

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 stimulants	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

Year	Males			Females		
	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 childbearing 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.