year		
Age	**1996**	**1997**	**1998**	**1996**	**1997**	**1998**
General population						
18–19	0.000	0.000	0.000	0.059	0.084	0.000
20–24	0.000	0.000	0.000	0.059	0.084	0.000
25–29	0.000	0.000	0.000	0.059	0.084	0.000
30–34	0.000	0.000	0.000	0.059	0.084	0.000
35–39	0.000	0.000	0.000	0.059	0.084	0.000
40–44	0.000	0.000	0.000	0.059	0.084	0.000
45–49	0.000	0.000	0.000	0.059	0.084	0.000
50–54	0.000	0.000	0.000	0.059	0.084	0.000
55–59	0.000	0.000	0.000	0.059	0.084	0.000
60–64	0.000	0.000	0.000	0.059	0.084	0.000
65–69	0.000	0.000	0.000	0.059	0.084	0.000
70–74	0.000	0.000	0.000	0.059	0.084	0.000
75–79	0.000	0.000	0.000	0.059	0.084	0.000
80+	0.000	0.000	0.000	0.059	0.084	0.000
Total (18+)	**0.000**	**0.000**	**0.000**	**0.059**	**0.084**	**0.000**

Note: Fractions for unspecified liver cirrhosis deaths are derived using the counts of deaths coded to alcoholic liver cirrhosis and to overall liver cirrhosis. Hence the fractions vary by year.

Unspecified liver cirrhosis: separations (ICD-9 code 571.5–571.9)

	Male			Female		
	Year			Year		
Age	**1996**	**1997**	**1998**	**1996**	**1997**	**1998**
General population						
18–19	0.000	0.000	0.000	0.206	0.212	0.205
20–24	0.000	0.000	0.000	0.206	0.212	0.205
25–29	0.000	0.000	0.000	0.206	0.212	0.205
30–34	0.000	0.000	0.000	0.206	0.212	0.205
35–39	0.000	0.000	0.000	0.206	0.212	0.205
40–44	0.000	0.000	0.000	0.206	0.212	0.205
45–49	0.000	0.000	0.000	0.206	0.212	0.205
50–54	0.000	0.000	0.000	0.206	0.212	0.205
55–59	0.000	0.000	0.000	0.206	0.212	0.205
60–64	0.000	0.000	0.000	0.206	0.212	0.205
65–69	0.000	0.000	0.000	0.206	0.212	0.205
70–74	0.000	0.000	0.000	0.206	0.212	0.205
75–79	0.000	0.000	0.000	0.206	0.212	0.205
80+	0.000	0.000	0.000	0.206	0.212	0.205
Total (18+)	**0.000**	**0.000**	**0.000**	**0.206**	**0.212**	**0.205**

Note: Fractions for unspecified liver cirrhosis separations are derived using the counts of separations coded to alcoholic liver cirrhosis and to overall liver cirrhosis. Hence the fractions vary by year.

Low birthweight

	Level of exposure	
	Low	**Hazardous or harmful**
Exposed population	–0.124	0.383
General population	–0.034	0.035

4 Tobacco

4.1 Introduction

As with alcohol and illicit drugs, we revised the aetiological fractions for tobacco to incorporate recent data on prevalence wherever such data were available. In addition, we revised the risk-ratio estimates for tobacco and cervix cancer and tobacco and peptic ulcer. Finally, we incorporated estimates of aetiological fractions associated with passive smoking, based on the National Health and Medical Research Council's report on passive smoking (NHMRC 1997).

As part of the revision of prevalence data, we also dealt with the question of the time lag between tobacco exposure and disease onset. This is discussed in detail in section 4.1.1; briefly it involves the derivation of a synthetic prevalence estimate which represents past exposure to tobacco rather than current exposure.

4.1.1 Aetiological fractions associated with cancer and chronic obstructive pulmonary disease

English et al. (1995) used an estimate of current smoking prevalence in their calculation of aetiological fractions for tobacco. But for many conditions there is a long time lag between exposure to tobacco smoke and the associated ill-effects—in the case of cancer it may be many decades—so for these conditions estimates of the current prevalence of smoking are not helpful in understanding the current associated disease burden.

We have followed the Australian Burden of Disease Study (Mathers et al. 1999) in using the method proposed by Peto et al. (1992) to adjust for the time lag. Peto et al. proposed using an artificial compound prevalence measure of tobacco exposure, derived from a comparison between lung cancer rates in the country of interest and lung cancer rates among non-smokers observed in a large long-term follow-up study in the United States. This method was used here to determine tobacco exposure for the cancers on our risk factor list and for chronic obstructive pulmonary disease. The mean time between tobacco exposure and the onset of the other illnesses and injuries discussed in this chapter is considerably shorter than that for cancer and chronic obstructive pulmonary disease, so the estimates of current tobacco exposure described in Chapter 1 were used for these other conditions.

4.2 Revised aetiological fractions for tobacco

4.2.1 Tobacco and cervix cancer

English et al. (1995) concluded that, while there is limited evidence that smoking causes cervix cancer, a causal interpretation of the association is credible, although confounding cannot be ruled out with confidence. Confounding could be due to known risk factors for cervix cancer, particularly the number of sexual partners and infection with the human papilloma virus (HPV). If confounding due to HPV infection were to explain the

relationship between cigarette smoking and the risk of cervix cancer, there would be little association between cigarette smoking and cervix cancer among women known to be infected with HPV.

Epidemiological evidence for reviewing the aetiological fraction for tobacco and cervix cancer

English et al. (1995) found, from a meta-analysis of 14 studies, that female ex-smokers had a relative risk of 1.31 (95% CI: 1.21–1.43) and female current smokers had a relative risk of 1.75 (95% CI: 1.66–1.85). The results for current smokers were broadly consistent with a previously published meta-analysis that reported a relative risk of 1.42 (95% CI: 1.33–1.51) for female smokers after adjusting for age and the number of sexual partners (Sood 1991). However, another meta-analysis, while confirming that female current smokers were at increased risk, did not confirm the significantly elevated risk for ex–smokers (Licciardone et al. 1990).

The overall aetiological fraction derived by English et al. for cervix cancer caused by smoking was estimated as 0.19. Thus 19% of cervix cancer was attributed to cigarette smoking.

The major risk factor for cervix cancer has been shown to be the human papilloma virus (Bosch et al. 1994b; Eluf Neto et al. 1994; Munoz et al. 1994), the association between HPV infection and cervix cancer being reflected in odds ratios ranging from 15 to 100 (Bosch et al. 1994a). Cervix cancer risk for a woman depends largely on the probability of being infected with some specific types of HPV (Bosch et al. 1994b).

Risk factors usually strongly associated with cervical neoplasia—such as number of sexual partners or age at first sexual intercourse—were no longer associated with cervix cancer among women who were HPV DNA positive, while the association persisted among women who were HPV DNA negative. Similarly, the odds ratio for the association between smoking status and cervix cancer ranged from 1.4 to 2.0 after adjustment for confounders including HPV. However, when HPV-positive women only were analysed (removing confounding due to undetected HPV), the odds ratio attributable to smoking was not statistically different from one (Bosch et al. 1994a).

Phillips and Davey Smith (1994) discussed the likelihood that the association between cigarette smoking and sexual activity makes the evaluation of the role of smoking difficult. This is because of confounding due to the presence of the aetiological pathogen (HPV), which is transmitted through sexual activity.

They noted that studies of the association between smoking and cervix cancer have adjusted for the lifetime number of sexual partners as a proxy measure of the presence of the aetiological pathogen and, in most cases, the association with smoking has diminished but remained significant. Use of a proxy tends, however, to result in an underestimation of the effect of the aetiological pathogen (HPV) on the risk of cervix cancer. Hence the adjustment is also likely to be insufficient, resulting in an overestimation of the adjusted or 'independent' effect of smoking. Using realistic estimates of the association between the presence of the aetiological pathogen (HPV) and both smoking and the risk of cervix cancer, Phillips and Davey Smith generated 'independent' relative risks for cigarette smoking of two and above. Thus they concluded that the observed 'independent' effect of cigarette smoking on cervical cancer arises because of residual confounding.

Revised aetiological fractions for tobacco and cervix cancer

Sexually transmitted viruses of the HPV type have been shown to be present in high grade squamous pre-cancer and cancer of the cervix. That the 'independent' effect of smoking with regard to cervix cancer as an outcome arises because of residual confounding does not support a causal relationship between smoking and cervix cancer; as a result the fraction should be zero.

4.2.2 Tobacco and peptic ulcer

English et al. (1995) and Ashley (1997) claimed that smoking increases the risk of the occurrence of peptic ulcer, delays healing (with or without treatment) and increases the risk of recurrence after healing. Thus they said the relationship between smoking and peptic ulcer is causal. English et al. found that 41% of peptic ulcer disease in males and 33% in females is caused by cigarette smoking. Studies published before that of English et al. have also published attributable fractions for cigarette smoking in the aetiology of peptic ulcer disease. Kurata et al. (1986) found that between 43% and 63% of duodenal ulcer mortality for males and between 25% and 50% for females could be attributed to smoking. Schoon et al. (1991) estimated that, among people aged 35–84 years, 24.4% of ulcers diagnosed for the first time and 42.0% of relapsing ulcers were caused by smoking. Johnsen et al. (1994) found that 53.0% of duodenal ulcer disease and 60.0% of gastric ulcer disease was attributable to daily cigarette smoking.

Epidemiological evidence for reviewing the aetiological fraction for tobacco and peptic ulcer

In the last 20 years there has been increasing recognition of the role that *Campylobacter pylori* or *Helicobacter pylori* infection may have as a major contributing factor to peptic ulcer disease (Everhart et al. 1998). In 1989 it was reported that the use of non-steroidal anti-inflammatory drugs and the presence of antibodies to *C. pylori* identified people at risk for peptic ulcer disease and that smoking increased this risk in subjects with *C. pylori* (Martin et al. 1989). An absence of a history of NSAID use and antibody to *C. pylori* therefore identifies individuals with a low probability of ulcer disease. This is confirmed in the more recent review by Blum (1996) that also reported that ulcer development in the absence of *H. pylori* is extremely rare in those not taking NSAIDs. While peptic ulcer disease is still described as a multifactorial condition that is influenced by a number of environmental factors (including smoking), in the absence of *H. pylori* infection these factors would not normally lead to ulcer formation (Blum 1996).

Gastritis induced by *H. pylori* is a powerful risk factor for peptic ulcer disease. The risk of developing peptic ulcer is at least 15 times higher in those infected with *H. pylori* when compared with those not infected with *H. pylori*. Furthermore, eradication of *C. pylori* or *H. pylori* leads to the cure of peptic ulcer disease and long-term remission (Blum 1996). Numerous studies have shown that eradication therapy significantly reduces the rate of relapse and complication associated with genuine non-NSAID induced duodenal ulcer (Chan et al. 1997; Hunt & Mohamed 1995; Labenz & Borsch 1994b; Labenz & Borsch 1994c; Marshall et al. 1988; Rauws & Tytgat 1990; Tytgat & Rauws 1990) or gastric ulcer (Chan et al. 1997; Labenz & Borsch 1994a; Labenz & Borsch 1994b; Labenz & Borsch 1994c). This marked decrease in the rate of recurrence of peptic ulcer disease following the eradication of infection provides the strongest evidence for the pathogenic role of *H. pylori* in peptic ulcer disease (NIH 1994).

Since there is no protection from reinfection after the cure of a first infection (Blum 1996), reinfection in adults can occur at a rate similar to the infection rate in adults. This is, however, rare (Cullen et al. 1993) and in industrialised countries amounts to about 1% a year. The four-year follow-up by Labenz (1994b) reported the reinfection rate one year after successful eradication was 2.6% (ulcer relapse 1.1%) and after two years reinfection had risen to 3.2% (ulcer relapse 1.6%). No further increase in reinfection was found during the subsequent two years. More recently, the 1% a year *H. pylori* infection rate among adults was confirmed by an Australian study of Sydney and Melbourne residents (Lin et al. 1998).

From the data available, a further difficulty in establishing a causal relationship between *H. pylori* and peptic ulcer disease is that only a small proportion of individuals harboring the organism develop ulceration (National Institute of Health 1994). It is hypothesised that diversity among *H. pylori* strains is in part responsible for the observed variability in the outcome of the infection (Blaser 1994). That only certain strains of *H. pylori* cause ulceration and that their ulcerogenic potential appears to be associated with the presence of strain-specific factors, such as the *cagA* gene (Blaser 1994), are further evidence for the pathogenic role of *H. pylori* in peptic ulcer disease (Blum 1996).

Studies used to revise the aetiological fraction for tobacco and peptic ulcer

The biological evidence cited in support of an aetiological role for cigarette smoking is that in general nicotine appears to act by potentiating the adverse effects of gastric aggressive factors such as acid and pepsin secretion, motility, duodenogastric reflux, the risk of *H. pylori* infection, levels of free radicals, vasopressin secretion, platelet activating factor generation, and endothelin generation. At the same time, nicotine attenuates defensive mechanisms by decreasing mucosal blood flow, prostaglandin synthesis, mucus secretion and epidermal growth factor secretion (Ashley 1997).

However, none of the studies used by English et al., and four of the six studies they excluded in deriving the aetiological fraction, considered the direct influence of *C. pylori* or *H. pylori* infection. Of the remaining two studies that did consider the influence of *H. pylori,* the most recent (Bateson 1993) found that, while the association of peptic ulcer disease with both *H. pylori* infection and cigarette smoking was confirmed, the excess of peptic ulcer disease in cigarette smokers may have been explained by their increased susceptibility to *H. pylori* infection. The other study—by Martin et al. 1989—found that, while smokers (>10 cigarettes/day) were more likely (41%; 11/27) to have an ulcer than non-smokers (20%; 16/80; $p < 0.05$), this was only because of the increased prevalence of ulcers in smokers who also had *C. pylori* (smokers: 73%; 11/15 in contrast with non-smokers: 29%; 13/45). Therefore, while smoking increased the risk in subjects with *C. pylori*, absence of a history of NSAID use and antibodies to *C. pylori* identified individuals with a low probability of ulcer disease (Martin et al. 1989).

The literature search found nine further studiesthat accounted for *H. pylori* infection and implicated smoking in the aetiology of peptic ulcer disease. Three studies were reviews (Eastwood 1997; Lam 1994b; Parsonnet 1998). Four papers (Archimandritis et al. 1995; Leoci et al. 1995; Menzel et al. 1995; Wang et al. 1996) gave estimates of relative risk for smoking status and *H. pylori* status. The remaining two papers presented summary (Lam 1994a) and pooled (Kurata & Nogawa 1997) estimates of relative risk; in one case the data were used to derive aetiological fractions (Kurata & Nogawa 1997).

Parsonnet (1998) concluded that *H. pylori* is the single most important cause of both duodenal ulcer disease and gastric ulcer disease and that in the United States it appears to be a causative factor in at least 50% to 65% of all duodenal ulcer. While the proportion of gastric ulcer disease attributable to *H. pylori* is thought to be lower than that for duodenal ulcer, this

could be because NSAIDs contribute disproportionately to gastric ulcer disease (Lam 1994a; Lam 1994b; McIntosh et al. 1985; Parsonnet 1998; Schubert et al. 1993).

Eastwood (1997) summarised the relationship between smoking and *H. pylori* infection in the pathogenesis of peptic ulcer disease as one where smoking appears to increase the risk for *H. pylori* infection and may also augment the harmful effects of *H. pylori* in the development of peptic ulceration. But smoking does not appear to delay ulcer healing or increase the risk of recurrence once *H. pylori* has been eradicated. Furthermore, while the adverse effects of smoking on aggressive and protective factors qualify it as an important contributor to the maintenance of peptic ulcer disease, these effects are transient and the affected physiological functions return to normal within minutes to hours after cessation of smoking.

Lam (1994b) concluded that circumstantial evidence is supportive of *H. pylori* playing a role in the aetiology of duodenal ulcer. Mucosal inflammation appears associated with peptic ulcer disease in many situations. Conditions associated with severe mucosal inflammation include the habitual use of NSAIDs, *H. pylori* infection and, to a lesser degree, cirrhosis of the liver and chronic renal failure, as well as conditions associated with minimal inflammation, such as cigarette smoking. Unlike Eastwood, who suggested that smoking appears to increase the risk of *H. pylori* infection, Lam cites Maxton et al. (1990) as finding that the ulcerogenic potential of NSAIDs and smoking is not mediated through a predisposition to *H. pylori* infection. A later study by Lee et al. (1994) also reported that there was no difference between people positive or negative to *H. pylori* in terms of their tobacco exposure. With regard to ulcer relapse or the recurrence of *H. pylori* infection after eradication, Chan et al. (1997) also found no significant difference between smokers (≥10 cigarettes/day) and non-smokers. They concluded that cigarette smoking does not appear to increase the recurrence of peptic ulcers after eradication of *H. pylori*.

The four studies published after 1993—Archimandritis et al. (1995), Leoci et al. (1995), Menzel et al. (1995) and Wang et al. (1996)—reported that, even after adjustment for *H. pylori* infection, smoking status also remained a significant predictor in the aetiology of peptic ulcer.

The prospective study by Archimandritis (1995) examined the impact of smoking and *H. pylori* on 166 duodenal ulcer disease patients and 75 gastric ulcer disease patients. Individuals having recently used NSAIDs were excluded. Univariate analysis found that 48% of duodenal ulcer and 37% of gastric ulcer patients had a positive family history. Furthermore, a majority of duodenal ulcer (63%) and gastric ulcer (67%) patients were smokers of more than 10 cigarettes a day and an even greater majority of duodenal ulcer (85%) and peptic ulcer (75%) patients were *H. pylori* positive.

In their prospective study evaluating the incidence and risk factors for duodenal ulcer, Leoci et al. (1995) found 41 cases of the disease among 526 individuals undergoing oesophagogastroduodenoscopy. Multiple logistic regression identified maximal acid output, a history of peptic ulcer in brothers, and smoking more than 10 cigarettes a day as significant predictors of peptic ulcer disease. Only a subgroup of 178 individuals had had gastric biopsies in this study, so adjustment for *H. pylori* infection was not possible in the analysis of all 526 individuals. Furthermore, this study was not able to use information on consumption of NSAIDs as an adjustment in the analysis because the data were considered unreliable. While maximal acid output and cigarette smoking were independent predictors of duodenal ulcer, Leoci et al. did not demonstrate any interaction between these variables; thus their findings are consistent with those of another report that found intragastric activity was not dependent on cigarette smoking (Kaufmann et al. 1990). In another report, however, Harris

et al. (1996) hypothesised that acid hypersecretion in duodenal ulcer disease is caused by *H. pylori* infection.

Menzel et al. (1995) reported on a study of 1299 individuals, of whom 310 had duodenal ulcer and 157 had gastric ulcer. They did not exclude individuals taking aspirin, steroids and/or NSAIDs and they used low and high urease activity as a marker for low and high *H. pylori* colonisation of the mucosa. (Low and high urease activity reflect low and high *H. pylori* colonisation of the mucosa.)

The analysis undertaken by Menzel et al. was by logistic regression and included all two-way-interactions between urease activity and the other factors considered. For the duodenal ulcer model there were interactions between urease activity and both presenting gastric symptoms and nationality. The interaction odds ratios of 87.4 (95% CI: 6.4–1187) for epigastric pain and high urease activity and 26.4 (95% CI: 9.0–78.0) for epigastric pain and low urease activity compare with that of 11.1 (95% CI: 4.8–25.9) for epigastric pain and no urease activity. This compares with an odds ratio of 2.2 (95% CI: 1.3–4.0) for smoking as an independent predictor of duodenal ulcer disease. Further, there was no apparent interaction between urease activity and smoking and there was no apparent dose–response relationship for those smoking more than 20 cigarettes a day and those smoking fewer than 20 cigarettes a day.

The regression results also showed that smoking was an independent predictor of disease (OR: 3.4; 95% CI: 2.0–5.7). However, this compared with the odds ratio of 3.4 (95% CI: 2.0–5.7) for low urease activity and 24.8 (95% CI: 8.5–72.3) for high urease activity when compared with no urease activity. Again, there was no evidence of a significant interaction between urease activity and smoking.

A case-control study of 500 factory workers in China with peptic ulcer (85% duodenal) and 500 employees selected from the same factories as controls was undertaken by Wang et al. (1996). Cases of peptic ulcer were confirmed, by endoscopy or gastrointestinal barium examination, as either new or recurrent (within the last two years). Cases due to NSAID use or Zellinger Ellison syndrome were excluded.

Among the cases there were more males (84%) than females (16%). There was a very high prevalence of smoking among the cases (67%), although there was only one female smoker. There was a very high prevalence of *H. pylori* infection among the cases (81.5%) when compared with the controls (69.9%) and a very high prevalence of a family history of peptic ulcer disease among the cases (50.4%) when compared with the controls (17.4%). Multivariate analysis of male workers identified age, family history, *H. pylori* infection and cigarette smoking as significant predictors for duodenal ulcer and peptic ulcer. However, among the females, where there was only one smoker, significant predictors were increasing age, family history and *H. pylori* infection.

Wang et al. purported to show that, despite *H. pylori* infection being almost ubiquitous within the population studied, male gender, increasing age, low socio-economic status, a family history of ulcer and cigarette smoking remain risk factors for peptic ulcer. But the high prevalence of *H. pylori* infection (81.5%) and cigarette smoking (up to 89% among male workers with ulcers) in the study may have rendered these factors less specific predictors of peptic ulcer (Rose 1985). Furthermore, Wang et al. did not examine the interaction between *H. pylori* and smoking, so as to assess any effect modification due to smoking between exposure to *H. pylori* infection and the outcome of peptic ulcer disease. A further limiting factor, not restricted to the Wang et al. study, is that, while adjustment was made for the presence of *H. pylori* infection, the adjustment did not extend to the specific *H. pylori* strain characteristics, which may have an important influence on clinical outcomes (Blaser 1994).

As did a number of others—such as Archimandritis et al. (1995) and Leoci et al. (1995)—Wang et al. identified a family history of ulcer as a strong predictive factor for peptic ulcer disease among both males and females. This is consistent with both the circumstantial evidence suggestive of person-to-person transmission of *H. pylori* (Lin et al. 1994; Mitchell et al. 1993b; Peach et al. 1997) and of genetic factors (Boren et al. 1994), which are evidenced by *H. pylori* concordance being higher in monozygotic than dizygotic twins (Parsonnet 1998).

Lam et al. (1994a) reported on summary estimates and Kurata and Nogawa (1997) reported on pooled estimates of relative risk for the three environmental risk factors—NSAID use, cigarette smoking and *H. pylori* infection—for peptic ulcer disease. Lam et al. reported that NSAID use (RR: 5.0) and cigarette smoking (RR: 5.0) carried a far higher risk for gastric ulceration than did infection with *H. pylori* (RR: 1.0). On the other hand, for duodenal ulcer disease it was cigarette smoking (RR: 2.0) and *H. pylori* infection (RR: 2.0) that carried a higher risk than NSAID use (RR: 1.0).

Kurata and Nogawa used meta-analysis techniques to determine overall risk ratios and 95% confidence intervals for each of the three main environmental risk factors for peptic ulcer disease. The outcomes of interest for the *H. pylori* studies incorporated in the meta-analysis were the presence or development of peptic ulcer and not past history or recurrence of ulcer.

Population-attributable risks were calculated for each of the major risk factors based on two hypothetical models—no interaction between risk factors and interaction between risk factors. The no-interaction model assumes that individuals are exposed to only one of the three risk factors at a time and that the estimates for the population-attributable risk percentages are additively combined.

The hypothetical interaction model assumes synergistic interaction and overlapping exposure, producing results consistent with the idea that there are two common forms of peptic ulcer: that associated with *H. pylori* infection and that associated with the use of NSAIDs. This is based on the assumption that NSAIDs do not interact with either cigarette smoking or *H. pylori* infection (Schubert et al. 1993) but that there is interaction between *H. pylori* infection and cigarette smoking. Therefore, non-smokers who are *H. pylori* positive are at increased risk and smokers who are *H. pylori* positive are at even greater risk. However, those who smoke and are *H. pylori* negative are not at increased risk.

Overall, the Kurata and Nogawa interaction model appears the most consistent with the literature review just discussed. This is supported by Borody et al. (1991), who examined 302 patients with an endoscopic diagnosis of duodenal ulcer and found 94% (284) to have associated *H. pylori* gastritis. Of the 18 who were *H. pylori* negative, eight had been taking NSAIDs and a further four had recently takeen of antibiotics. Similarly, in a later study of 115 patients with endoscopic diagnosis of gastric ulcer, 62% (71) had *H. pylori* infection (Borody et al. 1992). Of these patients, 30% (21) were taking NSAIDs. Of the 44 *H. pylori*-negative gastric ulcer cases, 66% (29) were taking NSAIDs. *H. pylori* infection and NSAID use accounted for 87% (100) of the 115 gastric ulcer cases.

Revised aetiological fractions for tobacco and the onset of peptic ulcer disease

The Kurata and Nogawa (1997) interaction model was the basis for calculation of the aetiological fraction. Data from Martin et al. (1989) were used to estimate relative risk for those exposed to both *H. pylori* and smoking. Those who were *H. pylori* positive and who smoked more than 10 cigarettes a day were 6.8 times more likely to develop peptic ulcer than *H. pylori*-positive individuals who did not smoke. This is derived from the fact that 11 out of 15 smokers with *H. pylori*, as opposed to 13 out of 45 non-smokers with *H. pylori*, had

duodenal and/or gastric ulcers. On the other hand, individuals who smoked and were *H. pylori* negative were not at increased risk.

The product of the prevalence of *H. pylori* infection in the general population and the smoking prevalence (10 or more cigarettes a day) for the general population provides an estimate of the proportion of the general population who smoke and who are *H. pylori* positive (Kurata & Nogawa 1997).

Table 4.1: Relative risk estimates for smoking and *H. pylori* exposure for peptic ulcer disease

		H. pylori +		Current smoker, ≥10 cigarettes per day		*H. pylori*+ and smoker	
Sex	**Age**	**RR**	**95% CI**	**RR**	**95% CI**	**RR**	**95% CI**
Male	All	3.3	2.6–4.4	1.9	1.7–2.1	6.8	1.8–25.2
Female	All	3.3	2.6–4.4	2.3	1.9–2.7	6.8	1.8–25.2

Sources: H. pylori—Kurata and Nogawa 1997; current smoker—Kurata and Nogawa 1997; *H. pylori* and smoker—Martin et al. 1989.

Lin et al. (1998) estimated that the overall prevalence of *H. pylori* in the population of Melbourne was 38% and increased with age from 18% at ages 20-30 years to 53% at ages over 70 years. The prevalence of *H. pylori* was 48% in men and 30% in women. The rate of acquisition of *H. pylori* infection was 1% per year.

Overall prevalence data from Lin et al. were disaggregated by sex so as to reflect the overall 48% prevalence in men and 30% prevalence in women across all the age groups examined in the study (Table 4.2).

Table 4.2: Prevalence of *H. pylori* infection among Australians, by age and sex, 1998

	Age						
Sex	**20–30**	**31–40**	**41–50**	**51–60**	**61–70**	**>70**	**20 and over**
				(per cent)			
Male	23	30	34	44	58	67	48
Female	14	19	21	28	36	42	30
Total	18	24	27	35	46	53	38

Source: Derived from Lin et al. 1998.

The estimates for the prevalence of current smokers (10 or more cigarettes a day) for the general population are derived from the 1995 National Smoking and Health Survey conducted by the Anti-cancer Council of Victoria and analysed by the Council's Centre for Behavioural Research in Cancer (Table 4.3).

Table 4.3: Proportion of the population smoking 10 or more cigarettes a day, by age and sex, 1995

	Age							
Sex	**18–19**	**20–29**	**30–39**	**40–49**	**50–59**	**60–69**	**70+**	**18 and over**
				(per cent)				
Male	13.8	22.2	23.7	18.6	18.9	10.6	7.2	17.8
Female	21.4	23.8	21.7	16.7	16.8	11.2	4.7	17.1

Source: Unpublished data from the Anti-Cancer Council of Victoria's Centre for Behavioural Research in Cancer.

We used the product of the *H. pylori* infection prevalence from Table 4.2 and the smoking prevalence from Table 4.3 to estimate the proportion of the population who smoke more than 10 cigarettes per day and are *H. pylori* positive. This is the population prevalence figure used in the aetiological fraction formula (Table 4.4).

The US National Institute of Health has reported 80% to 90% eradication of *H. pylori* as achievable with a multi-drug regimen lasting two weeks (NIH 1994). We took 80% as a conservative estimate of the proportion of people with H pylori infection who are successfully treated. Accordingly, before determining the aetiological fraction, the *H. pylori* prevalence cited above was scaled back by a factor of 0.20.

Overall, 9% of peptic ulcer disease among males and 6% among females is caused by the interaction between *H. pylori* and smoking 10 or more cigarettes a day (Table 4.5).

Table 4.4: Proportion of the population who smoke 10 or more cigarettes per day and who would remain *H. pylori* positive after therapy to eradicate *H. pylori* infection

	Age						
Sex	**20–30**	**31–40**	**41–50**	**51–60**	**61–70**	**>70**	**20 and over**
Male	0.010	0.014	0.013	0.017	0.012	0.010	0.017
Female	0.007	0.008	0.007	0.009	0.008	0.004	0.010

Source: AIHW analysis of data in Tables 4.2 and 4.3.

Table 4.5: Revised aetiological fractions for tobacco exposure and peptic ulcer disease

Age	Males	Females
Exposed		
All ages	0.853	0.853
General population		
20–24	0.056	0.037
25–29	0.056	0.037
30–34	0.076	0.046
35–39	0.076	0.046
40–44	0.068	0.039
45–49	0.068	0.039
50–54	0.088	0.052
55–59	0.088	0.052
60–64	0.067	0.045
65–69	0.067	0.045
70–74	0.053	0.022
75–79	0.053	0.022
80+	0.053	0.022
Total (20+)	**0.090**	**0.056**

Source: AIHW analysis of data in Tables 4.1 and 4.4.

Revised aetiological fractions for tobacco and death due to peptic ulcer disease

An Australian case-control study that examined the association between, on the one hand, individual co-existing illnesses, septicaemia, intra-abdominal abscess, marital status, smoking and alcohol use and, on the other, mortality following perforated peptic ulcer without pre-operative evidence of haemorrhage did not identify smoking as a risk factor for mortality (McIntosh et al. 1996). The study found co-existing illnesses, septicaemia and intra-abdominal abscess as the risk factors predictive of mortality following ulcer perforation.

More recently, in a study examining mortality within one month of peptic ulcer bleed, elderly patients, those undergoing surgery, or those who were current users of acid-suppressing drugs or NSAIDs were identified as at increased risk of mortality (Garcia Rodriguez et al. 1998). After *H. pylori*, NSAIDs are thought to be the most important cause of peptic ulcer disease and a major risk factor for ulcer complications and mortality (Kang 1995).

Overall, the mortality rate associated with peptic ulcer disease is in the main attributed to re-bleeding among the elderly population, who also have more co-existing systemic diseases. The majority of deaths, therefore, result from non-peptic ulcer diseases (Mueller et al. 1994). This is reflected in one report—by Ng et al. (1994)—on peptic ulcer disease among the people aged 60 years or more, which found that bleeding is a frequent and major complication occurring among 50% of cases and perforation occurs among 2% of cases. However, while mortality arising from bleeding peptic ulcers in this group is 11%, around 90% of this is due to concurrent medical conditions and only 10% arises directly from bleeding ulcer. The concurrent medical conditions most frequently encountered were hypertension and ischaemic heart disease (Ng et al. 1994).

We took 10% as our estimate of deaths coded to peptic ulcer disease that actually arose due to bleeding peptic ulcer. Hence the attributable fractions in Table 4.5 should be reduced by a factor of 10% when applied to deaths. This means that less than 1% of peptic ulcer deaths are attributable to tobacco smoking.

4.2.3 Passive exposure to tobacco smoke and its health effects in pregnancy and childhood

Environmental tobacco smoke (ETS) consists of exhaled mainstream and sidestream smoke. In 1993 the National Health and Medical Research Council established a working party to update the Council's 1986 report *Effects of Passive Smoking on Health*. The final review document (NHMRC 1997) made available a synthesis of relevant scientific knowledge on the health effects of passive smoking. It contains the estimates that formed the basis for quantifying and, where possible, revising the aetiological burden of ETS.

Exposure to passive smoking during pregnancy

Studies of women's exposure to passive smoking during pregnancy show a small detriment in birthweight among babies born to such women. The size of the effect is, however, small (30–40g) and so may have little clinical significance for most infants overall but a more marked significance among socially disadvantaged groups (NHMRC 1997).

Passive smoking and sudden infant death syndrome

Current evidence supports the conclusion that there is a causal association between sudden infant death syndrome (SIDS) and exposure to ETS. Nevertheless, the relative importance of maternal smoking during pregnancy and exposure to ETS after birth remains unclear (NHMRC 1997).

In examining whether or not postnatal exposure to ETS is associated with SIDS, the NHMRC report identified eight studies (Bergman & Wieser 1976; Blair et al. 1996; Brooke et al. 1997; Klonoff Cohen et al. 1995; McGlashan 1989; Mitchell et al. 1993a; Ponsonby et al. 1995; Schoendorf & Kiely 1992), of which seven were case-control studies. Pooled estimates of relative risk were, however, not reported. Consistent with later reports that parental smoking (Henderson Smart et al. 1998), and in particular maternal smoking (Mitchell et al. 1997), were significantly associated with SIDS, a pooled estimate of relative risk was determined from the study by Mitchell et al. (1997) and the three studies from the NHMRC report (Klonoff Cohen et al. 1995; Ponsonby et al. 1995; Schoendorf & Kiely 1992) that examined postnatal maternal smoking.

Table 4.6: Sudden infant death syndrome and maternal smoking

Study	Country	SIDS	Controls	Confounders controlled	OR (95% CI)
Schoendorf and Kiely 1992	US	234	6,000 random	Yes	1.75 (1.04–2.95
Klonoff Cohen et al. 1995	US	200	200 matched	Yes	2.28 (1.04–4.96)
Ponsonby et al. 1995	Australia	58	101 matched	Yes	3.82 (1.43–10.2)
Mitchell et al. 1997	NZ	232	1,200 random	Yes	5.01 (2.01–12.46)

The pooled relative risk estimate was 2.44 (95% CI: 1.69–3.51). This compares with a pooled estimate of relative risk of 2.76 (95% CI: 2.66–2.86) for any smoking during pregnancy, as determined by English et al. (1995). Based on an exposure prevalence of 0.29 for smoking during pregnancy, English et al. concluded that 0.338 or 34% of SIDS was attributable to this exposure.

Revised aetiological fraction for postnatal ETS and SIDS

The estimate of relative risk used was the pooled estimate of 2.44. The proportion of infants exposed to ETS was 0.126, based on the unpublished Victorian Anti-Cancer Council prevalence estimate of 12.6% of households where exposure to maternal smoke among children under 2 years old was likely to occur. This gave an aetiological fraction of 0.153, or around half that estimated by English et al. This halving of the aetiological fraction for SIDS is in the main due to the reduced prevalence of exposure to maternal smoking in the postnatal context. This is because around 50% of female smokers with a child under 2 years of age will not smoke in their home.

Passive smoking and asthma among children aged less than 15 years

The weight of evidence suggests that exposure to ETS is associated with asthma (ICD-9 code 493) in childhood (<15 years). In the main, it has been reported that exposure to ETS is associated with an increase in morbidity in young people with pre-existing asthma, although several studies have also reported an increase in the number of new cases of asthma among children. Furthermore, exposure to ETS is associated with increased

bronchial reactivity and the occurrence of atopy. The association of ETS with childhood asthma is most consistent at high exposures, and the evidence is supportive of a causal relationship (NHMRC 1997).

Aetiological fraction for passive smoking and asthma among children aged less than 15 years

Studies of childhood asthma and passive smoking have commonly reported on the association with heavy exposure (where mothers smoke more than 10 cigarettes a day). It is less clear whether exposure to lower levels of ETS increases the risk of asthma. Accordingly, children are defined as exposed if they had a mother who smoked more than 10 cigarettes a day. This is comparable with the definition of exposure applied in some of the studies reviewed in the NHMRC report. The 1989–90 ABS National Health Survey found that 22.2% of children aged less than 15 years had mothers who smoked more than 10 cigarettes a day. The NHMRC report based its aetiological fraction on this prevalence estimate. The corresponding estimate from the 1995 National Health Survey was 21.3%.

The median of the 50 estimates of relative risk summarised from the peer-reviewed literature in the NHMRC report was 1.40, with an inter-quartile range of 1.20–1.93. This estimate of 1.40 was used in the NHMRC report, along with the 1989–90 prevalence estimate of 0.222, to derive an aetiological fraction of 0.082 for childhood asthma and ETS (NHMRC 1997).

We used the relative risk estimate of 1.40, but our prevalence estimate was taken from unpublished results of the Victorian Anti-Cancer Council study described in Chapter 2. This shows that the proportion of children aged less than 16 years who live in a household with a female (assumed to be their mother) who smokes 10 or more cigarettes a day and who is not restricted to smoking outdoors is 0.054. The aetiological fraction derived from these estimates is 0.021. Again, the reduction in the fraction is a result of only some 25% of such households allowing smoking indoors.

Passive smoking and lower respiratory illness among children under 18 months

'Lower respiratory illness' refers to the illnesses principally affecting the respiratory tract below the epiglottis that correspond to ICD-9 codes 464, 466, 490, 480–486 and 487—but excluding asthma, which is considered separately. The NHMRC report indicates that, based on the peer-reviewed scientific literature, passive smoking is most strongly associated with lower respiratory illness in early life—in particular, the first 18 months after birth. The aetiological fraction calculation assumes that there is no increase in risk of lower respiratory illness for children exposed to ETS if they are over the age of 18 months (NHMRC 1997).

Aetiological fraction for passive smoking and lower respiratory illness among children aged less than 18 months

The median of the 26 estimates of relative risk from 25 published papers summarised in the NHMRC report was 1.60, with an inter-quartile range of 1.40–2.10. The report's estimate of the proportion of children aged less than 18 months and exposed to maternal smoking was 0.25. This was the mid-point of a range of estimates from the research literature. These estimates led to an aetiological fraction of 0.130 (NHMRC 1997).

We used the relative risk estimate of 1.60, but our exposure prevalence was based on the Victorian Anti-Cancer Council study results. This study showed that the proportion of children under 2 years who live in a household with a female (assumed to be their mother) who smokes one or more cigarettes a day and who is not restricted to smoking outdoors was

0.126. This led to an aetiological fraction of 0.070. Again, the reduction in the fraction is a result of only some 50% of such households allowing smoking indoors.

Passive smoking and lung cancer among adults

The NHMRC report concluded that, despite inherent limitations, the available data on ETS and lung cancer in humans were sufficiently strong to infer that ETS was a cause of the disease. Summarising the quantitative data on the relationship was made difficult by the differences in study methods adopted. However, restricting studies to those that examined lung cancer among people who never smoked but had a spouse who smoked yielded 34 studies with a median relative risk of 1.32 and an inter-quartile range of 1.10–1.69 (NHMRC (1997).

The 1998 European multicentre case-control study of lung cancer in non-smokers examined exposure to spousal smoke among 344 cases and 700 controls and yielded an odds ratio of 1.16 (95% CI: 0.93–1.44). When study subjects were stratified by gender, the odds ratio for exposure to spousal smoke was 1.47 (95% CI: 0.81–2.66) among men and 1.11 (95% CI: 0.88–1.39) among women (International Agency for Research on Cancer 1998). The study concluded there was some indication of an increasing risk of lung cancer with increasing cumulative exposure to ETS from the spouse. No clear trend emerged for average exposure or for years of exposure but, with duration of exposure expressed as the product of hours a day and years of exposure, a positive dose response was shown.

Aetiological fraction for passive smoking and lung cancer among adults

The formula for the aetiological fraction used in the NHMRC report is more complex than that described here in Chapter 2 and used throughout this report. The NHMRC (1997) formula is:

$$F = p_n p_s (RR_s - 1) \Big/ \{[p_s (RR_s - 1) + 1]\ [p_x (RR_x - 1) + p_c (RR_c - 1) + 1]\} \quad (1)$$

where

p_n = prevalence of people never having smoked

p_x = prevalence of ex-smokers

p_c = prevalence of current smokers

p_s = people never having smoked but who have spouses that are current smokers.

The relative risk estimates for ex-smokers (RR_x) and for current smokers (RR_c) were those derived by English et al. (1995). For males, these were $RR_x = 6.75$ and $RR_c = 13.0$; for females, they were $RR_x = 5.07$ and $RR_c = 11.4$. The relative risk for people who have never smoked but who have spouses that are current smokers (RR_s) was the median value found in the NHMRC report $RR_s = 1.32$.

Smoking prevalence data elsewhere in this report are based on the 1995 Victorian Anti-Cancer Council study. However, these data do not support an analysis of people who have never smoked but who have spouses who currently smoke. So the prevalence estimates for this aetiological fraction (p_n, p_x, p_c and p_s) are taken from the ABS 1995 National Health Survey. As with other cancer fractions calculated in this report, estimates are calculated only for ages 35 and over.

Table 4.7: Proportion of people who have never smoked but who have spouses that are current smokers

	Year			
	Males		Females	
Age	1989–90	1995	1989–90	1995
		(per cent)		
35–39	11.09	4.0	17.83	15.3
40–44	7.03	2.0	19.89	9.8
45–49	9.18	2.3	20.81	6.9
50–54	11.14	1.6	15.02	10.5
55–59	10.16	0.4	14.39	8.0
60–64	8.03	1.4	15.27	8.1
65–69	4.10	3.7	7.78	7.3
70–74	6.86	3.9	2.99	5.2
75 and over	4.50	3.1	2.04	2.9

Source: 1989–90 data from ABS National Health Survey, as reported in NHMRC (1997); 1995 data derived from 1995 ABS National Health Survey.

Table 4.8: Proportion of the population who are current smokers, ex-smokers or have never smoked, 1995

	Males			Females		
Age	Current smoker	Ex-smoker	Never smoked	Current smoker	Ex-smoker	Never smoked
			(per cent)			
35–39	36.8	25.6	37.6	26.1	18.1	55.8
40–44	31.3	29.4	39.3	23.8	17.5	58.6
45–49	33.3	31.9	34.8	24.4	20.6	55.0
50–54	30.5	33.7	35.8	22.1	16.6	61.3
55–59	29.5	41.2	29.3	20.7	17.9	61.4
60–64	26.9	45.8	27.4	17.9	18.4	63.6
65–69	22.2	53.4	24.3	14.0	21.7	64.3
70–74	16.3	54.1	29.7	13.5	21.0	65.4
75–79	11.7	55.9	32.3	9.8	18.6	71.6
80+	10.2	54.6	35.2	2.8	14.6	82.6

Source: Derived from 1995 ABS National Health Survey.

Passive smoking and cardiovascular disease among adults

The NHMRC report concluded that the evidence suggests that passive smoking increases the risk of ischaemic heart disease and that this excess risk is apparent in both men and women. While this relationship is unlikely to be explained by the confounding of passive smoking with other risk factors, some evidence of publication bias was evident and the excess risk of ischaemic heart disease in passive smokers appears large when compared with the excess risk in active smokers (NHMRC 1997). Because of these anomalies, although passive smoking appears to increase the risk of IHD, other non-causal explanations are possible. Furthermore, there was insufficient evidence to determine whether ETS affects the

risk of cerebrovascular disease or peripheral vascular disease. Therefore estimates of the effects of passive smoking are restricted to heart attacks (ICD-9 410) and other deaths from IHD (ICD-9 codes 411–414).

Table 4.9: People who have never smoked: revised aetiological fraction for lung cancer attributable to smoking by a spouse

Age	Males	Females
35–39	0.00083	0.00530
40–44	0.00041	0.00397
45–49	0.00048	0.00321
50–54	0.00028	0.00484
55–59	0.00007	0.00422
60–64	0.00021	0.00450
65–69	0.00051	0.00455
70–74	0.00050	0.00338
75–79	0.00016	0.00199
80 years and over	0.00103	0.00395

Source: AIHW analysis of prevalence data in Tables 4.7 and 4.8 and relative risk estimates reported by English et al. (1995).

Aetiological fraction for heart attacks and ischaemic heart disease among adults

The formula on page 83 was used for the aetiological fraction for heart attacks and IHD among adults. The prevalence estimates were those presented in Tables 4.7 and 4.8. The relative risk estimates for ex-smokers (RR_x) and for current smokers (RR_c) were those derived by English et al. (1995). For males aged more than 65 years these were $RR_x = 1.45$ and $RR_c = 3.06$; for females aged more than 65, they were $RR_x = 0.93$ and $RR_c = 1.67$.

Table 4.10: People who have never smoked: revised aetiological fractions for ischaemic heart disease attributable to smoking by a spouse

Age	Males	Females
30–34	0.0017	0.0143
35–39	0.0022	0.0113
40–44	0.0011	0.0081
45–49	0.0013	0.0063
50–54	0.0008	0.0095
55–59	0.0002	0.0080
60–64	0.0006	0.0084
65–69	0.0022	0.0096
70–74	0.0022	0.0071
75–79	0.0007	0.0037
80 years and over	0.0040	0.0059

Source: AIHW analysis of prevalence data in Tables 4.7 and 4.8 and relative risk estimates reported by English et al. (1995).

4.2.4 Aetiological fractions for tobacco updated with recent prevalence data

Where possible, all the aetiological fractions were revised to incorporate updated estimates of the prevalence of tobacco consumption. The conditions just discussed were also based on revised risk-ratio estimates. Table 4.11 lists the conditions that were revised to incorporate updated estimates of the prevalence of tobacco consumption but that were based on the risk-ratio estimates derived by English et al. (1995). Table 4.12 lists the values of the revised aetiological fractions.

Most of the fractions listed in Table 4.11 involve a straightforward application of the risk-ratio to the prevalence estimate derived from the Victorian Anti-Cancer Council study. However, some of the calculations are more complex and require some explanation.

Table 4.11: Conditions for which aetiological fractions were based on the English et al. risk-ratio estimates but were revised to incorporate updated estimates of prevalence

Condition	Source of prevalence data
Oropharyngeal cancer	Synthetic prevalence estimate
Oesophageal cancer	Synthetic prevalence estimate
Stomach cancer	Synthetic prevalence estimate
Anal cancer	Synthetic prevalence estimate
Pancreatic cancer	Synthetic prevalence estimate
Laryngeal cancer	Synthetic prevalence estimate
Lung cancer	Direct calculation
Endometrial cancer	Synthetic prevalence estimate
Vulvar cancer	Synthetic prevalence estimate
Penile cancer	Synthetic prevalence estimate
Bladder cancer	Synthetic prevalence estimate
Renal parenchymal cancer	Synthetic prevalence estimate
Renal pelvic cancer	Synthetic prevalence estimate
Respiratory carcinoma in situ	Synthetic prevalence estimate
Ischaemic heart disease	1995 Anti-Cancer Council Survey
Chronic obstructive pulmonary disease	Synthetic prevalence estimate
Parkinson's disease	1995 Anti-Cancer Council Survey
Pulmonary circulation disease	Same fraction as chronic obstructive pulmonary disease
Cardiac dysrhythmias	Same fraction as ischaemic heart disease
Heart failure	Derived from fraction for ischaemic heart disease
Stroke	1995 Anti-Cancer Council Survey
Atherosclerosis	1995 Anti-Cancer Council Survey
Pneumonia	1995 Anti-Cancer Council Survey
Crohn's disease	1995 Anti-Cancer Council Survey
Ulcerative colitis	1995 Anti-Cancer Council Survey
Ectopic pregnancy	1995 Anti-Cancer Council Survey
Spontaneous abortion	1998 National Drug Strategy Household Survey
Antepartum haemorrhage	1998 National Drug Strategy Household Survey
Hypertension in pregnancy	1998 National Drug Strategy Household Survey
Low birthweight	1998 National Drug Strategy Household Survey
Premature rupture of membranes	1998 National Drug Strategy Household Survey
SIDS (and smoking during pregnancy)	1998 National Drug Strategy Household Survey

Cancer and chronic obstructive pulmonary disease

Peto et al. (1992) derived an underlying rate of lung cancer among people who never smoked in the United States. We assumed that this rate applied in Australia and used it to derive the expected rate of lung cancer in the absence of smoking. Comparison of this with the observed lung cancer rate gave us the proportion of lung cancer attributable to smoking. We then used the Peto et al. estimates of lung cancer rates for smokers and non-smokers to derive a synthetic smoking prevalence rate that represented the historical prevalence which was consistent with the observed lung cancer rate. Finally, we combined this synthetic rate with the risk ratios from English et al. to derive aetiological fractions for the remaining cancers and chronic obstructive pulmonary disease.

Pulmonary circulation disease

We followed English et al. in applying the aetiological fraction for chronic obstructive pulmonary disease to ICD-9 codes 415.0, 416 and 417 but excluding 415.1 (pulmonary embolism). This approach is based on the assumption that chronic obstructive pulmonary disease is the underlying pathology in the vast majority of cases of pulmonary circulatory conditions other than embolism.

Cardiac dysrhythmias

We followed English et al. in assuming that most cardiac dysrhythmias in Australia—and especially those causing sudden death or significant morbidity—are the result of ischaemic heart disease.

Heart failure

We followed English et al. in apportioning heart failure conditions between ischaemic heart disease and other specific heart disease codes, in accordance with the proportional distribution of mortality or morbidity associated with the specific condition. The other specific heart conditions are not caused by cigarette smoking and so have aetiological fractions of zero. Hence the aetiological fraction for ischaemic heart disease was then applied to the relevant proportion of heart failure cases while the remainder were discarded.

Ectopic pregnancy

We followed English et al. in applying the prevalence of smoking in the general population rather among for pregnant women on the grounds that many women give up smoking after the time of conception.

Spontaneous abortion

English et al. derived a risk ratio and a fraction for the effect of tobacco on spontaneous abortion. We updated their fraction. However, during the period 1996 to 1998 there were no deaths coded to spontaneous abortion and no hospital stays longer than the average normal confinement. Hence if we do not count the separations, as recommended by English et al., this condition makes no contribution to the attributable mortality or hospital morbidity. The fractions are given in Table 4.12, but this condition has been excluded from the tables of results.

4.3 Unrevised aetiological fractions for tobacco

Only two conditions were left with unrevised fractions. The first was tobacco abuse, which has a fraction value of one by definition. The second was fire injuries. English et al. derived a fraction of 0.23 based on six case series of fire injuries. In the absence of better or more recent data, we used the same estimate.

Table 4.12: Revised values for tobacco fractions based on the English et al. risk-ratio estimates and updated prevalence data

1. Fractions updated with synthetic prevalence data derived using the Peto et al. method

Oropharyngeal cancer (ICD-9 codes 141, 143–146, 148, 149)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.43	0.78	0.71	0.92	0.83	0.43	0.78	0.71	0.92	0.83
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.302					0.280		
45–49			0.533					0.261		
50–54			0.479					0.412		
55–59			0.513					0.435		
60–64			0.570					0.443		
65–69			0.584					0.453		
70–74			0.583					0.523		
75–79			0.548					0.470		
80+			0.569					0.445		
Total (35+)			**0.464**					**0.361**		

Oesophageal cancer (ICD-9 code 150)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.44	0.75	0.69	0.75	0.80	0.44	0.75	0.69	0.75	0.80
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.269					0.248		
45–49			0.492					0.230		
50–54			0.438					0.373		
55–59			0.472					0.395		
60–64			0.529					0.403		
65–69			0.544					0.413		
70–74			0.543					0.482		
75–79			0.507					0.429		
80+			0.528					0.405		
Total (35+)			**0.423**					**0.324**		

Stomach cancer (ICD-9 code 151)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.10	0.29	0.61	0.55	0.68	0.10	0.29	0.61	0.55	0.68
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.048					0.043		
45–49			0.117					0.039		
50–54			0.096					0.075		
55–59			0.109					0.082		
60–64			0.133					0.084		
65–69			0.140					0.087		
70–74			0.139					0.112		
75–79			0.123					0.093		
80+			0.132					0.085		
Total (35+)			**0.091**					**0.061**		

Anal cancer (ICD-9 codes 154.2, 154.3)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.45	0.69	0.53		0.78	0.45	0.69	0.53		0.78
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.210					0.193		
45–49			0.412					0.178		
50–54			0.361					0.301		
55–59			0.393					0.321		
60–64			0.449					0.328		
65–69			0.463					0.337		
70–74			0.462					0.403		
75–79			0.427					0.352		
80+			0.448					0.330		
Total (35+)			**0.347**					**0.258**		

Note: There were no studies on which to base a risk-ratio estimate for people smoking 15–24 cigarettes a day.

Pancreatic cancer (ICD-9 code 157)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.13	0.46	0.39	0.41	0.49	0.13	0.46	0.39	0.41	0.49
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.095					0.086		
45–49			0.217					0.079		
50–54			0.182					0.145		
55–59			0.203					0.157		
60–64			0.243					0.162		
65–69			0.254					0.167		
70–74			0.253					0.210		
75–79			0.227					0.177		
80+			0.242					0.163		
Total (35+)			**0.173**					**0.120**		

Laryngeal cancer (ICD-9 code 161)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.65	0.87	0.77	0.83	0.92	0.65	0.87	0.77	0.83	0.92
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.442					0.415		
45–49			0.676					0.391		
50–54			0.626					0.561		
55–59			0.658					0.584		
60–64			0.708					0.592		
65–69			0.719					0.602		
70–74			0.719					0.667		
75–79			0.689					0.618		
80+			0.707					0.594		
Total (35+)			**0.613**					**0.508**		

Lung cancer (ICD-9 code 162)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.85	0.92	0.85	0.88	0.93	0.80	0.91	0.87	0.92	0.95
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.449					0.247		
45–49			0.659					0.528		
50–54			0.798					0.644		
55–59			0.866					0.732		
60–64			0.906					0.743		
65–69			0.920					0.781		
70–74			0.927					0.788		
75–79			0.917					0.743		
80+			0.903					0.646		
Total (35+)			**0.903**					**0.646**		

Endometrial cancer (ICD-9 codes 179, 182)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	—	—	—	—	—	–0.10	–0.89	—	—	—
General population		**All levels of exposure**					**All levels of exposure**			
35–39			–					0.000		
40–44			—					0.000		
45–49			—					0.000		
50–54			—					–0.102		
55–59			—					–0.114		
60–64			—					–0.118		
65–69			—					–0.123		
70–74			—					–0.170		
75–79			—					–0.133		
80+			—					–0.119		
Total (35+)			—					**–0.081**		

Note: The evidence for the effect of smoking on endometrial cancer supports only estimation of the effect for post-menopausal women. We followed English et al. in assuming that this corresponds to women aged 50 and over. There were no studies that allowed the separate estimation of risk ratios by numbers of cigarettes smoked per day for post-menopausal women.

Vulvar cancer (ICD-9 code 184.4)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	—	—	—	—	—	0.27	0.71	0.70	—	0.83
General population		**All levels of exposure**					**All levels of exposure**			
35–39			—					0.000		
40–44			—					0.209		
45–49			—					0.194		
50–54			—					0.323		
55–59			—					0.344		
60–64			—					0.352		
65–69			—					0.361		
70–74			—					0.428		
75–79			—					0.376		
80+			—					0.353		
Total (35+)			—					**0.278**		

Note: There were no studies on which to base a risk-ratio estimate for people smoking 15–24 cigarettes a day

Penile cancer (ICD-9 codes 187.1–187.4)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.38	0.44	0.15	0.55	0.68	—	—	—	—	—
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					—		
40–44			0.089					—		
45–49			0.205					—		
50–54			0.171					—		
55–59			0.192					—		
60–64			0.230					—		
65–69			0.241					—		
70–74			0.240					—		
75–79			0.215					—		
80+			0.229					—		
Total (35+)			**0.163**					—		

Bladder cancer (ICD-9 code 188)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.40	0.63	0.49	0.69	0.66	0.40	0.63	0.49	0.69	0.66
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.174					0.158		
45–49			0.356					0.146		
50–54			0.308					0.254		
55–59			0.338					0.272		
60–64			0.391					0.278		
65–69			0.405					0.286		
70–74			0.404					0.347		
75–79			0.370					0.300		
80+			0.390					0.280		
Total (35+)			0.296					**0.215**		

Renal parenchymal cancer (ICD-9 code 189.0)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.38	0.39	0.15	0.24	0.39	0.38	0.39	0.15	0.24	0.39
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.072					0.065		
45–49			0.171					0.060		
50–54			0.142					0.112		
55–59			0.160					0.122		
60–64			0.193					0.125		
65–69			0.202					0.130		
70–74			0.201					0.165		
75–79			0.180					0.138		
80+			0.192					0.126		
Total (35+)			**0.135**					**0.092**		

Renal pelvic cancer (ICD-9 code 189.1)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.49	0.75	—	—	—	0.49	0.75	—	—	—
General population		**All levels of exposure**					**All levels of exposure**			
35–39		0.000					0.000			
40–44		0.265					0.245			
45–49		0.488					0.227			
50–54		0.434					0.369			
55–59		0.468					0.391			
60–64		0.525					0.399			
65–69		0.540					0.408			
70–74		0.538					0.478			
75–79		0.503					0.425			
80+		0.524					0.401			
Total (35+)		**0.419**					**0.320**			

Respiratory carcinoma in situ (ICD-9 code 231)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.85	0.92	0.85	0.88	0.93	0.80	0.91	0.87	0.92	0.95
General population		**All levels of exposure**					**All levels of exposure**			
35–39		0.000					0.000			
40–44		0.449					0.247			
45–49		0.659					0.528			
50–54		0.798					0.644			
55–59		0.866					0.732			
60–64		0.906					0.743			
65–69		0.920					0.781			
70–74		0.927					0.788			
75–79		0.917					0.743			
80+		0.903					0.646			
Total (35+)		**0.903**					**0.646**			

Chronic obstructive pulmonary disease (ICD-9 codes 490–492, 496)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.85	0.90	0.85	0.79	0.79	0.85	0.90	0.85	0.79	0.79
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.518					0.491		
45–49			0.739					0.466		
50–54			0.695					0.635		
55–59			0.723					0.656		
60–64			0.767					0.664		
65–69			0.777					0.672		
70–74			0.776					0.731		
75–79			0.751					0.687		
80+			0.766					0.665		
Total (35+)			**0.682**					**0.583**		

Pulmonary circulation disease (ICD-9 codes 415.0, 416, 417)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.85	0.90	0.85	0.79	0.79	0.85	0.90	0.85	0.79	0.79
General population		**All levels of exposure**					**All levels of exposure**			
35–39			0.000					0.000		
40–44			0.518					0.491		
45–49			0.739					0.466		
50–54			0.695					0.635		
55–59			0.723					0.656		
60–64			0.767					0.664		
65–69			0.777					0.672		
70–74			0.776					0.731		
75–79			0.751					0.687		
80+			0.766					0.665		
Total (35+)			**0.682**					**0.583**		

2. Fractions updated with prevalence data derived from the 1995 Anti-Cancer Council Survey

Ischaemic heart disease (ICD-9 codes 410–414)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
Under 65	0.31	0.67	0.60	0.69	0.73	0.31	0.67	0.60	0.69	0.73
65 +	0.11	0.40	—	—	—	0.11	0.40	—	—	—
General population										
18–19	0.016	0.415	0.244	0.127	0.000	0.035	0.424	0.221	0.124	0.059
20–24	0.032	0.390	0.154	0.126	0.095	0.039	0.395	0.128	0.123	0.134
25–29	0.046	0.398	0.154	0.101	0.106	0.042	0.401	0.156	0.117	0.103
30–34	0.057	0.388	0.092	0.086	0.222	0.060	0.380	0.127	0.111	0.145
35–39	0.072	0.351	0.058	0.100	0.205	0.069	0.317	0.101	0.112	0.105
40–44	0.075	0.356	0.060	0.080	0.186	0.063	0.332	0.078	0.116	0.153
45–49	0.088	0.323	0.045	0.084	0.206	0.077	0.218	0.041	0.060	0.135
50–54	0.088	0.350	0.099	0.089	0.157	0.069	0.281	0.072	0.113	0.124
55–59	0.101	0.329	0.072	0.082	0.187	0.073	0.254	0.069	0.093	0.094
60–64	0.144	0.234	0.047	0.049	0.112	0.067	0.217	0.062	0.107	0.052
Total (<65)	**0.070**	**0.358**	**0.098**	**0.094**	**0.159**	**0.059**	**0.331**	**0.105**	**0.109**	**0.118**
65–69	0.052	0.102	—	—	—	0.022	0.086	—	—	—
70–74	0.056	0.081	—	—	—	0.029	0.049	—	—	—
75–79	0.056	0.081	—	—	—	0.029	0.049	—	—	—
80+	0.056	0.081	—	—	—	0.029	0.049	—	—	—
Total (65+)	**0.055**	**0.088**	—	—	—	**0.027**	**0.059**	—	—	—

Note: There were no data on which to base estimates of risk ratios for different levels of consumption at ages 65 and over.

Parkinson's disease (ICD-9 code 332)

	Male					Female				
Age	**Ever smoked cigarettes**					**Ever smoked cigarettes**				
Exposed										
All ages	—	—	–0.75	—	—	—	—	–0.75	—	—
General population										
18–19	—	—	–0.218	—	—	—	—	–0.291	—	—
20–24	—	—	–0.241	—	—	—	—	–0.268	—	—
25–29	—	—	–0.295	—	—	—	—	–0.287	—	—
30–34	—	—	–0.325	—	—	—	—	–0.322	—	—
35–39	—	—	–0.328	—	—	—	—	–0.273	—	—
40–44	—	—	–0.345	—	—	—	—	–0.272	—	—
45–49	—	—	–0.346	—	—	—	—	–0.202	—	—
50–54	—	—	–0.390	—	—	—	—	–0.237	—	—
55–59	—	—	–0.408	—	—	—	—	–0.222	—	—
60–64	—	—	–0.425	—	—	—	—	–0.181	—	—
65–69	—	—	–0.425	—	—	—	—	–0.181	—	—
70–74	—	—	–0.418	—	—	—	—	–0.171	—	—
75–79	—	—	–0.418	—	—	—	—	–0.171	—	—
80+	—	—	–0.418	—	—	—	—	–0.171	—	—
Total (18+)	—	—	**–0.327**	—	—	—	—	**–0.232**	—	—

Cardiac dysrhythmias (ICD-9 code 427)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
Under 65	0.31	0.67	0.60	0.69	0.73	0.31	0.67	0.60	0.69	0.73
65 +	0.11	0.40	—	—	—	0.11	0.40	—	—	—
General population										
18–19	0.016	0.415	0.244	0.127	0.000	0.035	0.424	0.221	0.124	0.059
20–24	0.032	0.390	0.154	0.126	0.095	0.039	0.395	0.128	0.123	0.134
25–29	0.046	0.398	0.154	0.101	0.106	0.042	0.401	0.156	0.117	0.103
30–34	0.057	0.388	0.092	0.086	0.222	0.060	0.380	0.127	0.111	0.145
35–39	0.072	0.351	0.058	0.100	0.205	0.069	0.317	0.101	0.112	0.105
40–44	0.075	0.356	0.060	0.080	0.186	0.063	0.332	0.078	0.116	0.153
45–49	0.088	0.323	0.045	0.084	0.206	0.077	0.218	0.041	0.060	0.135
50–54	0.088	0.350	0.099	0.089	0.157	0.069	0.281	0.072	0.113	0.124
55–59	0.101	0.329	0.072	0.082	0.187	0.073	0.254	0.069	0.093	0.094
60–64	0.144	0.234	0.047	0.049	0.112	0.067	0.217	0.062	0.107	0.052
Total (<65)	**0.070**	**0.358**	**0.098**	**0.094**	**0.159**	**0.059**	**0.331**	**0.105**	**0.109**	**0.118**
65–69	0.052	0.102	—	—	—	0.022	0.086	—	—	—
70–74	0.056	0.081	—	—	—	0.029	0.049	—	—	—
75–79	0.056	0.081	—	—	—	0.029	0.049	—	—	—
80+	0.056	0.081	—	—	—	0.029	0.049	—	—	—
Total (65+)	**0.055**	**0.088**	—	—	—	**0.027**	**0.059**	—	—	—

Atherosclerosis (ICD-9 codes 440–448)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.45	0.61	0.49	0.61	0.61	0.45	0.61	0.49	0.61	0.61
General population										
18–19	0.032	0.342	0.180	0.104	0.000	0.069	0.344	0.166	0.104	0.039
20–24	0.063	0.314	0.115	0.104	0.062	0.076	0.317	0.096	0.102	0.089
25–29	0.089	0.317	0.115	0.083	0.069	0.083	0.321	0.116	0.098	0.068
30–34	0.110	0.306	0.070	0.073	0.150	0.114	0.298	0.095	0.093	0.096
35–39	0.135	0.271	0.044	0.084	0.136	0.128	0.243	0.074	0.091	0.068
40–44	0.141	0.274	0.044	0.066	0.121	0.119	0.256	0.058	0.096	0.100
45–49	0.162	0.244	0.034	0.070	0.135	0.138	0.162	0.029	0.048	0.085
50–54	0.163	0.266	0.073	0.073	0.103	0.127	0.213	0.052	0.092	0.080
55–59	0.184	0.246	0.054	0.068	0.123	0.133	0.191	0.049	0.074	0.059
60–64	0.247	0.165	0.033	0.038	0.069	0.123	0.162	0.044	0.084	0.032
65–69	0.247	0.165	0.035	0.064	0.042	0.123	0.162	0.054	0.000	0.081
70–74	0.268	0.131	0.031	0.017	0.047	0.160	0.092	0.031	0.017	0.031
75–79	0.268	0.131	0.031	0.017	0.047	0.160	0.092	0.031	0.017	0.031
80+	0.268	0.131	0.031	0.017	0.047	0.160	0.092	0.031	0.017	0.031
Total (18+)	**0.145**	**0.257**	**0.063**	**0.068**	**0.096**	**0.112**	**0.232**	**0.071**	**0.077**	**0.070**

Pneumonia (ICD-9 codes 480–487)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.22	0.32	0.31	0.24	0.36	0.22	0.32	0.31	0.24	0.36
General population										
18–19	0.015	0.140	0.103	0.026	0.000	0.034	0.147	0.097	0.026	0.017
20–24	0.030	0.129	0.066	0.026	0.027	0.037	0.132	0.055	0.026	0.039
25–29	0.044	0.134	0.065	0.020	0.030	0.040	0.136	0.067	0.024	0.030
30–34	0.054	0.130	0.041	0.018	0.066	0.056	0.126	0.055	0.023	0.042
35–39	0.066	0.114	0.025	0.021	0.059	0.060	0.099	0.041	0.022	0.028
40–44	0.069	0.116	0.025	0.016	0.051	0.056	0.105	0.033	0.023	0.043
45–49	0.079	0.102	0.019	0.017	0.057	0.061	0.062	0.015	0.011	0.034
50–54	0.081	0.115	0.041	0.018	0.044	0.058	0.085	0.029	0.022	0.033
55–59	0.092	0.106	0.030	0.016	0.052	0.060	0.074	0.026	0.017	0.024
60–64	0.120	0.069	0.017	0.008	0.027	0.054	0.061	0.023	0.019	0.013
65–69	0.120	0.069	0.018	0.014	0.017	0.054	0.061	0.028	0.000	0.031
70–74	0.129	0.055	0.016	0.004	0.018	0.068	0.034	0.015	0.004	0.011
75–79	0.129	0.055	0.016	0.004	0.018	0.068	0.034	0.015	0.004	0.011
80+	0.129	0.055	0.016	0.004	0.018	0.068	0.034	0.015	0.004	0.011
Total (18+)	**0.070**	**0.108**	**0.035**	**0.016**	**0.040**	**0.052**	**0.092**	**0.039**	**0.018**	**0.029**

Crohn's disease (ICD-9 code 555)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.48	0.48	0.32	0.84	0.61	0.38	0.69	0.77	0.89	0.67
General population										
18–19	0.041	0.236	0.078	0.306	0.000	0.044	0.443	0.312	0.293	0.027
20–24	0.081	0.213	0.048	0.299	0.052	0.049	0.413	0.198	0.318	0.067
25–29	0.113	0.215	0.050	0.249	0.060	0.053	0.419	0.238	0.299	0.050
30–34	0.139	0.205	0.031	0.218	0.131	0.075	0.395	0.202	0.295	0.074
35–39	0.167	0.178	0.019	0.243	0.115	0.087	0.332	0.163	0.302	0.055
40–44	0.174	0.181	0.019	0.197	0.106	0.080	0.346	0.129	0.320	0.081
45–49	0.197	0.158	0.015	0.207	0.116	0.097	0.229	0.079	0.195	0.084
50–54	0.201	0.174	0.032	0.220	0.090	0.087	0.295	0.119	0.315	0.066
55–59	0.224	0.159	0.024	0.205	0.107	0.092	0.266	0.120	0.268	0.052
60–64	0.287	0.102	0.015	0.121	0.064	0.086	0.229	0.104	0.300	0.028
65–69	0.287	0.102	0.015	0.192	0.037	0.086	0.229	0.165	0.000	0.089
70–74	0.307	0.080	0.015	0.057	0.046	0.117	0.136	0.094	0.080	0.034
75–79	0.307	0.080	0.015	0.057	0.046	0.117	0.136	0.094	0.080	0.034
80+	0.307	0.080	0.015	0.057	0.046	0.117	0.136	0.094	0.080	0.034
Total (18+)	**0.178**	**0.168**	**0.028**	**0.206**	**0.085**	**0.076**	**0.317**	**0.165**	**0.266**	**0.059**

Ulcerative colitis (ICD-9 code 556)

	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
Age		**All**	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	0.42	–0.59	–0.20	–1.17	–9.00	0.42	–0.59	–0.20	–1.17	–9.00
General population										
18–19	0.048	–0.143	–0.049	–0.055	0.000	0.106	–0.147	–0.049	–0.059	–0.037
20–24	0.091	–0.125	–0.032	–0.057	–0.057	0.110	–0.128	–0.028	–0.057	–0.083
25–29	0.130	–0.128	–0.032	–0.044	–0.061	0.121	–0.131	–0.033	–0.053	–0.062
30–34	0.157	–0.121	–0.021	–0.042	–0.146	0.161	–0.116	–0.028	–0.052	–0.089
35–39	0.181	–0.101	–0.012	–0.046	–0.125	0.163	–0.086	–0.019	–0.045	–0.056
40–44	0.190	–0.103	–0.012	–0.033	–0.103	0.154	–0.092	–0.016	–0.051	–0.088
45–49	0.207	–0.087	–0.009	–0.036	–0.117	0.153	–0.050	–0.007	–0.021	–0.063
50–54	0.218	–0.099	–0.020	–0.038	–0.090	0.153	–0.071	–0.013	–0.046	–0.066
55–59	0.238	–0.088	–0.015	–0.036	–0.107	0.155	–0.061	–0.012	–0.034	–0.045
60–64	0.280	–0.052	–0.007	–0.016	–0.049	0.136	–0.050	–0.010	–0.036	–0.023
65–69	0.280	–0.052	–0.008	–0.027	–0.030	0.136	–0.050	–0.012	0.000	–0.057
70–74	0.290	–0.039	–0.006	–0.007	–0.031	0.160	–0.026	–0.006	–0.007	–0.020
75–79	0.290	–0.039	–0.006	–0.007	–0.031	0.160	–0.026	–0.006	–0.007	–0.020
80+	0.290	–0.039	–0.006	–0.007	–0.031	0.160	–0.026	–0.006	–0.007	–0.020
Total (18+)	**0.189**	**–0.093**	**–0.016**	**–0.034**	**–0.080**	**0.139**	**–0.080**	**–0.018**	**–0.037**	**–0.057**

Ectopic pregnancy (ICD-9 codes 633, 761.4)

Age	Male					Female				
	Ex-smoker	Current smoker (cigarettes per day)				Ex-smoker	Current smoker (cigarettes per day)			
		All	**1–14**	**15–24**	**25+**		**All**	**1–14**	**15–24**	**25+**
Exposed										
All ages	—	—	—	—	—	0.21	0.32	0.29	0.09	0.17
General population										
18–19	—	—	—	—	—	0.032	0.144	0.090	0.009	0.006
20–24	—	—	—	—	—	0.035	0.130	0.052	0.008	0.015
25–29	—	—	—	—	—	0.038	0.133	0.062	0.008	0.011
30–34	—	—	—	—	—	0.053	0.125	0.051	0.008	0.016
35–39	—	—	—	—	—	0.057	0.098	0.038	0.007	0.011
40–44	—	—	—	—	—	0.053	0.103	0.031	0.008	0.016
45–49	—	—	—	—	—	0.057	0.061	0.014	0.003	0.012
50–54	—	—	—	—	—	0.055	0.083	0.027	0.007	0.012
55–59	—	—	—	—	—	—	—	—	—	—
60–64	—	—	—	—	—	—	—	—	—	—
65–69	—	—	—	—	—	—	—	—	—	—
70–74	—	—	—	—	—	—	—	—	—	—
75–79	—	—	—	—	—	—	—	—	—	—
80+	—	—	—	—	—	—	—	—	—	—
Total (18+)	—	—	—	—	—	**0.048**	**0.108**	**0.038**	**0.008**	**0.014**

3. Fractions updated with prevalence data derived from the 1998 National Drug Strategy Household Survey

Spontaneous abortion: female only (ICD-9 codes 634, 761.8)	
	Any level of exposure
Exposed	
All ages	0.26
General population	
All ages	0.091

Antepartum haemorrhage: females and newborn males (ICD-9 codes 640, 641, 762.0, 762.1)	
	Any level of exposure
Exposed	
All ages	0.38
General population	
All ages	0.148

Hypertension in pregnancy: females and newborn males (ICD-9 codes 642, 760.0)	
	Any level of exposure
Exposed	
All ages	–0.28
General population	
All ages	–0.065

Low birthweight: females and newborn males (ICD-9 codes 656.5, 764, 765)	
	Any level of exposure
Exposed	
All ages	0.51
General population	
All ages	0.225

Premature rupture of membranes: females and newborn males (ICD-9 codes 658.1, 658.2, 761.1)	
	Any level of exposure
Exposed	
All ages	0.48
General population	
All ages	0.206

SIDS (and smoking during pregnancy), males and females aged less than 5 years (ICD-9 code 798.0)	
	Any level of exposure
Exposed	
All ages	0.64
General population	
All ages	0.329

5 Illicit drugs

5.1 Introduction

In this chapter we are concerned with quantifying morbidity and mortality in Australia caused by the following groups of illicit drugs:

- cannabis—for, example marijuana and hashish;
- opiates—for example heroin;
- stimulants—for, example cocaine and amphetamines;
- hallucinogens—for example LSD;
- anabolic steroids.

A full discussion of these drugs is beyond the scope of this report. We adopted the approach of English et al.—a full discussion of illicit drugs and aetiological fractions can be found in their report (1995, pp. 497–513).

The majority of conditions associated with illicit drugs have an aetiological fraction of one by definition. In other words, the illicit drug use is the only cause of the condition. This means that no fraction value needs to be estimated. These are conditions that are defined by association with an illicit drug—opiate dependence is an example. They are listed in Table 5.1. In cases where a fraction is estimated, the relevant prevalence data are presented along with the discussion of the fraction value.

The only condition related to illicit drug use for which we estimated a new risk-ratio was road injuries. English et al. did not calculate a fraction for road injuries because of the lack of Australian studies. They found that the great majority of relevant studies were conducted in the United States and were inappropriate as the basis of an Australian aetiological fraction because of the different prevalence and patterns of drug use. We found more recent Australian data on which to base risk-ratio and prevalence estimates.

We found prevalence data to update the fractions for drug-related cases of antepartum haemorrhage and low birthweight. We also updated the fraction for HIV/AIDS cases related to injecting drug use based on the most recent data from the *Australian HIV Surveillance Report* (National Centre in HIV Epidemiology and Clinical Research 1998). The remaining fractions were left at the values estimated by English et al.

Table 5.1: Conditions associated with illicit drug use that have an aetiological fraction of one

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)]
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)]
Other related causes	
Drug psychoses	292
Maternal drug dependence	648.3
Newborn drug toxicity	760.7, 779.5

(a) Chapter 17 code used only for calculating numbers of drug-caused hospital episodes and patient days.

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

5.2 Revised aetiological fractions for illicit drugs

5.2.1 Illicit drug use and road injuries

English et al. found several case series of road injuries that presented data on the proportion of cases exposed to illicit drugs. Only one paper (McLean et al. 1987) was of Australian origin, and it dealt with only a small number of exposed cases. English et al. noted,

> As the prevalence and patterns of drug use, particularly cocaine, are very different in the USA compared with Australia, it would be inappropriate to generalise the results of these studies to the Australian population…Therefore, no attempt has been made to pool study results, or to apply them to Australian road injury data. (1995, p. 574)

They did not derive an aetiological fraction for illicit drugs and road injury deaths.

Revised aetiological fractions for illicit drugs and road injuries

Responsibility analysis is a methodology used to make an assessment of the driver's culpability, or responsibility, in an accident. Factors (such as the condition of a road, adherence to road laws, and fatigue) mitigating a driver's responsibility in each accident are identified and scored. Given a sufficient number of mitigating circumstances, a driver could be found to be either partly or totally exonerated from blame and scored as either a contributory or a non-culpable driver. If drugs present in a driver contributed to accident causation, it would be expected that they would be over-represented among culpable drivers (those drivers whose culpability score does not exonerate them from blame) (Robertson & Drummer 1994).

Drummer (1994) combined death data from separate studies for New South Wales, Victoria and Western Australia covering the period 1990 to 1993 to determine the culpability of drivers killed in road traffic accidents, so as to determine if drug use by drivers contributed to accident causation. The basis of this analysis was to determine the culpability of drivers after the review of eight mitigating factors in the absence of knowledge of the involvement of drugs in the accident or the presence of drugs in the body fluids of the deceased. Drivers were grouped into categories, based on predetermined responsibility guidelines, as culpable, contributory and non-culpable. The *culpability ratio* is defined as the ratio of the number of drivers in the culpable group to the number of drivers in the non-culpable group.

Overall, 1,045 drivers were included in the analysis, representing 57% of all driver deaths occurring during the period. The exclusions were largely due to a lack of toxicology data or insufficient information for the purpose of assessing responsibility.

Alcohol was present in 36% of cases; illicit drugs were detected in 22% of cases. Of the illicit drugs, cannabis was the most common (11%), followed by amphetamines and related stimulants (3.7%), benzodiazepines (3.1%), and opiates (2.7%). In one case cocaine metabolites were detected in the urine.

Responsibility analysis showed that in 73% of the accidents the driver was culpable and in 18% not culpable. Drivers who had both alcohol and drugs of any type were more at risk than the control group, but no more so than the alcohol-only group. Drivers with drugs only had a slightly higher culpability ratio than the drug-free group, but this was not statistically significant. No differences were evident when the data were broken down by State.

Drivers aged less than 25 years and over 60 years had significantly higher ($p<0.05$) culpability ratios than did drivers aged 26–59 years. Culpability ratios for drug-free drivers aged 18–25 years and 26–59 years were 3.2 and 1.8, respectively, compared with an overall mean culpability ratio of 2.4 for drug-free drivers.

Table 5.2 provides a summary of the demographic information available for the drivers included in Drummer's responsibility analysis and gives a breakdown of both licit and illicit substances detected by toxicology. Alcohol, cannabis and stimulants were more frequently found among drivers aged less than 25 years.

Table 5.2: Age and sex of drivers included in responsibility analysis

Drug class	Mean age	Age range	Percentage of females
All drivers	34 ± 15	15 – 87	22.0
Alcohol	31 ± 12	16 – 78	10.0
Cannabis	25 ± 6	15 – 47	8.9
Benzodiazepines	40 ± 18	21 – 80	28.0
Amphetamines and related timulants	29 ± 11	18 – 73	13.0
Opiates	36 ± 14	16 – 75	32.0
Miscellaneous drugs	46 ± 20	16 – 87	29.0

Source: Drummer 1994.

The 138 cases involving drugs other than alcohol had a culpability ratio of 3.3. There were 112 cannabis cases in total. The 43 cannabis cases not involving alcohol or any other psychoactive drug had a culpability ratio of 1.5, which was half that for the control group ($p<0.05$).

Of the 39 cases involving amphetamines and related stimulants, 33 were culpable ($p<0.05$), although 10 of these cases also involved alcohol. Of the 21 drivers among whom only stimulants were detected as the psychoactive drug, the culpability ratio of 4.0 was not significantly different from that of the control group ($p>0.05$).

Of the 28 cases involving opiates, only 13 did not involve another psychoactive drug. For the opiate-only cases, the culpability ratio was 5.5 which again was not significantly different from that of the control group ($p>0.05$).

Culpability ratios showed an age dependence. For example, for drug-free drivers, the highest culpability ratios were among the under 25 and over 60 age groups, whereas those aged 26–35 and 35–39 years had less than average culpability scores.

Table 5.3: Culpability score for drivers involved in motor vehicle accidents, by drug class

Drug class	Number	Culpable	Contributor	Nonculpable	Ratio
Drug free	532	339	53	140	2.4
Cannabis only	43	21	8	14	1.5
Stimulants only	21	16	1	4	4.0
Opiates only	13	11	0	2	5.5

Source: Drummer 1994.

Drummer calculated relative risk and confidence intervals for all accident deaths by dividing the culpability ratio of the drug group by the culpability ratio of the drug-free

(control) group. The statistical methodology used was that of Fischer's exact test. The results are shown in Table 5.4.

Table 5.4: Relative risk and confidence intervals for drivers involved in motor vehicle accidents

Drug group	Number	Culpability ratio	Relative risk	95% CI
Drug free	532	2.4	1.0	—
Cannabis only	43	1.5	0.6	0.3–1.2
Stimulants only	21	4.0	1.6	0.5–5.0
Opiates only	13	5.5	2.3	0.5–10.0

Source: Drummer 1994.

None of the risk estimates in Table 5.4 is statistically significant. For both stimulants and opiates, this may simply be a function of the small numbers of cases available, leading to an inability to detect a significant difference. Sample size calculations suggest that for opiates a sample size of 1,500 cases would be required to show a significant increase in the relative risk, whereas for stimulants the sample size required would be 6,000 (Drummer 1994).

The apparent protective effect attributed to cannabis-only use may be a result of the fact that the measurement of inactive carboxy-THC, which can persist in blood for several days or in urine for several weeks, is a poor proxy for the assessment of psychoactive THC, which is more difficult to measure. Furthermore, it is generally accepted that the use of cannabis can cause impairment for up to two to four hours and that there is little compelling evidence that impairment lasts beyond this time, even among regular users (Drummer 1994).

For the calculation of the aetiological fraction, the prevalence of exposure among cases was combined with the relative risk estimates derived by Drummer. Despite the relative risk of less than one, no protective effect has been ascribed to cannabis use, given the limitations described.

For stimulant-only use the prevalence was 0.020 (21 out of 1,045). Based on the relative risk estimate of 1.6, this gave an aetiological fraction of 0.008. For opiate-only use the prevalence was 0.012 (13 out of 1,045). Based on the relative risk estimate of 2.3, this gave an aetiological fraction of 0.007.

As noted, the culpability ratios were age dependent, so if the fractions were to be applied to deaths or hospital separations among drivers, the estimates should vary by age. But the fractions are intended for application to all deaths and injuries arising from road traffic accidents so the single estimate of the fraction applied to all ages is more appropriate.

5.2.2 Illicit drug use and HIV/AIDS

Infection with the human immunodeficiency virus—the virus that causes acquired immune deficiency syndrome—and new cases of AIDS are notifiable in Australia. A standard set of information is collected on each notification. All identified cases of HIV and AIDS are then reported to the National Centre in HIV Epidemiology and Clinical Research, which produces the *Australian HIV Surveillance Report*, presenting data on HIV infection and cases of AIDS. We extracted data for 1996, 1997 and 1998. These were reported under a number of exposure categories, including 'male homosexual/bisexual contact and injecting drug use' and 'injecting drug use'. We followed English et al. (1995) in attributing most of the cases in the first category to sexual contact rather than drug use and including only the second category in our calculations. The results are presented in Table 5.5.

Table 5.5: New cases of HIV infection and AIDS, by sex and exposure category, 1996 to 1998

	Males			Females		
Year	Injecting drug use	All exposure categories	Proportion attributable to injecting drug use	Injecting drug use	All exposure categories	Proportion attributable to injecting drug use
HIV						
1996	2	165	0.012	1	7	0.143
1997	2	147	0.014	1	9	0.111
1998	1	141	0.007	2	3	0.667
AIDS						
1996	19	629	0.030	5	33	0.152
1997	10	343	0.029	6	26	0.231
1998	15	330	0.045	3	16	0.188

Source: NCHECR (1998).

We followed English et al. in using the reported proportions for AIDS as the aetiological fraction for both deaths and hospitalisations. Under-reporting of injecting drug use probably means these are underestimates.

5.2.3 Illicit drug use and antepartum haemorrhage

Opiates

English et al. (1995) derived a relative risk estimate of 2.36 for opiate use and antepartum haemorrhage, with a 95% confidence interval of 1.35–4.12. A relative risk estimate of this size is consistent with a moderately strong association between opiate use in pregnancy and antepartum haemorrhage, although English et al. noted that this result is not adjusted for any potential confounders. They did not, however, find any reliable Australian data on illicit drug use during pregnancy, so they used the prevalence of illicit opiate use in women of child-bearing age. They derived an estimated aetiological fraction of 0.002 but noted that because of the dearth of prevalence data it is probably inaccurate. Because of this and the fact that the estimate is substantially less than 1%, they did not apply this fraction to Australian morbidity or mortality data.

The 1998 National Drug Strategy Household Survey collected data that would enable estimation of the proportion of pregnant women who take opiates. The final sample count was, however, too small to allow a meaningful estimate, so we followed English et al. in using as a prevalence estimate the proportion of women of child-bearing age (14–39 years) who use opiates. We derived an estimate from the 1995 and 1998 National Drug Strategy Household Surveys and used linear interpolation to derive estimates for 1996 and 1997 (Table 5.6). These were then combined with the risk-ratio estimate of 2.36 from English et al. to derive the aetiological fractions (Table 5.7). The fraction estimates for 1996 to 1998 are higher than 1% because of the higher prevalence estimates. Hence, although we still have poor prevalence data, we do apply the fractions to the data on mortality and hospital separations.

Cocaine

English et al. (1995) derived a relative risk estimate of 3.89 for cocaine use and antepartum haemorrhage, with a 95% confidence interval of 2.80–5.35. Although this estimate is quite

high, English et al. noted that it is possible that other maternal factors—such as use of other drugs, inadequate antenatal care, and infection—could account for part of the observed association. As with opiates, they did not find any reliable Australian data on illicit drug use during pregnancy. Instead, they used the prevalence of cocaine use in women of child-bearing age as a basis for the fraction's estimation. Their estimate of the fraction was 0.02. Although they noted that this value was probably inaccurate, they did apply it to their data on mortality and morbidity.

The 1998 National Drug Strategy Household Survey collected data that would enable estimation of the proportion of pregnant women who take cocaine, but again the sample size was too small to allow a meaningful estimate. Instead, we again followed English et al. in using as a prevalence estimate the proportion of women of childbearing age (14–39 years) who use cocaine. We derived an estimate from the 1995 and 1998 National Drug Strategy Household Surveys and used linear interpolation to derive estimates for 1996 and 1997 (Table 5.6). These were then combined with the risk ratio estimate of 3.89 from English et al. to derive the aetiological fractions (Table 5.7).

Table 5.6: Proportion of women aged 14–39 years using opiates or cocaine, 1995 to 1998

Year	Proportion using opiates	Proportion using cocaine
	(per cent)	
1995	0.90	1.14
1996	0.92	1.26
1997	0.93	1.37
1998	0.96	1.60

Source: AIHW analysis of the 1995 and 1998 National Drug Strategy Household Survey data.

Table 5.7: Revised aetiological fractions for antepartum haemorrhage and opiate or cocaine use

Year	Aetiological fraction opiates	Aetiological fraction cocaine
Exposed		
All years	0.58	0.74
General population		
1995	0.012	0.032
1996	0.012	0.035
1997	0.012	0.038
1998	0.013	0.044

Source: AIHW analysis of prevalence data in Table 5.6 and estimates of relative risk reported by English et al. (1995).

5.2.4 Illicit drug use and low birthweight

Because of the significant overlap between low birthweight, prematurity and intrauterine growth retardation, and the difficulty in relating each of these features or combinations of two or three of these features to specific ICD-9 codes—low birthweight was selected as the outcome most representative of conditions covered by ICD-9 codes 656.5, 764 and 765.

Opiates

English et al. (1995) derived a relative risk estimate of 3.34 for maternal opiate use in pregnancy and low birthweight, with a 95% confidence interval of 3.07–3.64. As with antepartum haemorrhage, they used the prevalence of illicit opiate use in women of child-bearing age as a proxy. They derived an estimated aetiological fraction of 0.004 but noted that because of the dearth of prevalence data it is probably inaccurate. Because of this and the fact that the estimate is substantially less than 1%, they did not apply this fraction to Australian morbidity or mortality data.

We followed English et al. in using as a prevalence estimate the proportion of women of child-bearing age (14 to 39 years) who use opiates (Table 5.6). This was combined with the risk-ratio estimate of 3.34 from English et al. to derive the aetiological fractions (Table 5.8). The fraction estimates for 1996 to 1998 are higher than 1% because of the higher prevalence estimates. Hence, although we still have poor prevalence data, we applied the fractions to our data on mortality and hospital separations.

Cocaine

English et al. derived a relative risk estimate of 2.97 for maternal cocaine use in pregnancy and low birthweight, with a 95% confidence interval of 2.31–3.80. As with antepartum haemorrhage, they used the prevalence of illicit cocaine use in women of child-bearing age as a proxy. They derived an estimated aetiological fraction of 0.015 but noted that because of the dearth of prevalence data it is likely to be inaccurate.

We followed English et al. in using the proportion of women of child-bearing age (14 to 39 years) who use cocaine as a prevalence estimate (Table 5.6). This was combined with the risk-ratio estimate of 2.97 from English et al. to derive the aetiological fractions (Table 5.8).

Table 5.8: Revised aetiological fractions for low birthweight and opiate or cocaine use

Year	Aetiological fraction opiates	Aetiological fraction cocaine
Exposed		
All years	0.70	0.66
General population		
1995	0.021	0.022
1996	0.021	0.024
1997	0.021	0.026
1998	0.022	0.031

Source: AIHW analysis of prevalence data in Table 5.6 and estimates of relative risk reported by English et al. (1995).

5.3 Unrevised aetiological fractions for illicit drugs

5.3.1 Opiates and suicide

Unlike suicide and self-inflicted poisoning by barbiturates and by other sedatives, ICD-9 does not distinguish between suicide by opiate overdose and suicide by using any type of

analgesic, antipyretic or antirheumatic drug. It is therefore necessary to derive an aetiological fraction for all suicides due to opiates if this cause of mortality is to be quantified.

Both Holman et al. (1990) and English et al. (1995) used a review of death certificates and coronial records of suicides in Western Australia from 1974 to 1984 (Swensen 1988) to estimate the fraction of suicides caused by opiates in Australia. This fraction was 0.09 and since we found no more recent Australian data, we used the same fraction.

5.3.2 Injecting drug use and viral hepatitis

English et al. derived separate fractions for hepatitis B and hepatitis non-A, non-B. The viral agent responsible for most non-A, non-B hepatitis has been identified and named the hepatitis C virus. But hepatitis C is not distinguished from other types of non-A, non-B hepatitis in mortality data, so English et al. calculated pooled estimates and aetiological fractions for non-A, non-B hepatitis rather than hepatitis C.

We did not revise this fraction and used the English et al. estimates (Table 5.9).

Table 5.9: Aetiological fractions for injecting drug use and viral hepatitis

Condition	Males	Females
Exposed		
Hepatitis B	0.98	0.98
Hepatitis, non-A, non-B	0.98	0.98
General population		
Hepatitis B	0.29	0.29
Hepatitis, non-A, non-B	0.42	0.42

Source: English et al. (1995).

5.3.3 Injecting drug use and infective endocarditis

Holman et al. derived an estimated aetiological fraction of 0.14 for injecting drug use and infective endocarditis. English et al. found no additional studies on this association that met their inclusion criteria and so retained 0.14 as the value for their fraction estimate. We found no additional studies either, so we also used 0.14 as the aetiological fraction.

6 Attributable mortality in 1998

Tables 6.1 to 6.3 show the number of deaths attributable to tobacco, alcohol and illicit drugs in 1998, classified by age, the drug involved and the cause of death, for males, females and persons. Tables 6.4 to 6.6 show the number of PYLL attributable to tobacco, alcohol and illicit drugs in 1998, classified by age, the drug involved and the cause of death, for males, females and persons. These are summary results for groups of causes of death. Detailed tables for individual causes of death are available from the Institute on request.

6.1 Alcohol

Alcohol has both a causative and an apparent preventive effect on deaths. The largest number of alcohol-related deaths among men are due to alcoholism and alcoholic liver cirrhosis. The second largest number are due to cancer. Close to half (44%) of the male alcohol-related cancer deaths are due to oesophageal cancer, while around a quarter (24%) are due to liver cancer.

The pattern is different for women. The largest number of female alcohol-related deaths are due to cancer. More than half of these deaths (55%) are due to breast cancer; oesophageal cancer and liver cancer account for 24% and 13% of alcohol-related cancer deaths respectively. Alcoholism and alcoholic liver cirrhosis account for the second-largest number of alcohol-related deaths among females.

For both men and women the third largest category of alcohol-related deaths is road injuries. The number of alcohol-related road injury deaths is much larger for men than for women, with the majority of these deaths occurring in the 15 to 34 years age range.

Alcohol has an apparent protective effect against some conditions classified in the 'other' category for both men and women. It should be noted, however, that this estimate is a net figure that includes an increased death rate due to some causes offset by a reduced death rate from other causes. The protective effect of alcohol relates mainly to a reduced risk of death from ischaemic heart disease and stroke with moderate alcohol consumption. However, as noted in Appendix A, high alcohol consumption is related to an increased risk of death from a number of causes in this category—including an increased risk of death from stroke.

The largest cause of alcohol-related PYLL for men is alcoholism and alcoholic liver cirrhosis. However, the second largest cause of male alcohol-related PYLL is road injuries rather than cancer. This reflects the fact that, although there are more alcohol-related cancer deaths than road injury deaths among males, the road injury deaths tend to happen at a younger age and hence contribute more PYLL.

Alcohol is related to a net increase in PYLL in the 'other 'category for men, despite being related to a net decrease in deaths. This is because of causes such as suicide and assault: they contribute a moderate number of male alcohol-related deaths at young ages but a large number of PYLL, which offsets the reduced number of PYLL at older ages related to ischaemic heart disease and stroke.

The pattern of alcohol-related PYLL for women is similar to that for alcohol-related deaths for women, with cancer contributing the largest number, followed by alcoholism and

alcoholic liver cirrhosis. As with female alcohol-related deaths, the net effect of alcohol on the causes of death in the 'other' category is a reduction in PYLL.

6.2 Tobacco

For men, the largest number of tobacco-related deaths are caused by cancer, which is responsible for around 43% of all male tobacco-related deaths. These cancer deaths are dominated by lung cancer, which accounts for 77% of male tobacco-related cancer deaths. The remaining tobacco-related male cancer deaths are caused by several different types of cancer—including oesophageal cancer (6%), oropharyngeal cancer (4%), bladder cancer (4%) and pancreatic cancer (3%).

The second- and third- largest causes of male tobacco-related deaths are ischaemic heart disease and chronic obstructive pulmonary disease, which account for 22% and 19% of male tobacco-related deaths respectively. The 'other direct smoking' category accounts for 15% of male tobacco-related deaths. This category comprises a variety of causes, the largest contributors being stroke (43% of the 'other' category), atherosclerosis (29%) and pneumonia (19%).

For women the pattern is a little different. As with men, the largest number of tobacco-related deaths for women are caused by cancer, which is responsible for around 32% of all female tobacco-related deaths. This category is also dominated by lung cancer, which accounts for 75% of female tobacco-related cancer deaths. The remaining tobacco-related female cancer deaths are caused by several different types of cancer—including oesophageal cancer (7%), pancreatic cancer (7%), bladder cancer (4%) and oropharyngeal cancer (3%).

Chronic obstructive pulmonary disease accounts for a larger proportion of female tobacco-related deaths (22%) than does ischaemic heart disease (20%). However, the 'other direct smoking' category is larger than either of these, accounting for 25% of female tobacco-related deaths. Again, this category comprises a variety of causes, the largest contributors being stroke (46% of the 'other' category), atherosclerosis (23%) and pneumonia (17%).

The tobacco-related PYLL for both men and women follow the same pattern as the tobacco-related deaths. Hence the largest number of tobacco-related PYLL for both men and women is due to cancer. However, while more male tobacco-related PYLL are due to ischaemic heart disease than chronic obstructive pulmonary disease, the reverse is true for female tobacco-related PYLL.

6.3 Illicit drugs

The patterns of illicit drug-related deaths for men and women are similar. The largest proportion of these deaths is directly related to opiate dependence, abuse or poisoning (79% for men and 69% for women). The second-largest proportion is due to suicide (13% for both men and women). Hepatitis is a slightly larger cause of illicit drug-related deaths for women (10%) than for men (4%), as are road traffic accidents (4% for women and 2% for men). However, the absolute number of illicit drug-related hepatitis and road traffic accident deaths is small, so these proportions should be interpreted with caution.

The pattern of illicit drug-related PYLL is similar to that for illicit drug-related deaths. The largest proportion of these PYLL is directly related to opiate dependence, abuse or poisoning (79% for men and 73% for women). The second-largest proportion is due to suicide (14% for both men and women). Hepatitis is a slightly larger cause of illicit drug-related deaths for

women (5%) than for men (2%), as are road traffic accidents (3% for women and 2% for men). However, the proportions for PYLL due to illicit drug-related hepatitis and road traffic accident deaths should be interpreted with caution because of the small number of deaths on which they are based.

Table 6.1: Deaths attributable to drug use, by drug involved, cause of death and age: males, 1998

	Age				
Drug involved/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	3	245	423	672
Alcoholism and alcoholic liver cirrhosis	0	26	451	226	703
Road injuries	12	245	111	19	387
Other	3	406	129	–1,667	–1,129
Total alcohol	**15**	**680**	**936**	**–998**	**633**
Tobacco					
Direct smoking					
Cancer	0	0	1,375	4,207	5,582
Ischaemic heart disease	0	27	1,114	1,707	2,848
Chronic obstructive pulmonary disease	0	0	229	2,275	2,504
Other	43	27	326	1,565	1,961
Environmental tobacco smoke	14	0	2	32	48
Total tobacco	**57**	**54**	**3,047**	**9,787**	**12,944**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	0	0	0	0
Opiates	0	315	151	0	466
Cocaine	0	3	0	0	3
Amphetamines	0	1	1	0	2
Hallucinogens	0	1	0	0	1
Poisoning	0	0	0	0	0
Opiates	0	126	64	2	192
Psychostimulants	0	2	1	0	3
Hallucinogens	0	0	0	0	0
Suicide	0	86	25	0	111
Antepartum haemorrhage	2	0	0	0	2
Low birthweight	1	0	0	0	1
Hepatitis B	0	0	8	4	12
Hepatitis, non-A, non-B	0	1	12	5	19
AIDS	0	2	4	0	6
Infective endocarditis	0	1	0	0	1
Drug psychoses	0	0	0	0	0
Maternal drug dependence	0	0	0	0	0
Newborn toxicity	1	0	0	0	1
Road traffic accidents	1	9	6	2	18
Total illicit drugs	**5**	**547**	**272**	**14**	**838**
Total all drugs	**77**	**1,280**	**4,254**	**8,802**	**14,414**

Note: Columns may not add exactly to totals due to rounding.

Table 6.2: Deaths attributable to drug use, by drug involved, cause of death and age: females, 1998

	Age				
Drug involved/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	8	177	301	485
Alcoholism and alcoholic liver cirrhosis	0	18	132	74	224
Road injuries	3	28	20	3	53
Other	3	80	–34	–3,815	–3,766
Total alcohol	**6**	**134**	**294**	**–3,438**	**–3,004**
Tobacco					
Direct smoking					
Cancer	0	0	454	1,506	1,960
Ischaemic heart disease	0	7	225	954	1,186
Chronic obstructive pulmonary disease	0	0	130	1,205	1,335
Other	33	19	179	1,285	1,515
Environmental tobacco smoke	9	0	8	63	80
Total tobacco	**42**	**26**	**995**	**5,012**	**6,075**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	0	0	0	0
Opiates	0	67	32	2	101
Cocaine	0	1	0	0	1
Amphetamine	0	1	0	0	1
Hallucinogens	0	0	0	0	0
Poisoning	0	0	0	0	0
Opiates	0	12	13	2	27
Psychostimulants	0	0	0	0	0
Hallucinogens	0	0	0	0	0
Suicide	0	17	7	0	24
Antepartum haemorrhage	2	0	0	0	2
Low birthweight	1	0	0	0	1
Hepatitis B	0	0	1	2	4
Hepatitis, non-A, non-B	0	0	4	11	15
AIDS	0	0	1	0	1
Infective endocarditis	0	0	0	0	0
Drug psychoses	0	0	0	0	0
Maternal drug dependence	0	0	0	0	0
Newborn toxicity	0	0	0	0	0
Road traffic accidents	1	3	2	2	7
Total illicit drugs	**4**	**102**	**60**	**19**	**185**
Total all drugs	**51**	**262**	**1,350**	**1,593**	**3,256**

Note: Columns may not add exactly to totals due to rounding.

Table 6.3: Deaths attributable to drug use, by drug involved, cause of death and age: persons, 1998

	Age				
Drug involved/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	11	422	724	1,157
Alcoholism and alcoholic liver cirrhosis	0	44	583	300	927
Road injuries	15	273	130	22	440
Other	6	486	95	–5,482	–4,895
Total alcohol	**21**	**814**	**1230**	**–4,436**	**–2,371**
Tobacco					
Direct smoking					
Cancer	0	0	1,829	5,713	7,542
Ischaemic heart disease	0	34	1,339	2,661	4,034
Chronic obstructive pulmonary disease	0	0	359	3,480	3,839
Other	76	46	505	2,849	3,476
Environmental tobacco smoke	23	0	10	95	128
Total tobacco	**99**	**80**	**4,042**	**14,799**	**19,019**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	0	0	0	0
Opiates	0	382	183	2	567
Cocaine	0	4	0	0	4
Amphetamine	0	2	1	0	3
Hallucinogens	0	1	0	0	1
Poisoning	0	0	0	0	0
Opiates	0	138	77	4	219
Psychostimulants	0	2	1	0	3
Hallucinogens	0	0	0	0	0
Suicide	0	103	32	0	135
Antepartum haemorrhage	4	0	0	0	4
Low birthweight	2	0	0	0	2
Hepatitis B	0	1	9	6	16
Hepatitis, non-A, non-B	0	2	16	16	34
AIDS	0	2	5	0	7
Infective endocarditis	0	1	0	0	1
Drug psychoses	0	0	0	0	0
Maternal drug dependence	0	0	0	0	0
Newborn toxicity	1	0	0	0	1
Road traffic accidents	2	12	8	4	25
Total illicit drugs	**9**	**649**	**332**	**33**	**1,023**
Total all drugs	**129**	**1,542**	**5,604**	**10,396**	**17,671**

Note: Columns may not add exactly to totals due to rounding.

Table 6.4: PYLL attributable to drug use, by drug involved, cause of death and age: males, 1998

	Age				
Drug involved/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	88	4,215	3,695	7,998
Alcoholism and alcoholic liver cirrhosis	0	675	8,418	2,225	11,317
Road injuries	356	6,637	2,418	160	9,570
Other	101	10,845	3,762	–11,841	2,867
Total alcohol	**457**	**18,244**	**18,812**	**–5,761**	**31,752**
Tobacco					
Direct smoking					
Cancer	0	0	22,499	36,169	58,667
Ischaemic heart disease	0	690	19,443	12,667	32,800
Chronic obstructive pulmonary disease	0	0	3,556	17,159	20,715
Other	1,312	720	5,712	10,447	18,191
Environmental tobacco smoke	413	2	39	229	683
Total tobacco	**1,725**	**1,411**	**51,249**	**76,670**	**131,055**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	0	0	0	0
Opiates	0	8,365	3,489	0	11,853
Cocaine	0	78	0	0	78
Amphetamine	0	28	24	0	52
Hallucinogens	0	28	0	0	28
Poisoning	0	0	0	0	0
Opiates	0	3,354	1,445	12	4,811
Psychostimulants	0	54	23	0	77
Hallucinogens	0	0	0	0	0
Suicide	0	2,297	594	0	2,891
Antepartum haemorrhage	63	0	0	0	63
Low birthweight	29	0	0	0	29
Hepatitis B	0	8	161	36	204
Hepatitis, non-A, non-B	0	32	237	45	315
AIDS	0	44	92	3	139
Infective endocarditis	0	15	7	0	22
Drug psychoses	0	0	0	0	0
Maternal drug dependence	0	0	0	0	0
Newborn toxicity	30	0	0	0	30
Road traffic accidents	28	245	114	20	407
Total illicit drugs	**149**	**14,548**	**6,185**	**116**	**20,998**
Total all drugs	**2,332**	**34,203**	**76,245**	**71,025**	**183,805**

Note: Columns may not add exactly to totals due to rounding.

Table 6.5: PYLL attributable to drug use, by drug involved, cause of death and age: females, 1998

	Age				
Drug involved/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	208	3,533	2,701	6,443
Alcoholism and alcoholic liver cirrhosis	0	486	2,779	831	4,095
Road injuries	91	782	473	24	1,369
Other	89	2,221	–288	–24,534	–22,513
Total alcohol	**180**	**3,696**	**6,496**	**–20,978**	**–10,605**
Tobacco					
Direct smoking					
Cancer	0	0	7,532	12,336	19,867
Ischaemic heart disease	0	184	3,849	5,816	9,850
Chronic obstructive pulmonary disease	0	0	2,064	8,861	10,925
Other	995	498	3,261	7,296	12,051
Environmental tobacco smoke	278	2	137	413	830
Total tobacco	**1,273**	**685**	**16,843**	**34,723**	**53,523**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	0	0	0	0
Opiates	0	1,802	734	20	2,555
Cocaine	0	27	0	0	27
Amphetamine	0	28	0	0	28
Hallucinogens	0	0	0	0	0
Poisoning	0	0	0	0	0
Opiates	0	327	282	14	623
Psychostimulants	0	0	0	0	0
Hallucinogens	0	0	0	0	0
Suicide	0	462	168	0	630
Antepartum haemorrhage	74	0	0	0	74
Low birthweight	19	0	0	0	19
Hepatitis B	0	8	21	22	50
Hepatitis, non-A, non-B	0	11	76	97	184
AIDS	0	5	21	0	26
Infective endocarditis	0	8	3	0	11
Drug psychoses	0	0	0	0	0
Maternal drug dependence	0	0	0	0	0
Newborn toxicity	0	0	0	0	0
Road traffic accidents	18	71	47	14	151
Total illicit drugs	**112**	**2,748**	**1,352**	**166**	**4,377**
Total all drugs	**1,565**	**7,129**	**24,691**	**13,911**	**47,295**

Note: Columns may not add exactly to totals due to rounding.

Table 6.6: PYLL attributable to drug use, by drug involved, cause of death and age: persons, 1998

	Age				
Drug involved/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	296	7,748	6,397	14,441
Alcoholism and alcoholic liver cirrhosis	0	1,161	11,196	3,055	15,413
Road injuries	447	7,418	2,890	184	10,940
Other	190	13,066	3,473	–36,375	–19,646
Total alcohol	**637**	**21,941**	**25,309**	**–26,739**	**21,147**
Tobacco					
Direct smoking					
Cancer	0	0	30,030	48,504	78,534
Ischaemic heart disease	0	874	23,292	18,484	42,650
Chronic obstructive pulmonary disease	0	0	5,620	26,020	31,640
Other	2,307	1,218	8,973	17,743	30,241
Environmental tobacco smoke	691	4	176	642	1,513
Total tobacco	**2,998**	**2,096**	**68,091**	**111,393**	**184,579**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	0	0	0	0
Opiates	0	10,166	4,222	20	14,408
Cocaine	0	104	0	0	104
Amphetamine	0	56	24	0	80
Hallucinogens	0	28	0	0	28
Poisoning	0	0	0	0	0
Opiates	0	3,681	1,727	25	5,434
Psychostimulants	0	54	23	0	77
Hallucinogens	0	0	0	0	0
Suicide	0	2,760	762	0	3,522
Antepartum haemorrhage	137	0	0	0	137
Low birthweight	48	0	0	0	48
Hepatitis B	0	15	181	58	254
Hepatitis, non-A, non-B	0	44	313	142	499
AIDS	0	49	112	3	164
Infective endocarditis	0	23	10	0	33
Drug psychoses	0	0	0	0	0
Maternal drug dependence	0	0	0	0	0
Newborn toxicity	30	0	0	0	30
Road traffic accidents	46	316	161	34	557
Total illicit drugs	**261**	**17,296**	**7,536**	**282**	**25,375**
Total all drugs	**3,896**	**41,332**	**100,936**	**84,936**	**231,100**

Note: Columns may not add exactly to totals due to rounding.

7 Attributable hospital separations in 1998

Tables 7.1 to 7.3 show the number of hospital separations attributable to tobacco, alcohol and illicit drugs in 1997–98 classified by age, the drug involved and the reason for separation, for males, females and persons. Tables 7.4 to 7.6 present the number of patient days attributable to tobacco, alcohol and illicit drugs in 1997–98 classified by age, the drug involved and the reason for separation, for males, females and persons. These are summary results for groups of reasons for separation. Detailed tables for individual causes of death are available from the Institute on request.

7.1 Alcohol

Alcohol has both a causative and an apparent preventive effect on hospital separations. The largest number of alcohol-related separations among both men and women is due to alcoholism and alcoholic liver cirrhosis. The second-largest number is due to road injuries for men and cancer for women. The male cancer separations are due to oesophageal cancer (37% of attributable cancer separations), oropharyngeal cancer (30%), laryngeal cancer (20%) and liver cancer (13%). The female cancer separations are mainly due to breast cancer (67% of attributable cancer separations); the remainder are attributable to oesophageal cancer (17%), oropharyngeal cancer (9%), liver cancer (4%) and laryngeal cancer (2%).

Alcohol has an apparent protective effect against some conditions classified in the 'other' category for both men and women. It should be noted, owever, that the estimate for this category is a net figure that includes an increased separation rate due to some causes offset by a reduced separation rate due to other causes. The protective effect of alcohol relates mainly to a reduced risk of hospitalisation from ischaemic heart disease, stroke, cholelithiasis and hypertension with moderate alcohol consumption. However, as noted in Appendix A, high alcohol consumption is related to an increased risk of hospitalisation from a number of causes in this category—including stroke.

The overall net effect of alcohol on the conditions in the 'other' category is to increase hospitalisations for men. This is because the number of separations averted due to the protective effect of alcohol is offset by an increase in separations mainly due to unintentional injuries, assault and attempted suicide among younger men and pancreatitis and supraventricular cardiac dysrhythmias among middle-aged and older men. The overall effect for women is a decrease in the number of separations because of the smaller numbers having these conditions.

7.2 Tobacco

The largest specific cause of hospital separations for males attributable to tobacco is ischaemic heart disease, with 29% of the attributable separations. This is followed by cancer (21%) and chronic obstructive pulmonary disease (19%). The cancer category is dominated by lung cancer (50% of attributable cancer separations) and bladder cancer (23%). Oropharyngeal and oesophageal cancer each account for 7% of the attributable cancer

separations; the remaining cancers each account for less than 5% of the attributable cancer separations.

The 'other direct smoking' category accounts for 30% of the attributable separations for males. No single cause dominates this group, but the largest contributors are atherosclerosis (27% of other attributable separations), cardiac dysrhythmias (20%), stroke (20%) and pneumonia (18%).

The largest specific cause of hospital separations for females attributable to tobacco is chronic obstructive pulmonary disease, with 22% of the attributable separations. This is followed by ischaemic heart disease (19%) and cancer (14%). The cancer category is dominated by lung cancer (59% of attributable cancer separations) and bladder cancer (17%). Oesophageal cancer accounts for 9% of the attributable cancer separations and oropharyngeal cancer accounts for 6%. The remaining cancers each account for less than 5% of the attributable cancer separations.

The 'other direct smoking' category accounts for 43% of the attributable separations for females. Again, no single cause dominates this group, but the largest contributors are atherosclerosis (17% of other attributable separations), stroke (17%), cardiac dysrhythmias (16%) and pneumonia (16%).

7.3 Illicit drugs

As with the mortality data, the hospital separations attributable to use of illicit drugs are dominated by opiate dependence, abuse or poisoning, which accounts for 52% of attributable separations for males and 40% for females. The next-largest category of attributable separations is due to drug psychoses, which accounts for 30% of attributable separations for males and 25% for females. For males, cannabis dependence or abuse contributes 6% of the attributable separations and road traffic accidents contribute 4%; all other causes contribute less than 3% each. For females, antepartum haemorrhage and maternal drug dependence each contribute 8% each of the attributable separations; all other causes contribute less than 3% each.

Table 7.1: Hospital separations attributable to drug use, by drug, reason for separation and age: males, 1997–98

	Age				
Drug involved/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	20	1,460	1,691	3,171
Alcoholism and alcoholic liver cirrhosis	143	3,783	11,564	2,162	17,652
Road injuries	329	3,083	1,145	233	4,790
Other	229	11,813	2,419	–4,873	9,588
Total alcohol	**701**	**18,699**	**16,588**	**–786**	**35,201**
Tobacco					
Direct smoking					
Cancer	0	0	6,961	13,718	20,679
Ischaemic heart disease	0	302	20,685	7,712	28,699
Chronic obstructive pulmonary disease	0	0	3,613	14,656	18,268
Other	94	1,892	11,851	14,917	28,755
Environmental tobacco smoke	873	1	36	127	1,037
Total tobacco	**967**	**2,195**	**43,146**	**51,130**	**97,438**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	421	47	7	475
Opiates	0	2,363	824	6	3,193
Cocaine	0	21	17	1	39
Amphetamines	0	236	35	0	271
Hallucinogens	0	35	1	0	36
Poisoning	0	0	0	0	0
Opiates	0	814	278	11	1,103
Psychostimulants	0	183	35	0	218
Hallucinogens	0	87	16	1	104
Other psychotropic drugs	0	45	46	5	96
Anabolic steriods	0	1	1	0	2
Antepartum haemorrhage	0	0	0	0	0
Low birthweight	0	0	0	0	0
Hepatitis B and non-A, non-B	0	0	0	0	0
AIDS	0	0	0	0	1
Infective endocarditis	0	15	9	0	24
Drug psychoses	0	1,890	439	144	2,473
Newborn toxicity	0	0	0	0	0
Road traffic accidents	28	160	81	26	295
Total illicit drugs	**28**	**6,271**	**1,829**	**201**	**8,329**
Total all drugs	**1,696**	**27,166**	**61,563**	**50,545**	**140,969**

Note: Columns may not add exactly to totals due to rounding.

Table 7.2: Hospital separations attributable to drug use, by drug, reason for separation and age: females, 1997–98

	Age				
Drug involved/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	93	1,619	1,158	2,869
Alcoholism and alcoholic liver cirrhosis	135	2,081	5,162	728	8,107
Road injuries	81	628	296	50	1,056
Other	118	3,705	–71	–7,953	–4,200
Total alcohol	**334**	**6,508**	**7,006**	**–6,016**	**7,831**
Tobacco					
Direct smoking					
Cancer	0	0	1,965	4,328	6,293
Ischaemic heart disease	0	96	5,077	3,248	8,421
Chronic obstructive pulmonary disease	0	0	2,286	7,714	10,000
Other	48	4,894	6,779	7,720	19,442
Environmental tobacco smoke	555	2	136	238	931
Total tobacco	**603**	**4,992**	**16,243**	**23,249**	**45,087**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	153	24	0	177
Opiates	0	1492	466	9	1,967
Cocaine	0	17	3	0	20
Amphetamines	0	126	12	0	138
Hallucinogens	0	15	5	0	20
Poisoning	0	0	0	0	0
Opiates	0	405	97	4	506
Psychostimulants	0	144	21	0	165
Hallucinogens	0	59	15	0	74
Other psychotropic drugs	0	77	70	24	171
Anabolic steriods	0	0	0	0	0
Antepartum haemorrhage	0	549	78	0	627
Low birthweight	0	54	5	0	59
Hepatitis B, and non-A, non-B	0	0	0	0	0
AIDS	0	3	1	1	4
Infective endocarditis	0	11	3	0	14
Drug psychoses	0	958	335	225	1,518
Maternal drug dependence	0	471	40	0	511
Newborn toxicity	0	0	0	0	0
Road traffic accidents	16	71	53	30	170
Total illicit drugs	**16**	**4,605**	**1,228**	**293**	**6,142**
Total all drugs	**953**	**16,105**	**24,477**	**17,525**	**59,060**

Note: Columns may not add exactly to totals due to rounding.

Table 7.3: Hospital separations attributable to drug use, by drug, reason for separation and age: persons, 1997–98

	Age				
Drug involved/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	113	3,078	2,849	6,040
Alcoholism and alcoholic liver cirrhosis	278	5,864	16,726	2,890	25,759
Road injuries	410	3,711	1,442	283	5,846
Other	346	15,519	2,348	–12,825	5,388
Total alcohol	**1,034**	**25,207**	**23,594**	**–6,802**	**43,033**
Tobacco					
Direct smoking					
Cancer	0	0	8,926	18,046	26,972
Ischaemic heart disease	0	398	25,762	10,960	37,120
Chronic obstructive pulmonary disease	0	0	5,899	22,370	28,268
Other	142	6,787	18,630	22,638	48,196
Environmental tobacco smoke	1,428	2	172	365	1,968
Total tobacco	**1,570**	**7,187**	**59,389**	**74,379**	**142,525**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	574	71	7	652
Opiates	0	3,855	1,290	15	5,160
Cocaine	0	38	20	1	59
Amphetamines	0	362	47	0	409
Hallucinogens	0	50	6	0	56
Poisoning	0	0	0	0	0
Opiates	0	1,219	375	15	1,609
Psychostimulants	0	327	56	0	383
Hallucinogens	0	146	31	1	178
Other psychotropic drugs	0	122	116	29	267
Anabolic steriods	0	1	1	0	2
Antepartum haemorrhage	0	549	78	0	627
Low birthweight	0	54	5	0	59
Hepatitis B and non-A, non-B	0	0	0	0	0
AIDS	0	3	1	2	5
Infective endocarditis	0	26	12	0	38
Drug psychoses	0	2,848	774	369	3,991
Maternal drug dependence	0	471	40	0	511
Newborn toxicity	0	0	0	0	0
Road traffic accidents	44	231	134	55	465
Total illicit drugs	**44**	**10,876**	**3,057**	**494**	**14,471**
Total all drugs	**2,604**	**32,394**	**82,983**	**67,576**	**185,558**

Note: Columns may not add exactly to totals due to rounding.

Table 7.4: Hospital patient days attributable to drug use, by drug, reason for separation and age: males, 1997–98

	Age				
Drug involved/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	144	10,321	14,383	24,848
Alcoholism and alcoholic liver cirrhosis	164	15,719	146,136	49,969	211,988
Road injuries	1,187	16,651	7,842	2,286	27,967
Other	938	30,856	10,126	–15,397	26,524
Total alcohol	**2,289**	**63,371**	**174,425**	**51,241**	**291,327**
Tobacco					
Direct smoking					
Cancer	0	0	40,828	96,422	137,249
Ischaemic heart disease	0	1,018	80,705	39,379	121,102
Chronic obstructive pulmonary disease	0	0	24,802	124,576	149,378
Other	28,744	7,841	61,370	112,806	210,760
Environmental tobacco smoke	2,737	3	139	670	3,549
Total tobacco	**31,481**	**8,861**	**207,844**	**373,852**	**622,038**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	3,118	399	7	3,524
Opiates	0	10,136	4,421	30	14,587
Cocaine	0	109	67	6	182
Amphetamines	0	1,535	251	0	1,786
Hallucinogens	0	86	43	0	129
Poisoning	0	0	0	0	0
Opiates	0	2,082	748	23	2,853
Psychostimulants	0	243	66	0	309
Hallucinogens	0	119	18	1	138
Other psychotropic drugs	0	92	151	42	285
Anabolic steriods	0	2	1	0	3
Antepartum haemorrhage	1	0	0	0	1
Low birthweight	6,578	0	0	0	6,578
Hepatitis B and non-A, non-B	0	0	0	0	0
AIDS	0	5	0	2	7
Infective endocarditis	0	302	106	0	408
Drug psychoses	0	14,212	2,905	1,608	18,725
Newborn toxicity	2,216	0	0	0	2,216
Road traffic accidents	115	831	526	228	1,700
Total illicit drugs	**8,909**	**32,871**	**9,703**	**1,948**	**53,430**
Total all drugs	**42,680**	**105,102**	**391,972**	**427,041**	**966,795**

Note: Columns may not add exactly to totals due to rounding.

Table 7.5: Hospital patient days attributable to drug use, by drug, reason for separation and age: females, 1997–98

	Age				
Drug involved/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	346	8,116	9,176	17,638
Alcoholism and alcoholic liver cirrhosis	149	7,756	30,202	10,342	48,449
Road injuries	319	3,231	1,667	456	5,673
Other	524	9,751	–3,123	–82,742	–75,590
Total alcohol	**991**	**21,084**	**36,863**	**–62,768**	**–3,830**
Tobacco					
Direct smoking					
Cancer	0	0	12,632	37,151	49,783
Ischaemic heart disease	0	356	19,325	17,377	37,058
Chronic obstructive pulmonary disease	0	0	14,945	72,813	87,757
Other	25,181	15,695	34,021	65,344	140,241
Environmental tobacco smoke	1,738	6	545	1,278	3,567
Total tobacco	**26,919**	**16,057**	**81,467**	**193,963**	**318,406**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	1,081	156	0	1237
Opiates	0	7,905	2,922	97	10,924
Cocaine	0	132	9	0	141
Amphetamines	0	1,179	105	0	1,284
Hallucinogens	0	56	20	0	76
Poisoning	0	0	0	0	0
Opiates	0	834	256	23	1,113
Psychostimulants	0	220	27	0	247
Hallucinogens	0	82	38	0	120
Other psychotropic drugs	0	255	360	253	868
Anabolic steriods	0	0	0	0	0
Antepartum haemorrhage	2	1,868	452	0	2,322
Low birthweight	5,827	336	53	0	6,216
Hepatitis B and non-A, non-B	0	0	0	0	0
AIDS	0	3	1	9	13
Infective endocarditis	0	229	46	0	275
Drug psychoses	0	5,109	2,415	2,945	10,469
Maternal drug dependence	0	2,782	237	0	3,019
Newborn toxicity	2,449	0	0	0	2,449
Road traffic accidents	74	340	302	263	979
Total illicit drugs	**8,352**	**22,410**	**7,399**	**3,590**	**41,752**
Total all drugs	**36,262**	**59,551**	**125,729**	**134,785**	**356,327**

Note: Columns may not add exactly to totals due to rounding.

Table 7.6: Hospital patient days attributable to drug use, by drug, reason for separation and age: persons, 1997–98

	Age				
Drug involved/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
Alcohol					
Cancer	0	490	18,438	23,558	42,486
Alcoholism and alcoholic liver cirrhosis	313	23,475	176,338	60,311	260,437
Road injuries	1,506	19,883	9,509	2,742	33,640
Other	1,462	40,607	7,003	–98,138	–49,066
Total alcohol	**3,281**	**84,455**	**211,288**	**–11,527**	**287,497**
Tobacco					
Direct smoking					
Cancer	0	0	53,459	133,573	187,032
Ischaemic heart disease	0	1,373	100,030	56,756	158,160
Chronic obstructive pulmonary disease	0	0	39,747	197,388	237,136
Other	53,925	23,536	95,390	178,150	351,001
Environmental tobacco smoke	4,475	9	685	1,948	7,116
Total tobacco	**58,399**	**24,918**	**289,311**	**567,815**	**940,444**
Illicit drugs					
Drug dependence and abuse					
Cannabis	0	4,199	555	7	4,761
Opiates	0	18,041	7,343	127	25,511
Cocaine	0	241	76	6	323
Amphetamines	0	2,714	356	0	3,070
Hallucinogens	0	142	63	0	205
Poisoning	0	0	0	0	0
Opiates	0	2,916	1,004	46	3,966
Psychostimulants	0	463	93	0	556
Hallucinogens	0	201	56	1	258
Other psychotropic drugs	0	347	511	295	1,153
Anabolic steriods	0	2	1	0	3
Antepartum haemorrhage	3	1,868	452	0	2,323
Low birthweight	12,405	336	53	0	12,794
Hepatitis B and non-A, non-B	0	0	0	0	0
AIDS	0	7	1	11	20
Infective endocarditis	0	531	152	0	683
Drug psychoses	0	19,321	5,320	4,553	29,194
Maternal drug dependence	0	2,782	237	0	3,019
Newborn toxicity	4,665	0	0	0	4,665
Road traffic accidents	189	1,170	828	491	2,678
Total illicit drugs	**17,262**	**55,281**	**17,102**	**5,537**	**95,182**
Total all drugs	**61,680**	**109,373**	**500,599**	**556,288**	**1,227,940**

Note: Columns may not add exactly to totals due to rounding.

Appendix A Partial aetiological fractions for alcohol using low consumption as the reference level

English et al. (1995) calculated aetiological fractions for hazardous and harmful alcohol consumption (as defined by the National Health and Medical Research Council) relative to low alcohol consumption. Their rationale was to reflect more accurately the idea that unsafe drinking—as opposed to low alcohol consumption, which may be protective—is the cause for concern. The alcohol fractions presented in the body of our report reflect the earlier approach of Holman et al. (1990) and reflect both the risks and benefits of alcohol at all levels of consumption relative to abstaining from alcohol. This appendix presents revised estimates of the alcohol fractions using the approach taken by English et al.

The data in this appendix represent the extra effect of alcohol consumption for the 'unsafe' drinker compared with the 'responsible' drinker (English et al. 1995, p. 58), where unsafe and responsible consumption are defined by the NHMRC guidelines for responsible drinking (NHMRC 1992). The data are presented here in recognition of the fact that public health efforts in Australia are generally directed towards the reduction of unsafe alcohol consumption and not alcohol consumption per se. The data may thus be useful in supporting that policy.

A.1 Methods

The partial fraction for unsafe alcohol consumption is calculated in a similar way to the partial fractions used in the body of the report, but with a different reference level. We designated p_0, p_1, p_2 and p_3 as the population prevalence of abstinence, 'low', 'hazardous' and 'harmful' drinking respectively and RR_0, RR_2 and RR_3 as the relative risk associated with abstinence, hazardous and harmful drinking relative to low drinking. By definition, $RR_1 = 1$, so the equation for the partial fraction for unsafe drinking (hazardous and harmful combined) relative to low drinking is

$$F_u = \sum_{i=2}^{3}\left[p_i\left(RR_i - 1\right)\right] \Big/ \sum_{i=0}^{3}\left[p_i\left(RR_i - 1\right)\right] + 1$$

It is also possible to formulate an aetiological fraction, F_0, for the contribution of abstinence to morbidity and mortality. Its formula is

$$F_0 = \left[p_0\left(RR_0 - 1\right)\right] \Big/ \sum_{i=0}^{3}\left[p_i\left(RR_i - 1\right)\right] + 1$$

This report follows English et al. (1995) and does not calculate F_0. The main reason is that the results in this appendix are presented to support public policy relating to alcohol: abstinence is not currently an object of public policy. But this does mean that the results presented in

this appendix represent only a partial view of the total effect of alcohol. In particular, they do not take account of the benefits ascribed to moderate alcohol consumption or the risks associated with moderate alcohol consumption and some conditions.

A number of the fractions presented in the body of the report are based on case series using formula (6) in Chapter 2. These case series did not allow a partitioning of risk into that due to moderate alcohol intake and that due to high intake. Hence the data do not support the direct estimation of a fraction for hazardous and harmful levels of drinking. We have followed English et al. in using these fractions directly as the fractions for hazardous and harmful levels of drinking. The assumption implicit in this is that most of the alcohol harm relating to the conditions associated with these fractions arises from hazardous and harmful levels of drinking.

A.2 Results

Table A1 presents the age- and sex-specific partial aetiological fractions for unsafe alcohol consumption relative to low alcohol consumption. Tables A2 to A13 present the deaths, PYLL, hospital separations and patient days attributable to unsafe alcohol consumption relative to low alcohol consumption for the period 1996 to 1998.

English et al. (1995) found inadequate evidence that the marginal exposure between low and hazardous or harmful alcohol intake is either a cause of or protective against ischaemic heart disease. As a result, this condition is omitted from Table A1. English et al. also recommended the exclusion of heart failure from the analysis since its predominant cause is ischaemic heart disease, so this condition has also been omitted from Table A1. The fractions for unspecified liver cirrhosis deaths and separations are derived using the counts of deaths or separations coded to alcoholic liver cirrhosis and to overall liver cirrhosis. As a result, both these fractions vary by year. The values presented in Table A1 are those for 1998 for deaths and those for 1997–98 for hospital separations.

Table A1: Partial aetiological fractions for harmful and hazardous levels of alcohol consumption relative to moderate levels of consumption, by condition, age and sex

A1.1: Males

	Age																	
Condition	**0–4**	**5–9**	**10–14**	**15–19**	**20–24**	**25–29**	**30–34**	**35–39**	**40–44**	**45–49**	**50–54**	**55–59**	**60–64**	**65–69**	**70–74**	**75–79**	**80–84**	**85+**
Oropharyngeal cancer				0.24	0.24	0.22	0.21	0.17	0.19	0.19	0.20	0.13	0.22	0.15	0.19	0.19	0.11	0.11
Oesophageal cancer				0.16	0.16	0.15	0.15	0.12	0.13	0.14	0.14	0.10	0.16	0.11	0.13	0.13	0.08	0.08
Liver cancer				0.20	0.20	0.20	0.20	0.17	0.19	0.20	0.19	0.15	0.22	0.16	0.17	0.14	0.12	0.12
Laryngeal cancer				0.24	0.24	0.24	0.24	0.20	0.23	0.24	0.23	0.18	0.26	0.19	0.20	0.18	0.14	0.14
Female breast cancer	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Alcoholic psychosis	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Alcohol dependence	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Alcohol abuse	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Epilepsy				0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Alcoholic polyneuropathy	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Hypertension				0.10	0.10	0.10	0.10	0.08	0.09	0.09	0.09	0.07	0.10	0.07	0.08	0.07	0.05	0.05
Alcoholic cardiomyopathy	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Supraventricular cardiac dysrhythmias				0.09	0.09	0.09	0.09	0.07	0.09	0.09	0.08	0.07	0.10	0.07	0.07	0.06	0.05	0.05
Haemorrhagic stroke				0.12	0.12	0.12	0.13	0.10	0.12	0.13	0.12	0.10	0.14	0.10	0.10	0.08	0.07	0.07
Ischaemic stroke				0.08	0.08	0.08	0.08	0.06	0.07	0.08	0.07	0.06	0.09	0.06	0.06	0.06	0.04	0.04
Oesophageal varices				0.54	0.54	0.53	0.55	0.49	0.54	0.56	0.53	0.48	0.57	0.48	0.48	0.42	0.39	0.39
Gastro-oesophageal haemorrhage	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47
Alcoholic gastritis	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Alcoholic liver cirrhosis	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Unspecified liver cirrhosis (deaths 1998) [(a)]					0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Unspecified liver cirrhosis (separations 1997–98) [(a)]					0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

(continued)

Table A1 (continued): Partial aetiological fractions for harmful and hazardous levels of alcohol consumption relative to moderate levels of consumption, by condition, age and sex

A1.1 (continued): Males

	Age																
Condition	**0–4**	**5–9**	**10–14**	**15–19**	**20–24**	**25–29**	**30–34**	**35–39**	**40–44**	**45–49**	**50–54**	**55–59**	**60–64**	**65–69**	**70–74**	**75–79**	**80–84**	**85+**
Cholelithiasis				–0.04	–0.04	–0.04	–0.04	–0.03	–0.04	–0.04	–0.03	–0.03	–0.04	–0.03	–0.03	–0.03	–0.02	–0.02
Acute pancreatitis	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24
Chronic pancreatitis	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84
Low birthweight																		
Psoriasis				0.04	0.04	0.04	0.04	0.03	0.03	0.03	0.03	0.02	0.04	0.02	0.03	0.03	0.02	0.02
Ethanol toxicity [b]				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Methanol toxicity [b]				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Road injury																		
Driver etc. death [b]	0.33	0.33	0.33	0.30	0.39	0.39	0.40	0.40	0.40	0.40	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10
Pedestrian death [b]				0.75	0.61	0.61	0.53	0.53	0.53	0.53	0.19	0.19	0.19	0.19	0.19	0.19	0.19	0.19
Driver etc. hospitalisation [c]	0.25	0.25	0.25	0.20	0.33	0.33	0.24	0.24	0.24	0.24	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09
Pedestrian hospitalisation [c]				0.42	0.52	0.52	0.49	0.49	0.49	0.49	0.31	0.31	0.31	0.31	0.31	0.31	0.31	0.31
Alcoholic beverage poisoning [c]	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Other ethanol and methanol poisoning [c]				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Fall injuries				0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.12	0.12	0.12	0.12	0.12
Fire injuries				0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44
Drowning			0.03	0.32	0.32	0.32	0.50	0.50	0.50	0.42	0.42	0.42	0.31	0.31	0.31	0.31	0.31	0.31
Aspiration				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Occupational/machine injuries				0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07
Suicide				0.12	0.12	0.12	0.12	0.10	0.12	0.13	0.11	0.09	0.13	0.10	0.10	0.08	0.07	0.07
Assault	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47
Child abuse	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16

(continued)

Table A1 (continued): Partial aetiological fractions for harmful and hazardous levels of alcohol consumption relative to moderate levels of consumption, by condition, age and sex

A1.2: Females

	Age																	
Condition	**0–4**	**5–9**	**10–14**	**15–19**	**20–24**	**25–29**	**30–34**	**35–39**	**40–44**	**45–49**	**50–54**	**55–59**	**60–64**	**65–69**	**70–74**	**75–79**	**80–84**	**85+**
Oropharyngeal cancer				0.12	0.12	0.09	0.09	0.10	0.09	0.11	0.14	0.10	0.07	0.08	0.06	0.04	0.06	0.06
Oesophageal cancer				0.10	0.10	0.08	0.07	0.08	0.07	0.08	0.10	0.08	0.06	0.07	0.05	0.03	0.04	0.04
Liver cancer				0.18	0.18	0.14	0.14	0.16	0.14	0.15	0.17	0.16	0.15	0.17	0.11	0.07	0.08	0.08
Laryngeal cancer				0.21	0.21	0.17	0.16	0.18	0.16	0.17	0.20	0.18	0.17	0.19	0.13	0.07	0.09	0.09
Female breast cancer				0.04	0.04	0.03	0.03	0.04	0.03	0.03	0.04	0.04	0.03	0.04	0.03	0.01	0.02	0.02
Alcoholic psychosis	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Alcohol dependence	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Alcohol abuse	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Epilepsy				0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Alcoholic polyneuropathy	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Hypertension				0.08	0.08	0.06	0.06	0.07	0.06	0.07	0.08	0.07	0.06	0.07	0.05	0.03	0.03	0.03
Alcoholic cardiomyopathy	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Supraventricular cardiac dysrhythmias				0.09	0.09	0.07	0.06	0.07	0.06	0.07	0.08	0.07	0.07	0.08	0.05	0.03	0.03	0.03
Haemorrhagic stroke				0.20	0.20	0.16	0.15	0.18	0.16	0.23	0.27	0.20	0.10	0.13	0.10	0.08	0.13	0.13
Ischaemic stroke				0.04	0.04	0.03	0.03	0.03	0.03	0.04	0.04	0.04	0.03	0.03	0.02	0.01	0.02	0.02
Oesophageal varices				0.54	0.54	0.47	0.46	0.50	0.46	0.47	0.53	0.51	0.49	0.53	0.41	0.27	0.30	0.30
Gastro-oesophageal	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47
Alcoholic gastritis	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Alcoholic liver cirrhosis	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Unspecified liver cirrhosis (deaths 1998) [(a)]					0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Unspecified liver cirrhosis (separations 1997–98) [(a)]					0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03

(continued)

Table A1 (continued): Partial aetiological fractions for harmful and hazardous levels of alcohol consumption relative to moderate levels of consumption, by condition, age and sex

A1.2 (continued): Females

	Age																	
Condition	**0–4**	**5–9**	**10–14**	**15–19**	**20–24**	**25–29**	**30–34**	**35–39**	**40–44**	**45–49**	**50–54**	**55–59**	**60–64**	**65–69**	**70–74**	**75–79**	**80–84**	**85+**
Cholelithiasis				–0.03	–0.03	–0.02	–0.02	–0.03	–0.02	–0.03	–0.03	–0.03	–0.02	–0.03	–0.02	–0.01	–0.01	–0.01
Acute pancreatitis	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24
Chronic pancreatitis	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84
Low birthweight																		
Psoriasis				0.02	0.02	0.01	0.01	0.01	0.01	0.02	0.02	0.01	0.01	0.01	0.01	0.00	0.01	0.01
Ethanol toxicity [(b)]				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Methanol toxicity [(b)]				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Road injury																		
Driver etc. death [(b)]	0.11	0.11	0.11	0.08	0.16	0.16	0.18	0.18	0.18	0.18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Pedestrian death [(b)]				0.50	0.22	0.22	0.42	0.42	0.42	0.42	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06
Driver etc. hospitalisation [(c)]	0.11	0.11	0.11	0.09	0.15	0.15	0.12	0.12	0.12	0.12	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03
Pedestrian hospitalisation [(c)]				0.23	0.24	0.24	0.24	0.24	0.24	0.24	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03
Alcoholic beverage poisoning [(c)]	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Other ethanol and methanol				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Fall injuries				0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.04	0.04	0.04	0.04	0.04
Fire injuries				0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44	0.44
Drowning			0.03	0.32	0.32	0.32	0.50	0.50	0.50	0.42	0.42	0.42	0.31	0.31	0.31	0.31	0.31	0.31
Aspiration				1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Occupational/machine injuries				0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.07
Suicide				0.12	0.12	0.09	0.09	0.10	0.08	0.09	0.11	0.10	0.09	0.11	0.07	0.04	0.05	0.05
Assault	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47	0.47
Child abuse	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16	0.16

n.a. Not available

(a) This fraction depends on the counts coded to alcoholic liver cirrhosis and to overall liver cirrhosis. Hence it is different for separations and deaths and varies by year. The values presented here are those for 1998 for deaths and those for 1997–98 for hospital separations.

(b) Fractions for this condition apply only to deaths or PYLL.

(c) Fractions for this condition apply only to hospital separations or patient days.

Table A2: Deaths attributable to hazardous and harmful alcohol consumption relative to low consumption, by cause of death, age and year: males, 1996 to 1998

	Age				
Year/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1996					
Cancer	0	2	98	151	250
Alcoholism and alcoholic liver cirrhosis	0	32	481	240	753
Road injuries	20	266	116	23	425
Other	9	209	372	455	1,046
Total 1996	**30**	**509**	**1,067**	**869**	**2,474**
1997					
Cancer	0	2	103	149	254
Alcoholism and alcoholic liver cirrhosis	0	27	512	255	794
Road injuries	16	254	100	21	391
Other	7	220	358	444	1,029
Total 1997	**23**	**503**	**1,072**	**870**	**2,468**
1998					
Cancer	0	2	100	140	241
Alcoholism and alcoholic liver cirrhosis	0	26	451	226	703
Road injuries	12	245	111	19	387
Other	4	213	355	431	1,004
Total 1998	**16**	**485**	**1,017**	**816**	**2,335**

Note: Columns may not add exactly to totals due to rounding.

Table A3: Deaths attributable to hazardous and harmful alcohol consumption relative to low consumption, by cause of death, age and year: females, 1996 to 1998

	Age				
Year/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1996					
Cancer	0	1	59	70	130
Alcoholism and alcoholic liver cirrhosis	0	8	138	64	210
Road injuries	4	30	20	2	56
Other	9	46	123	350	528
Total 1996	**13**	**85**	**339**	**485**	**923**
1997					
Cancer	0	2	58	69	130
Alcoholism and alcoholic liver cirrhosis	0	13	142	57	213
Road injuries	3	37	21	3	63
Other	5	55	130	347	537
Total 1997	**8**	**107**	**351**	**477**	**943**
1998					
Cancer	0	2	58	66	126
Alcoholism and alcoholic liver cirrhosis	0	18	132	74	224
Road injuries	3	28	20	3	53
Other	3	53	124	353	533
Total 1998	**6**	**101**	**334**	**495**	**936**

Note: Columns may not add exactly to totals due to rounding.

Table A4: Deaths attributable to hazardous and harmful alcohol consumption relative to low consumption, by cause of death, age and year: persons, 1996 to 1998

	Age				
Year/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1996					
Cancer	0	3	157	221	380
Alcoholism and alcoholic liver cirrhosis	0	40	619	304	963
Road injuries	24	296	136	25	481
Other	18	255	495	804	1,573
Total 1996	**42**	**594**	**1,407**	**1,355**	**3,398**
1997					
Cancer	0	4	161	218	384
Alcoholism and alcoholic liver cirrhosis	0	40	654	313	1,007
Road injuries	19	291	121	24	454
Other	12	275	487	792	1,566
Total 1997	**31**	**610**	**1,423**	**1,346**	**3,411**
1998					
Cancer	0	4	158	205	367
Alcoholism and alcoholic liver cirrhosis	0	44	583	300	927
Road injuries	15	273	130	22	440
Other	8	266	479	784	1,537
Total 1998	**23**	**586**	**1,351**	**1,311**	**3,271**

Note: Columns may not add exactly to totals due to rounding.

Table A5: PYLL attributable to hazardous and harmful alcohol consumption relative to low consumption, by cause of death, age and year: males, 1996 to 1998

	Age				
Year/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1996					
Cancer	0	43	1,649	1,382	3,074
Alcoholism and alcoholic liver cirrhosis	0	837	8,952	2,314	12,103
Road injuries	595	7,218	2,557	186	10,556
Other	286	5,580	7,288	3,494	16,647
Total 1996	**881**	**13,678**	**20,446**	**7,376**	**42,381**
1997					
Cancer	0	46	1,748	1,366	3,160
Alcoholism and alcoholic liver cirrhosis	0	714	9,594	2,532	12,840
Road injuries	472	6,891	2,197	177	9,737
Other	202	5,887	6,988	3,428	16,505
Total 1997	**674**	**13,538**	**20,527**	**7,503**	**42,242**
1998					
Cancer	0	45	1,717	1,253	3,014
Alcoholism and alcoholic liver cirrhosis	0	675	8,418	2,225	11,317
Road injuries	356	6,637	2,418	160	9,570
Other	125	5,683	6,920	3,247	15,975
Total 1998	**481**	**13,039**	**19,472**	**6,885**	**39,877**

Note: Columns may not add exactly to totals due to rounding.

Table A6: PYLL attributable to hazardous and harmful alcohol consumption relative to low consumption, by cause of death, age and year: females, 1996 to 1998

	Age				
Year/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1996					
Cancer	0	36	1,188	717	1,940
Alcoholism and alcoholic liver cirrhosis	0	217	2,849	719	3,785
Road injuries	114	820	475	20	1,428
Other	277	1,283	2,583	2,680	6,822
Total 1996	**390**	**2,356**	**7,094**	**4,135**	**13,976**
1997					
Cancer	0	63	1,175	708	1,946
Alcoholism and alcoholic liver cirrhosis	0	348	3,019	682	4,049
Road injuries	98	1,024	484	25	1,631
Other	155	1,517	2,760	2,628	7,061
Total 1997	**253**	**2,952**	**7,438**	**4,043**	**14,687**
1998					
Cancer	0	61	1,165	644	1,870
Alcoholism and alcoholic liver cirrhosis	0	486	2,779	831	4,095
Road injuries	91	782	473	24	1,369
Other	105	1,464	2,611	2,697	6,876
Total 1998	**196**	**2,792**	**7,027**	**4,195**	**14,210**

Note: Columns may not add exactly to totals due to rounding.

Table A7: PYLL attributable to hazardous and harmful alcohol consumption relative to low consumption, by cause of death, age and year: persons, 1996 to 1998

	Age				
Year/cause of death	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1996					
Cancer	0	79	2,837	2,099	5,015
Alcoholism and alcoholic liver cirrhosis	0	1,054	11,801	3,032	15,888
Road injuries	709	8,038	3,032	206	11,984
Other	563	6,863	9,870	6,174	23,469
Total 1996	**1,271**	**16,034**	**27,540**	**11,511**	**56,357**
1997					
Cancer	0	109	2,923	2,075	5,107
Alcoholism and alcoholic liver cirrhosis	0	1,062	12,613	3,213	16,889
Road injuries	570	7,915	2,681	202	11,368
Other	357	7,404	9,748	6,057	23,566
Total 1997	**927**	**16,490**	**27,965**	**11,547**	**56,929**
1998					
Cancer	0	106	2,882	1,897	4,884
Alcoholism and alcoholic liver cirrhosis	0	1,161	11,196	3,055	15,413
Road injuries	447	7,418	2,890	184	10,940
Other	229	7,147	9,531	5,944	22,851
Total 1998	**676**	**15,831**	**26,499**	**11,080**	**54,087**

Note: Columns may not add exactly to totals due to rounding.

Table A8: Separations attributable to hazardous and harmful alcohol consumption relative to low consumption, by reason for separation, age and year: males, 1995–96 to 1997–98

	Age				
Year/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1995–96					
Cancer	0	14	539	536	1,089
Alcoholism and alcoholic liver cirrhosis	127	4,167	11,158	2,226	17,678
Road injuries	336	3,431	1,173	245	5,184
Other	253	11,184	9,840	4,485	25,762
Total 1995–96	**716**	**18,796**	**22,709**	**7,492**	**49,713**
1996–97					
Cancer	0	15	573	535	1,122
Alcoholism and alcoholic liver cirrhosis	129	3,930	11,232	2,324	17,615
Road injuries	318	3,273	1,224	255	5,071
Other	253	11,014	9,965	4,736	25,968
Total 1996–97	**700**	**18,232**	**22,994**	**7,850**	**49,776**
1997–98					
Cancer	0	10	607	579	1,196
Alcoholism and alcoholic liver cirrhosis	143	3,783	11,564	2,162	17,652
Road injuries	329	3,083	1,145	233	4,790
Other	229	10,765	9,966	4,819	25,779
Total 1997–98	**701**	**17,640**	**23,283**	**7,793**	**49,417**

Note: Columns may not add exactly to totals due to rounding.

Table A9: Separations attributable to hazardous and harmful alcohol consumption relative to low consumption, by reason for separation, age and year: females, 1995–96 to 1997–98

	Age				
Year/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1995–96					
Cancer	0	25	482	248	755
Alcoholism and alcoholic liver cirrhosis	139	2,006	4,000	621	6,766
Road injuries	82	715	330	55	1,182
Other	124	4,032	4,592	3,465	12,213
Total 1995–96	**346**	**6,777**	**9,404**	**4,389**	**20,917**
1996–97					
Cancer	0	25	482	245	751
Alcoholism and alcoholic liver cirrhosis	124	2,119	4,428	668	7,339
Road injuries	84	661	313	55	1,113
Other	106	3,900	4,499	3,648	12,154
Total 1996–97	**314**	**6,705**	**9,722**	**4,616**	**21,357**
1997–98					
Cancer	0	25	519	265	808
Alcoholism and alcoholic liver cirrhosis	135	2,071	5,074	668	7,948
Road injuries	81	628	296	50	1,056
Other	118	3,789	4,528	3,759	12,193
Total 1997–98	**334**	**6,513**	**10,417**	**4,742**	**22,005**

Note: Columns may not add exactly to totals due to rounding.

Table A10: Separations attributable to hazardous and harmful alcohol consumption relative to low consumption, by reason for separation, age and year: persons, 1995–96 to 1997–98

	Age				
Year/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1995–96					
Cancer	0	39	1,021	784	1,844
Alcoholism and alcoholic liver cirrhosis	266	6,173	15,158	2,847	24,444
Road injuries	419	4,146	1,503	299	6,366
Other	378	15,216	14,431	7,950	37,975
Total 1995–96	**1,062**	**25,574**	**32,113**	**11,881**	**70,630**
1996–97					
Cancer	0	40	1,055	780	1,874
Alcoholism and alcoholic liver cirrhosis	253	6,049	15,660	2,992	24,954
Road injuries	402	3,935	1,537	310	6,184
Other	359	14,914	14,465	8,384	38,121
Total 1996–97	**1,015**	**24,937**	**32,716**	**12,466**	**71,133**
1997–98					
Cancer	0	35	1,126	843	2,004
Alcoholism and alcoholic liver cirrhosis	278	5,854	16,638	2,830	25,600
Road injuries	410	3,711	1,442	283	5,846
Other	346	14,553	14,494	8,578	37,971
Total 1997–98	**1,034**	**24,153**	**33,700**	**12,535**	**71,422**

Note: Columns may not add exactly to totals due to rounding.

Table A11: Patient days attributable to hazardous and harmful alcohol consumption relative to low consumption, by reason for separation, age and year: males, 1995–96 to 1997–98

	Age				
Year/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1995–96					
Cancer	0	80	4,652	5,190	9,922
Alcoholism and alcoholic liver cirrhosis	145	18,191	89,467	43,782	151,586
Road injuries	1,496	19,671	8,091	2,874	32,132
Other	5,380	30,334	48,033	47,222	130,970
Total 1995–96	**7,022**	**68,277**	**150,243**	**99,069**	**324,610**
1996–97					
Cancer	0	104	4,449	4,963	9,516
Alcoholism and alcoholic liver cirrhosis	8,576	16,959	118,226	39,357	183,117
Road injuries	1,279	18,482	9,003	3,053	31,817
Other	5,836	36,914	47,185	45,903	135,839
Total 1996–97	**15,691**	**72,459**	**178,863**	**93,276**	**360,289**
1997–98					
Cancer	0	73	4,376	5,042	9,490
Alcoholism and alcoholic liver cirrhosis	164	15,719	146,136	49,969	211,988
Road injuries	1,187	16,651	7,842	2,286	27,967
Other	6,311	27,387	42,198	44,178	120,073
Total 1997–98	**7,662**	**59,830**	**200,551**	**101,475**	**369,518**

Note: Columns may not add exactly to totals due to rounding.

Table A12: Patient days attributable to hazardous and harmful alcohol consumption relative to low consumption, by reason for separation, age and year: females, 1995–96 to 1997–98

	Age				
Year/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1995–96					
Cancer	0	94	2,692	2,111	4,896
Alcoholism and alcoholic liver cirrhosis	162	8,466	31,611	15,788	56,028
Road injuries	361	3,448	1,955	563	6,327
Other	4,712	11,710	23,131	37,649	77,202
Total 1995–96	**5,234**	**23,719**	**59,389**	**56,110**	**144,453**
1996–97					
Cancer	0	102	2,381	2,027	4,511
Alcoholism and alcoholic liver cirrhosis	155	8,733	32,115	12,129	53,132
Road injuries	299	3,459	1,864	536	6,157
Other	5,214	12,590	26,140	37,390	81,334
Total 1996–97	**5,667**	**24,884**	**62,500**	**52,082**	**145,134**
1997–98					
Cancer	0	98	2,610	2,027	4,735
Alcoholism and alcoholic liver cirrhosis	149	7,756	30,202	10,342	48,449
Road injuries	319	3,231	1,667	456	5,673
Other	5,283	10,478	21,199	35,590	72,550
Total 1997–98	**5,751**	**21,563**	**55,679**	**48,415**	**131,407**

Note: Columns may not add exactly to totals due to rounding.

Table A13: Patient days attributable to hazardous and harmful alcohol consumption relative to low consumption, by reason for separation, age and year: persons, 1995–96 to 1997–98

	Age				
Year/reason for separation	**0–14**	**15–34**	**35–64**	**65 and over**	**All ages**
1995–96					
Cancer	0	174	7,343	7,301	14,818
Alcoholism and alcoholic liver cirrhosis	307	26,658	121,079	59,571	207,614
Road injuries	1,857	23,119	10,046	3,437	38,459
Other	10,092	42,045	71,164	84,871	208,172
Total 1995–96	**12,256**	**91,996**	**209,632**	**155,180**	**469,063**
1996–97					
Cancer	0	205	6,831	6,990	14,026
Alcoholism and alcoholic liver cirrhosis	8,731	25,692	150,341	51,486	236,249
Road injuries	1,577	21,941	10,867	3,589	37,974
Other	11,050	49,505	73,325	83,293	217,173
Total 1996–97	**21,358**	**97,343**	**241,363**	**145,358**	**505,423**
1997–98					
Cancer	0	171	6,986	7,068	14,225
Alcoholism and alcoholic liver cirrhosis	313	23,475	176,338	60,311	260,437
Road injuries	1,506	19,883	9,509	2,742	33,640
Other	11,594	37,865	63,397	79,768	192,623
Total 1997–98	**13,412**	**81,394**	**256,230**	**149,889**	**500,925**

Note: Columns may not add exactly to totals due to rounding.

Appendix B Studies reviewed in revising relative risk estimates

This appendix lists the studies reviewed in revising the aetiological fractions for alcohol and female breast cancer, stroke and fall injuries.

Risk-ratio estimates were also revised for alcohol and road injuries, tobacco and peptic ulcer, and illicit drugs and road injuries. However, each of these was based on only a small number of studies which were listed in the relevant sections of Chapters 3 and 5. Recent research results do not support a causal relationship between smoking and cervical cancer, so no new relative risk estimate was calculated.

Table B.1: Studies used by English et al. to revise aetiological fractions for female breast cancer attributable to alcohol

Reference	Age	Cases[a]	Outcome	Country	Subjects[b]	Adjustments[c]	Study type[d]
Chu et al. 1989	20–54	I	Breast cancer	US	3,217/2,945	Age, T	C-C
Ewertz 1991	<70	I	Breast cancer	Denmark	1,486/1,336	Age	C-C
Ferraroni et al. 1991	30–65	I	Breast cancer	Italy	214/215	Age	C-C
Franceschi et al. 1991	<75	I	Breast cancer	Italy	132/499	Age	C-C
Harvey et al. 1987	All	I	Breast cancer	US	1,524/1,896	Age	C-C
Hiatt & Bawol 1984	15+	I	Breast cancer	US	838/838	Age, T	Coh
Hiatt et al. 1988		I	Breast cancer	US	303/69,303	Age, T	Coh
La Vecchia et al. 1989	23–74	I	Breast cancer	Italy	2,402/2,020	Age, T	C-C
La Vecchia et al. 1985	26–74	I	Breast cancer	Italy	437/437	Age, T	C-C
Le et al. 1984		I	Breast cancer	France	500/945	Age	C-C
Nasca et al. 1990	20–79	I	Breast cancer	US	1,617/1,617	Age	C-C
O'Connell et al. 1987		I	Breast cancer	US	275/1,519	Age, T	C-C
Rosenberg et al. 1990	<70	I	Breast cancer	Canada	607/1,214	Age, T	C-C
Schatzkin et al. 1987	25–74	I	Breast cancer	US	121/7,188	Age	Coh
	31–64	I	Breast cancer	US	143/2,636	Age, T	Coh
Simon et al. 1991	21+	I	Breast cancer	US	87/87	Age, T	Coh
Toniolo et al. 1989	<75	I	Breast cancer	Italy	250/499	Age	C-C
Webster et al. 1983	20–54	I	Breast cancer	US	1,226/1,279	Age	C-C
Adami et al. 1988	<45	I	Breast cancer	Sweden	422/527	Age, T	C-C

(continued)

Table B.1 (continued): Studies used by English et al. to revise aetiological fractions for female breast cancer attributable to alcohol

Reference	Age	Cases[a]	Outcome	Country	Subjects[b]	Adjustments[c]	Study type[d]
Friedenreich et al. 1993	Pre-menopausal	I	Breast cancer	Canada	284/691	Age, T	C-C
Harris et al. 1992	Pre-menopausal	I	Breast cancer	US	192/184	Age, T	C-C
Martin Moreno et al. 1993	Pre-menopausal	I	Breast cancer	Spain	247/356	Age	C-C
Meara et al. 1989	25–44	I	Breast cancer	UK	351/351 (hosp. study)	T	C-C
Richardson et al. 1989	25–45	I	Breast cancer	France	78/140		C-C
Rohan & McMichael 1988	Pre-menopausal	I	Breast cancer	Australia	146/132	Age, T	C-C
Sneyd et al. 1991	25–34	I	Breast cancer	NZ	64/423	Age, T	C-C
	35–44	I	Breast cancer	NZ	323/804	Age, T	C-C
Willett et al. 1987	34–39	I	Breast cancer	US	71/20,230	Age	Coh
	40–44	I	Breast cancer	US	92/18,175	Age	Coh
van't Veer et al. 1989	25–44	I	Breast cancer	Netherlands	47/89	Age, T	C-C
Friedenreich et al. 1993	Post-menopausal	I	Breast cancer	Canada	284/691	Age, T	C-C
Gapstur et al. 1992	55–69	I	Breast cancer	US	459/37,059	Age, T	Coh
Garfinkel et al. 1988	45+	D	Breast cancer	US	2,933/581,321	Age, T	Coh
Harris et al. 1992	Post-menopausal	I	Breast cancer	US	412/336	Age, T	C-C
Martin Moreno et al. 1993	Post-menopausal	I	Breast cancer	Spain	515/632	Age	C-C
Meara et al. 1989	45–69	I	Breast cancer	UK	647/647 (hosp. study)	T	C-C
Meara et al. 1989	45–69	I	Breast cancer	UK	647/647 (screening study)	T	C-C
Richardson et al. 1989	46–55	I	Breast cancer	France	126/165		C-C
Richardson et al. 1989	56–65	I	Breast cancer	France	145/154		C-C

(Continued)

Table B.1 (continued): Studies used by English et al. to revise aetiological fractions for female breast cancer attributable to alcohol

Reference	Age	Cases[(b)]	Outcome	Country	Subjects[(b)]	Adjustments[(c)]	Study Type[(d)]
Rohan & McMichael 1988	Post-menopausal	I	Breast cancer	Australia	281/288	Age, T	C-C
Sneyd et al. 1991	45–54	I	Breast cancer	NZ	501/649	Age, T	C-C
Willett et al. 1987	45–49	I	Breast cancer	US	153/18,661	Age	Coh
	50–54	I	Breast cancer	US	146/17,949	Age	Coh
	55–59	I	Breast cancer	US	139/14,523	Age	Coh
van't Veer et al. 1989	55–64	I	Breast cancer	Netherlands	73/79	Age, T	C-C

(a) Cases are classified as incident cases of breast cancer (I) or deaths from breast cancer (D).

(b) Numbers of subjects are presented as m/n, where m is the number of cases and n is the number of controls in a case-control study or the total number of subjects in a cohort study.

(c) This column shows whether results are adjusted for potential confounding by age or tobacco consumption (T).

(d) Studies are classified as either case-control studies (C-C) or cohort studies (Coh).

Table B.2: Studies used by English et al. and relative effect measures for aetiological fractions for female breast cancer and alcohol

Reference	Unit[a]	Level 1[b]	RR	95% CI	Level 2[b]	RR	95% CI	Level 3[b]	RR	95% CI	Level 4[b]	RR	95% CI	Level 5[b]	RR	95% CI	Level 6[b]	RR	95% CI
Chu et al. 1989	d/w	<1	1.00	0.80–1.10	1–3	1.00	0.80–1.20	4–7	0.90	0.70–1.10	8–14	1.10	0.90–1.30	15–21	1.00	0.80–1.40	22+	1.20	0.90–1.60
Ewertz 1991	g/d	1–23	0.74	0.47–1.15	24+	0.63	0.34–1.17												
Ferraroni et al. 1991	g/d	0.1–5.3	1.10	0.50–2.20	5.3–13.1	1.5	0.80–2.80	13.1–24.3	1.20	0.60–2.40	24.35+	2.10	1.10–3.90						
Franceschi et al. 1991	d/d	1	1.30	0.70–2.60	2	1.40	0.80–2.70	3+	1.70	0.90–3.20									
Harvey et al. 1987	g/w	1–13	1.12	0.90–1.30	14–91	1.06	0.90–1.30	92–182	1.31	1.00–1.70	183+	1.66	1.20–2.40						
Hiatt & Bawol 1984	d/d	≤2	1.19	1.02–1.38	3–5	1.67	1.28–2.18	6+	1.50	0.86–2.62									
Hiatt et al. 1988	d/d	1–2	1.50	0.98–2.29	3–5	1.47	0.78–2.79	6+	3.30	1.18–9.28									
La Vecchia et al. 1989	d/d	<1	1.30	1.10–1.60	1–2	1.30	1.10–1.50	2–3	1.40	1.20–2.70	>3	2.20	1.70–2.70						
La Vecchia et al. 1985	d/d	≤3	1.25	0.91–1.73	>3	2.10	1.12–3.95												
Le et al. 1984	g/w	1–79	1.00	0.70–1.40	80–159	1.40	1.00–2.00	160–239	1.50	1.00–2.10	240+	1.20	0.70–2.00						
Nasca et al. 1990	g/d	<1.4	1.07	0.83–1.36	1.5–4.9	1.04	0.78–1.39	5–14.9	1.10	0.87–1.39	15+	1.26	0.98–1.64						
O'Connell et al. 1987	d/w	1+	1.45	0.99–2.12															
Rosenberg et al. 1990	d/w	1–3/m	0.60	0.40–0.80	1–3	1.0	0.70–1.40	4–6	0.80	0.60–1.20	1/d	0.80	0.50–1.10	2+/d	1.00	0.70–1.50			

(continued)

Table B.2 (continued): Studies used by English et al. and relative effect measures for aetiological fractions for female breast cancer and alcohol

Reference	Unit[a]	Level 1[b]	RR	95% CI	Level 2[b]	RR	95% CI	Level 3[b]	RR	95% CI	Level 4[b]	RR	95% CI	Level 5[b]	RR	95% CI	Level 6[b]	RR	95% CI
Schatzkin et al. 1987	g/d	<1.2	1.40	0.80–2.50	1.3–4.9	1.60	0.90–3.10	5+	2.0	1.10–3.70									
	g/d	<1.4	1.00	0.60–1.50	1.5–4.9	0.70	0.40–1.10	5+	0.60	0.40–1.00									
Simon et al. 1991	d/d	<1	1.08	0.64–1.82	1<2	1.23	0.49–3.10	2+	1.12	0.25–5.01									
Toniolo et al. 1989	g/d	1–10	0.90	0.50–1.50	11–20	1.20	0.80–1.90	21–30	1.10	0.70–1.80	31–40	1.30	0.70–2.70	41+	1.90	1.10–3.30			
Webster et al. 1983	g/w	<50	0.90	0.70–1.20	50–149	0.90	0.70–1.20	150–199	1.10	0.70–1.70	200–249	1.10	0.70–1.90	250–299	1.00	0.50–1.70	300+	1.10	0.60–1.80
Adami et al. 1988	g/d	0.1–1.2	1.10	0.50–2.40	1.30–4.90	0.80	0.60–1.20	5–14.9	0.60	0.40–0.90	15+	0.50	0.20–1.30						
Friedenreich et al. 1993	g/d	1–9	1.11	0.71–1.71	10–19	1.37	0.79–2.30	20–29	1.51	0.80–2.86	30+	1.88	0.96–3.66						
Harris et al. 1992	g/d	1–15	1.20	0.70–1.90	16+	0.70	0.30–1.50												
Martin Moreno et al. 1993	g/d	<3.01	1.10	0.60–1.80	3.01–9.20	1.50	0.90–2.50	9.21–23.0	1.30	0.70–2.20	>23	1.60	0.90–2.80						
Meara et al. 1989	g/d	3–12	1.20	0.70–2.10	13–27	0.70	0.30–1.40	28+	0.70	0.30–1.70									
Richardson et al. 1989	d/w	1–7	2.00	1.00–3.80	>7	1.60	0.80–3.20												
Rohan & McMichael 1988	g/d	<2.51	0.77	0.28–2.12	2.51–9.30	1.64	0.62–4.36	>9.3	2.33	0.85–6.37									

(continued)

Table B.2 (continued): Studies used by English et al. and relative effect measures for aetiological fractions for female breast cancer and alcohol

Reference	Unit[a]	Level 1[b]	RR	95% CI	Level 2[b]	RR	95% CI	Level 3[b]	RR	95% CI	Level 4[b]	RR	95% CI	Level 5[b]	RR	95% CI	Level 6[b]	RR	95% CI
Sneyd et al. 1991	d/w	1–7	0.66	0.37–1.19	8+	1.10	0.36–3.34												
	d/w	1–7	0.67	0.50–0.89	8+	0.77	0.49–1.21												
Willett et al. 1987	g/d	<1.5	1.63	0.82–3.25	1.5–4.9	1.06	0.54–2.08	5–14.9	1.27	0.65–2.50	15+	1.35	0.59–3.08						
	g/d	<1.5	1.14	0.57–2.28	1.5–4.9	0.98	0.53–1.82	5–14.9	0.81	0.41–1.59	15+	2.35	1.35–4.08						
van't Veer et al. 1989	g/d	1–4	0.30	0.00–1.70	5–14	0.50	0.10–2.90	15–29	0.80	0.10–4.90	30+	2.30	0.30–19.1						
Friedenreich et al. 1993	g/d	1–9	1.02	0.72–1.43	10–19	0.77	0.47–1.26	20–29	1.16	0.64–2.12	30+	0.86	0.46–1.59						
Gapstur et al. 1992	g/d	<1.5	1.18	0.86–1.61	1.5–4.9	1.20	0.93–1.56	5–14.9	1.25	0.93–1.68	15+	1.46	1.04–2.04						
Garfinkel et al. 1988	d/d	<1	0.96	0.82–1.13	1	1.18	1.03–1.36	2	1.06	0.86–1.30	3	1.28	0.95–1.74	4	1.36	0.90–2.07	5	2.10	1.18–3.27
Harris et al. 1992	g/d	1–15	1.1	0.80–1.60	16+	0.80	0.50–1.30												
Martin Moreno et al. 1993	g/d	<1.81	1.2	0.80–1.70	1.81–6.60	1.60	1.10–2.40	6.61–18.8	1.80	1.30–2.70	>18.8	1.90	1.30–2.80						
Meara et al. 1989	g/d	<3	1.20	0.40–3.60	3–12	1.10	0.30–3.50	13–27	0.70	0.20–2.90	28+	1.20	0.10–9.40						
Meara et al. 1989	g/d	<3	1.20	0.40–3.60	3–12	1.10	0.30–3.50	13–27	0.70	0.20–2.90	28+	1.20	0.10–9.40						
Richardson et al. 1989	d/w	1–7	1.30	0.70–2.20	>7	3.30	1.80–5.60												

(continued)

Table B.2 (continued): Studies used by English et al. and relative effect measures for aetiological fractions for female breast cancer and alcohol

Reference	Unit[a]	Level 1[b]	RR	95% CI	Level 2[b]	RR	95% CI	Level 3[b]	RR	95% CI	Level 4[b]	RR	95% CI	Level 5[b]	RR	95% CI	Level 6[b]	RR	95% CI
Richardson et al. 1989	d/w	1–7	2.40	1.40–4.10	>7	3.20	1.70–6.10												
Rohan & McMichael 1988	g/d	<2.51	0.84	0.46–1.53	2.51–9.30	1.12	0.59–2.15	>9.30	1.27	0.69–2.33									
Sneyd et al. 1991	d/w	1–7	0.96	0.74–1.24	8+	1.20	0.77–1.87												
Willett et al. 1987	g/d	<1.5	0.83	0.45–1.54	1.5–4.9	0.61	0.34–1.08	5–14.9	1.58	1.03–2.41	15+	1.88	1.22–2.90						
	g/d	<1.5	1.00	0.56–1.80	1.5–4.9	0.88	0.53–1.47	5–14.9	1.63	1.07–2.49	15+	1.14	0.69–1.87						
	g/d	<1.5	0.78	0.41–1.48	1.5–4.9	1.23	0.77.1.98	5–14.9	0.66	0.37–1.17	15+	1.48	0.93–2.35						
van't Veer et al. 1989	g/d	1–4	0.80	0.30–2.30	5–14	1.00	0.03–3.60	15–29	1.10	0.30–4.30	30+	0.90	0.20–4.50						

(a) Unit of measurement used in the study—drinks per week (d/w), grams per day (g/d) or drinks per day (d/d).

(b) Studies used different classification schemes for levels of alcohol consumption. These columns show the definitions of these levels for each study, from the lowest (level 1) to the highest (level 6).

Table B.3: Studies reviewed to revise aetiological fractions for female breast cancer attributable to alcohol

Reference	Age	Cases[(a)]	Outcome	Country	Subjects[(b)]	Adjustments[(c)]	Study type[(d)]
Ferraroni et al. 1998	23–74	I	Breast cancer	Italy	2,569/2,588	Age, P,AR,AB,AM, BMI, F	C-C
Bowlin et al. 1997	20–79	I	Breast cancer	US	1,214/1,214	Age,R,I,MS,F,AM,P,A	C-C
Royo Bordonada et al. 1997	50–74	I	Breast cancer	Europe	315/364	Age,BMI,T,AM,P,HRT,+	C-C
Swanson et al. 1997	<45	I	Breast cancer	US	1,645/1,497	Age,R,P,OCP,	C-C
Thun et al. 1997	30–104	D	Breast cancer	US	691/230,552	Adjusted	Coh (prospective)
Haile et al. 1996	<50	I	Bilateral breast cancer	US	144/232	Age,E,AM,AB,HRT,OCP, +	C-C
Boice et al. 1995	>30	I	Breast cancer	US	528/2,640	Age,AM,AB,P,+	C-C
Freudenheim et al. 1995	40–85	I	Breast cancer	US	740/810	Age,E,BMI,MS,AM, +	C-C
Holmberg et al. 1995	40–70	I	Breast cancer	Sweden	380/525	F,P,AB,E,BMI	C-C
Longnecker et al. 1995a	<75	I	Breast cancer	US	6,888/9,424	Age,AB,P,BMI,AM,E+	C-C
Longnecker et al. 1995b	55–64	I	Breast cancer	US	1,431/1,431	Age matched,AM,P,BMI,+	C-C
van den Brandt et al. 1995	55–69	I	Breast cancer	Netherlands	422/62,573	Age,AM,F,OCP,P,AB,E,+	Coh
Katsouyanni et al. 1994		I	Breast cancer	Greece	820/1,548	Age, AB,P,AM, MS,+	C-C
Nasca et al. 1994	20–79	I	Breast cancer (Estrogen receptor +)	US	1,152 (792 ER+)/1,617	Age	C-C
Begg et al. 1983		I	Breast cancer	US/Can	75/75	Age, T	C-C
Byers & Funch 1982	30–69	I	Breast cancer	US	1,314/770	Age	

(a) Cases are classified as incident cases of breast cancer (I) or deaths from breast cancer (D).

(b) Numbers of subjects are presented as m/n, where m is the number of cases and n is the number of controls in a case-control study or the total number of subjects in a cohort study.

(c) This column shows whether results are adjusted for potential confounding. Potential confounders are age, parity (P), age at first birth (AB), age at menarche (AM), body mass index (BMI), family history (F), area of residence (AR), education (E), energy intake (EI), physical activity (PA), religion (R), income (I), marital status (MS), tobacco (T), hormone replacement therapy (HRT), oral contraceptive pill (OCP), race (R), and menopausal status (MS).

(d) Studies are classified as either case-control studies (C-C) or cohort studies (Coh).

Table B.4: Risk estimates for studies reviewed to revise aetiological fractions for female breast cancer attributable to alcohol

Reference	Age	Unit[a]	Level 1[b]	RR	95% CI	Level 2[b]	RR	95% CI	Level 3[b]	RR	95% CI	Level 4[b]	RR	95% CI	Level 5[b]	RR	95% CI	Level 6[b]	RR	95% CI
Ferraroni et al. 1998	23–74	g/d	1.00–5.87	1.21	1.00–1.47	5.88–13.4	1.23	1.02–1.50	13.4–24.55	1.19	0.98–1.45	24.6–27.6	1.21	0.99–1.47	>27.60	1.41	1.17–1.71			
	Pre-men	g/d	1.00–5.87	1.45	1.06–1.97	5.88–13.4	1.12	0.80–1.55	13.4–24.55	1.55	1.10–2.18	24.6–27.6	1.47	1.04–2.08	>27.60	1.80	1.30–2.50			
	Post-men	g/d	1.00–5.87	1.01	0.79–1.30	5.88–13.4	1.23	0.97–1.56	13.4–24.55	0.98	0.77–1.25	24.6–27.6	1.03	0.81–1.30	>27.60	1.13	0.89–1.44			
Bowlin et al. 1997	20–79	g/d	>0<5	1.29	1.00–1.65	≥5	1.46	1.13–1.89												
	Pre-men	g/d	>0<5	1.26	0.71–2.22	≥5	1.54	0.87–2.74												
	Post-men	g/d	>0<5	1.32	0.97–1.80	≥5	1.51	1.09–2.08												
Thun et al. 1997	30–104	d/d	<1	1.10	0.90–1.30	1	1.20	1.00–1.60	2–3	1.50	1.20–1.90	≥4	1.00	0.70–1.40						
Boice et al. 1995	>30	d/w	<1	0.86	0.67–1.10	1–6	0.91	0.69–1.20	7–13	0.86	0.61–1.22	≥14	2.12	1.06–4.27						
Holmberg et al. 1995	40–70	g/d	<=0.75	1.2	0.80–1.80	0.76–2.00	1.90	1.20–2.90	>=2.00	1.60	1.00–2.40									
	<=50	g/d	<=0.75	0.40	0.10–1.50	0.76–2.00	0.70	0.30–1.80	>=2.00	0.80	0.40–1.40									
	>50	g/d	<=0.75	1.4	0.90–2.30	0.76–2.00	2.10	1.30–3.40	>=2.00	1.80	1.10–2.90									
Longnecker et al. 1995a	<75	g/d	≤5	1.08	0.98–1.19	6–11	1.09	0.96–1.23	12–18	1.17	1.01–1.37	19–32	1.49	1.24–1.79	33–45	1.95	1.42–2.66	≥46	1.96	1.43–2.67
	Pre-men	g/d	≤5	1.25	0.97–1.61	6–11	1.25	0.93–1.67	12–18	1.18	0.83–1.67	19–32	1.43	0.96–2.13	33–45	1.65	0.88–3.10	≥46	1.61	0.90–2.86
	Post-men	g/d	≤5	1.05	0.94–1.17	6–11	1.07	0.92–1.24	12–18	1.20	1.00–1.44	19–32	1.59	1.28–1.98	33–45	2.01	1.37–2.95	≥46	2.28	1.51–3.44

(continued)

Table B.4 (continued): Risk estimates for studies reviewed to revise aetiological fractions for female breast cancer attributable to alcohol

Reference	Age	Unit[a]	Level 1[b]	RR	95% CI	Level 2[b]	RR	95% CI	Level 3[b]	RR	95% CI	Level 4[b]	RR	95% CI	Level 5[b]	RR	95% CI	Level 6[b]	RR	95% CI
Longnecker et al. 1995b	55–64	d/w	>0–5	1.01	0.84–1.22	6–11	1.21	0.95–1.55	12–18	0.94	0.69–1.44	19–32	1.63	1.14–2.33	33–45	2.45	1.22–4.93	≥46	0.94	0.46–1.93
Katsouyanni et al. 1994		d/w	≤1	1.30	1.01–1.67	2–6	1.11	0.86–1.43	7–13	0.95	0.65–1.38	14–20	1.29	0.66–2.52	21–27	3.01	1.14–7.95	28+	3.79	1.05–13.7
Nasca et al. 1994																				
Oestrogen receptor +	20–79	d/d	<1.5	1.18	0.88–1.57	1.5–4.9	1.28	0.91–1.80	5.0–14.9	1.28	0.96–1.70	≥15	1.35	0.99–1.85						
Oestrogen receptor –	20–79	d/d	<1.5	0.92	0.62–1.36	1.5–4.9	1.19	0.77–1.83	5.0–14.9	0.94	0.64–1.35	≥15	1.05	0.70–1.59						
Swanson et al. 1997	<45	d/w	<1	1.35	1.10–1.70	1–2.9	1.01	0.80–1.20	3–6.9	1.03	0.80–1.30	7–13.9	1.10	0.80–1.50	≥14	1.79	1.20–2.60			
Haile et al. 1996	<50	d/w	1–3	1.20	0.60–2.30	>3	1.80	1.00–3.40												
Royo Bordonada et al. 1997	50–74	g/d	1.7	1.00	0.60–1.67	6.0	1.01	0.60–1.73	20.0	1.18	0.69–2.03									
Freudenheim et al. 1995	40–85	d/w	<1	0.90	0.65–1.25	1–4	0.85	0.61–1.18	>4<7	0.91	0.55–1.50	≥8	0.89	0.62–1.30						
van den Brandt et al. 1995	55–69	g/d	>0<5	1.30	0.96–1.95	5–14	1.29	0.89–1.85	15–29	1.28	0.81–2.03	≥30	1.72	0.90–3.28						
Begg et al. 1983		d/w	1–7	0.90	0.80–1.10	>7	1.40	0.90–2.00												
Byers & Funch 1982	30–69	d/m	<3	1.11	0.85–1.44	3–8	1.02	0.76–1.33	9–25	1.09	0.77–1.44	26+	1.13	0.88–1.44						

(a) Unit of measurement used in the study—drinks per week (d/w), grams per day (g/d), drinks per day (d/d) or drinks per month (d/m).

(b) Studies used different classification schemes for levels of alcohol consumption. These columns present the definitions of these levels for each study, from the lowest (level 1) up to the highest (level 6).

Table B.5: Studies reviewed to revise aetiological fractions for alcohol exposure and stroke

Reference	Age	Sex	Cases[a]	Outcome	Country	Subjects[b]	Adjustments[c]	Study type[d]
Numminen et al. 1996	35–74	M+F	I	All stroke	Finland	426/157		C-C
Wannamethee & Shaper 1996	Middle age	M	I+D	All stroke	UK	216	Age	Coh (prospective)
Beghi et al. 1995	24–87	M+F	I	All stroke	Italy	200/602	Age, sex, pst stk, HT, D, T	C-C
Iso et al. 1995	40–69	M	I+D	All stroke	Japan	178	Age adjusted	Coh (prospective)
Lee et al. 1995	>65	M+F	I	All stroke	Taiwan	155	Age, T, HT,D,Chol	Coh (retrospective)
	>65	M	I	All stroke	Taiwan	91	Age, T, HT, D, Chol	Coh (retrospective)
Donahue et al. 1986	45+	M	I+D	All stroke	US	290	Age, HT, Chol, BMI, T, +	Coh (prospective)
Sacco et al. 1999	> 39	M+F	I+D	First ischaemic stroke	US	677/1,139	Matched age, sex, race	C-C
	>39	M	I+D	First ischaemic stroke	US	299/447	Age, sex, race, HT,D,IHD+	C-C
	>39	F	I+D	First ischaemic stroke	US	378/692	Age, sex, race, HT,D,IHD+	C-C
Haapaniemi et al. 1996	16–60	M+F	I	First ischaemic cerebral	Finland	535	Unadjusted	Case series
Goldberg et al. 1995	55–64	M	I+D	Thromboembolic stroke	US	184	HR, BP, S, Chol, BMI,+	Coh (prospective)
Hillbom et al. 1995	16–40	M+F	I	First ischaemic stroke	Finland	74/133	Age, sex, acuteness, +	C-C
	16–40	M	I	First ischaemic stroke	Finland	47/83	Age, sex, acuteness, +	C-C
	16–40	F	I	First ischaemic stroke	Finland	27/50	Age, sex, acuteness, +	C-C
Iso et al. 1995	40–69	M	I+D	First non-haemorrhagic stroke	Japan	104	Age adjusted	Coh (prospective)
Kiyohara et al. 1995	≥40	M+F	I	Cerebral infarction	Japan	244	Age, sex	Coh (prospective)
Lee et al. 1995	>65	M+F	I	Cerebral infarction	Taiwan	155	Age, T, HT, D, Chol	Coh (retrospective)
	>65	M	I	Cerebral infarction	Taiwan	91	Age, T, HT, D, Chol	Coh (retrospective)

(continued)

Table B.5 (continued): Studies reviewed to revise aetiological fractions for alcohol exposure and stroke

Reference	Age	Sex	Cases[a]	Outcome	Country	Subjects[b]	Adjustments[c]	Study type[d]
Donahue et al. 1986	45+	M	I+D	Thromboembolic stroke	US	190	Age, HT, Chol, BMI, T, +	Coh (prospective)
Giroud et al. 1995		M+F	I	Primary cerebral haemorrhage	France	130/130	Matched age, sex	C-C
Iso et al. 1995	40–69	M	I+D	First haemorrhagic stroke	Japan	58	Age adjusted	Coh (prospective)
Juvela et al. 1995	16–60	M+F	I+D	First intracerebral haemorrhage	Finland	156/332	Age, sex, BMI, T, HT	C-C
	16–60	M	I+D	First intracerebral haemorrhage	Finland	96/192	Age, sex, BMI, T, HT	C-C
	16–60	F	I+D	First intracerebral haemorrhage	Finland	60/140	Age, sex, BMI, T, HT	C-C
Kiyohara et al. 1995	≥40	M+F	I	Cerebral haemorrhage	Japan	60	Age, sex	Coh (prospective)
Longstreth et al. 1992	>18	M+F	I+D	Subarachnoid haemorrhage	US	149/298	Age, sex, respondent type	C-C
Donahue et al. 1986	45+	M	I+D	Total haemorrhagic stroke	US	76	Age, HT, Chol, BMI, T, +	Coh (prospective)

(a) Cases are classified as incident cases of breast cancer (I) or deaths from breast cancer (D).

(b) Numbers of subjects are presented as m/n, where m is the number of cases and n is the number of controls in a case-control study or the total number of subjects in a cohort study.

(c) This column shows whether results are adjusted for potential confounding. Potential confounders are age, sex, past stroke (Pst stk), Hypertension (HT), Diabetes (D), Tobacco use (T), Cholesterol level (Chol), body mass index (BMI), previous ischaemic heart disease (IHD) and acuteness of stroke.

(d) Studies are classified as either case-control studies (C-C), cohort studies (Coh) or case series.

Table B.6: Studies used by English et al. to revise aetiological fractions for alcohol exposure and stroke

Reference	Age	Sex	Cases[(a)]	Outcome	Country	Subjects[(b)]	Adjustments[(c)]	Study type[(d)]
Klatsky et al. 1981a	Mean (43.3)	M+F	D	All stroke	US	50/8,060		Coh
Klatsky et al. 1981b		M+F	I	All stroke	US	121/5,535		Coh
Herman et al. 1983	40–74	M+F	I	All stroke	Netherlands	132/239	Age, sex	C-C
von Arbin et al. 1985	52–96	M+F	I	All stroke	Sweden	209/209		C-C
Gill et al. 1986	20–70	M	I	All stroke	UK	143/143	T	C-C
Gill et al. 1986	20–70	F	I	All stroke	UK	87/87	T	C-C
Gordon & Doyle 1987	All (38–55)	M	D	All stroke	US	33/1,762		Coh
Oleckno 1988	15–40	M+F	I	All stroke	US	54/864	Age, sex, T	C-C
Shaper et al. 1991	40–59	M	I	All stroke	UK	110/7,735	Age, sex, T	Coh
Ben–Shlomo et al. 1992	15–69	M+F	I	All stroke	UK	115/84	Age, sex, T	C-C
Shinton et al. 1993	35–74	M	I	All stroke	UK		Age, T	C-C
Shinton et al. 1993	35–74	F	I	All stroke	UK		Age, T	C-C
Stampfer et al. 1988	34–59	F	I	Ischaemic stroke	US	76/87,526	Age, T	Coh
Gorelick et al. 1989	Middle age	M	I	Ischaemic stroke	US		Age	C-C
Gorelick et al. 1989	Middle age	F	I	Ischaemic stroke	US		Age	C-C
Henrich and Morwitz 1989	15–65	M+F	I	Ischaemic stroke	US	89/178	Age, sex	C-C
Klatsky et al. 1989		M	I	Ischaemic stroke	US	162/10,552	Age, T	Coh
Klatsky et al. 1989		F	I	Ischaemic stroke	US	130/10,552	Age, T	Coh
Gill et al. 1991	20–70	M	I	Ischaemic stroke	UK		Age, T	C-C
Gill et al. 1991	20–70	F	I	Ischaemic stroke	UK		Age, T	C-C
al-Roomi et al. 1992	35–69	M+F	I	Ischaemic stroke	Australia	91/480	Age, T, sex	C-C

(continued)

Table B.6 (continued): Studies used by English et al. to revise aetiological fractions for alcohol exposure and stroke

Reference	Age	Sex	Cases[a]	Outcome	Country	Subjects[b]	Adjustments[c]	Study type[d]
Marini et al. 1993	15–44	M+F	I	Ischaemic stroke	Italy	308/616	Age, T, sex	C-C
Palomaki & Kaste 1993	<60	M	I	Ischaemic stroke	Finland	156/153	Age, T	C-C
Rogers et al. 1993		M	I	Ischaemic stroke	UK	137/137	Age, T	C-C
Rogers et al. 1993		F	I	Ischaemic stroke	UK	172/172	Age, T	C-C
Jamrozik et al. 1994		M+F	I	Ischaemic stroke	Australia	360/518	Age, T, sex	C-C
Stampfer et al. 1988	34–59	F	I	Haemorrhagic stroke	US	35/87,526	Age, T	Coh
Klatsky et al. 1989		M+F	I	Haemorrhagic stroke	US	69/10,459	Age, T, sex	Coh
Gill et al. 1991	20–70	M	I	Haemorrhagic stroke	UK		Age, T	C-C
Gill et al. 1991	20–70	F	I	Haemorrhagic stroke	UK		Age, T	C-C
Gill et al. 1991	20–70	M	I	Intracerebral haemorrhage	UK		Age, T	C-C
Gill et al. 1991	20–70	F	I	Intracerebral haemorrhage	UK		Age, T	C-C
al-Roomi et al. 1992	35–69	M+F	I	Haemorrhagic stroke	Australia	31/480	Age, T, sex	C-C
Juvela et al. 1993	15–60	M	I	Haemorrhagic stroke	Finland	145/164	Age, T	C-C
Juvela et al. 1993	15–60	F	I	Haemorrhagic stroke	Finland	133/150	Age, T	C-C
Jamrozik et al. 1994		M+F	I	Haemorrhagic stroke	Australia	59/279	Age, T, sex	C-C

(a) Cases are classified as incident cases of breast cancer (I) or deaths from breast cancer (D).

(b) Numbers of subjects are presented as m/n, where m is the number of cases and n is the number of controls in a case-control study or the total number of subjects in a cohort study.

(c) This column shows whether results are adjusted for potential confounding. Potential confounders are: age, sex, and tobacco use (T).

(d) Studies are classified as either case-control studies (C-C) or cohort studies (Coh).

Table B.7: Studies used to revise aetiological fractions for alcohol exposure and stroke with associated relative effect measures

Reference[a]	Age	Sex	Outcome	Unit[b]	Level 1[c]	RR	95% CI	Level 2[c]	RR	95% CI	Level 3[c]	RR	95% CI	Level 4[c]	RR	95% CI	Level 5[c]	RR	95% CI
Donahue et al. 1986	45+	M	Thromboembolic	oz/m	None	1.0		1–14	1.0	0.9–1.5	15–39	1.3	0.9–1.4	≥40	1.3	0.9–1.7			
	45+	M	Haemorrhagic	oz/m	None	1.0		1–14	2.2	1.1–4.2	15–39	2.9	1.4–5.9	≥40	4.7	2.4–9.5			
Stampfer et al. 1988	34–59	F	Ischaemic	g/d	< 1.5	0.70	0.40–1.60	1.5–4.9	0.40	0.20–0.90	5–14.9	0.30	0.10–0.70	≥ 15	0.50	0.20–1.10			
Gorelick et al. 1989	Middle–age	M	Ischaemic	g/w	1–99	2.20	0.95–5.13	100–299	1.86	0.89–3.92	≥ 300	1.68	0.79–3.56						
	Middle–age	F	Ischaemic	g/w	1–99	1.06	0.23–4.86	100–299	2.70	0.75–9.77	≥ 300	1.77	0.23–13.4						
Klatsky et al. 1989		M	Ischaemic	d/d	< 1	0.58	0.34–0.98	1–2	0.48	0.27–0.87	≥ 3	0.50	0.25–0.98						
		F	Ischaemic	d/d	< 1	0.63	0.40–0.98	1–2	0.66	0.36–1.22	≥ 3	0.11	0.02–0.84						
Gill et al. 1991	20–70	M	Haemorrhagic	g/w	10–90	0.78	0.30–1.80	100–390	0.57	0.20–1.30	≥ 400	1.48	0.60–3.80						
	20–70	F	Haemorrhagic	g/w	10–90	0.71	0.40–1.30	100–390	0.34	0.10–0.90	≥ 400	0.00	0.00–0.00						
	20–70	M	Ischaemic	g/w	10–90	0.50	0.20–1.10	100–390	0.77	0.40–1.50	≥ 400	2.07	0.90–4.70						
	20–70	F	Ischaemic	g/w	10–90	0.71	0.40–1.30	100–390	0.45	0.20–1.20	≥ 400	4.98	0.40–67.9						
Palomaki & Kaste 1993	<60	M	Ischaemic	g/w	> 0–150	0.54	0.28–1.05	>150–300	0.86	0.34–2.18	>300	4.41	1.09–17.8						
Rogers et al. 1993		M	Ischaemic	g/w	< 8.5	0.21	0.08–0.55	8.5–180	0.31	0.16–0.59	181–300	0.65	0.24–1.79	301–430	0.79	0.21–3.04	≥ 431	1.50	0.37–6.11
		F	Ischaemic	g/w	< 8.5	0.37	0.21–0.66	8.5–120	0.28	0.16–0.48	121–220	0.00	0.00–0.00	221–300	0.00	0.00–0.00	≥ 301	0.00	0.00–0.00

(continued)

Table B.7 (continued): Studies used to revise aetiological fractions for alcohol exposure and stroke with associated relative effect measures (continued)

Reference[a]	Age	Sex	Outcome	Unit[b]	Level 1[c]	RR	95% CI	Level 2[c]	RR	95% CI	Level 3[c]	RR	95% CI	Level 4[c]	RR	95% CI	Level 5[c]	RR	95% CI
Juvela et al. 1993	15–60	M	Haemorrhagic	g/d	1–40	0.34	0.14–0.81	41–120	2.45	1.10–5.47	> 120	4.45	1.54–12.9						
	15–60	F	Haemorrhagic	g/d	1–40	0.35	0.16–0.80	> 40	6.36	2.26–17.9									
Goldberg et al. 1995	55–64	M	Thromboembolic	mL/m	None	1.0		≤ 111	1.07	0.66–1.73	114–714	1.12	0.69–1.84	≥717	1.18	0.73–1.91			
Hillbom et al. 1995	16–40	M	First ischaemic	g/w	None	1.0		1–150	0.95	0.39–2.33	151–300	1.20	0.35–4.16	>300	2.57	0.76–8.75			
	16–40	F	First ischaemic	g/w	None	1.0		1–150	1.40	0.52–3.73	151–300	2.36	0.29–19.0	>300	No	controls			
Juvela et al. 1995	16–60	M	Intracerebral haemorrhage	g/w	None	1.0		1–150	1.74	0.87–3.46	151–300	2.47	0.93–6.53	>300	16.86	7.21–39.4			
	16–60	F	Intracerebral haemorrhage	g/w	None	1.0		1–150	0.94	0.49–1.81	151–300	4.17	1.3–13.8	>300	5.21	0.4–59.8			
Sacco et al. 1999	> 39	M	First ischaemic	d/d	None	1.0		≤2	0.54	0.36–0.80	>2 – <5	0.72	0.38–1.36	≥5	1.33	0.56–3.17			
	> 39	F	First ischaemic	d/d	None	1.0		≤2	0.49	0.34–0.71	>2 – <5	0.23	0.05–1.08	≥5	5.35	0.51–56.7			

(a) Studies used by English et al. (1995) are listed in bold.

(b) Unit of measurement used in the study—grams per week (g/w), grams per day (g/d), drinks per day (d/d), ounces per month (oz/m) and millilitres per month (ml/m).

(c) Studies used different classification schemes for levels of alcohol consumption. These columns present the definitions of these levels for each study, from the lowest (level 1) up to the highest.

Table B.8: Studies used to revise aetiological fractions for alcohol exposure and fall injuries

Study	Age	Sex	Subjects	Cases	Exposure criteria	Country	Outcome	Residence
Allander et al. 1998 [a][b]	>49	M	730	72	Clinical judgment alcohol immediate contributor	Europe	Falls resulting in hip fracture	All
	>49	F	2,086	50				
Hutchison et al. 1998	All	M+F	2,416	269	Clinically assessed alcohol <4 hrs before injury	UK	Falls resulting in facial injury	All
Hartshorne et al. 1997	All	M	33	19	Ethyl alcohol with toxicology or noted in records	US	Fatal head injury due to ground-level fall	All
	All	F	15	4				
Mosenthal et al. 1995 [a]	18–64	M+F	131	68	Blood alcohol analysis or toxicology +ve alcohol	US	Fall injuries (non-occupational)	All
Borges et al. 1994	15+	M+F	73	8	Breathalyser reading >100 mg/100mL	Mexico	Fall injuries A&E patients	All
Grisso et al. 1994 [a]	>44	F	144	37	Alcohol consumed last year = >2 drinks per week	US	First hip fracture among black women due to falls	All
Hussain et al. 1994	15+	M+F	389	109	Clinical notation significant alcohol consumption	UK	Fall-related craniofacial trauma	All
O' Loughlin et al. 1994 [b]	>64	M+F	470	79	Self-report—daily alcohol consumption	Canada	Indoor or outdoor falls (non-injurious & injurious)	Non-inst.
Malmivaara et al. 1993 [a][b]	20–44[c]	M	131	22	Self-report (M ≥1,000g/mth; F≥500 g/mth)	Finland	Injurious falls leading to hospitalisation or death	All
		F	61	1				
	45–64[c]	M	124	8				
		F	124	4				
	>64	M	50	5				
		F	138	1				
O'Loughlin et al. 1993 [b]	>64	M+F	197	12	Self-report (M+F= low ≤ 100g/wk; haz/harm >110 ≤700g/wk)	Canada	Non-injurious falls	Non-inst.
	>64	M+F	91	9	Self-report (M+F= low ≤ 100g/wk; haz/harm >110 ≤700g/wk)	Canada	Injurious falls	Non-inst.
Rivara et al. 1993	18+	M+F	398	151	Admission BAC ≥ 100mg/100 mL	US	A&E admission with trauma due to fall	All

(continued)

Table B.8 (continued): Studies used to revise aetiological fractions for alcohol exposure and fall injuries

Study	Age	Sex	Subjects	Cases	Exposure criteria	Country	Outcome	Residence
Adams et al. 1992 [b]	>64	M+F	13	0	Self-report intake within 24 hours before A&E visit	US	Falls presenting as medical problem to A&E	Non-inst
Nelson et al. 1992 [b]	>64	M+F	320	3	Self-report (current consumption ≥ 140 g/wk)	US	Fall injuries	Non-inst
Rutledge & Messick 1992	All	M+F	142	41	BAC ≥100mg/100 mL	US	Death within 24 hours of fall	
Honkanen & Smith 1991 [a]	15–64	M	587	124	Clinically assessed intoxication or breath test (BAC ≥0.5 g/l)	Finland	Falls resulting in injury and hospitalisation	All
Felson et al. 1988 [a][b]	28–64	M	11	8	Self-report ounces of alcohol per week (≥7 oz=haz/harm) last examination before fracture	US	Falls resulting in hip fracture	All
		F	30	7				
	65–74[c]	M	12	4				
		F	50	5				
	>74[c]	M	20	3				
		F	94	5				
Centers for Disease Control 1984	15+	M+F	52	11	BAC ≥0.1g/l	US	Death within 8 hours of fall	All
Wechsler et al. 1969	>15	M+F	272	62	Breathalyser reading >0.01%	US	Falls injury and admission to hospital A&E	All

(a) Study used in revising fraction for ages under 65.

(b) Study used in revising fraction for ages 65 and over.

Notes

Age groups aggregated as weighted averages.

Studies were excluded from revision where data did not allow relevant age and/or sex categorisation.

Studies used by English et al. (1995) are listed in bold.

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