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 $\beta=+0.0067$, 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

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

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