Illicit drug use in Australia: Epidemiology, use patterns and associated harm. (2nd Edition)

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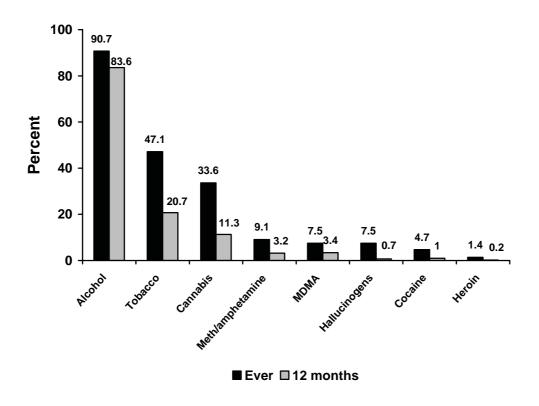
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Executive Summary

Overview of Illicit Drug Use in Australia

The overall prevalence of use of each drug, based upon the 2004 NDS household survey, is presented below. The table presents both lifetime prevalence and prevalence within the preceding 12 months. In order to provide a context for illicit drug use, comparable prevalence figures are also presented for alcohol and tobacco.

Prevalence of drug use in Australia, 2004 NDS Survey



The only illicit drug that approaches the prevalence of licit drugs is cannabis. The prevalence of both lifetime and current use of the other illicit drug classes is uniformly low.

Cannabis

Cannabis remains the most widely used illicit drug in Australia. In 2004 33.6% of the population aged 14 years and over had ever used cannabis, with 11.3% having used it in the preceding 12 months. Adolescents aged 14-19 years are more likely to have ever tried cannabis (25.5%) than tobacco (16.2%).

Recent (past year) cannabis use is most common among males in the 20-29 year age group. Males also tend to use more frequently, and use the more potent parts of the cannabis plant. People with lower education are more likely to become regular cannabis users, and cannabis dependence has been associated with unemployment.

Population surveys indicate that the most common way of using cannabis in Australia is smoking the heads (76.2%) and leaf (43.7%) of the plant. The use of other drugs in conjunction with cannabis, particularly alcohol, is common.

Harms associated with cannabis use

Cannabis use may precipitate schizophrenia in people who have a personal or family history the disorder. There is also some research evidence that cannabis in those who have psychosis vulnerability increases the risk of experiencing psychotic symptoms. Cannabis use may also exacerbate symptoms of schizophrenia, but it remains unclear whether or not cannabis causes additional cases of schizophrenia.

Cannabis use also poses a moderate risk for later depression, with heavy cannabis use possibly posing a small additional risk of suicide. While cannabis use and anxiety disorders co-occur at a rate greater than chance, the relationship appears to be largely mediated by factors other than cannabis use.

One of the most common harms associated with cannabis use is dependence. A population based study conducted in 1997 indicated that approximately one in three people who had used cannabis in the preceding 12 months either abused or were dependent on the drug. With increasing levels of cannabis use in the community, there has been an increase in rates of treatment provision for cannabis dependence.

Cannabis also has physical health effects. Evidence suggests that cannabis smoke is carcinogenic, and raises the risk of cancer development in areas of the body that are exposed to cannabis smoke. Despite this, the link between cannabis use and cancer is still debated. Smoking of cannabis during pregnancy has also been linked to a minor reduction in birth-weight.

Cannabis intoxication causes impaired cognitive and psychomotor functioning. There is some evidence that cannabis use produces minor impairments in driving performance, which increases the risk of involvement in traffic accidents. This is particularly true when cannabis is combined with alcohol.

Trends in cannabis use

Cannabis remains the most commonly used illicit drug in Australia. A need for increased treatment provision is likely to be largely driven by an ageing cohort of cannabis users, with daily smoking of cannabis now most commonly reported among 30-39 year olds. This group is likely to be experiencing significant physical and psychological harms as a result of their long-term, regular use. The increasing rates of cannabis use in Aboriginal and Torres Strait Islander communities are also cause for concern.

Meth/amphetamine

Meth/amphetamine is the most widely used illicit drug after cannabis, with an estimated 9.1% of the population having used it. Use is particularly common among young adults aged 20-29, with one-in-five (21.1%) have used meth/amphetamine in their lifetime, and 10.7% have taken the drug recently.

Meth/amphetamine users vary from young novice and 'recreational' drug users from various demographic backgrounds, who may be highly functional, through to chronic dependent users of the drug, who are more likely to be injecting drug users with low socioeconomic status and a range of comorbid mental and physical health problems.

The majority of meth/amphetamine users are young adults whose social contacts also use the drug. They often live in share accommodation or with their parents, and tend to have a lower income than people of a similar age within the general population – often being dependent on government allowances (disability pensions or unemployment benefits).

The onset of meth/amphetamine use typically occurs in the mid-to-late teens. Patterns of methamphetamine use vary from infrequent snorting or swallowing of the drug, which is typical of younger novice or occasional users of the drug, through to daily injection of methamphetamine. The crystalline form of methamphetamine can also be smoked.

The use of meth/amphetamines often occurs within a context of wider polydrug use, with heavy cannabis use being particularly common among this group.

Harms associated with meth/amphetamine use

Methamphetamine can induce a brief paranoid psychosis in some people who take the drug. Indeed, among methamphetamine users who take the drug monthly or more often, the prevalence of psychosis is 11 times higher than among the general population. Other forms of psychopathology that frequently co-occur with meth/amphetamine use include depression, increased aggression, anxiety disorders (including post traumatic stress disorder), and personality disorders.

As with all psychoactive drugs, methamphetamine use also carries the risk of dependence, with dependence strongly associated with injecting and smoking methamphetamine, and using the drug more than weekly.

The large proportions of meth/amphetamine users who inject the drug places this group at risk of blood borne infections through needle sharing. Meth/amphetamine users are as likely as opioid users to share injection equipment. Furthermore, as a young sexually active group meth/amphetamine users are also at risk of sexually transmitted diseases.

While fatalities associated with methamphetamine use are far less common than with opioid drugs, methamphetamine use has been associated with a range of fatal and non-fatal cardiac pathology, an increased risk of stroke, and other pathology, including renal failure and pulmonary oedema.

Trends in meth/amphetamine use

Despite the stable prevalence of methamphetamine use over recent years, several indicators of methamphetamine-related problems have shown steady increases since the mid-to-late 1990s (e.g., increases in methamphetamine-related arrests and hospital admissions). This increase in methamphetamine-related problems is likely to reflect several trends in the drug market including: a growing number of chronic users who initiated use in the mid-to-late 1990s; the shift from amphetamine to methamphetamine supply, which occurred in the mid-1990s; the emergence of imported high purity crystalline methamphetamine since 1999; and the up-take of crystalline methamphetamine use among injecting heroin users in the wake of the 2001 heroin shortage.

Ecstasy

Population based surveys indicate that MDMA has been used by approximately 7.5% of the population, with 3.4% having used it in the preceding 12 months.

Regular ecstasy users in Australia tend to be male, aged in their mid-twenties, from English speaking backgrounds, well-educated, engaged in either full-time employment or tertiary education. They tend to have little contact with law enforcement and very few report being in drug treatment.

Among regular users of ecstasy, the age of first use appears to begin in late adolescence while regular (monthly) use occurs soon after. Ecstasy is primarily taken orally, though snorting the drug is also quite common, with small proportions of regular users reporting smoking or injecting the drug.

The use of other drugs is common among ecstasy users, most notably alcohol, cannabis and methamphetamine.

Harms associated with Ecstasy use

There is little research in Australia investigating the association between mental health problems and ecstasy use, and this remains an area of further research need.

Ecstasy users perceive a range of adverse effects related to the drug including physical harms, such as non-fatal overdose and dehydration; neurological harms, such as memory impairment and damage to brain function; and psychological harms, such as depression and anxiety. Users have also reported concerns regarding unknown long-term effects which may stem from their ecstasy use, such as, alterations in brain structure and neurotransmitter actions.

While injection is not a common route of administration among ecstasy users, there is a risk of blood borne virus transmission as a result of needle sharing among the minority who do inject the drug.

Ecstasy has been implicated in a number of deaths in Australia. During 2000 to 2004 the National Coronial Information System (NCIS) recorded 112 ecstasy-related deaths (Kinner *et al*, 2005), with ecstasy deemed to be the primary contributor in 51 (46%) of these cases. MDMA was the sole drug present in only six (5%) of these deaths, reflecting the polydrug using nature of MDMA users (Kinner *et al*, 2005).

Trends in Ecstasy use

Patterns of ecstasy use appear to have remained stable over time. Those who use ecstasy appear to identify a range of risks which can result from ecstasy use, and do exhibit concern regarding long-term consequences. A minority of users report seeking help for such problems as dependence, depression and associated psychological issues.

LSD

Overall, the lifetime prevalence of hallucinogen use is 7.5%, with 0.7% of the population aged 14 years or older having used these drugs in the preceding 12 months.

There has been very little research conducted specifically on LSD users, so the demographics described here pertain to hallucinogen users in general. Hallucinogen users are most commonly male, polydrug users in their twenties, who were born in Australia or the British Isles. LSD appears to be a drug that is used infrequently, mainly for recreational purposes and in the company of others. While LSD is usually swallowed it may also be injected. Injectors of LSD are also likely to have injected other drugs. Hallucinogens (including LSD) appear to be commonly used in conjunction with other psychoactive drugs.

Harms associated with LSD use

As very little research has been conducted on LSD use, there is limited information available regarding the harms. Work conducted in the United States indicates an association between LSD use and panic reactions, prolonged schizoaffective psychoses, and post-hallucinogen perceptual disorder.

Trends in LSD use

Unfortunately population based surveys have tended to ask about hallucinogens generally, rather than LSD specifically. The lifetime prevalence of hallucinogen use appears to have fallen since 1998, a finding that is supported by a reduction in arrests for the consumption and provision of hallucinogens.

Cocaine

The lifetime prevalence of cocaine use is 4.7%, having remained relatively stable across the past three NDS household surveys. Use in the preceding 12 months in all surveys has been stable at approximately 1%.

Males are generally more likely than females to report having ever used cocaine, and to have done so in the past 12 months, although the reverse is true in the 14-19 year age group. Cocaine users are typically in their twenties, with the average age of initiation being 23.5 years. There appear to be two types of cocaine users, differentiated according to their socio-economic status (SES). Those of a lower SES have low levels of education, are more likely to be unemployed, and are typical 'hard-core' drug users. Those of higher SES are on regular or above average incomes, and come from a range of educational and occupational backgrounds.

Patterns of cocaine use appear to vary according to the SES of the user, with those of a lower SES being more likely to exclusively use cocaine by injection, to use the drug as often as possible and to inject other drugs. Those of a higher SES are more likely to primarily administer cocaine intra-nasally. Polydrug use is common in both groups.

Harms associated with Cocaine use

Cocaine use can produce psychological symptoms, such as anxiety, panic, dysphoria, severe depression, paranoia, hallucinations, delirium, and violent agitation, particularly in high doses and during withdrawal. Binge use of cocaine typically induces such symptoms and can lead to a state known as "cocaine psychosis".

Due to its vasoconstrictive effects, cocaine has the potential to cause a wide range of physical problems including: cardiovascular (e.g. chest pain, cardiac arrhythmias, myocardial infarction and ischemia, and cardiomyopathy); neurological (e.g. cerebrovascular accidents i.e. strokes, seizures, and headaches); gastrointestinal (e.g. abdominal pain, vomiting, colitis, and bowel infarction); and respiratory symptoms (e.g. exacerbation of asthma, rapid and/or irregular breathing, pulmonary oedema, and bronchitis). Cocaine can also cause obstetric complications, such as irregularities in placental blood flow, premature labour, and low neonate birth weights. Deaths due to cocaine toxicity are due primarily to cardiovascular complications, such as myocardial ischaemia and infarction and cardiac arrhythmias. In 2004 there were 20 accidental drug-induced deaths in Australia where cocaine was mentioned as either the primary cause of death While higher doses and a greater frequency of use increase the likelihood of adverse reactions to cocaine, the thresholds over which complications occur can vary widely between individuals.

In Australia, and internationally, cocaine injecting has been associated with higher levels of HIV risk-taking behaviours than seen among opioid users. Cocaine also has a high dependence liability. Indeed, cocaine dependence can develop after a relatively short history of use and is associated with a range of use patterns.

Trends in cocaine use

Whilst cocaine use among the general population has remained low and stable, The IDRS detected an increase in the use of cocaine in the preceding six months among IDU in NSW from 34% in 1997 to 84% in 2001. In 2006 67% of IDU had used cocaine. Much of the increase in cocaine use in NSW was reported among heroin users and the sharp increase in 2001 was attributed to the reduction of the availability of heroin in late 2000/early 2001.

Heroin

Approximately 1.4% of the population have tried heroin in their lifetime, with 0.2% having used during the preceding 12 months.

Overall males appear more likely to have ever used heroin, with lifetime heroin use most commonly reported among the 20-29 year age group. The 2004 NDS household survey suggests that the mean age of initiation to heroin use is approximately 21 years. While a reduction in the age of initiation was been noted in the literature prior to the Australian heroin shortage in 2001, there is evidence that the shortage resulted in a reduction in the number of new heroin injectors in Australia.

While the overwhelming majority of heroin users inject the drug, smoking of the drug has gained popularity in the past decade. This was first noted among the Indochinese communities in the 1990s, and spread from there to younger Caucasian heroin users. Following the heroin shortage, however, heroin smoking has dramatically reduced among IDU.

Polydrug use is the norm among heroin users, and large proportions of Australian heroin users also meet the criteria for dependence on other drugs, such as, alcohol, meth/amphetamines and benzodiazepines. The injection of oral preparations represents a particularly high risk aspect of the polydrug use surrounding primary heroin use. The removal of temazepam gel capsules from the market has resulted in a welcome reduction in the injection of oral benzodiazepine formulations among IDU.

Harms associated with heroin use

It has been repeatedly demonstrated that heroin users have high rates of psychiatric morbidity, with the most common diagnoses being mood disorders, anxiety disorders, and Anti-Social Personality Disorder (ASPD). The completed suicide rate among heroin users is estimated to at 14 times that of the general population. In addition, high rates of post traumatic stress disorder (PTSD) and borderline personality disorder (BPD) have been reported among dependent heroin users.

The major harm associated with heroin use is the risk of fatal and non-fatal overdose. The 'typical' fatal heroin overdose case has been described as a long-term opiate dependent male in his early 30s, who is not in treatment for heroin dependence, and has consumed other drugs in combination with heroin, primarily other central nervous system depressants. While the 1990s saw a dramatic increase in the number of Australian heroin overdose fatalities, following the 2001 reduction in the heroin supply, there was a 63% reduction in NSW ambulance callouts to suspected heroin overdoses. Similarly, data on suspected drug-related deaths between January 1995 and June 2003 showed a 43% decrease in the number of drug-related deaths after the

reduction in the supply of heroin. The largest decreases were seen among the younger age groups, suggesting that younger users were more likely to cease or reduce their use in response to the heroin shortage.

Heroin dependence is a major health risk associated with use. While it has been argued that route of administration is a significant predictor of dependence, it is noteworthy that one study found that one in ten entrants to treatment for heroin dependence were heroin smokers, yet experienced dependence symptoms severe enough to seek treatment.

Blood borne viruses also represent a significant harm associated with the injection of heroin and other drugs, with 61% of those who use needle and syringe programmes being HCV positive. High levels of the hepatitis B infection have also been reported among cohorts of IDU, yet the uptake of the HBV vaccine among this group appears poor. Despite the risks involved, the 2005 IDRS indicates that 11% of the national IDU sample reported having used a needle after someone else in the past month, and 17% reported that someone else had used a needle after them in that time.

Trends in heroin use

Since the 2001 heroin shortage the number of regular heroin users in Australia has reduced dramatically, and opioid deaths have declined from 938 in 2000 to 357 in 2004. The greatest reduction in heroin use and opioid fatalities is evident among the younger age groups.

Chapter 1: Introduction

In 2000 a report prepared by the National Drug and Alcohol Research Centre (NDARC), entitled *Illicit Drug Use in Australia: Epidemiology, Use Patterns and Associated Harm,* was published as a National Drug Strategy Monograph, and was used as the primary evidence base for the National Drugs Campaign.

In 2006 the Australian Government announced additional funding for a further phase of the National Drugs Campaign, which was to focus on emerging trends in illicit drug use, as well as a campaign to raise public awareness of links between illicit drug use and mental health problems.

In order to support the development of these campaigns, the Australian Department of Health and Ageing requested that NDARC update the original report, including any changes in the epidemiology, use patterns and harms associated with illicit drug use.

Aims:

- To provide an evidence base for the National Drugs Campaign.
- To examine the Australian epidemiology, use patterns and associated harm of heroin, meth/amphetamines, cocaine, cannabis, ecstasy and LSD.

Chapter 2: Method

As indicated above, the major illicit drug classes that NDARC was commissioned to examine in this report were heroin, meth/amphetamine, cocaine, cannabis, ecstasy and LSD. For each drug class the major literature pertaining to Australian use patterns and harms was examined. Within each drug class, information is presented on the following areas:

1. Epidemiology

Estimates of the number of lifetime and current users of each drug class, based upon household surveys and studies specifically designed to estimate the number of drug users.

2. Characteristics of users of the drug

A review of what is known of the demographic characteristics of the users of each drug based upon studies conducted in Australia.

3. Drug use patterns

Examines the patterns of drug use in Australia, including frequency of use, routes of administration, and associated polydrug use.

4. Associated psychopathology

Explores the relationship between each drug class and mental health problems.

5. Other harms associated with the use of the drug

Provides a review of what is currently known about the specific harms associated with each drug, including overdose, blood borne viruses and dependence.

6. Current trends in the use of the drug

Examines what is known about trends in the use of each of these drugs in Australia, including trends in the prevalence of use, the demographic characteristics of users and routes of administration.

7. Summary

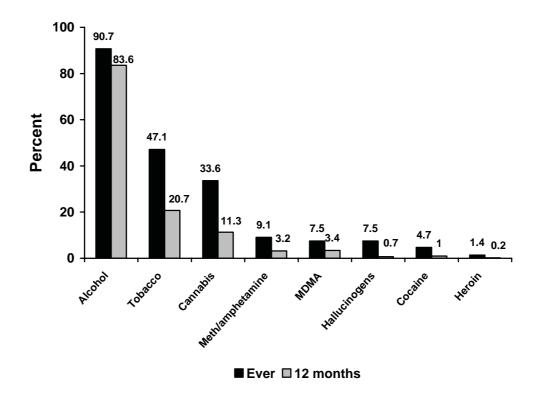
Presents a brief summary of the major features of the use of each drug in Australia, and any projected trends in such use.

Chapter 3: Overview of illicit drug use in Australia

Before examining the major illicit drug classes individually, it is useful to present the overall prevalence of use of each drug. Figure 3.1 presents the lifetime prevalence and prevalence within the preceding 12 months from the 2004 NDS household survey. In order to provide a context for illicit drug use, prevalence figures for tobacco and alcohol are also presented.

As can be seen, the only drug that approaches the prevalence of licit drugs is cannabis. The prevalence of both lifetime and current use of the other illicit drug classes is uniformly low.

Figure 3.1: Prevalence of drug use in Australia, 2004 NDS Survey



The following chapters discuss each drug type in descending order of prevalence. The prevalence of use for each of the illicit drugs is presented by age and gender (in tabular form) in Appendix A.

Chapter 4: Cannabis

Jan Copeland

Summary of key changes in cannabis use over the past decade:

- Levels of cannabis use in Australia have decreased with lifetime prevalence reduced from 39% to 33% overall.
- Among 14-19 year olds, lifetime use of cannabis has reduced from 45% in 1998 to 25.5% in 2004.
- Rates of cannabis use among indigenous Australians, however, appear to have increased over this time but the data is of poor quality.
- While the age of initiation to cannabis use if generally falling, the heaviest use cohort are now those in the 30-39 years age range.
- A higher proportion of specialist alcohol and other drug treatment services are now provided to individuals with cannabis as the principal drug of concern (now have NMDS data).
- There have been significant developments in the evidence base on interventions for cannabis use disorders, including Australian research.
- There have been a number of longitudinal cohort studies that have identified the relationship between cannabis use and psychosis, including the putative role of genetic predisposition. There is not yet convincing evidence of causality but there is growing consensus that cannabis use may precipitate an episode in those vulnerable to psychotic disorders.
- There is now evidence to support the concern that cannabis use poses a moderate risk for later depression, particularly among adolescent girls.
- There is a better understanding of the negative effect of cannabis use on educational outcomes for adolescents arising from longitudinal birth cohort studies.

Epidemiology of cannabis use

Widespread cannabis use emerged in Australia in the early 1970s and has been rising since. The most recent NDS survey revealed that in 2004, cannabis remained the most commonly used illicit drug in Australia with an estimated five and a half million people aged 14 and above having tried the drug and more than three quarters of a million people having used cannabis in the previous week. Typically, people begin using cannabis in the late teens or early twenties and use steadily decreases thereafter (Chen & Kandel, 1995). While most cannabis use is experimental or intermittent, it has been estimated that around one in ten people who try it become dependent (Hall, Degenhardt & Lynskey, 2001).

According to the 2004 National Household Survey, around one third (33.6%) of Australians aged 14 yrs and over reported that they had used cannabis at some point in their lives. This proportion has remained relatively stable since 1993 but was marginally higher in 1998. Cannabis use in Australia is most prevalent among young adults aged 20-39 yrs (Figure 4.1). In 2004, more than one in two young adults (54.5%) and one in four adolescents (25.5%) reported using cannabis at least once in their life. Adolescents aged 14-19 years are now more likely to have ever tried cannabis (25.5%) than tobacco (16.2%).

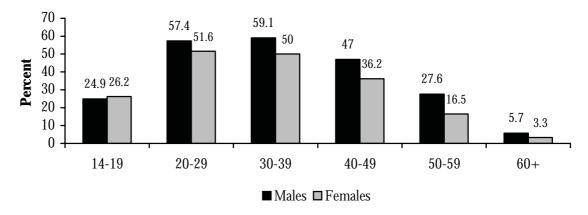
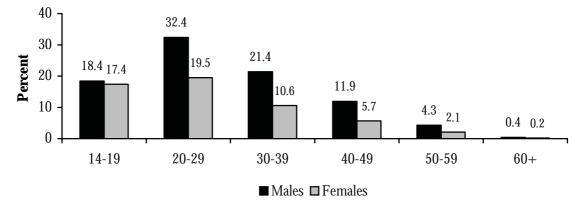


Figure 4.1: Prevalence of lifetime cannabis use in Australia by age and gender, 2004

Source: National Drug Strategy Household Survey, 2004

More than one in four young adults aged 20-29 years (26%) and more than one in six adolescents (17.9%) reported using the drug recently (in the past 12 months). Overall, 11.3% of the population aged 14 years and over reported recent cannabis use. Males in all age groups appear more likely to have used cannabis in the past year, with the gender difference being less distinctive among the 14-19 year age group (Figure 4.2). Generally, around one in five recent users reported using the drug at least weekly with the rates of daily use peaking in the 30-39 years age group (21.2%). Moreover, among the 14-19 year age group, the average age at which people are beginning to use cannabis in Australia has dropped from 14.8 years in 1995 to 14.6 years in 2001.





Source: National Drug Strategy Household Survey, 2004

Consistent with global trends and National Household Survey data, surveys of Australian secondary school students indicate a rise in cannabis use in the 1990s. In a review of the literature, Donnelly and Hall (1994) found that between 25-30% of school aged children (12-17 yrs) reported having used cannabis in the early 1990s. While there appeared to be a slight increase in the 1996 Australian School Student's Alcohol and Drugs Survey (ASSADS), the most recent 2002 ASSADS found that the prevalence of cannabis use decreased to levels observed in the early 1990s.

Evidence suggests that higher proportions of people are beginning to use cannabis in their teens. Whereas around one fifth of cannabis users born between 1940 and 1949 first used by age 18, about three quarters of cannabis users born between 1970 and 1979 had used by the same age (Coffey *et al.*, 2000). This drop in the age of initiation is concerning because an earlier age of cannabis initiation is associated with an increased likelihood of regular use. In turn, regular use at a young age is a risk factor for dependence and other problems such as criminal activity and the use of other drugs of dependence (see Copeland, Gerber & Swift, 2006 for review).

A further issue of concern is the growing rate of cannabis use and associated problems in the indigenous Australian communities (Clough *et al.*, 2002). The 2001 National Household Survey found that cannabis use among Aboriginal and Torres Strait Islanders (ATSI) was higher than for the non indigenous population: 27% of ATSI respondents reported using cannabis in the last 12 months compared to 13 % of non-indigenous Australians. Cannabis use in indigenous communities has received little research attention and the social and health impacts of cannabis use in such communities remain largely unknown. Surveys in the mid-1980s in the Northern Territories "Top End" did not detect cannabis use among indigenous peoples (Watson, Fleming & Alexander, 1988). By 1999, however, cannabis use was reported by 55% of males and 13% of females in the same region (Clough *et al.*, 2002). A random sample of indigenous peoples in two contiguous communities in Arnhem Land recently reported that 67% of males and 22% of females were current cannabis users (Clough *et al.*, 2004).

On a broader level, this highlights the importance of investigating cannabis use in ethnically diverse groups within the Australian population. There is some recent research suggesting that cannabis use is higher in Sydney youth of English speaking background compared to those from Vietnamese and Arabic speaking backgrounds (Rissell, McLelland & Bauman, 2000).

More than one in four Australians aged 14 years and over approve of personal cannabis use (27.4%) according to the 2004 NDS. This figure climbs to in excess of three quarters of regular cannabis users, approving of their personal use of the drug. This approval of cannabis use is in contrast to more than one in five (22%) believing cannabis use to be associated with a "drug problem" in the same survey. Cannabis is widely available in the community with more than one in two respondents (58.2%) reporting that they had the opportunity to use cannabis in the previous year.

The group in the community with the highest average levels of cannabis use is injecting drug users (IDU). The 2005 report of the Illicit Drug Reporting system found that 95% of the sample of IDU had ever used cannabis and 82% had used in the previous 6 months, typically at least once daily (Stafford *et al.*, 2006). That survey of illicit drug users reported that the price and potency of cannabis remained stable across the country with hydroponic cannabis, dominating the market.

Finally, the groups representing the most problematic levels of cannabis use are those presenting to treatment. More disability-adjusted *healthy* years of life were estimated to have been lost in 1996 due to cannabis use and dependence (4,416 years) than to HIV, Hepatitis B and Hepatitis C combined (2,189 years) (Mathers, Vos & Stevenson, 1999). Although, unlike these blood-borne viruses, cannabis has not caused any deaths, the disability caused by dependence and the extent of use in Australia means that the burden of disease is greater (Mathers, Vos & Stevenson, 1999). There is an increasing demand for interventions for cannabis-related problems with around one in five treatment episodes of alcohol and other drug treatment being for a primary cannabis use disorder (AIHW, 2006). In 2000-01, the first year of the alcohol and other drugs national minimum data set, cannabis (14%) was the third most common principal drug of concern, after alcohol (34%) and heroin (28%). In each subsequent year, however, cannabis was

the second most common principal drug of concern (21% in 2001-02, 22% in 2002-03 and 2003-04 and 23% 2004-5). The mean duration of a cannabis treatment episode in 2003-4 was 12 days, varying from 9 days for withdrawal management, 41 days for counseling and 28 days for residential rehabilitation. The most common treatment type was counseling (32.7%) followed by information and education (24%), withdrawal management (13.9%), support and case management (10.8%) and residential rehabilitation (6.1%). Consistent with other drug classes, 53.8% of those seeking treatment with cannabis as their principal drug of concern had another drug of concern (highest at 67.4% for amphetamines as a principal drug of concern and lowest at 42.8% for alcohol) (AIHW, 2005).

Characteristics of cannabis users

In addition to age, there are other factors that increase the risks of using cannabis. As with most drugs, males are more likely than females to have tried cannabis and use it at a greater frequency (Coffey *et al.*, 2003). The NDS (2004) reported that 37.4% of males compared with 29.9% of females had ever used cannabis in their lifetime. Males were twice as likely as females to report having used cannabis in the previous week (6.2% versus 3.0%). Males are also more likely to report using the more potent parts of the cannabis plant, such as the heads (78.9% versus 71.5%) and hashish oil (6.0% versus 3.6%). Males are also more likely to usually source their cannabis from dealers (18.7% versus 14%) thus increasing their potential exposure to other drug markets and contact with law enforcement. While males and females equally commonly report that they usually use cannabis in a private home, males are more likely to report using cannabis in a public place than are females (20.3% versus 15.1%).

Few studies describe the characteristics of adolescent cannabis users in Australia. One sample of one hundred adolescents aged between 15 and 19 years who had used cannabis at least once in the previous year, were recruited from all the main regions across Sydney (Copeland, Swift & Reid, 2003). The majority of participants (92%) were currently attending a public secondary school and all had attended public secondary schools in the preceding twelve months. One quarter of this sample (26.3%), predominantly females (39.5% vs. 16.1% of males), claimed one or both of their parents used cannabis, and two thirds of these had seen them using it. The young people interviewed for this study had first tried cannabis at about 14 years of age (mean=13.9 yrs, SD=1.6; range=7-17; mean of 14 years for males and 13.8 years for females). This experience had predominantly been with friends (90%), although a small proportion (<10%) reported being with others, such as relatives and strangers; one person had been alone. Curiosity was the most common reason for first use (71%), followed by the observation that "everyone else was doing it" (19%) and peer pressure (16%). In the past month, approximately one half of the sample (49%) were smoking on about a weekly or more frequent basis (i.e., used 3-5 times); 13% had smoked on a daily/near daily (20+ times) basis. Males were more likely to have daily use patterns than females (17.6% smoked 20+ times compared to 7% of females) (Copeland, Swift & Reid, 2003).

A study of the feasibility of an Adolescent Cannabis Check-up recruited individuals aged 14-19 who self-referred, or were referred by their parents or school, to participate in the study (Martin, Copeland & Swift, 2005). On average, participants were 16 years old (mean=16.4, sd=1.5, range=14-19). The majority lived at home with their parent(s), were still at school or studying elsewhere (n=46; 63%) or in fulltime employment (n=10; 13.7%). A small minority were currently unemployed (n=6; 8.2%). The mean age of first cannabis use was 13.2 years (sd=1.7; range=9-17 yrs). The majority of participants (80.8%) reported having used on a daily or near daily basis at some time, with this pattern of use beginning on average at age 15 years (sd=1.4; range=12-18). Participants varied widely in their reported quantity and frequency of cannabis use, with a mean of 55/90 using days (range=2-90), and an average consumption of 465 cones (sd=466; range= 9-2145) in the 90 day period. The mean number of hours spent stoned on a

typical day was 4 (sd=2.4) with a range of 0.45 to 10 hours. The overwhelming majority of participants (94.5%) obtained their supply from friends or acquaintances, spending a mean of \$A50.7 (sd=41.7; range: \$0-175) per week. The great majority of the sample (94.5%) met DSM-IV diagnostic criteria for a cannabis use disorder (Martin, Copeland & Swift, 2005).

Two further groups are more likely to use cannabis than others in the community. People with lower education are more likely to become regular users (Gruber *et al.*, 2003; Johnson, O'Malley & Bachman, 2001). A review of the literature also highlighted that unemployment is associated with an increased risk of cannabis dependence (Hall, Degenhardt & Lynskey, 2001). Among those that started using cannabis in early adolescence this lack of employment may be related to the substantial evidence that cannabis use is associated with impaired academic performance, higher rates of absenteeism and earlier school leaving (Copeland, Gerber & Swift, 2006). Crosssectional findings that cannabis is associated with poorer educational outcomes have been supported by longitudinal data internationally.

The most extensive evidence comes from a 25 year longitudinal study conducted by Fergusson and colleagues in a large New Zealand birth cohort. After controlling for mental health, family dysfunction, delinquent and drug-using peer affiliations and educational achievement, early initiators were 3.1 times more likely to leave school as compared to non-users (Fergusson, Lynskey & Horwood, 1996). A later follow-up of this cohort found that those who had started using cannabis by age 16 were at substantially higher risk of leaving school without formal qualifications (Fergusson & Horwood, 1997). By the time this cohort reached age 25 years, increasing cannabis use was associated with a higher risk of leaving school without qualifications, failure to enter university and failure to secure a university degree (Fergusson, Horwood & Lynskey, 2003). Consistent with these data was that the association between cannabis use and poor educational outcomes was not due to cannabis impairing cognitive functioning or producing an 'amotivational syndrome', but rather, to the social context in which cannabis is used, encouraging attitudes that promote school dropout and diminish further education opportunities (Fergusson, Horwood & Lynskey, 2003).

Patterns of cannabis use

Types of cannabis used

There are three main forms of cannabis; herbal cannabis (marijuana), 'hashish' and 'hash oil'. Marijuana is the dried matter from the flowering tops and leaves of the plant. Levels of delta-9-tetrahydrocannabinol (THC), the main psychoactive component of cannabis, depend on the growing conditions and genetic strain of the plant. The flowering tops or 'heads' generally have 5-14% THC (ElSohly & Ross, 2003). Leaf has lower THC levels, ranging from 0.5-4%, and cannabis resin, or 'hash', has 2-10% THC, while 'hash oil' contains 16.5-60% THC (ElSohly & Ross, 2003). Hash and hash oil are not commonly used in Australia with rates of 13.4% and 5.1% respectively (AIHW, 2004).

According to the 2004 National Household Survey, the most common way of using cannabis in Australia is smoking the heads (76.2%) and leaf (43.7%) of the plant. 'Skunk', a particularly potent form of heads, was reported to be smoked by around one in four cannabis users (19.3%), although it is not clear how they could be sure they were smoking this form. In the 2001 NDS those aged between 14-39 yrs were more likely to smoke the heads of the plant whereas older people (40 + yrs) were more likely to choose leaf. This is evidence for the shifting preference for using the stronger parts of the plant in the younger generation of users.

Polydrug use

The 2004 National Household Survey data indicated that cannabis is rarely used alone. Alcohol was very commonly used in conjunction with cannabis (86.2%), with concurrent amphetamines (27.9%) and ecstasy (24.2%) use also quite frequent. Only 10.8% reported not having used any other drug with cannabis. While this survey did not assess concurrent tobacco use, cannabis users are more likely to be tobacco smokers than non-users.

While there has been research on the combined effects of alcohol and cannabis on performing various tasks (eg. driving), little research has been conducted on the reasons why this combination is so popular. Evidence from one controlled experimental study suggests that alcohol causes THC to be absorbed more rapidly into the blood plasma of the user (Lukas & Orozco, 2001).

Cannabis users often report substituting cannabis for other drugs. The 2004 NDS found 60.4% of recent cannabis users reported using alcohol when cannabis was not available. The 1997 National Survey of Mental Health and Well-being survey, and other research among adolescent cannabis users, found that current tobacco use was associated with cannabis abuse/dependence (Copeland, Gerber & Swift, 2006).

Effects of different routes of cannabis administration

In Australia, cannabis is most often smoked. Within minutes of smoking, THC is absorbed into the bloodstream from the lungs (Grotenhermen & Russo, 2002). This results in a fairly immediate onset of the 'high'. The intensity of this 'high' depends on several factors including the potency of cannabis, the method of use and experience of the user.

Cannabis can be smoked through a 'joint', pipe or waterpipe. A 'joint' is a hand rolled cigarette containing matter from the leaf, heads or resin of the cannabis plant. Other substances such as tobacco are often added to improve burning. A waterpipe or 'bong' uses water to cool the smoke before it is inhaled through a mouthpiece. While this minimises the amount of wasted smoke and enables maximum THC from the plant material to be recovered, larger amounts of carbon monoxide and tar are inhaled using this method compared to smoking a joint (AGAL, 1996). Compared to smoking, eating cannabis produces a less intense high of longer duration. After eating, it can take 1-3 hours for psychoactive effects to occur because of the longer time it takes for THC to enter the bloodstream (Grotenhermen & Russo, 2002). The delay between ingestion and the onset of acute effects makes it difficult for users to control the dose of THC consumed as compared to smoking where the psychoactive effects are more immediate. Adding cannabis to infusions is also possible, but uncommon in Australia.

With rising concerns about the health effect of cannabis smoke, new methods of cannabis use have emerged that do not involve smoke inhalation such as vaporisation(Taylor *et al.*, 2002), however, this method is rarely reported by Australian users.

Social factors

Data from the 2004 National Household Survey highlight the important role of the social group in the use of cannabis. For people who had tried cannabis at least once, the majority reported that up to a half of their friends or acquaintances had also tried the drug. The most common place to use cannabis for recent users was either their own home or a friend's house (89.1%), with private parties being the second most common (49.1%). For those who had used recently, cannabis was usually obtained from friends or acquaintances (69.4%) compared with about one in seven who bought it from a dealer. Similarly, data from a Western Australian sample of 68 individuals with prior convictions for minor cannabis offences showed that family and friends were a common main source of supply for cannabis (44.8%), while purchasing from a dealer was less common (8.6%) (Lenton, Bennett & Heale, 1999).

Psychopathology associated with of cannabis use

The relationship between cannabis use and mental health disorders is affected by several factors with a number of possible causal relationships (Copeland, Gerber & Swift, 2006). This section will discuss cannabis and the most common mental health problems, depression and anxiety, and the most debilitating problem of psychosis.

Relationship with psychosis

The nature of the demonstrated association between cannabis use and psychosis is yet to be persuasively determined. Cannabis use may cause symptoms similar to those observed in psychotic disorders like schizophrenia where individuals experience delusional thoughts, hallucinations and impaired reality testing (Copeland, Gerber & Swift, 2006). This is plausible because THC stimulates increases production of dopamine (Wenger, Moldrich, & Furst, 2003) a neurotransmitter also implicated in psychotic disturbances. A 1996 community study in New Zealand reported that one in seven (14%) of cannabis users reported 'strange, unpleasant experiences such as hearing voices' or 'becoming convinced that someone is trying to harm you' after using cannabis (Thomas, 1996). The 1997 National Survey of Mental Health and Wellbeing reported that after adjusting for confounding factors a diagnosis of cannabis dependence doubled the odds of reporting psychotic symptoms (Degenhardt & Hall, 2001).

There are several arguments for the association between cannabis and psychosis in the literature. Some believe that heavy cannabis use can lead to a temporary 'cannabis psychosis' or toxic psychosis which remits after cessation of use (Chopra & Smith, 1974). A Danish study has reported that of those presenting with cannabis-induced psychosis, almost one half received a subsequent schizophrenia spectrum disorder diagnosis (Arendt *et al.*, 2005).

It is also argued that cannabis use is a causal factor in the formation of schizophrenia, an often chronic psychotic disorder (Zammitt *et al.*, 2002). Research has also examined whether cannabis use exacerbates the symptoms of schizophrenia (Van Os *et al.*, 2002) or triggers the disorder in psychosis-vulnerable individuals (Verdoux *et al.*, 2003).

The Australian Low Prevalence Study, conducted in Perth, Melbourne, Brisbane and Canberra found that 25% of people who screened positive for a psychotic disorder also met the criteria for cannabis abuse or dependence at some point in their lives (Jablensky *et al.*, 2000). A large scale population survey in the US, known as the Epidemiological Catchment Area (ECA) study, found that cannabis was the second most frequently used drug (23%) besides alcohol (37%) among persons with schizophrenia (Anthony & Helzer, 1991).

While cross-sectional population surveys provide evidence that cannabis use is associated with schizophrenia, this does not support the conclusion that cannabis use causes schizophrenia. Studies that follow samples of people over multiple time points are better able to disentangle the sequence of events leading to the development of psychosis. The largest longitudinal study examining the link between cannabis and psychosis followed approximately 50,000 Swedish army conscripts for 15 years (Andreasson, Allebeck & Rydberg, 1989). After controlling for other factors such as parental mental illness or a pre-existing psychotic illness at conscription, the odds of developing schizophrenia later in life were 1.5 times higher for those who had used cannabis one to ten times, and 2.3 times more likely for those who had used cannabis ten times or more, compared with those who had not used. A longer term follow-up of this cohort ruled out the argument that the use of other drugs or the existence of personality traits that pose a risk of schizophrenia may have been responsible for this association (Zammitt *et al.*, 2002). A

longitudinal study of a birth cohort in Dunedin, New Zealand supports and extends the Swedish cohort findings (Arsenault *et al.*, 2002). After controlling for other drug use and the presence of psychiatric symptoms at age 11 years, cannabis use at age 18 years was associated with significantly higher rates of schizophrenia, especially for those who initiated cannabis before 15 years of age.

Several cross-sectional studies have examined whether cannabis use predicted later development of individual psychotic symptoms such as hallucinations or delusional beliefs (Copeland, Gerber & Swift, 2006). In a study using ECA data, Tien and Anthony (1990) compared people who had recently experienced one or more psychotic symptoms (within the last 12 months) with those who had not had such symptoms. After controlling for demographic factors and other mental health problems, cannabis use doubled the risk of reporting psychotic symptoms.

The most extensive longitudinal research in this area was conducted by Fergusson and colleagues (2003) in the Christchurch Health and Development Study (CHDS) which followed a New Zealand birth cohort over 21 years. A large range of possible confounding factors were considered such as previous psychotic symptoms, other drug use, anxiety, depression, criminality and demographic variables. Cannabis dependence at age 18 was associated with an approximate doubling of risk for developing psychotic symptoms at age 21, compared to those with no dependence diagnosis, after adjusting for all confounds measured in the study.

A growing body of literature has specifically investigated whether those who are already prone to psychosis are at higher risk of developing a psychotic disorder after cannabis use. In a general population sample followed over three years, Van Os and colleagues (2002) found that as cannabis use levels increased, so did risk for psychosis incidence. Moreover, an additive effect was evident for cannabis use and psychosis vulnerability in terms of later psychosis development. That is, cannabis use placed those with underlying psychotic tendencies at greater risk for psychosis than those without such tendencies. Cannabis use was also associated with a poorer prognosis for those with an existing psychosis. Verdoux and colleagues (2003) conducted a unique study where college students kept a personal record before and after cannabis use. After controlling for other drug use, age and gender it was found that cannabis use posed a significant risk for experiencing psychotic symptoms in the period of hours after using cannabis. This risk was higher for individuals deemed to be prone to psychosis. Only one small study by McGuire and colleagues (1995) has directly examined the role of family history in schizophrenia and cannabis use. It was found that schizophrenic patients with a history of cannabis use were ten times more likely to have a family history of schizophrenia compared with those who had not used the substance (McGuire et al., 1995). The authors concluded that a shared genetic predisposition to schizophrenia is associated with the disorder in the context of cannabis use.

While studies have indicated that regular cannabis use increases the likelihood of experiencing psychotic symptoms among vulnerable individuals and worsens the prognosis of schizophrenia, they are unable to provide direct evidence that cannabis use causes new cases of clinical psychoses in the population. This question was addressed by Degenhardt and colleagues using modelling techniques to assess whether rising cannabis use over the past 30 yrs in Australia corresponded with rising levels of schizophrenia. It found that despite significant increases in cannabis use, levels of schizophrenia in the population had slightly decreased or remained stable over this time period. The authors concluded that cannabis is unlikely to cause schizophrenia, but rather precipitate it in vulnerable people (Degenhradt, Hall & Lynskey, 2003).

This view is supported by some evidence that cannabis users with schizophrenia have their first episode at a younger age than non-users (DiMaggio *et al.*, 2001). A recent Greek cross-sectional survey has reported on subclinical positive and negative psychosis dimensions and depression

(Stefanis *et al.*, 2004). The authors reported that the use of cannabis was positively associated with negative and positive dimensions of psychosis, independent of each other, and depression. These results suggest that cannabis use may be contributing to the population level of subclinical psychosis expression.

A recently published study using the Dunedin birth cohort data found that genetic predisposition moderated the effect of cannabis on psychosis such that adolescent cannabis users with functional polymorphism of a gene (catechol-O-methyltransferase, or COMT) involved in the dopamine system (the neurotransmitter system implicated in schizophrenia) were at risk of developing schizophreniform disorder by the age of 26, whereas adolescent cannabis users who did not have this genetic polymorphism were not at an increased risk of being diagnosed with schizophreniform disorder (Caspi *et al.*, 2005).

The association between cannabis use and psychotic symptoms has also been reported among the Australian Aboriginal communities in Arnhem Land (Clough *et al.*, 2006). This study followed a sample of cannabis users and found that those using cannabis at baseline and three years later reported high levels of auditory hallucinations, suicidal ideation and imprisonment than those not using cannabis.

In summary, evidence indicates that cannabis use may precipitate schizophrenia in people who are vulnerable due to a personal or family history of schizophrenia. New research also suggests that cannabis use in those who have psychosis vulnerability increases the chances of experiencing psychotic symptoms, especially when this use is regular. In addition, cannabis use appears to exacerbate the symptoms of schizophrenia. Whether using cannabis causes additional cases of schizophrenia is undecided.

Relationship with depression

In comparison to psychosis, there has been far less attention given to the association between cannabis use and depression. One reason may be that depressed cannabis users are less likely than those with psychosis to access treatment and, if they do, treatment providers are unlikely to enquire about depressive symptoms (Degenhardt, Hall & Lynskey, 2001).

Cross-sectional epidemiological evidence indicates that cannabis use and depression occur together at a frequency greater than chance. It remains unclear, however, whether this association is due to other variables that are also correlated with depression such as age, gender and other drug use. Chen and colleagues (2002) re-analysed the US National Comorbidity Survey (NCS) to examine the relationship between cannabis use and major depressive episode, and found that having been cannabis dependent at some point in life was associated with a 3.4 times greater risk of major depression.

Longitudinal research on the association between cannabis use and depression has also produced mixed results (Copeland, Gerber & Swift, 2006). Of the more recent studies, Patton and colleagues followed a sample of Australian secondary students (aged 14-15 years) over seven years and found that 68% of daily cannabis users suffered from a mixed state of depression and anxiety. This translated to a fourfold increased risk of depression and anxiety compared to non-users after controlling for other drug use, pre-existing symptoms and antisocial behaviour. These findings, however, were only evident for females in the sample (Patton *et al.*, 2002).

Research findings have not supported a self-medication hypothesis that depressed people are more likely than others to use cannabis to improve their mood (Copeland, Gerber & Swift, 2006). Given the high rate of suicide in young Australian males and the common use of cannabis in this group, the issue of suicide risk and cannabis use is important. A small body of

research has examined whether cannabis use heightens the risk of suicide or attempted suicide, however, there is no persuasive evidence of a causal relationship (Copeland, Gerber & Swift, 2006).

In summary, cross sectional studies have generally indicated that the association between cannabis use and depression is partly explained by factors such as the use of other drugs, family structure, marital status and personality characteristics. Longitudinal research has consistently found that depression does not predict cannabis use, but that cannabis use poses a moderate risk for later depression after accounting for other influencing factors, especially for adolescent girls. It has been asserted that 9% of the cases of depression among past-year cannabis users in the US population might be prevented by eliminating cannabis use (Harder, Morral & Arkes, 2006). Although the literature on cannabis use and suicide risk has produced mixed results, there is reason to believe that heavy cannabis use may pose a small additional risk of suicide. There is also sufficient evidence, particularly among females, to provide support the provision of information in public health campaigns on cannabis use about the elevated risk of depression and suicidality (Copeland, 2006).

Relationship with anxiety

There is little research examining the relationship between cannabis use and anxiety. While experiencing individual symptoms of anxiety is a fairly normal human experience, when several symptoms cluster together this may form an anxiety disorder. This disorder may encompass several syndromes such as panic disorder, obsessive compulsive disorder and phobias.

Like depression and schizophrenia, anxiety disorders occur at higher rates in frequent users of cannabis, however, it is unclear whether this association is due to factors such as other drug use, personal and peer characteristics. In a survey of Australian adolescents, Rey and colleagues found that those who reported cannabis use in the past month experienced more anxiety symptoms than those who did not (Rey *et al.*, 2002). Unfortunately, it is unknown whether this relationship would have persisted after controlling for other factors.

Longitudinal research has generally found no relationship between cannabis use and anxiety (McGee *et al.*, 2000) or that other factors account for this relationship (Fergusson and colleagues, 1997; 2002; Goodwin, Fergusson & Horwood, 2004), however, there have been exceptions (Patton *et al.*, 2002). The Dunedin longitudinal study found no relationship between cannabis use and anxiety at ages 15, 18 and 21 years (McGee *et al.*, 2000). In the Christchurch cohort study, Fergusson and Horwood found that those who use cannabis 10 or more times by the age of 15-16 years were more likely to have screened positively for an anxiety disorder at age 16-18 years; 31% of those who had used 10 times or more, 19% of those who had used one to nine times and 15% of those who had never used cannabis. After controlling for individual, familial, peer and socio-demographic variables, this relationship became non-significant. The most recently released findings from the cohort at 21 years indicated that substance abuse and dependence was substantially higher among those with anxiety, however, this could be explained by childhood and family factors, prior substance dependence, comorbid depression and peer affiliations 9Goodwin, Fergusson & Horwood, 2004). The authors concluded that the relationship between anxiety and illicit substance use is either largely or wholly non-causal.

In summary, research suggests that cannabis use and anxiety disorders occur together at a rate greater than chance. This relationship, however, seems to be largely mediated by other variables such as childhood and family factors, other drug use and peer affiliations, rather than cannabis use per se.

Other harms associated with cannabis use

Intoxication

Unlike other drugs of abuse such as heroin or cocaine, there have been no documented cases of death from cannabis overdose. Adverse cardiovascular consequences arising from occasional use in healthy individuals are also unlikely. This may be dangerous, however, for persons who are predisposed to cardiovascular problems or have respiratory pathology.

While naïve users may experience anxiety, dysphoria, panic and paranoia which may contribute to panic attacks, the most serious effects of intoxication are cognitive and psychomotor impairment (Swift, Copeland & Lenton, 2000). Cannabis intoxication causes disruption of cognitive function (e.g., memory, learning and temporal processing) and psychomotor impairment (Beardsley & Kelly, 1998). Cannabis use is viewed as potentially disruptive of everyday behaviours reliant on complex cognitive processing. The most particular concern is the effects of cannabis on driving. As there has been no reliable method to measure cannabis intoxication analogous to random breath testing, information on the prevalence of people driving under the influence of cannabis has often relied on self-report or analysis of the urine or blood of people involved in car accidents (Copeland, Gerber & Swift, 2006). A large review of fatal and non-fatal vehicle accident studies shows that between 1.4% and 27.5% (average 11.9%) of accidents involved drivers who tested positive for cannabis in their urine or blood (McDonald *et al.*, 2003).

Among cannabis users, the majority (90%) of a sample of long-term cannabis users from the north coast of NSW reported driving at least occasionally after using cannabis and 70% reported driving sometimes while using it (Reilly *et al.*, 2998). A subsequent telephone survey of 502 18-29 yr old cannabis users recruited from the general population in the same area revealed that 11% had ever driven within an hour of using cannabis, while 7% had done so in the past 12 months (Jones, Freeman & Weatherburn, 2003). In that study, among regular cannabis users 41% had driven within an hour of using cannabis in the past 12 months.

Several studies have examined whether cannabis-using drivers were more culpable in fatal and non-fatal accidents than those who had not used the drug. A recent review concluded that most culpability studies have not found that cannabis alone predicted crash culpability (Shannon *et al.*, 1993). These studies, however, only tested urine or blood for an inactive THC metabolite. This makes it difficult to ascertain whether drivers were impaired at the time of the accident. In contrast, the few studies that have tested for active THC in the blood have found that its presence is associated with an approximate 3-fold risk of accident (Shannon *et al.*, 1993). This risk is elevated when larger quantities of cannabis are consumed or when cannabis is used with alcohol.

There have only been two controlled studies examining rates of cannabis use in drivers involved in fatal and non-fatal car accidents. The first study found that people testing positive for cannabis in their urine were no more likely to be involved in a collision than controls (Marquet *et al.*, 1998). When only women were analysed, however, cannabis intoxication was associated with an increased accident risk. A larger study using blood sampling found that after matching for sex and age, people testing positive for cannabis were 2.5 times more likely to be involved in a collision than those who were drug free (Mura *et al.*, 2003). In that study, people who had combined cannabis with alcohol were 4.6 times more likely to be involved in a collision than controls.

In summary, the evidence suggests that cannabis use does produce minor impairments in driving performance. In turn, this increases the chances of being involved in, and being culpable for, traffic accidents. This is especially the case when cannabis is combined with alcohol.

Cannabis and reproductive health

Cannabis is the most commonly used illicit drug amongst women of reproductive age and levels of self-reported cannabis use during pregnancy are relatively high compared with other illicit drugs (Copeland, Gerber & Swift, 2006). There is firm evidence that cannabis use during pregnancy is not associated with higher rates of foetal mortality (Fried, 2002). The most robust finding in the literature, however, is that cannabis use during pregnancy is associated with a minor reduction in birth weight, smaller than that related to maternal tobacco use (Zuckerman *et al.*, 1989; Fergusson, Horwood & Northstone, 2002).

Cannabis dependence

The most common consequence of prolonged, regular cannabis use is dependence. The Australian National Survey of Mental Health and Wellbeing, estimated that approximately 300,000 people or 2.2% of the Australian adult population either abused or where dependent upon cannabis (Swift, Hall & Teesson, 2001). This translates to roughly one in three individuals who had used cannabis in the past twelve months. The top four symptoms reported by dependent adults were withdrawal or using cannabis as withdrawal relief (88.8%), persistent desire or unsuccessful efforts to control use (86.9%), tolerance (72.6%) and using cannabis in larger amounts or for a longer time than intended (62.8%) (Swift, Hall & Teesson, 2001a). More recently, an Australian epidemiological survey of young adults found the most common symptoms experienced by dependent cannabis users were a persistent desire for the drug (91%), using for longer or in larger amounts than intended (84%), withdrawal (74%) and spending excessive time obtaining or using the drug (74%) (Coffey, *et al.*, 2002). In that study, around one in five cannabis-dependent young people also reported a tolerance to the drug.

One of the consequences of increasing levels of cannabis dependence in the community is the concomitant increase in rates of treatment provision for cannabis dependence that have been discussed previously.

Cannabis and crime

In addition to the association between cannabis use and adverse social outcomes such as poor educational attainment, evidence suggests that cannabis use is related to delinquency and crime. Despite this, there remains uncertainty as to how cannabis use and crime are associated. There is some longitudinal evidence that drug users had higher rates of criminal activity even before initiating substance use (including cannabis) and that subsequent drug use, with the exception of heroin, had little effect on crime (Johnston, O'Malley & Eveland, 1978). This has been supported by other findings that after controlling for demographic and psychosocial variables, adolescent drug use failed to predict crime (Newcomb & Bentler, 1988). In contrast, a longitudinal study by Fergusson and Horwood(1997) found that cannabis use predicted criminal behaviour at age 16 years in a New Zealand sample after statistically adjusting for a range of factors including peer criminal behaviour and substance use. This relationship persisted at age 21 years, especially in early initiators. Another prospective New Zealand study found that cannabis dependence, alcohol dependence, and schizophrenia were strongly associated with officially recorded violence but this relationship disappeared when confounding factors were taken into account(Arseneault et al., 2000). There is also recent evidence, however, that deviant peer affiliations explain at least some of the link between drug use and crime (Fergusson & Horwood, 2002).

In addition to population-based studies linking the early and/or heavy use of cannabis with increased participation in crime, surveys of young people involved with the juvenile justice system report high levels of cannabis use and a relationship between frequent cannabis use and offending behaviour (Copeland, Gerber & Swift, 2006). For example, young NSW detainees report much higher rates of regular (at least weekly) cannabis use and a lower age of initiation

than their Australian peers at a population level (Copeland *et al.*, 2003). There was a significant increase in the proportion of detainees reporting use at this level between 1996 and 1999 (83% vs. 71%). One in three (30%) male detainees has reported that they felt their cannabis use was a problem (Copeland, Howard & Fleischmann, 1998).

Salmelainen's (1995) study of 247 NSW juvenile detainees found that those offenders who reported higher levels of cannabis consumption were more likely to be frequent offenders, particularly those involving motor vehicle theft and break and enter offences. While these studies have focused on young people in custodial care, a 1996 NSW secondary schools survey also found cannabis use predicted criminal involvement, with the odds of participation in assault, malicious damage and acquisitive property crime between two and fives times greater among frequent cannabis users than non-users, after controlling for drug use and developmental factors (Baker, 1998).

Cannabis and respiratory health

Evidence suggests that cannabis smoke is carcinogenic and causes mutations in cells, raising the risks of cancer development in the areas of the body that are exposed to cannabis smoke (Tashkin, 1999). Despite this, the link between cannabis use and cancer is still debated. The effect of cannabis smoke on the airway system has received significant attention in the medical research literature. One reason for this is that the smoke of cannabis is similar to the smoke of tobacco in several ways (Copeland, Gerber & Swift, 2006).

A number of studies have indicated that cannabis smoke causes respiratory inflammation even in relatively young habitual cannabis smokers who do not use tobacco (Copeland, Gerber & Swift, 2006). In those studies, the effects of cannabis dependence were roughly comparable to smoking 1-10 cigarettes per day. In comparison to the non-smoking group, cannabis dependent individuals had higher rates of shortness of breath, wheezing, bronchial sputum and chest tightness after controlling for tobacco use. Taylor and colleagues (2002) conducted a further follow-up on this group at age 26 years and found that cannabis smoking produced minor decreases in lung function after controlling for the possible confounders of tobacco use, age and weight. Although these findings were marginal, the authors believed that with continued cannabis use, lung functioning could further deteriorate.

Trends in cannabis use

Cannabis remains to be the most commonly used illicit drug in Australia with an estimated five and a half million people aged 14 and above having tried the drug and more than three quarters of a million people having used cannabis in the previous week. While cannabis use in the Australian community rose during the 1970s and peaked in the 1990s, levels of use have slightly decreased and stabilized since 1998. Similarly, analyses of the cannabis market continue to show it is easily available and the price is stable.

One of the concerning patterns of cannabis use is the declining age of initiation to cannabis use and the use of the more potent parts of the cannabis plants among younger smokers. As adolescents are more likely to develop cannabis dependence for a given exposure than are adults this pattern will continue to feed the increase in rates of cannabis treatment seeking currently being observed in Australia and internationally. This need for greater treatment provision will be further driven by the ageing of the cannabis smoking cohort. The 30-39 year age group is now reporting the highest levels of daily cannabis smoking and is likely to be experiencing significant physical and psychological harms as a result of their long-term, regular use.

The most concerning effects of regular cannabis for young people are the risk of dependence, poor educational outcomes and increased risk of criminal offending such as property crime. For

adults, the greatest risks from regular cannabis use are dependence, mental health problems, respiratory effects, and reduced birth weight for the babies of pregnant smokers. For clinicians and policy makers the increasing rates of cannabis use in Aboriginal and Torres Strait Islander communities and the increased demand for cannabis treatment are emerging issues.

Summary of the cannabis use in Australia

Population based research indicates that cannabis remains the most commonly used illicit drug in Australia, with 33.6% having tried cannabis and 11.3% having used it in the preceding 12 months. Males are more likely than females to use cannabis, to use with greater frequency and to use the more potent parts of the plant. The demand for treatment provision has increased in recent years, a demand that is likely to continue, driven by an ageing cohort of regular heroin smokers.

References

AGAL. (1996). Report on cannabis smoking project. Report prepared for the Department of Human Services and Health. Melbourne: Australia: Australian Government Analytical Laboratories, Victoria.

Andreasson, S., Allebeck, P., & Rydberg, U. (1989). Schizophrenia in users and nonusers of cannabis: a longitudinal study in Stockholm County. *Acta Psychiatrica Scandinavica*, 79(5), 505-510.

Arendt, M., Rosenberg, R., Foldager, L., Perto, G. & Munk-Jorgensen, P. (2005). Cannabisinduced psychosis and subsequent schizophrenia-spectrum disorders: follow-up study of 535 incident cases. *British Journal of Psychiatry*, 187, 510-515.

Arseneault, L., Cannon, M., Poulton, R., Murray, R., Caspi, A., & Moffitt, T. E. (2002). Cannabis use in adolescence and risk for adult psychosis: Longitudinal prospective study. *British Medical Journal*, *325*(7374), 1212-1213.

Arseneault, L., Moffitt, T., Caspi, A., Taylor, A., & Silva, P. A. (2000). Mental disorders and violence in a total birth cohort: results from the Dunedin study. *Archives of General Psychiatry*, *57*, 979-986.

Anthony, J. C., & Helzer, J. (Eds.). (1991). *Syndromes Of Drug Abuse And Dependence*. New York: Free Press, Macmillan.

Baker, J. (1998). *Juveniles in crime – Part 1: Participation rates and risk factors*. Sydney: NSW Bureau of Crime Statistics and Research and NSW Crime Prevention Division.

Beardsley, P.M. & Kelly, T.H. (1999). Acute effects of cannabis on human behavior and central nervous system functions. In: Kalant H, Corrigall W, Hall W, Smart R. eds. *The Health Effects of Cannabis*. Toronto: Centre for Addiction and Mental Health, 129-169.

Caspi, A., Moffitt, T., Cannon, M., McClay, J., Murray, R., Harrington, *et al.*, (2005). Moderation of the effect of adolescent-onset cannabis use on adult psychosis by the functional polymorphism in the catechol-O-methyltransferase gene: longitudinal evidence of a gene X environment interaction. *Biological Psychiatry*, *57*(*10*), 1117-1127.

Chen, B. C., & Kandel, D. B. (1995). The natural history of drug use from adolescence to the mid-thirties in a general population sample. *American Journal of Public Health*, *85*, 41-47.

Chen, C.-Y., Wagner, F. A., & Anthony, J. C. (2002). Marijuana use and the risk of major depressive episode: Epidemiological evidence from the United States National Comorbidity Survey. *Social Psychiatry & Psychiatric Epidemiology*, *37*(*5*), 199-206.

Chopra, G. S., & Smith, J. W. (1974). Psychotic reactions following cannabis use in East Indians. *Archives of General Psychiatry*, *30*, 24-27.

Clough, A. R., d'Abbs, P., Cairnley, S., Gray, D., Maruff, P., Parker, R. & O'Reily, B. (2004). Emerging patterns of cannabis and other substance use in Aboriginal communities in Arnhem Land, Northern Territory: a study of two communities. *Drug & Alcohol Review, 23*(4), 381-390.

Clough, A. R., Cairney, S. J., Maruff, P., & Parker, R. M. (2002). Rising cannabis use in Indigenous communities. *Medical Journal of Australia*, 177(7), 395-396.

Clough, A. R., Guyula, T., Yunupingu, M., & Burns, C. B. (2002). Diversity of substance use in eastern Arnhem Land (Australia): Patterns and recent changes. *Drug & Alcohol Review*, 21(4), 349-356.

Clough, A.R., Lee K.S.K., Cairney, S., Maruff, P., O'Reilly, B., d'Abbs, P. & Conigrave, K. (2006). Changes in cannabis use and its consequences over 3 years in a remote indigenous population in northern Australia. *Addiction*, *101*, 696-705.

Coffey, C., Carlin, J. B., Degenhardt, L., Lynskey, M., Sanci, L., & Patton, G. C. (2002). Cannabis dependence in young adults: An Australian population study. *Addiction*, 97(2), 187-194.

Coffey, C., Carlin, J. B., Lynskey, M., Li, N., & Patton, G. C. (2003). Adolescent precursors of cannabis dependence: Findings from the Victorian Adolescent Health Cohort Study. *British Journal of Psychiatry*, 182(4), 330-336.

Coffey, C., Lynskey, M., Wolfe, R., & Patton, G. C. (2000). Initiation and progression of cannabis use in a population-based Australian adolescent longitudinal study. *Addiction*, 95(11), 1679-1690.

Copeland, J. (2006). (Invited editorial). Cannabis use, depression and public health. *Addiction*, *101(10)*, 1380.

Copeland, J. Gerber, S. & Swift, W. *Evidence-Based Answers To Cannabis Questions*. Canberra: Australian National Council on Drugs.

Copeland, J., Howard, J. & Fleischmann, S. (1998). Gender, HIV knowledge and risk-taking behaviour among substance using adolescents in custody in New South Wales. *Journal of Substance Misuse* 3, 206-212.

Copeland, J., Howard, J., Keogh, T. & Seidler, K. (2003). Patterns and correlates of substance use amongst juvenile detainees in New south Wales 1989-99. *Drug and Alcohol Review* 22, 15-20.

Copeland, J., Swift, W., & Reid, A. (2003). Young cannabis users' attitudes and beliefs about cannabis drug education. *Journal of Drug Education and Awareness*, 1(2), 119-127.

Degenhardt, L., & Hall, W. (2001). The association between psychosis and problematical drug use among Australian adults: Findings from the National Survey of Mental Health and Well-Being. *Psychological Medicine*, *31*(*4*), 659-668.

Degenhardt, L., Hall, W., & Lynskey, M. (2001). The relationship between cannabis use, depression and anxiety among Australian adults: Findings from the National Survey of Mental Health and Well-Being. *Social Psychiatry & Psychiatric Epidemiology*, *36*(5), 219-227.

Degenhardt, L., Hall, W., & Lynskey, M. (2003). Testing hypotheses about the relationship between cannabis use and psychosis. *Drug & Alcohol Dependence*, *71(1)*, 37-48.

DiMaggio, C., Martinez, M., Menard, J. F., Petit, M., & Thibout, F. (2001). Evidence for a cohort effect for age of onset of schizophrenia. *American Journal of Psychiatry*, 158(3), 489-492.

ElSohly, M. A., & Ross, S. A. (2003). *Quarterly Report Potency Monitoring Project: Report* 82. Mississippi: National Centre for Natural Products Research: University of Mississippi.

Fergusson, D. M., & Horwood, L. J. (1997). Early onset cannabis use and psychosocial adjustment in young adults. *Addiction*, 92(3), 279-296.

Fergusson, D. M. & Horwood, J. (2002). Deviant Perr Affiliations, Crime and Substance Use: A Fixed Effect Regression Analysis. *Journal of Abnormal Child Psychology*, *30*(4), 419-430.

Fergusson, D. M., Horwood, L. J., & Lynskey, M. (2003). Cannabis and educational achievement. *Addiction*, *98*, 1681-1692.

Fergusson, D. M., Horwood, L.J. & Northstone, K. (2002). Maternal use of cannabis and pregnacy outcome. *International Journal of Obstetrics & Gynecology*, *109*, 21-27.

Fergusson, D. M., Lynskey, M., & Horwood, L. J. (1996). The short-term consequences of early onset cannabise use. *Journal of Abnormal Child Psychology*, 24(4), 499-512.

Fergusson, D. M., Horwood, L. J., & Swain-Campbell, N. R. (2003). Cannabis dependence and psychotic symptoms in young people. *Psychological Medicine*, *33*(1), 15-21.

Goodwin, R. D., Fergusson, D. M., & Horwood, L. J. (2004). Association between anxiety disorders and substance use disorders among young persons: results of a 21 year longitudinal study. *Journal of Psychiatric Research*, *38*, 295-304.

Fried. (2002). The consequences of marijuana use during pregnancy: A review of the human literature, *Russo, Ethan (Ed); Dreher, Melanie (Ed); et al. (2002). Women and cannabis: Medicine, science, and sociology. Journal of cannabis therapeutics, Vol. 2 (3-4) 2002.* (pp. 85-104). New York, NY, US: Haworth Press, Inc.

Goodwin, R. D., Fergusson, D. M., & Horwood, L. J. (2004). Association between anxiety disorders and substance use disorders among young persons: results of a 21 year longitudinal study. *Journal of Psychiatric Research*, *38*, 295-304.

Grotenhermen, F., & Russo, E. (2002). *Cannabis and Cannabinoids: Pharmacology, Toxicology and Therapeutic Potential*. The Haworth Integrative Healing Press: NY. *Cannabis and Cannabinoids: Pharmacology, Toxicology and Therapeutic Potential*. The Haworth Integrative Healing Press: NY.

Gruber, A. J., Pope, H. G., Hudson, J. I., & Yurgelun-Todd, D. (2003). Attributes of long-term heavy cannabis users: a case control study. *Psychological Medicine*, *33*, 1415-1422.

Hall, W., Degenhardt, L., & Lynskey, M. (2001). *The health and psychological effects of cannabis use*. Sydney: National Drug Strategy: Monograph No. 44.

Harder, V.S., Morral, A.R., & Arkes, J. (2006). Marijuana use and depression amongst adults: testing for causal association. *Addiction*, *101(10)*, 1463-1472.

Jablensky, A., McGrath, J., Herrman, H., Castle, D., Gureje, O., Evans, M., Carr, V., Morgan, V., Korten, A., & Harvey, C. (2000). Psychotic disorders in urban areas: An overview of the Study on Low Prevalence Disorders. *Australian & New Zealand Journal of Psychiatry*, 34(2), 221-236.

Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (2001). *Monitoring The Future: national results on Drug Use: 1975-2001. Volume II: College Students and Adults Aged 19-40.* Bethesda, MD: National Institute on Drug Abuse.

Johnston, L. D., O'Malley, P. M., & Eveland, L. K. (Eds.). (1978). *Drugs and delinquency: a search for causal connections*. New York: John Wiley.

Jones, C., Freeman, K. & Weatherburn, D. (2003). Driving Under the Influence of Cannabis in a NSW Rural Area. *Crime and Justice Bulletin, May,* NSW Bureaof Crime Statistics and Research: Sydney.

Lenton, S., Bennett, A., & Heale, P. (1999). *The Social Impact of a Minor Cannabis Offence Under Strict Prohibition - The Case of Western Australia*. Perth: National Centre for Research into the Prevention of Drug Abuse.

Lukas, S. E., & Orozco, S. (2001). Ethanol increases plasma Delta-9 tetrahydrocannabinol (THC) levels and subjective effects after marihuana smoking in human volunteers. *Drug & Alcohol Dependence*, 64(2), 143-149.

Marquet, P., Delpha, P. A., Kerguelen, S., Bremend, J., Facey, F., Garnier, M., Guery, B., Lhermitte, M., D., M., Pellisier, A. L., Renaudeau, C., Vest, P., & Seguela, J. P. (1998). Prevalence of drugs of abuse in urine of drivers involved in road accidents in France: a collaborative study. *Journal of Forensic Sciences*, 43(4), 806-811.

Martin, G., Copeland, J., & Swift, W. (2005). Adolescent Cannabis Check-up: feasibility study *Journal of Substance Abuse Treatment*, 29(3), 207-213.

Mathers, C., Vos, T. and Stevenson, C. (1999) *The Burden Of Disease And Injury In Australia*. Canberra, Australian Institute of Health and Welfare.

Macdonald, S., Anglin-Bodrug, K., Mann, R. E., Erickson, P., Hathaway, A. D., Chipman, M., & Rylett, M. (2003). Injury risk associated with cannabis and cocaine use. *Drug & Alcohol Review*, 72(2), 99-115.

McGee, R., Williams, S., Poulton, R., & Moffitt, T. (2000). A longitudinal study of cannabis use and mental health from adolescence to early adulthood. *Addiction*, *95*(*4*), 491-503.

McGuire, P. K., Jones, P., Harvey, I., Williams, M., & et al. (1995). Morbid risk of schizophrenia for relatives of patients with cannabis-associated psychosis. *Schizophrenia Research*, 15(3), 277-281.

Mura, P., Kintz, P., Ludes, B., Gaulier, J. M., Marquet, P., Martin-Dupont, S., Vincent, F., Kaddour, A., Goulle, J. P., Nouveau, J., Moulsma, M., Tilhet-Coartet, S., & Pourrat, O. (2003). Comparison of the prevalence of alcohol, cannabis and other drugs between 900 injured drivers and 900 control subjects: Results of a French collaborative study. *Forensic Science International*, 133(1-2), 79-85.

Newcomb, M. D., & Bentler, P. M. (1988). Consequences of adolescent drug use: impact on the lives of young adults. CA: Sage.

Patton, G. C., Coffey, C., Carlin, J. B., Degenhardt, L., Lynskey, M., & Hall, W. (2002). Cannabis use and mental health in younger people: Cohort study. *British Medical Journal*, *325*(7374), 1195-1198.

Reilly, D., Didcott, P., Swift, W., & Hall, W. (1998). Long-term cannabis use: characteristics of users in an Australian rural area. *Addiction*, *93*(6), 837-846.

Rey, J. M., Sawyer, M. G., Raphael, B., Patton, G. C., & Lynskey, M. (2002). Mental health of teenagers who use cannabis: Results of an Australian survey. *British Journal of Psychiatry*, 180(3), 216-221.

Rissel, C., McLellan, L., & Bauman, A. (2000). Social factors associated with ethnic differences in alcohol and marijuana use by Vietnamese-, Arabic- and English-speaking youths in Sydney, Australia. *Journal of Paediatrics & Child Health.*, *36*(2), 145-152.

Salmelainen, P. (1995). *The correlates of offending frequency: A study of juvenile theft offenders in detention.* Sydney: NSW Bureau of Crime Statistics and Research.

Shannon, H. S., Hope, L., Griffith, L., & Stieb, D. (1993). Fatal occupational accidents in Ontario, 1986-1989. *American Journal of Individual Medicine*, 23, 253-264.

Stafford, J., Degenhardt, L., Black, E., Brunio, R., Buckingham, K., Fetherston, J., Jenkinson, R., Kinner, S., Newman, J. & Weekley, J. (2006). *Australian Drug Trends 2005. Findings of the Illicit Drug Reporting System (IDRS).* NDARC Monograph No. 59. Sydney: National Drug and Alcohol Research Centre.

Stefanis, N.C., Delespaul, P., Henquet, C., Bakoula, C., Stefanis, C.N., & Van Os, J. (2004). Early adolescent cannabis exposure and positive and negative dimensions of psychosis. *Addiction*, *99*(*10*), 1333-1341.

Swift, W., Copeland, J., & Lenton, S. (2000). Cannabis and harm reduction. Harm Reduction Digest. *Drug and Alcohol Review, 19*, 101-112.

Swift, W., Hall, W., & Teesson, M. (2001). Cannabis use and dependence among Australian adults: Results from the National Survey of Mental Health and Wellbeing. *Addiction*, *96*(5), 737-748.

Swift, W., Hall, W., & Teesson, M. (2001a). Characteristics of DSM-IV and ICD-10 cannabis dependence among Australian adults: Results from the National Survey of Mental Health and Wellbeing. *Drug & Alcohol Dependence*, *63*(2), 147-153.

Taylor, D. R., Fergusson, D. M., Milne, B. J., Horwood, L. J., Moffitt, T. E., Sears, M. R., & Poulton, R. (2002). A longitudinal study of the effects of tobacco and cannabis exposure on lung function in young adults. *Addiction*, *97*(8), 1055-1061.

Thomas, H. (1996). Psychiatric symptoms in cannabis users. Drug & Alcohol Dependence, 42(3), 201-207

Tien, A. Y., & Anthony, J. C. (1990). Epidemiological analysis of alcohol and drug use as risk factors for psychotic experiences. *The Journal of Nervous and Mental Disease*, *178*(8), 473-480. Van Os, J., Bak, M., Hanssen, M., Bijl, R. V., De Graaf, R., & Verdoux, H. (2002). Cannabis use and psychosis: A longitudinal population-based study. *American Journal of Epidemiology*, *156*(4), 319-327.

Verdoux, H., Gindre, C., Sorbara, F., Tournier, M., & Swendsen, J. D. (2003). Effects of cannabis and psychosis vulnerability in daily life: An experience sampling test study. *Psychological Medicine*, 33(1), 23-32.

Watson, C., Fleming, J. & Alexander, K. (1988). A Survey Of Drug Use Patterns In Northern Territory Aboriginal Communities 1986-1987. Darwin: Northern Territory Department of Health and Community Services, Drug and Alcohol Bureau.

Wenger, T., Moldrich, G., & Furst, S. (2003). Neuromorphological background of cannabis addiction. *Brain Research Bulletin*, 61, 125.

White, V. & Hayman, J. (2004). Australian Secondary Students Use of Over-the-Counter and illicit substances in 2002. National Drug Strategy Monograph Series No. 56. Canberra. Australian Government Department of Health and Ageing.

Zammit, S., Allebeck, P., Andreasson, S., Lundberg, I., & Lewis, G. (2002). Self reported cannabis use as a risk factor for schizophrenia in Swedish conscripts of 1969: Historical cohort study. *British Medical Journal*, *325*(7374), 1199-1201.

Zuckerman, B., Frank, D. A., Hingson, R., Amaro, H., Levenson, S. M., Kayne, H., Parker, S., Vinci, R., Aboagye, K., Fried, L. E., & et al. (1989). Effects of maternal marijuana and cocaine use on fetal growth. *New England Journal of Medicine.*, *320*(12), 762-768

Chapter 5: Meth/amphetamine

Rebecca McKetin

Summary of key changes in meth/amphetamine use over the past decade:

- The number of current meth/amphetamine users in Australia increased during the late 1990s, and has since shown a stable to declining trend.
- There has been a shift from the production and supply of amphetamine to its more potent analogue, methamphetamine, from the mid 1990s.
- Imported high purity crystalline methamphetamine has become a significant feature of the Australian drug market.
- Smoking crystalline methamphetamine has emerged as a new pattern of drug use, alongside the increased availability of crystalline methamphetamine.
- Problems related to methamphetamine have increased from the late 1990s, including methamphetamine-related arrests and hospital admissions for methamphetamine psychosis.
- Methamphetamine injection has been taken-up among a proportion of former heroin injectors subsequent to the 2001 heroin shortage.

Meth/amphetamine use in Australia

The use of 'amphetamines' is a long-standing feature of the Australian drug-scape, with concern over epidemic use levels among young Australians being signalled in the mid-to-late 1980s (Hall & Hando, 1993). Since this time, several changes have occurred in the nature of the amphetamine problem in Australia, which need to be made clear before describing the epidemiology of use. First, in the mid 1990s there was a shift in the manufacture and supply of amphetamine, to its more potent analogue, methamphetamine, which now accounts for over 90% of the amphetamines seized in Australia. Second, Australia has seen the introduction of imported high purity crystalline methamphetamine, or 'ice', since the late 1990s. The emergence of 'ice' has been associated with increased harms among already existing users of the drug (Topp et al., 2002), and the up-take of smoking methamphetamine among a broader group of so-called 'recreational' drug users (McKetin et al., 2005a). Around the same time that crystalline methamphetamine emerged on the Australia drug market, domestically produced methamphetamine started to be marketed as a high purity damp or oily powder called 'base'. It should also be recognized that many of the ecstasy pills available on the Australian drug market contain methamphetamine, either alone or in combination with ketamine (although this market for the drug will not be discussed in the current chapter). The current market for methamphetamine is entrenched, broad and dynamic, encompassing patterns of use that range from infrequent recreational use through to heavy dependent injecting use of the drug.

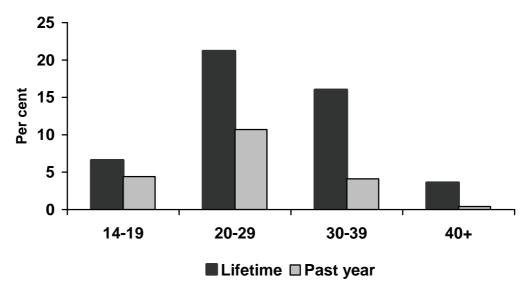
Epidemiology of meth/amphetamine use

Almost one in ten Australians (9.1%) have tried methamphetamine or amphetamine - the drugs sold on the street under a variety of pseudonyms such as 'speed', 'base', 'ice', 'crystal meth' and 'amphetamines'. These findings are from the National Drug Strategy Household Survey, which

also shows that 3.2% of Australians ages 14 and over, or around half-a-million Australians, have used these drugs in the past year (Australian Institute of Health & Welfare, 2005a).

Statistics on the overall prevalence of meth/amphetamine in the general community mask the worryingly high prevalence among young adults in Australia. Among those aged 20-29, one-in-five (21.1%) have used meth/amphetamine in their lifetime, and 10.7% have taken the drug recently (Figure 5.1). Most of these people who take the drug do so infrequently (e.g., 89% use monthly or less often) and are unlikely to experience substantial harms related to their meth/amphetamine use.

Figure 5.1: Lifetime and past year prevalence of meth/amphetamine use in Australia by age, 2004



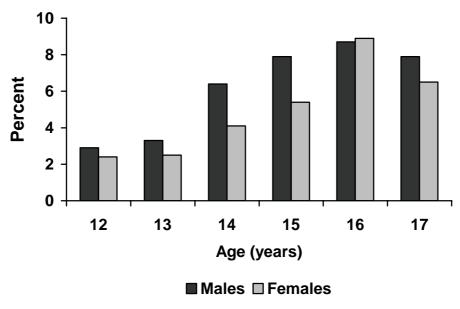
Source: National Drug Strategy Household Survey, 2004

Harms from meth/amphetamine use are seen disproportionately among people who take the drug weekly or more often. According to the 2004 National Drug Strategy Household Survey, around 57,000 Australians fell into this category (Australian Institute of Health & Welfare, 2005a). This figure represents a slight decrease from 2001, when around 64,000 Australians used meth/amphetamine weekly or more often (Australian Institute of Health & Welfare, 2002).

Household surveys typically under-estimate the size of heavy illicit drug using populations, and in-direct prevalence estimation techniques provide an alternative means of estimating the size of these populations. Using such techniques, the estimated number of dependent methamphetamine users in 2002 was slightly higher than the estimate provided by the household survey, at 73,000 or 0.7% of the population aged 15-45 years (McKetin *et al.*, 2005b).

Early onset of drug use is an important predictor of later problematic use patterns. The onset of meth/amphetamine use typically occurs in the mid-to-late teens, and this is reflected in the lifetime prevalence of 8% among 16-17 year old secondary school students (White & Hayman, 2006). Earlier onset of use is less common (Figure 5.2) and is more likely to occur among male students. It is noteworthy that there is little gender difference in the lifetime prevalence of meth/amphetamine among secondary school students by the age of 16-17 years (7% vs. 5%).

Figure 5.2. Lifetime prevalence of meth/amphetamine use among secondary school students (12-17 years) in Australia, 2005



Source: White & Hayman, 2006

Characteristics of meth/amphetamine users

Around 1.5 million Australians have ever tried meth/amphetamine (Australian Institute of Health & Welfare, 2005a). For this reason, it is difficult to define the typical characteristics of those who use the drug. Surveys of methamphetamine users in the community provide a clearer picture of the demographic characteristics of regular methamphetamine users, that is, those people who take the drug at least monthly. Even within this group, the types of users vary from young novice and 'recreational' drug users from various demographic backgrounds, who may be highly functional, through to chronic dependent users of the drug, who are more likely to be injecting drug users with low socioeconomic status and a range of comorbid mental and physical health problems.

Recent community surveys of methamphetamine users have found that the majority of users are young adults who are socially networked with other users of the drug (Kaye & Darke, 2000; Lynch *et al.*, 2003; McKetin *et al.*, 2005a). They often live in share accommodation or with their parents (Lynch *et al.*, 2003; McKetin *et al.*, 2005a), and they tend to have a lower income than people of a similar age within the general population – often being dependent on government allowances (disability pensions or unemployment benefits; Lynch *et al.*, 2003; McKetin *et al.*, 2003; McKetin *et al.*, 2003; McKetin *et al.*, 2005a). Those who are employed tend to work in a variety of non-skilled and semi-skilled occupations (e.g., clerical, sales, service, trades, labour). Less common are highly educated people employed in high-level management and professional occupations (McKetin *et al.*, 2005a), although this subgroup does exist, and is likely to be under-represented in community surveys of drug users because of the way in which they are conducted.

Patterns of meth/amphetamine use

A typical 'hit' of methamphetamine consists of a 'point' (approximately 0.1 grams) of the socalled 'base' methamphetamine or crystalline methamphetamine ('ice'), or half-to-one gram of the low purity powder form of the drug ('speed'). Patterns of methamphetamine use vary from infrequent snorting or swallowing of the drug, which is typical of younger novice or occasional users of the drug, through to daily injection of methamphetamine.

Injecting methamphetamine users typically take the drug two-to-four days a week, injecting once or twice on these days. There is a smaller sub-set of injectors who inject methamphetamine daily and take high doses of the drug several times a day. The crystalline form of methamphetamine can also be smoked using a glass pipe, or less commonly, using a water pipe or 'bong' or using an aluminum foil (McKetin *et al.*, 2005a). The methamphetamine pipe is often shared among friends in social situations, and patterns of smoking methamphetamine range from occasional use through to frequent daily use.

Polydrug use

Methamphetamine users dabble in a variety of other drugs, but heavy cannabis use is particularly common, with over one-third (42%) reporting daily cannabis use (McKetin *et al.*, 2005a), and a similarly high proportion of methamphetamine treatment entrants reporting concurrent cannabis problems (McKetin *et al.*, 2005c). The majority smoke tobacco and drink alcohol, although only a small proportion drink heavily. A substantial proportion have a history of heroin use, including people who are enrolled in opioid maintenance therapy (McKetin *et al.*, 2005a).

Psychopathology associated with of meth/amphetamine use

Psychosis

Methamphetamine can induce a brief paranoid psychosis in some people who take the drug. Symptoms typically include paranoia and hallucinations, which can be accompanied by repetitive stereotyped behaviour, social withdrawal and increased philosophical concern (Davis & Schlemmer, 1980). Methamphetamine psychosis can also be accompanied by an emotionally brisk or labile state, agitation, and sometimes seemingly irrational hostile behaviour (Ellinwood, 1971; Szuster *et al.*, 1990). Symptoms of methamphetamine psychosis usually last hours to days, and in severe cases, can lead to hospitalization and require sedation and/or antipsychotic medication. In rare cases, the condition can last weeks to months, or can remit and recur over a longer period of time, contiguous with drug use and other life stressors (Sato *et al.*, 1992).

The prevalence of psychosis is 11 times higher among methamphetamine users who take the drug monthly or more often than among the general population (McKetin *et al.*, 2006a). Symptoms of psychosis are most likely to occur among chronic dependent users of the drug, rather than infrequent users (in the latter case, the likelihood of psychotic symptoms is more contingent on an underlying vulnerability to psychosis). For example, the relative prevalence of psychotic symptoms among dependent methamphetamine users from the community is 31% versus 13% among their non-dependent counterparts (McKetin *et al.*, 2006a). Even after excluding methamphetamine users with a history of chronic psychotic disorders (e.g., schizophrenia or mania) the past year prevalence of psychotic symptoms among dependent methamphetamine users is still 27%.

Methamphetamine users are at a particularly high risk of experiencing psychotic symptoms from methamphetamine use if they suffer from schizophrenia, mania or other psychotic disorders. Among this group, the drug can precipitate or exacerbate psychotic episodes (as distinct from brief psychotic symptoms that occur in response to intoxication, Curran *et al.*, 2004). Conversely, it is important to appreciate that the majority of methamphetamine users who

experience psychotic symptoms after taking the drug have no known history of schizophrenia, mania or other chronic psychotic disorders (McKetin *et al.*, 2006a).

Other psychopathology

Although methamphetamine psychosis is the most conspicuous form of psychopathology associated with methamphetamine use, other forms of psychopathology are also common – and in some cases more prevalent, chronic and debilitating than methamphetamine psychosis. Depressive symptoms are particularly common among dependent methamphetamine users (Copeland & Sorenson, 2001). Depression often precedes the onset of drug dependence, although depression is also part of the methamphetamine withdrawal syndrome (McGregor *et al.*, 2005). Regular methamphetamine users also report high levels of aggression (Hall *et al.*, 1996), and problems controlling aggressive behaviour (Zweben *et al.*, 2004). Other psychopathologies that need to be considered among this population are anxiety disorders, post-traumatic stress disorder, and personality disorders that often co-occur with dependent drug use.

Other harms associated with meth/amphetamine use

Dependence

Dependence on methamphetamine, like all psychoactive drugs, is defined by tolerance, withdrawal, pre-occupation with the drug, not being able to reduce use, and using the drug despite significant social, health or psychological impairment. Tolerance and withdrawal from methamphetamine are less marked than for depressant drugs, like alcohol and opioids, but these symptoms are still a key feature of the dependence syndrome (Topp & Mattick, 1997). Tolerance is evidenced by the progression from non-injecting routes of administration to injecting methamphetamine use, using higher doses of the drug per hit (e.g., 2-3 'points' per hit) several times per day, and a preference for high purity crystalline methamphetamine. Withdrawal symptoms consist of lethargy, depressed mood, irritability, appetite and sleep disturbances, and strong cravings for the drug (McGregor *et al.*, 2005; Topp & Mattick, 1997). Dependence is strongly associated with injecting and smoking methamphetamine and using the drug more than weekly (McKetin *et al.*, 2006b).

BBV risk

The high prevalence of injection among dependent methamphetamine users places this group at a risk of blood borne virus transmission. Currently levels of HIV are low and stable among injecting drug users in Australia, but Hepatitis C infection is endemic, and there has been a modest increase in hepatitis C prevalence among injectors surveyed through the Australian Needle and Syringe Program Survey in recent years (61% in 2005, National Centre in HIV Epidemiology & Clinical Research, 2006).

Needle sharing behaviour among methamphetamine injectors is similar to that seen among opioid injectors (Kaye & Darke, 2000; McKetin *et al.*, 2005a); however, methamphetamine users are at higher risk than opioid users of contracting HIV through sexual transmission (Molitor *et al.*, 1999; Zule & Desmond, 1999). Methamphetamine users are a younger and more sexually active population than opioid injectors, while it has been argued that the drug's ability to increase libido (Rawson *et al.*, 2002) further enhances the risk of sexual disease transmission among this group.

Toxicity

Methamphetamine use has been associated with a range of fatal and non-fatal cardiac pathology (Kaye & McKetin, 2006); it increases the risk of stroke (Petitti *et al.*, 1998); and, it has been associated with other pathology, including renal failure (Richards *et al.*, 1999) and pulmonary oedema (Nestor *et al.*, 1989). Fatalities from methamphetamine use do occur, but are far less

common than with opioid drugs (Bartu *et al.*, 2004), with around 50 deaths a year caused by psychostimulant drugs, including methamphetamine (Degenhardt *et al.*, 2003).

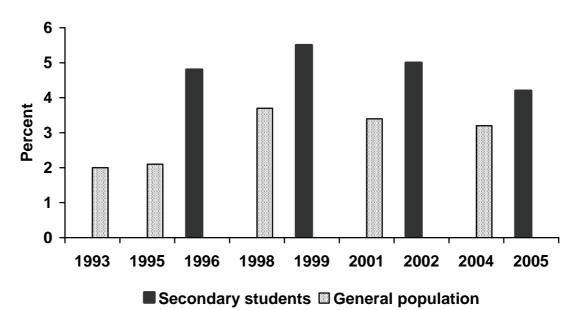
Symptoms of acute overdose are not well documented, but usually include palpitations and tachycardia, sweating, shallow breathing, excessive pupil dilation, hyperthermia, and possibly nausea and diarrhoea. These symptoms may occur in the context of agitation, panic/anxiety, psychosis and aggression, which can also be brought on by excessive doses of the drug (MIMS Online, 2006).

Trends in methamphetamine use

Epidemics of methamphetamine use in Australia have been reported since the 1970s (Hall & Hando, 1993), although national surveys through which we can document the prevalence of use have only been in place over the last twenty years. Even then, the small sample size of early surveys, coupled with methodological changes between surveys, means that robust analysis of trends is only possible over the past decade.

According to the triennial National Drug Strategy Household Surveys, the prevalence of methamphetamine use increased in Australia during the late 1990s (Figure 5.3). At this time, the past year prevalence almost doubled from 2.1% in 1995 to 3.7% in 1998, while there was a corresponding jump in lifetime prevalence (5.7% to 8.8%) (Australian Institute of Health & Welfare, 1999, 2002, 2005a, 2005b). Since this time, the lifetime and current prevalence of methamphetamine use has not changed significantly – if anything, there has been a slight decline in prevalence. Trends in the prevalence of methamphetamine use among secondary school students showed a similar trend, peaking in 1999, and subsequently declining (Figure 5.2, Letcher & White, 1999; White, 2001; White & Hayman, 2004; White & Hayman, 2006).

Figure 5.3. Trends in the past year prevalence of methamphetamine use among secondary school students (aged 12-17 years) and the general population (aged 14 year or over)



Despite the stable prevalence of methamphetamine use over recent years, several indicators of methamphetamine-related problems have shown steady increases since the mid-to-late 1990s (e.g., methamphetamine-related arrests and hospital admissions, Figure 5.4; Australian Bureau

of Criminal Intelligence, 2000, 2001, 2002; Australian Crime Commission, 2003, 2004, 2005, 2006; Australian Institute of Health & Welfare, 2005c). The increase in methamphetamine-related problems seen since the late 1990s is likely to reflect several trends in the drug market (McKetin *et al.*, 2006c), including:

- a) a growing number of chronic users who initiated use in the mid-to-late 1990s;
- b) the shift from amphetamine to methamphetamine supply, which occurred in the mid-1990s;
- c) the emergence of imported high purity crystalline methamphetamine since 1999; and
- d) the up-take of crystalline methamphetamine use among injecting heroin users in the wake of the 2001 heroin shortage.

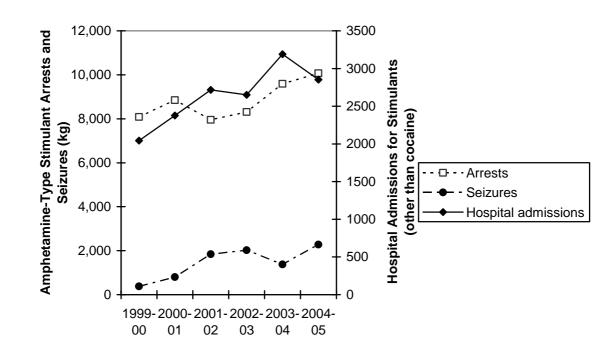


Figure 5.4. Trends in Amphetamine-type Stimulant arrests, seizures and hospital admissions¹

The emergence of high purity crystalline methamphetamine, or 'ice', is a particular concern because it has been associated with higher levels of dependence than other forms of methamphetamine (e.g., low purity powder methamphetamine or 'speed').

Crystalline methamphetamine was first reported through the Illicit Drug Reporting System (IDRS) in 1999 (McKetin *et al.*, 2000). In 2000, quantification of crystalline methamphetamine use through the IDRS revealed that 15% of injecting drug users had recently taken the drug (Topp *et al.*, 2003). Recent use varied between 6-17% of injecting drug users in most jurisdictions, with unusually high levels of recent use in Western Australia (51%). Use peaked in 2001, around the time of the Australian heroin shortage. Despite showing a slight recession in

¹ Amphetamine-type stimulant arrests and seizure data were sourced from the Australian Crime Commission's Illicit Drug Data Reports from 2000 to 2006. Hospital data represent separations from public and private acute care and psychiatric facilities where stimulants other than cocaine were the primary reason for admission (ICD-10 codes F15).

2002, the recent use of crystalline methamphetamine among injecting drug users remained high over the following five years, and recent use of the drug among injecting drug users surveyed across Australia is now 57% (varying between 29% and 88% across jurisdictions). Clear increases have been seen in New South Wales and the Australian Capital Territory over this time, although trends in other jurisdictions are more erratic. Similarly high exposure to crystalline methamphetamine can be seen among ecstasy users, with 49% of regular ecstasy users having recently used the drug (National Drug and Alcohol Research Centre, 2006).

Summary of the methamphetamine use in Australia

Methamphetamine use in Australia is widespread, and is characterized by a range of use patterns and a strong demand for high purity crystalline methamphetamine. Although the prevalence of methamphetamine use has stabilized in recent years, increased up-take of use in the 1990s, coupled with recent increases in the use of crystalline methamphetamine, has been associated with an increase in problems from the drug. Symptoms of psychosis are one of the particularly worrying consequences of methamphetamine use, although dependent methamphetamine users also suffer from a range of comorbid mental health problems, which are often more prevalent and chronic than psychosis. Fatalities are not common in comparison with opioid drugs, although methamphetamine use does increase the risk of morbidity and mortality from cardiovascular pathology. A further concern is the risk of HIV and other blood borne virus transmission among people who inject the drug, while the possibility for secondary sexual transmission of HIV among this group needs to be carefully monitored. Responding to the methamphetamine use situation needs to embrace the broad range of use patterns and types of people who use the drug, and an understanding that harms are disproportionately represented among people who are dependent on the drug.

Crystalline methamphetamine or 'ice'

What is ice?

Crystalline methamphetamine, or 'ice', is a highly purified form of methamphetamine that has a crystal-like appearance. It is sold on the street as 'ice' or 'crystal meth', or less commonly as 'skates', 'shabu', 'shabs' or 'tina'. Ice contains the same chemical as the street drugs 'speed' and 'base', only in a more pure form. These drugs are made through a similar chemical reaction, using the same types of precursor chemicals and reagents. The only difference in the manufacture process is that the ice undergoes additional refinement to remove impurities and allow it to develop a crystalline texture.

How is ice different from 'speed' or amphetamine?

Ice only differs from the powdery street drug 'speed' in terms of its purity. People often think that 'speed' is amphetamine. This was true in the early 1990s, but since the mid-1990s, producers of the drug switched from manufacturing amphetamine to its more potent chemical cousin, methamphetamine. These chemicals have an almost identical pharmacological action on the brain, only methamphetamine is reported to provide a more intense drug effect. While the chemical composition of 'speed' changed from amphetamine to methamphetamine in the mid-1990s, the street term 'speed' remained. Since the late 1990s, almost all street seizures of the drug (irrespective of whether they have a powdery, damp or crystalline appearance) contain the chemical methamphetamine.

How is ice taken?

Ice can be easily injected because it is readily soluble in water. Crystalline methamphetamine can also be smoked using a glass pipe, and when inhaled, the vapourised methamphetamine is rapidly absorbed into the blood stream, yielding an almost instant drug effect (Cook *et al.*, 1993). Methamphetamine can also be smoked using a 'bong' (water pipe) or by heating the drug on a non-flammable surface. While other forms of methamphetamine are smokable in principle (e.g., smoking methamphetamine pills is common in several Southeast Asian countries), the high purity crystalline methamphetamine lends itself to smoking because of the lack of impurities.

Where does ice come from?

It is believed that most of the crystalline methamphetamine available in Australia is imported from East Asia. This view has stemmed from the emergence of large scale border detections of crystalline methamphetamine from 2000, coinciding with increased availability of crystalline methamphetamine on the Australian drug market. Crystalline methamphetamine is now readily available to regular drug users in Australia, and there is evidence that it is also being manufactured domestically, although the extent to which this is occurring is not clear.

Who uses ice?

Most people who use crystalline methamphetamine, or 'ice', also use other forms of methamphetamine, such as 'speed' and 'base'. People who have a preference for ice over 'speed' or 'base' tend to be either older experienced methamphetamine users who prefer the stronger, longer-lasting high that they get from taking this high purity form of the drug. Or, they are people who like to smoke methamphetamine, and use the crystalline form of the drug for this reason.

In recent years, there has been a noticeable up-take of crystalline methamphetamine injection among former heroin users, and smoking crystalline methamphetamine among younger non-injecting drug users. These groups are distinct in terms of their demographic characteristics and their polydrug use. Essentially, crystalline methamphetamine injectors are heavy polydrug users with a long history of contact with drug treatment services and the criminal justice system. In contrast, crystalline methamphetamine smokers are a younger group (median age 22 years vs. 33 years for injectors), who have very limited contact with either drug treatment services or with the criminal justice system, and their polydrug use consists primarily of ecstasy and cannabis use (McKetin *et al.*, 2005a).

Both smoking and injecting methamphetamine are associated with high levels of methamphetamine dependence. In fact, the level of methamphetamine use among smokers rivals that seen among methamphetamine injectors, and methamphetamine smokers are over twice as likely to report symptoms of methamphetamine dependence than other non-injecting methamphetamine users (i.e., those who snort or swallow the drug, McKetin *et al.*, 2006b).

What harms are associated with ice use?

The harms associated with crystalline methamphetamine are the same as those associated with other forms of methamphetamine, such as 'speed' and 'base'. This is because they are essentially the same drug. What dictates the level of harm among users is dependence on methamphetamine. People who use crystalline methamphetamine tend to have higher levels of dependence than people who use only use the low purity powder form of the drug ('speed'), they are more likely to smoke or inject methamphetamine, and they are therefore more likely to suffer harms from their methamphetamine use. In addition, crystalline methamphetamine is a high dosage form of the drug, and this can increase the risk of acute toxicity

References

Australian Bureau of Criminal Intelligence (2000). *Australian Illicit Drug Report 1998-1999*. Commonwealth of Australia, Canberra.

Australian Bureau of Criminal Intelligence (2001). *Australian Illicit Drug Report 1999-2000*. Commonwealth of Australia, Canberra.

Australian Bureau of Criminal Intelligence (2002). *Australian Illicit Drug Report 2000-2001*. Commonwealth of Australia, Canberra.

Australian Crime Commission (2003). *Illicit Drug Data Report 2001-02*. Commonwealth of Australia, Canberra.

Australian Crime Commission (2004). *Illicit Drug Data Report 2002-03*. Commonwealth of Australia, Canberra.

Australian Crime Commission (2005). *Illicit Drug Data Report 2003-04*. Commonwealth of Australia, Canberra.

Australian Crime Commission (2006). *Illicit Drug Data Report 2004-05*. Commonwealth of Australia, Canberra.

Australian Institute of Health and Welfare (1999). 1998 National Drug Strategy Household Survey: Detailed Findings. Drug Statistics Series 6, AIHW cat. no. PHE 27. Australian Institute of Health and Welfare, Canberra.

Australian Institute of Health and Welfare (2002). 2001 National Drug Strategy Household Survey: Detailed Findings. Drug Statistics Series 11, AIHW cat. no. PHE 41. Australian Institute of Health and Welfare, Canberra.

Australian Institute of Health and Welfare (2002). 2001 National Drug Strategy Household Survey: Detailed Findings. AIHW cat. no. PHE 41. Australian Institute of Health and Welfare, Canberra.

Australian Institute of Health and Welfare (2005a). 2004 National Drug Strategy Household Survey: Detailed Findings. Drug Statistics Series 16, AIHW cat. no. PHE 66. Australian Institute of Health and Welfare, Canberra.

Australian Institute of Health and Welfare (2005b). Statistics on drug use in Australia 2004. Drug Statistics Series 15, AIHW cat. no. PHE62. Australian Institute of Health and Welfare, Canberra.

Australian Institute of Health and Welfare (2005c). *Principal Diagnosis Data Cubes, Separation statistics by principal diagnosis in ICD-10-AM, Australia, 1998-99 to 2003-04*. Interactive national hospital morbidity data, Australian Institute of Health and Welfare, Canberra, viewed October 27 2006, http://www.aihw.gov.au/cognos/cgibin/ppdscgi.exe?DC=Q&E=/AHS/principaldiagnosis0304>.

Bartu A., Freeman, N. C., Gawthorne, G. S., Codde, J. P. & Holman, C. D. (2004). Mortality in a cohort of opiate and amphetamine users in Perth, Western Australia. Addiction, 99, 53-60.

Cook, C.E., Jeffcoat, A.R., Hill, J.M., Pugh, D.E., Patetta, P.K., Sadler, B.M., White, W.R., Perez-Reyes, M., (1993). Pharmacokinetics of methamphetamine self-administered to human subjects by smoking s-(+)-methamphetamine hydrochloride. Drug Metab. Dispos. 21, 717-723.

Copeland, A. L. & Sorenson, J. L. (2001). Differences between methamphetamine users and cocaine users in treatment. Drug and Alcohol Dependence, 62, 91-95.

Curran C., Byrappa N., & McBride A. (2004). Stimulant psychosis: systematic review. British Journal of Psychiatry, 185, 196-204.

Davis, J. M. & Schlemmer, R. F. (1980). The amphetamine psychosis. In: Caldwell, J., (Ed), Amphetamine and Related Stimulants: Chemical, Biological, Clinical and Sociological Aspects, pp. 161-173. CRC Press, Florida.

Degenhardt, L., Roxburgh, A. & Black, E. (2003). Cocaine and amphetamine mentions in accidental drug-induced deaths in Australia 1997-2003. National Drug and Alcohol Research Centre, Sydney.

Ellinwood, E. H. (1971). Assault and homicide associated with amphetamine abuse. American Journal of Psychiatry, 127, 90-95.

Hall, W. & Hando, J. (1993). Patterns of illicit psychostimulant use in Australia. In: Burrows, D., Flaherty, B., MacAvoy, M. (Eds.). Psychostimulant use in Australia. Australian Government Publications Service, 53-70.

Hall, W., Hando, J., Darke, S. & Ross, J. (1996) Psychological morbidity and route of administration among amphetamine users in Sydney, Australia. *Addiction*, *91*, 81-87.

Hall, W., Darke, S., Ross, M. & Wodak, A. (1993). Patterns of drug use and risk-taking among injecting amphetamine and opioid drug users in Sydney, Australia. Addiction, 88, 509-16.

Kaye, S. & Darke, S. (2000). A comparison of the harms associated with the injection of heroin and amphetamines. Drug and Alcohol Dependence, 58, 189-195.

Kaye, S., McKetin, R. (2006). Cardiotoxicity associated with methamphetamine use and signs of cardiovascular pathology among methamphetamine users. National Drug and Alcohol Research Centre Technical Report No. 238. National Drug and Alcohol Research Centre, Sydney.

Letcher, T., & White, V. (1999). Australian secondary students' use of over-the-counter and illicit substances in 1996. National Drug Strategy Monograph series No. 33. Commonwealth of Australia, Canberra.

Lynch, M., Kemp, R., Krenske, L., Conroy, A. & Webster, J. (2003). Patterns of amphetamine use. Initial findings from the Amphetamines in Queensland research project. Crime and Misconduct Commission, Brisbane.

McGregor, C., Srisurapanont, M., Jittiwutikarn, J., Laobhripatr, S., Wongtan, T., & White J. M. (2005). The nature, time course and severity of methamphetamine withdrawal. Addiction, 100,1320-9.

McKetin, R., Darke, S., Bruno, R., Dwyer, R., S., Kinner, S., Fleming, J., Hargreaves, K., Humeniuk, R., Rysavy, P. (2000). Australian Drug Trends 1999. Findings from the Illicit Drug

Reporting System (IDRS). NDARC Monograph No.43. National Drug and Alcohol Research Centre, Sydney.

McKetin, R., McLaren, J. & Kelly, E. (2005a). The dynamics of the Sydney methamphetamine market: an overview of supply, use patterns, harms and social consequences. National Drug Law Enforcement Research Fund Monograph Series No. 13. Adelaide: Australasian Centre for Policing Research.

McKetin, R., Kelly, E. & McLaren, J. (2005c). The characteristics of treatment provided for amphetamine use in New South Wales, Australia. Drug and Alcohol Review, 24, 433-436.

McKetin, R., McLaren, J., Kelly, E. (2006b). The relationship between crystalline methamphetamine use and dependence. Drug and Alcohol Dependence, 58, 198-204.

McKetin, R., McLaren, J., Kelly, E., Hall, W. & Hickman, M. (2005b). Estimating the number of regular and dependent methamphetamine users in Australia. National Drug and Alcohol Research Centre Technical Report no. 230. National Drug and Alcohol Research Centre, Sydney.

McKetin, R., McLaren, J., Kelly, E., Lubman, D. & Hides, L. (2006a). The prevalence of psychotic symptoms among methamphetamine users, Addiction, 101, 1473-1478.

McKetin, R., McLaren, J., Riddell, S. & Robins, L. (2006c). The relationship between methamphetamine use and violent behaviour. NSW Bureau of Crime Statistics and Research Crime and Justice Bulletin No. 97. August 2006. NSW Bureau of Crime Statistics and Research, Sydney.

MIMS online. (2003). http://www.mims.com.au. Viewed 8/12/2006. MIMS Australia Pty Ltd.

Molitor, F., Ruiz, J. D., Flynn, N., Mikanda, J. N., Sun, R. K. & Anderson R. (1999). Methamphetamine use and sexual and injection risk behaviors among out-of-treatment injection drug users. American Journal of Drug & Alcohol Abuse, 25, 475-493.

National Centre in HIV Epidemiology and Clinical Research (2006). HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia. 2006 Annual Surveillance Report (Australian Institute of Health and Welfare cat. no. PHE78). National Centre in HIV Epidemiology and Clinical Research, Sydney.

National Drug and Alcohol Research Centre (2006). IDRS – Methamphetamine Findings (Press Release) National Drug and Alcohol Research Centre. Viewed December 12, 2006.<http://notes.med.unsw.edu.au/NDARCWeb.nsf/resources/PR_3/\$file/IDRS+EDRS+PR+FINAL+2006.pdf>

Nestor, T. A., Tamamoto, W. I., Kam, T. H., Schultz, T. (1989). Acute pulmonary oedema caused by crystalline methamphetamine. Lancet. 2,1277-8.1989 Nov 25.

Petitti, D. B., Sidney, S., Quesenberry, C. & Bernstein, A. (1998). Stroke and cocaine or amphetamine use. Epidemiology, 9, 596-600.

Rawson, R. A., Washton, A., Domier, C., & Reiber, C. (2002). Drugs and sexual effects: role of drug type and gender. Journal of Substance Abuse Treatment, 22, 103-108.

Richards, J. R., Johnson, E. B., Stark, R. W. Derlet, R. W. (1999). Methamphetamine abuse and rhabdomyolysis in the ED: a 5-year study. American Journal of Emergency Medicine. 17, 681-685.

Sato, M., Numachi, Y. & Hamamura, T. (1992). Relapse of paranoid psychotic state in methamphetamine model of schizophrenia. *Schizophrenia Bulletin*, 18, 115-122.

Sommers, I., Baskin, D. Baskin-Sommers, A. (2006). Methamphetamine use among young adults: Health and Social Consequences. Addictive Behaviours, 31, 1469-1476.

Szuster, RR 1990, 'Methamphetamine in psychiatric emergencies', Hawaii Medical Journal, vol. 49, pp. 389-391.

Topp, L., & Mattick, R. (1997). Validation of the amphetamine dependence syndrome and the SamDQ. Addiction, 92, 839-845.

Topp, L., Degenhardt, L., Roxburgh, A., Bruno, R., Duquemin, A., Fetherston, J., Fischer, J., Jenkinson, R., Kinner, S., Longo, M. & Rushforth, C. (2003). Australian Drug Trends 2002. Findings from the Illicit Drug Reporting System (IDRS). NDARC Monograph No. 50. National Drug and Alcohol Research Centre, Sydney.

Topp, L., Degenhardt, L., Kaye, S. & Darke, S. (2002). The emergence of potent forms of methamphetamine in Sydney, Australia: A case study of the IDRS as a strategic early warning system. *Drug and Alcohol Review*, 21, 341-348.

White, V. (2001). Australian secondary students' use of over-the-counter and illicit substances in 1999. National Drug Strategy Monograph series No. 46. Commonwealth of Australia, Canberra.

White, V. & Hayman, J. (2004) Australian secondary students' use of over-the-counter and illicit substances in 2002. Canberra: Commonwealth Department of Health and Aged Care.

White, V. & Hayman, J. (2006). Australian secondary school students' use of over-the-counter and illicit substances in 2005. National Drug Strategy Monograph series No. 60. The Cancer Council, Victoria.

Zule, W. A. & Desmond, D. P. (1999). An ethnographic comparison of HIV risk behaviors among heroin and methamphetamine injectors. American Journal of Drug & Alcohol Abuse, 25, 1-23.

Zweben, J. E., Cohen, J. B., Christian, D., Galloway, G. P., Salinardi, M., Parent D. & Iguchi, M. (2004). Psychiatric symptoms in methamphetamine users. American Journal on Addictions, 13, 181-90.

Chapter 6: Ecstasy

Louisa Degenhardt & Matthew Dunn

Summary of key changes in ecstasy use over the past decade:

- The lifetime prevalence of ecstasy use increased from 4.8% in 1998 to 7.5% in 2004. Recent (past year) use increased from 2.4% to 3.4% across that period.
- Among community samples of ecstasy users, the proportion using ecstasy at least weekly has remained stable over the past four years, as has the number of tablets used in a typical session. The past 6 month prevalence of intravenous ecstasy use among this group has decreased (10% in 2003, 5% in 2004, 4% in 2005 and 2006).
- The Secondary Student's Alcohol and Drug Survey (ASSAD) has shown small increases in the proportion of 16-17 year old students indicating past-month ecstasy use between 1996 and 2005.
- Over the past ten years very little research has been conducted in Australia examining the relationship between ecstasy use and mental health. Future research is needed to explore this issue in greater depth.

"Ecstasy" is a "street" term commonly used for tablets containing MDMA, or 3,4methylendioxymethamphetamine. MDMA is a stimulant with hallucinogenic properties. It produces such short-term effects as euphoria and a feeling of well-being, increased selfconfidence, lack of inhibitions, sweating, inability to sleep and increased body temperature. Little is known about the long-term effects, however at high doses these can include memory and cognition problems, depression and neurotoxicity.

Tablets sold as ecstasy may contain a range of substances that do not include MDMA. These can include methamphetamine or ketamine, as well as other substances which are part of the phenethylamine family, such as 3,4-methylenedioxyamphetamine (MDA), para-methoxyamphetamine (PMA) or 3,4-methylenedioxyethylamphetamine (MDEA) (Quinn, Breen & White, 2004). Tablets may also contain licit substances such as caffeine or paracetamol. The term "ecstasy" is used in this chapter with the understanding that drugs sold and consumed as ecstasy may not contain MDMA. Users are well aware of this variability (Johnston, Barrett, Fry *et al*, 2006; Stafford, Degenhardt, Dunn, 2006) and terms such as "pills" are also used to describe ecstasy tablets (perhaps in acknowledgment of the variable quality).

Epidemiology of ecstasy use

Since ecstasy was first included in the National Drug Strategy Household Survey (NDSHS) in 1988, the prevalence of lifetime use among the general population aged 14 and above has increased. The proportions reporting lifetime use rose from 1% of the population in 1988 to 7.5% in the 2004 survey (Australian Institute of Health & Welfare, 2005). Similarly, an upward trend has been observed in the use of ecstasy within the past 12 months. This figure remained at 1% from 1988 to 1995, before increasing to 3.4% in the 2004 survey (Figure 6.1).

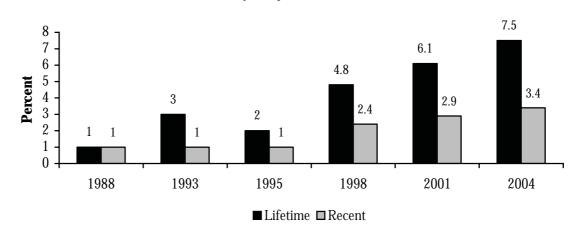
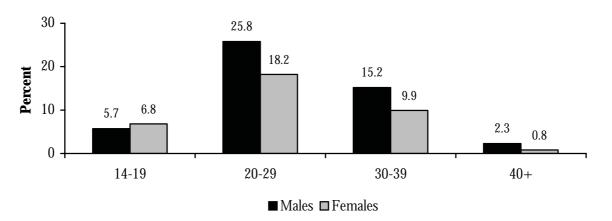


Figure 6.1: Prevalence of lifetime and past year MDMA use in Australia, 1988-2004

Source: National Drug Strategy Household Surveys, 1988-2004 Note: In the 2001 and earlier NDS household surveys, ecstasy was analysed as ecstasy/designer drugs, the term 'designer drugs' never being defined in the survey. The 2004 survey separated out ecstasy, ketamine and GHB and did not cover any other 'designer' drugs.

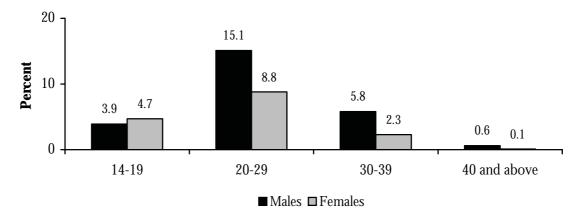
The prevalence of ecstasy use varies slightly by gender and age. In the 2004 survey, 9.1% of males reported lifetime ecstasy use compared to 6% of females (Figure 6.2), while 4.4% of males and 2.4% of females reported recent use (Figure 6.3). Use in the past 12 months was most common among those aged 20-29 years, with 15% of males and 8.8% of females reporting use in this period (Figure 6.3).

Figure 6.2: Prevalence of lifetime MDMA use in Australia by age and gender, 2004



Source: National Drug Strategy Household Survey, 2004



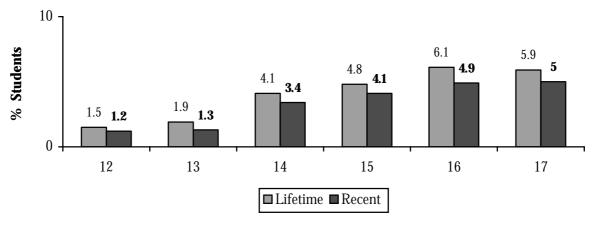


Source: National Drug Strategy Household Survey, 2004

Data collected as part of the 2005 Australian Secondary Student's Alcohol and Drug Survey (ASSAD) show that 3.9% of students aged 12-17 had ever used ecstasy, with 3.2% using ecstasy in the past year (White & Hayman, 2006). Lifetime and past month use appears to increase with age (Figure 6.4); 1.5% of those aged 12 had ever used ecstasy compared with 5.9% of those aged 17. In relation to past month use, there appear to be gender differences according to age: more males reported recent ecstasy use at all ages compared with females (Figure 6.5).

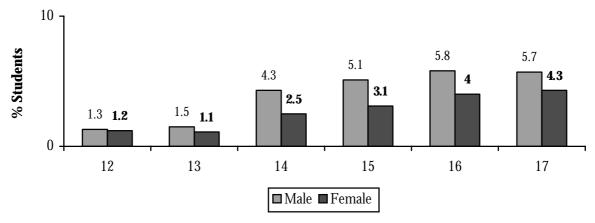
Results from the ASSAD survey over time have indicated no change among younger students indicating either lifetime or past-month ecstasy use (White & Hayman, 2006). Small increases were found in the proportion of 16-17 year old students indicating past-month ecstasy use between 1996 and 2005 (White & Hayman, 2006).





Source: White & Hayman, 2006

Figure 6.5: Prevalence of past month MDMA use in secondary school students by gender, 2005



Source: White & Hayman, 2006

Characteristics of ecstasy users

General population data suggest that ecstasy use is concentrated among young adults. Findings from the 2001 NDS household survey indicate that approximately two-thirds (68%) of those who had ever used ecstasy were aged between 14 and 29 years of age and three-quarters (80%) of those that reported use in the past year (Degenhardt, Barker & Topp, 2004). Users aged 20-29 years of age were more likely to be male, single, students, living in a shared household, or households with parents and children, than non-users of the same age (Degenhardt, Barker & Topp, 2004).

Findings from the Ecstasy and Related Drugs Reporting System (EDRS) support those presented from the general population. Regular ecstasy users in Australia tend to be male, aged in their mid-twenties, from an English speaking background, well-educated, engaged in either full-time employment or tertiary education. They tend to have little contact with law enforcement and very few report being in drug treatment (Dunn, Degenhardt, Campbell *et al*, 2007). These characteristics have remained stable over time.

Patterns of ecstasy use

Among the general population, only a small proportion of recent users report using ecstasy on a weekly basis; in the 2004 NDSHS, 6.3% of those who reported using ecstasy in the past year did so at least once per week. The highest rate of weekly or daily use was found among those aged 14-19 years, with no users aged 40 years and older reporting weekly or daily use (AIHW, 2005).

On a day when ecstasy was used, three-quarters (76%) of recent ecstasy users consumed an average of 1.5 pills. Recent ecstasy users reported concurrently using other drugs with ecstasy, the most commonly reported included alcohol, cannabis and meth/amphetamine (AIHW, 2005).

Findings from the EDRS give detailed patterns of ecstasy use amongst those who report regular use of ecstasy. Age of first ecstasy use appears to begin in late adolescence (mean = 19 yrs) while regular (monthly) use of ecstasy occurs soon after (mean = 21yrs). Females report first using ecstasy at a significantly younger age than males (mean 19 vs. 20) and start using ecstasy regularly at a significantly younger age than males (20 vs. 21).

In 2006, EDRS participants had used ecstasy on a median of 12 days in the past six months (range 4-120); 31% reported using once per week or more. This proportion has remained stable from 2003 to 2006 (Dunn, Campbell & Degenhardt, 2006). The median number of days of use has also remained stable across this time (Dunn, Campbell & Degenhardt, 2006). Participants reported using a median of 2 tablets (range 0.5-20) in a typical use session and a median of 4 tablets (range 1-35) in a heavy use session. The number of tablets used in a typical use session has remained consistent from 2003-2006 (Dunn, Campbell & Degenhardt, 2006); however, 8% reported using four or more tablets in a typical use session.

Ecstasy was primarily taken orally, though other routes of administration were reported: 65% reported recently snorting ecstasy, 6% reported smoking ecstasy and 4% reported using ecstasy intravenously. The prevalence of intravenous use of ecstasy in the past six months has decreased in the past four years (10% in 2003, 5% in 2004, 4% in 2005 and 2006), and remains low in comparison to the intravenous use of other drugs, such as methamphetamine speed (8% in 2006) and crystal methamphetamine (10% in 2006).

Data from the Illicit Drug Reporting System provides information on the use of ecstasy by regular injecting drug users (IDU). In 2006, these surveys found that two-thirds (68%) of regular IDU had ever used ecstasy, while one-quarter (26%) had used ecstasy in the past six months (O'Brien, Black, Degenhardt *et al*, 2007). Of those IDU who had used ecstasy in the last six months, use occurred on a median of 3 days (range 1-180); 12% reported using ecstasy once per week or more in the past six months. Swallowing was the most common route of administration among the IDU surveyed.

Regular ecstasy users are likely to report extended periods of drug taking, sometimes occurring over multiple days (Stafford, Degenhardt, Dunn *et al*, 2006). In the 2006 EDRS, half of the regular ecstasy users surveyed (49%) had "binged" (used continuously without sleep for 48 hours or more) on ecstasy and other drugs. Among this group, 90% had used ecstasy while doing so. Other drugs reported during a "binge" period were alcohol, methamphetamine ("speed" powder and crystal methamphetamine), and cannabis.

Polydrug use

Almost all (93%) of the sample for the 2006 EDRS reported usually using other drugs with ecstasy, most commonly alcohol, cannabis and methamphetamine speed. More than threequarters (80%) reported usually using other drugs when they 'come down' from ecstasy, most commonly alcohol and cannabis (Dunn, Degenhardt, Campbell *et al*, 2007). In the 2001 household survey data, the use of other drugs, particularly amphetamines, cocaine, LSD and cocaine, were the strongest correlates for ecstasy use in young adults (Degenhardt, Barker & Topp, 2004). This finding suggests that ecstasy users are highly likely to be polydrug users, a finding that has also been made in the EDRS.

In 2006, EDRS participants reported having ever used a median of 9 drug types and using a median of 7 drug types in the last six months. Alcohol, cannabis and tobacco were the drugs most likely to have ever been used and to have been used in the past six months. Other drugs such as methamphetamine and ketamine were used by significant proportions of the samples.

Psychopathology associated with ecstasy use

There is little research in Australia investigating the association between mental health problems and ecstasy use, and this remains an area of further research need.

The 2006 EDRS administered the Kessler Psychological Distress Scale (K10) to more than 700 regular ecstasy users. More than one-third (38%) reported being at no or low risk of

psychological distress, half (54%) being at medium risk of psychological distress and 7% reported being at a high risk for psychological distress (Dunn, Degenhardt, Campbell *et al*, 2007).

When asked to identify risks associated with the use of ecstasy, regular ecstasy users report concern about a range of psychological harms, including such problems as dependence and psychosis. Small numbers report accessing services such as counsellors and psychologists for these issues which they attribute specifically to their ecstasy use. What is unclear, however, is the extent to which users perceive these effects as acute and short-term or emerging after longterm use. Users appear to anticipate acute effects during the 'comedown' phase from ecstasy and devise their own methods for coping with these effects.

Other harms associated with ecstasy use

Ecstasy users perceive a range of adverse effects related to ecstasy. These adverse effects include physical harms, such as non-fatal overdose and dehydration; neurological harm, such as memory impairment and damage to brain function; and psychological harms, such as depression and anxiety. Users also displayed concerns regarding unknown long-term effects which may stem from their ecstasy use. Such effects included altering brain structure as well as altering neurotransmitter actions.

A survey of regular ecstasy users was conducted to examine the benefits and risks perceived to be associated with ecstasy and related drugs. Almost all participants perceived risks associated with the use of ecstasy, with the most prevalent perceived risk related to ecstasy use being the use of an illicit substance of unknown content (White *et al*, 2005). Harms related to physical health, including dehydration and non-fatal overdose, were also commonly mentioned (White *et al*, 2005).

Risks which may stem from the drug itself have also been identified by users. Ecstasy users have identified that risks from ecstasy use can include being unaware of the pharmacological make-up of ecstasy tablets, which can include such substances as other illicit drugs (such as amphetamine and ketamine) to caffeine. Furthermore, as previously mentioned, regular ecstasy users have been found to be polydrug users, and these users have identified harms which may stem from their polydrug use, including the combination of taking ecstasy with alcohol.

It is unclear to what extent these risks are perceived as likely to occur to the users themselves.

Ecstasy users report a range of problems associated with their drug use. Similar proportions have nominated ecstasy as the primary drug of concern implicated in their financial (19%), occupational (18%) and social (16%) problems; 2% identified ecstasy as being the primary drug of concern for recent problems concerning law enforcement (Dunn, Degenhardt, Campbell *et al*, 2007).

Financial problems include having no money for recreational activities or luxuries; being in debt and owing money, or having no money for food or rent. Occupational problems include having a lack of motivation, trouble concentrating, and reduced work performance. Social problems include mistrust, arguments, and ending relationships. Law enforcement problems include being arrested and being cautioned by the police (Dunn, Degenhardt, Campbell *et al*, 2007).

An analysis of mortality related to ecstasy during 2000 to 2004 was conducted using data from the National Coronial Information System (NCIS). Over this four year period NCIS identified 112 ecstasy-related deaths (Kinner *et al*, 2005). Ecstasy was deemed to be the primary

contributor in 51 (46%) of these cases; however, MDMA was the sole drug present in only six (5%) of these deaths (Kinner *et al*, 2005).

Some ecstasy users have concerns about their ecstasy use. It is unlikely that there is a classic "dependence syndrome" for MDMA, but the EDRS has documented proportions of regular ecstasy users who endorse questions from the Severity of Dependence Scale that reflect concern about use (Stafford, Degenhardt, Dunn *et al*, 2006). Data from the Australian Minimum Dataset have also suggested that small numbers of clients are presenting for treatment for ecstasy (although many appear to be very brief in duration) (unpublished data).

A minority of regular ecstasy users report injecting drugs. In 2004, 23% of the sample interviewed for the EDRS reported having ever injected a drug and 15% reported injecting in the six months prior to interview (White, etc 2006); only 9% of those who had injected in the past six months reported daily injecting drug use (White *et al*, 2006). Older age, being unemployed and having ever been in prison were significant predictors of having ever injected a drug, while completion of secondary school and identifying as heterosexual was associated with a lower likelihood of having ever injected (White *et al*, 2006).

The majority of those who had injected in the past six months reported obtaining their needles from NSPs (67%) and/or pharmacies (38%). Other sources of needles and syringes included a friend (26%), a dealer (11%), a partner (5%) and vending machines (4%). The proportion reporting needle sharing in the past month was low; this remained constant when examining needle sharing in the past six months (White *et al*, 2006). Half did report sharing other injecting equipment, which places them at increased risk for Hepatitis C infection.

Current trends in ecstasy use

From 1995 to 2004 there was an increase in the population prevalence of lifetime (2% v. 7.5%) and recent (1% v. 3.4%) ecstasy use. Among community samples of ecstasy users the proportion using ecstasy at least weekly has remained stable over the past four years, as has the number of tablets used in a typical session. The prevalence of injecting ecstasy among this group has decreased from 10% in 2003 to 4% in 2006.

Summary of ecstasy use in Australia

Ecstasy has been used by approximately 8% of the population, with approximately 3% having used in the preceding 12 months. Users are typically male, aged in their twenties, and in full-time employment or engaged in full-time study. Users of ecstasy are also more likely to be users of other substances. There is a minority who report injecting ecstasy, though the proportion reporting intravenous ecstasy use is less than that of other drug types.

Patterns of ecstasy use appear to have remained stable over time. Those who use ecstasy appear to identify a range of risks which can result from ecstasy use, and do exhibit concern regarding long-term consequences. While a minority of users report seeking help for such problems as dependence, depression and associated psychological issues which they attribute to their ecstasy use, at present there is a lack of research being conducted in Australia examining the relationship between ecstasy use and mental health. Future research is needed to explore this issue in greater depth.

References

Australian Institute of Health and Welfare (2005). 2004 National Drug Strategy Household Survey: Detailed findings. Canberra: Australian Institute of Heath and Welfare.

Degenhardt, L., Barker, B. & Topp, L. (2004). Patterns of ecstasy use in Australia: findings from a national household survey. *Addiction*, *99*, 187-195.

Dunn, M., Campbell, G. & Degenhardt, L. (2006). An overview of the 2006 EDRS: the regular ecstasy users survey findings. EDRS Drug Trends Bulletin, December 2006. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Dunn, M., Degenhardt, L., Campbell, G., George, J., Johnston, J., Kinner, S., Matthews, A., Newman, J., White, N. (2007). Australian Trends in Ecstasy and Related Drug Markets 2006: Findings from the Ecstasy and Related Drugs Reporting System (EDRS). Sydney: National Drug and Alcohol Research Centre.

Johnston, J., Barrett, M.J., Fry, C.L., Kinner, S., Stoové, M., Degenhardt, L., George, J., Jenkinson, R., Dunn, M. & Bruno, R. (2006). A survey of regular ecstasy users' knowledge and practices around determining pill content and purity: Implications for policy and practice. *International Journal of Drug Policy*, *17*, 464-472.

Kinner, S., Fowler, G., Fischer, J. & Degenhardt, L. (2005). Monitoring the ecstasy market in Australia – Challenges and successes. PDI Drug Trends Bulletin, April 2005. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

O'Brien, S, Black, E., Degenhardt, L., Roxburgh, A, Campbell, G., Fetherston, J., de Graaff, B., Jenkinson, R., Kinner, S., Moon, C. & White, N. (2007). Australian Drug Trends 2006: Findings from the Illicit Drug Reporting System (IDRS). Sydney: National Drug and Alcohol Research Centre.

Quinn, K., Breen, C. & White, B. (2004). Illicit tablet markets in Victoria. PDI Drug Trends Bulletin, June 2004. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Stafford, J., Degenhardt, L., Dunn, M., Fischer, J., George, J., Johnston, J., Matthews, A., Newman, J., Proudfoot, P. & Weekley, J. (2006). Australian trends in ecstasy and related drug markets 2005. NDARC Monograph Number 58. Sydney: National Drug and Alcohol Research Centre

White, B., Day, C., Degenhardt, L., Kinner, S., Fry, C., Bruno, R. & Johnston, J. (2006). Prevalence of injecting drug use and associated risk behaviour among regular ecstasy users in Australia. *Drug and Alcohol Dependence*, *83*, 210-217.

White, B., Degenhardt, C., Breen, C., Bruno, R., Newman, J. & Proudfoot, P. (2005). Risk and benefit perceptions of party drug use. *Addictive Behaviors*, *31*, *3*, 137-142.

White, V. & Hayman, J. (2006). Australian secondary school students' use of over-the-counter and illicit substances in 2005. Canberra, ACT: Australian Government Department of Health and Ageing.

Chapter 7: LSD

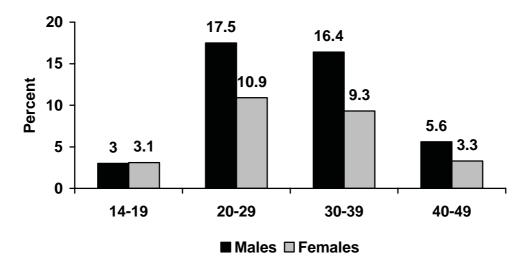
Joanne Ross

Summary of key changes in LSD use over the past decade:

- No information is specifically collected on LSD use in Australia.
- Population based surveys which ask about hallucinogens generally, indicate that the lifetime prevalence of hallucinogen use has fallen from 9.9% in 1998 to 7.5% in 2004. This finding is supported by a reduction in arrests for the consumption and provision of hallucinogens.
- Research describing the characteristics of LSD users, related psychopathology, and other harms associated with LSD use, is severely lacking.

Epidemiology of LSD use

The NDS surveys do not specifically ask about the use of LSD (lysergic acid diethylamide), but they do ask about synthetic and naturally occurring hallucinogens generally. Overall, the lifetime prevalence of hallucinogen use remained fairly stable at 7% from 1985 to 1993, dropping to 6% in 1995, prior to a peak of 9.9% in 1998 (Darke *et al*, 2000). In 2001 the lifetime prevalence dropped back to 7.6% and remained stable at 7.5% in 2004 (AIHW, 2005). The lifetime prevalence of hallucinogen use appears highest in the 20-29 and 30-39 year age groups, with males more likely to have used than females (Figure 7.1). No gender difference appears to exist among the 14-19 year age group.





Use of hallucinogens in the 12 months preceding interview fluctuated between one and two percent from 1988 to 1995, with 3% reporting use in the 1998 NDS survey (Darke *et al*, 2000).

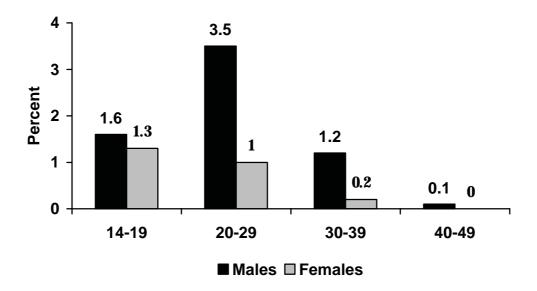
Source: National Drug Strategy Household Survey, 2004

In 2001 the prevalence of recent use dropped to 1.1%, declining further to 0.7% in 2004 (AIHW, 2005). As with lifetime hallucinogen use, the 12 month prevalence was highest among the 20-29 year age group (Figure 7.2). In contrast, however, recent use was less apparent among the 30-39 year age group.

According to the 2005 Australian Secondary Student's Alcohol and Drug Survey (ASSAD), 3% of all secondary school students had ever used hallucinogens, with the proportion increasing significantly with age, from 1% of 12-year olds to 5% of 16 year olds. (White & Hayman, 2006).

The reduction in the population prevalence of hallucinogen use since 1998 is also reflected in the number of arrests for hallucinogen consumption or provision. According to arrest data from the Australian Crime Commission (ACC), in 1998-99 there were 618 arrests relating to hallucinogens compared to 124 in 2003-04 (AIHW, 2005).

Figure 7.2: Prevalence of past year hallucinogen use by age and gender.



Source: National Drug Strategy Household Survey, 2004

The availability of LSD/synthetic hallucinogens was also reported to have decreased slightly, with 2.2% of people aged 14 years and over having been offered or having had the opportunity to use these drugs in 2004 versus 3.4% in 2001 (AIHW, 2005).

Findings from the Ecstasy and Related Drugs Reporting System (EDRS) indicate that, in 2005, 64% of regular ecstasy users had ever used LSD, approximately a third (32%) having done so in the preceding 6 months (Stafford *et al*, 2006). Data from the ERDS, suggests that there was a slight increase in the use of LSD among ecstasy users in 2005 (Stafford, Degenhardt, Dunn *et al*, 2006).

Characteristics of LSD users

The information available regarding the characteristics of LSD users is extremely limited. To the knowledge of the author, no recent studies have specifically examined the characteristics of LSD users in Australia.

According to the 2004 NDS household survey, hallucinogen use is most common among males in the 20-29 year age group, and the average age at which Australians first used hallucinogens was 19.5 years (AIHW, 2005). Males are more likely to have been offered hallucinogens (3.8% v. 2.3%), to have tried these drugs (9.4% v. 5.6%), and to have used them in the 12 months preceding interview (1.1% v. 0.4%). Pooled data from the 1985-1995 NDS household surveys suggested that respondents born in Australia or the British Isles were more likely to report having used hallucinogens than 'NES Europe' or Asia (16% v. 5%) (Makkai & McAllister, 1998).

Reflecting the situation in the general population, findings from the ASSAD (Stafford *et al*, 2006) indicate that hallucinogen use is generally more common among male than female secondary school students (lifetime: 4.3% v. 2.1%; past year: 3.5% v. 1.5%; past month: 2.0% v. 0.7%).

Patterns of LSD use

In examining pooled data from the 1985-1995 NDS household surveys, Makkai and McAllister (1998) report that among those respondents who had ever used LSD, nearly seven out of ten say that they used it more than a year ago, or that they no longer use it. A further 17% report having used it a few times in the preceding year, with only 14% having used it several times a month or more. This pattern of use supports the image of LSD as a drug that is used mainly for recreational purposes, usually in the company of others, and often in large-scale social situations such as parties (Makkai & McAllister, 1998). Infrequent LSD use was also reported by ecstasy users in the EDRS, having been used on a median of two days in the preceding 6 months (Stafford, Degenhardt, Dunn *et al*, 2006). The EDRS sample reported most commonly using LSD at friend's homes, or the users' own home, followed by raves and nightclubs. Among Australian secondary school students infrequent hallucinogen was also the norm, with less than 1% having used these drugs 10 or more times in the preceding year (White & Hayman, 2006).

LSD is usually swallowed, but may also be injected (Moore *et al*, 1992; Stafford *et al*, 2006). LSD had been injected at some stage by 3% of the ecstasy users in the EDRS (Stafford, Degenhardt, Dunn *et al*, 2006), and hallucinogens (including LSD) were reported to have ever been injected by 16% of the IDU in the 2005 IDRS sample (Stafford, Degenhardt, Black *et al*, 2006).

Recent hallucinogen users in the 2004 NDS survey most commonly reported using LSD tabs (72.2%). On a day when hallucinogens were used, 70.5% had one trip only. The overwhelming majority of hallucinogen users at all ages and both genders were relatively infrequent users, with 69% reporting having used hallucinogens only once or twice a year (AIHW, 2005).

Polydrug use

The 2004 NDS survey found that almost three quarters (71.6%) of recent users of hallucinogens had used these drugs in conjunction with alcohol, and 67% had used in conjunction with cannabis (AIHW, 2005). A study of 30 hallucinogen users in Melbourne (Fitzgerald & Hamilton, 1994) also reported that LSD is rarely used alone, with users often co-administering a variety of other psychoactive substances (e.g. cannabis and amphetamines).

Psychopathology associated with LSD use

Psychological disturbances from LSD use occur frequently, including depression, paranoia, and psychosis (Makkai & McAllister, 1998). An American review of several clinical and neuropharmacological reports on LSD (Abraham and Aldridge, 1993) reports an association between LSD use and panic reactions, prolonged schizoaffective psychoses, and post-hallucinogen perceptual disorder, the latter being present for as long as five years.

Other harms associated with LSD use

As with other aspects of LSD use, there is a scarcity of information regarding the harms associated with the use of this drug. An ethnographic study of 30 recreational drug users that was conducted in Perth, Western Australia, makes particular reference to LSD use (Moore *et al*, 1992). Based on observation and interviews with LSD users, the authors describe the problems associated with 'coming down' (i.e. problems in the period immediately following intoxication), and the side effects of the drug. 'Coming down' from LSD is generally associated with lethargy, some degree of depression, an aching body and insomnia. The negative effects of 'coming down' are exacerbated by the use of alcohol and other drugs. Unfortunately, LSD (and MDMA) greatly increases the number of drinks required for a person to experience a subjective feeling of intoxication. The side effects resulting from LSD include 'a bad trip' (i.e. the inability to interact coherently with others and to deal with the sometimes disorienting visual and/or mental hallucinations), 'flashbacks' to the tripping state (such as seeing colours or objects moving – although this seems to be a characteristic of more regular LSD users), paranoia, painful introspection, and generally unpredictable behaviour (Moore *et al*, 1992).

Current trends in LSD use

Given that population based surveys have tended to ask about hallucinogen use in general rather than LSD use specifically, it is not possible to discuss trends in LSD use here. Overall, the lifetime prevalence of hallucinogen use has reduced from a peak of 9.9% in 1998, to 7.5% in 2004. There is also some evidence that the availability of hallucinogens may have decreased since 1998.

Summary of LSD use in Australia

Hallucinogens have been used by 7.5% of the population, with 0.7% having used them in the preceding 12 months. The 'typical' hallucinogen user is a polydrug using male, in his twenties. Hallucinogen use tends to be infrequent, and injectors of LSD are likely to have injected other drugs.

References

Abraham, H.D. & Aldridge, A.M. (1993) Adverse consequences of lysergic acid diethylamide. *Addiction*, *88*, 1327-1334.

Australian Institute of Health and Welfare (2005) 2004 National Drug Strategy Household Survey: First Results. AIHW cat. no. PHE 57. Canberra: AIHW (Drug Statistics Series No. 13).

Darke, S., Ross, J., Hando, J., Hall, W. & Degenhardt, L. (2000) Illicit Drug Use in Australia: Epidemiology, use patterns and associated harms. Monograph series no. 43. Canberra: Commonwealth Department of Health and Aged Care.

Fitzgerald, J. & Hamilton, M. (1994) An exploratory study of hallucinogen use in Melbourne. A report to the Drug Rehabilitation and Research Fund. Melbourne: The University of Melbourne.

Makkai, T. & McAllister, I. (1998). *Patterns of drug use in Australia, 1985–95.* Canberra: Australian Government Publishing Service.

Moore, D., Saunders, B. & Hawks, D. (1992) Recreational drug use, with particular reference to methamphetamines, ecstasy and LSD, amongst a social network of young people in Perth, Western Australia. Perth: Curtin University of Technology.

Stafford, J., Degenhardt, L., Black, E., Bruno, R., Buckingham, K., Fetherston, J., Jenkinson, R., Kinner, S., Newman, J., & Weekley, J. (2005). *Australian Drug Trends 2005: Findings from the Illicit Drug Reporting System (IDRS)*. National Drug and Alcohol Research Centre.

Stafford, J., Degenhardt, L., Dunn, M., Fischer, J., George, J., Johnston, J., Matthews, A., Newman, J., Proudfoot, P., & Weekley, J. (2005). *Australian trends in ecstasy and related drug markets 2005: Findings from the Party Drugs Initiative*. National Drug and Alcohol Research Centre.

White, V. & Hayman, J. (2006). Australian secondary school students' use of over-the-counter and illicit substances in 2005. Canberra, ACT: Australian Government Department of Health and Ageing.

Chapter 8: Cocaine

Sharlene Kaye

Summary of key changes in cocaine use over the past decade:

- The prevalence of lifetime and recent use of cocaine among the general population has remained relatively low and stable at around 4.5% and 1%, respectively.
- The average age of initiation of cocaine use among the general population has increased marginally from 22.3 years in 1998 to 23.5 years in 2004.
- The use and availability of cocaine remains most prevalent in NSW.
- There has been a fluctuation in the prevalence and frequency of cocaine use among IDU in NSW since 1998. A marked increase was observed between 1998 and 2001, at which point the prevalence and frequency of cocaine use was at a peak. Following the 2001 peak in use, there was an overall decline in use until 2004. Increases in cocaine use were reported in 2005 and 2006, although not to the levels seen in 2001.
- Nationally, the prevalence of cocaine use among IDU decreased substantially between 2001 and 2004, increased in 2005, again not to the levels seen in 2001, and remained relatively stable in 2006. The frequency of cocaine use in Australian jurisdictions other than NSW has remained low and sporadic.
- The prevalence of recent cocaine use among regular ecstasy users across Australia increased between 2003 and 2005, and then decreased slightly in 2006. In NSW, a similar trend was observed.
- The availability of cocaine remained stable between 2002 and 2005 and was generally regarded by IDU in NSW as "easy" to "very easy" to obtain.
- Between 1993 and 2005, the number of cocaine-related hospital separations fluctuated, with sharp increases in 1998/99, 2001/02 and 2004/05, the 2004/05 increase due primarily to an increase in cocaine dependence separations.
- There has been an overall decrease in the number of mentions of cocaine in accidental drug-induced deaths in Australia between 1998 and 2004.
- Recent research has provided a better understanding of the demographic characteristics and drug use patterns of cocaine users in Australia and of the physical and psychological morbidity associated with cocaine use.
- Studies of cocaine-related fatalities have provided a better understanding of the nature of death due to cocaine toxicity, particularly with respect to the demographics, toxicology and underlying organ pathology of decedents.

Epidemiology of cocaine use

The 2004 NDS household survey (Australian Institute of Health and Welfare [AIHW], 2005) revealed that 4.7% of persons reported lifetime use of cocaine. This proportion is similar to those reported in the 2001 (4.4%) and 1998 (4.3%) NDS household surveys (Adhikari & Summerill, 2000; AIHW, 2002), and slightly higher than the 3% reported consistently in the NDS surveys conducted between 1985 and 1995 (Makkai & McAllister, 1998).

Males in 2004 (5.8%) were generally more likely to report lifetime use than females (3.7%) (AIHW, 2005). These figures were similar to those reported in the 2001 (5.3% of males vs 3.5%

of females) and 1998 (5.3% of males vs 3.3% of females) surveys (Adhikari & Summerill, 2000; AIHW, 2002) and slightly higher than those reported in the 1985-1995 NDS household surveys (4% of males vs 2% of females) (Makkai & McAllister, 1998). Although males in 2004 were generally more likely to report lifetime use than females, the reverse was true in the 14-19 year age group (Figure 8.1).

Lifetime use in 2004 was most prevalent among those aged 20-39 years (Figure 8.1), with similar proportions of the 20–29 and 30-39 year age groups (8.9% and 8.8%, respectively) reporting such use. These proportions were higher than those among the 14-19 (1.9%) and 40+ (2.6%) year age groups.

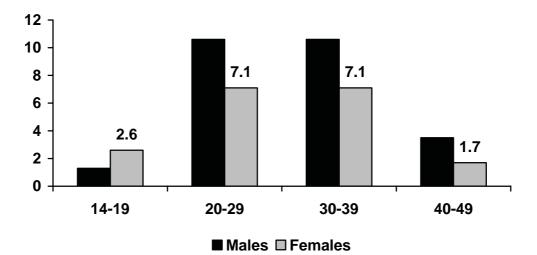


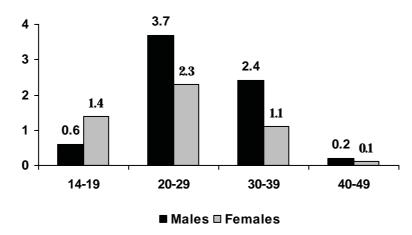
Figure 8.1: Prevalence of lifetime cocaine use by age and gender.

Source: National Drug Strategy Household Survey, 2004

The 2004 NDS household survey revealed that 1% of the population had used cocaine in the previous 12 months, a finding consistent with those of previous NDS household surveys (Adhikari & Summerill, 2000; AIHW, 2002; Makkai & McAllister, 1998). Recent use (i.e. in the last 12 months) in 2004 was slightly more common among males (1.3% of males vs 0.8% of females) (AIHW, 2005), these figures being similar to those reported in the 2001 (1.6% of males vs 1.0% of females) and 1998 (1.9% of males vs 0.9% of females) NDS household surveys (Adhikari & Summerill, 2000; AIHW, 2002), as well as those in the 1985-1995 NDS household surveys (1% of males vs 1% of females) (Makkai & McAllister, 1998). Although males were generally more likely to report recent use than females, females were more likely than males to report recent use in the 14-19 year age group (Figure 8.2).

Cocaine use in the past 12 months was most common among those aged 20–29 years (Figure 8.2), with 3% of this age group reporting such use. Recent cocaine use was reported by 1% of those aged 14-19 years, 1.8% of the 30-39 year age group, and 0.2% of those aged 40+ years.

Figure 8.2: Prevalence of past year cocaine use by age and gender

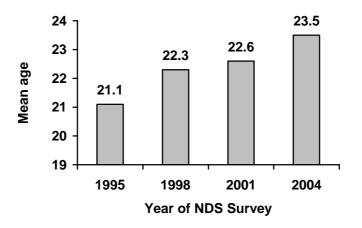


Source: National Drug Strategy Household Survey, 2004

Characteristics of cocaine users

The 2004 NDS household survey found that the average age at which cocaine was used for the first time was 23.5 years, a slight increase from the age of initiation reported in previous NDS household surveys (Figure 8.3). Overall, the average ages of first use of cocaine reported in the NDS household surveys are consistent with those of previous studies of Australian cocaine users, where the average age of initiation is 21-23 years (Kaye & Darke, 2004a,b).

Figure 8.3: Age of initiation of lifetime cocaine use, 1995-2004



Cross-sectional studies of Australian current cocaine users have consistently reported a mean age of 28-30 years, with males generally comprising two thirds of the sample (Kaye & Darke, 2002; Kaye & Darke, 2004a,b; Williamson *et al.*, 2006). The 2004 NDS household survey revealed that males were more likely than females to have been offered cocaine or had the opportunity to use cocaine in the preceding 12 months (3.6% males vs 2.6% females). This finding is consistent with those of the 2001 NDS household survey (4.3% males vs 2.6% females) and previous NDS household surveys (males 8% vs females 5%) (Makkai & McAllister, 1998)

A study conducted for the World Health Organisation during 1993-1994 identified two distinct groups of cocaine users in Sydney – a group with a higher socio-economic status (SES) who typically administered the drug intra-nasally (snorted), and another with a lower SES who

injected cocaine (Hando *et al.*, 1997). The lower SES users were aged 14–45 years (with the majority in their late teens and 20s), were more likely to be unemployed or, in the case of females, working in the sex industry, had lower levels of education, were more criminally active, and more likely to be enrolled in a methadone maintenance program. The preferred drug of this group was reported to be either heroin or cocaine. The higher SES group identified by Hando and colleagues (1997) were aged 17–50 years (with the majority in their 20s and 30s), had regular or above average incomes, came from a range of educational backgrounds and occupations, and their preferred drug was reported to be either cocaine or alcohol. It should be borne in mind that this study is based on a small sample size (29 key informant interviews in Sydney and eight in Melbourne), and the sampling frame was restricted to the inner city in both cities.

Subsequent studies of injecting and non-injecting cocaine users across the Sydney metropolitan area have also found higher levels of unemployment and lower levels of education among cocaine injectors than among non-injectors, with the former group also more likely to be male, have a prison history and be enrolled in a methadone maintenance program (Kaye & Darke, 2004a,b). A recent study of the Australian cocaine mark*et also* identified two distinct types of cocaine users (Shearer *et al.*, 2005). The first group was a high SES group of cocaine users who typically snorted cocaine, and usually did so in conjunction with other licit and illicit drugs, such as alcohol and ecstasy. The second group was of lower SES who injected cocaine alone or in combination with heroin or benzodiazepines and used more frequently than the high SES users (Shearer *et al.*, 2005).

Pooled data from the 1998-1995 NDS household surveys indicate that those who are unemployed are more likely than manual and non-manual workers to have been offered cocaine (14% vs 8% and 7%, respectively), to have ever used it (8% vs 4%), and to have used it in the preceding 12 months (3% v. 1%) (Makkai & McAllister, 1998). A study of cocaine users in Sydney and Melbourne, which was designed to recruit a broad spectrum of users, found that approximately two-thirds of cocaine users were in full-time, part-time, casual or self employment (Shearer *et al.*, 2005). In studies examining cocaine use among illicit drug users in Sydney, however, approximately 60% of cocaine users were unemployed (Kaye & Darke, 2002; Kaye & Darke, 2004a,b), although levels of unemployment were typically much higher among cocaine injectors (85%) than among non-injectors (8-23%).

Patterns of cocaine use

According to the 2004 NDS household survey, of those who had used cocaine in the preceding 12 months, 15.4 % had used it at least once a month, 21.9% had used cocaine every few months, with the majority (62.7%) using cocaine only once or twice a year. The use of cocaine once a month or more in the previous 12 months was most common among 14-19 (21%) and 20-29 (20%) year olds. Those aged 30-39 years of age were most likely to have only used once or twice in the preceding year (75.8%). The majority of recent users (93.7%) survey in the 2004 NDS household survey had used cocaine intra-nasally ("snorted"), with 7.6% reporting that they had injected cocaine.

Among the national sample of injecting drug users (IDU) surveyed for the Illicit Drug Reporting System (IDRS) in 2006, the median days of cocaine use in the preceding six months was 5.5 days (Black *et al.*, 2006). Cocaine users in NSW reported the highest frequency of use, with a median of 20 days of use during that period. Among regular ecstasy users (REU) surveyed for the Ecstasy and Related Drugs Reporting System (EDRS) who had recently used cocaine, a median of 2 days of use was reported (Dunn *et al.*, 2006).

Hando and colleagues (1997) found that the patterns of cocaine use in their study differed according to SES. Cocaine users in the lower SES group typically injected cocaine, as well as other substances, such as heroin, and used more heavily than those in the higher SES group. More recent studies of cocaine users have found similar differences in use patterns, with lower SES users being more likely to inject cocaine and use more frequently than higher SES users (Kaye & Darke, 2004a,b; Shearer *et al.*, 2005).

The form of cocaine used by respondents in the 2004 NDS household survey was overwhelmingly powder (98.9%), with 12.1% reporting the use of crack cocaine. This finding is consistent with cocaine use among IDU and REU who primarily use cocaine in the powdered form and reflects the limited availability of crack in Australia (Stafford *et al.*, 2006a).

Polydrug use

The majority of cocaine users use other drugs in conjunction with cocaine. The 2004 NDS household survey indicates that alcohol is the most common substance to be used with cocaine, with 84.4% of recent cocaine users reporting concurrent use of alcohol with cocaine. Ecstasy and cannabis use in conjunction with cocaine use was reported by approximately half of the recent cocaine users. The type of polydrug use associated with cocaine use varies according to the type of cocaine user. Injecting cocaine users, who are often existing heroin users, typically use heroin, cannabis, benzodiazepines and alcohol (Kaye & Darke, 2004a,b; Shearer *et al.*, 2005). Cocaine injectors often combine heroin with cocaine, with the resultant mixture referred to as a 'CC (cocaine cocktail)' or 'speedball'. The rapid sequential injection of heroin and cocaine is also a common practice among IDU (Darke *et al.*, 2002). Non-injecting cocaine users typically use ecstasy, methamphetamine, cannabis and alcohol with cocaine (Kaye & Darke, 2004a,b; Shearer *et al.*, 2004a,b; Shearer *et al.*, 2005).

Psychopathology associated with cocaine use

The psychological effects of cocaine vary according to dose, route of administration, the context in which the drug is taken, and individual characteristics and experience (American Psychiatric Association [APA], 2000; Johanson, 1986). Although cocaine can produce feelings of pleasure or euphoria, it can also produce aversive psychological symptoms, such as anxiety, panic, dysphoria, severe depression, paranoia, hallucinations, delirium, and violent agitation, particularly in high doses and during withdrawal (APA, 2000; Platt, 1997).

Binge use of cocaine typically induces such symptoms and can lead to a state known as "cocaine psychosis". Cocaine psychosis is essentially a schizophreniform paranoid psychosis where the user experiences paranoid delusions, mania, delirium, and hallucinations. Paranoid psychosis induced by cocaine is typically transient and limited to the episode of use, as opposed to the more persistent psychosis induced by methamphetamine. With continued use of cocaine, however, paranoia can become more intense and develop a more rapid onset (Satel *et al.*, 1991). It is difficult to predict who will develop cocaine-induced psychosis as it is not directly related to dose or route of administration. It would appear that some users are more susceptible to develop psychosis than others (Karch, 2002; Satel *et al.*, 1991).

Psychological morbidity has been demonstrated among cocaine users in Australia, with dependent users, injectors in particular, most likely to report psychological symptoms, typically anxiety, depression and paranoia (Hando *et al.*, 1997; Kaye & Darke, 2004b; van Beek *et al.*, 2001; Williamson *et al.*, 2006). Injecting cocaine use has been associated with a greater risk of psychological symptomatology than either intranasal use (Hando *et al.*, 1997; Kaye & Darke, 2004b) or smoking (Lexau *et al.*, 1998). In turn, smoking cocaine is associated with a higher prevalence of psychological symptoms than intranasal use (Ferri & Gossop, 1999; Washton & Gold, 1987).

Psychiatric comorbidity among dependent cocaine users is well-documented (Majewska, 1996; Nnadi *et al.*, 2005; Platt, 1997). Chronic cocaine use is often associated with problems such as anxiety, paranoia, and affective disorders, such as depression and bipolar mood disorder. Such comorbid conditions may increase the risk of suicide and trigger craving and relapse, thereby complicating the treatment for cocaine dependence and worsening the prognosis for recovery (APA, 2000; Majewska, 1996; Nnadi *et al.*, 2005; Platt, 1997). Whether comorbid psychiatric disorders are an antecedent to, or a consequence of, cocaine use has been the subject of much debate. It is thought that cocaine may be used to self-medicate premorbid symptoms of anxiety or depression. Among cocaine users with an anxiety disorder, the anxiety is often found to precede regular cocaine use (Halikas *et al.*, 1994; Rounsaville *et al.*, 1991). Alternatively, cocaine use may induce symptoms of anxiety and depression, particularly during withdrawal. It is likely that cocaine may both precipitate and be a consequence of these disorders (Majewska, 1996; Platt, 1997).

Other harms associated with the use of cocaine

Physical harms

Many of the physical harms associated with cocaine use are due to its vasoconstrictive effects, which may manifest in several of the body's physiological systems. Consequently, cocaine has the potential to cause a wide range of problems including: cardiovascular (e.g. chest pain, cardiac arrhythmias, myocardial infarction and ischemia, and cardiomyopathy); neurological (e.g. cerebrovascular accidents, i.e. strokes, seizures, and headaches); gastrointestinal (e.g. abdominal pain, vomiting, colitis, and bowel infarction); and respiratory symptoms (e.g. exacerbation of asthma, rapid and/or irregular breathing, pulmonary oedema, and bronchitis). Cocaine can also cause obstetric complications, such as irregularities in placental blood flow, premature labour, and low neonate birth weights (Platt, 1997; Warner, 1993).

Some of the adverse physical effects of cocaine are specific to the route of administration (Cregler & Mark, 1986; Yeager *et al.*, 1987). The intranasal use of cocaine is associated with a number of nasal symptoms, such as nasal congestion, rhinitis, bleeding, ulceration and perforation of the nasal septum, as well as a loss of the sense of smell. Injecting cocaine use is associated with the problems related to injecting drug use *per se*, such as vascular damage, abscesses, bacterial infections (e.g. endocarditis). Due to the higher frequency of injecting that is associated with cocaine use, however, injection-related problems are more prevalent among cocaine injectors than among injectors of other illicit drugs, such as heroin (Chaisson *et al.*, 1989; Schoenbaum *et al.*, 1989; Bux *et al.*, 1995; Darke, *et al.*, 2002; van Beek *et al.*, 2001). Indeed, injecting cocaine users in Australia report more vascular damage, abscesses, and infections than other IDU (Darke *et al.*, 2002; van Beek *et al.*, 2001). Overall, injecting cocaine use has been associated with a greater number and severity of hams than the intranasal use of cocaine. Cocaine injectors (Hando *et al.*, 1997; Kaye & Darke, 2004a,b; Shearer *et al.*, 2005).

Two of the most serious consequences of cocaine use are cardiovascular and cerebrovascular complications. These can occur regardless of administration route (Cregler, 1994; Egred & Davis, 2005). Chest pains, palpitations and seizures are among the most common complaints among cocaine users presenting to emergency departments in the US (Derlet & Albertson, 1989) and cardiovascular complications account for the majority of cocaine-related deaths (Advisory Council on the Misuse of Drugs, 2000). In Australia, palpitations are among the most common physical side effect reported by cocaine users (van Beek *et al.*, 2001; Kaye & Darke, 2004b).

Cocaine is a cardiotoxic drug. Chronic cocaine use is associated with the accelerated and premature development of coronary artery disease, which increases the risk of acute myocardial infarction. Underlying coronary artery disease is well-documented among both patients with cocaine-associated myocardial infarction and cocaine-related fatalities (Wilson, 1998; Lange & Hillis, 2001; Karch, 2002; Kontos *et al.*, 2003). A recent fatality study found a higher prevalence and greater severity of coronary artery disease among cocaine-related fatalities than among opioid-related fatalities and non-drug related fatalities (Darke *et al.*, 2006). Chronic cocaine use has also been associated with ventricular hypertrophy (enlargement of the ventricle wall), a condition that can increase the risk of myocardial infarction and/or arrhythmia (Benzaquen *et al.*, 2001; Karch, 2002; Darke *et al.*, 2005).

Although cocaine-induced strokes may be less common than seizures, their prevalence has increased in the US since the early 1980s as cocaine use has become more prevalent (Klonoff *et al.*, 1989). A study assessing the risk of stroke associated with cocaine use found a 14-fold increase in risk among cocaine users compared to matched controls (Pettiti *et al.*, 1998).

The above complications are potentially fatal and can occur among young, healthy users without any previous risk factors (Platt, 1997; Vasica & Tennant, 2002). Moreover, there is no specific combination of conditions under which such outcomes can be predicted. They can occur with small amounts of cocaine and on the first occasion of use (Cregler, 1994). While higher doses and a greater frequency of use increase the likelihood of adverse reactions to cocaine, the thresholds over which complications occur can vary widely between individuals (Platt, 1997). Thus, while toxic reactions to cocaine are usually dose-related, they are not dose-specific (Sbriglio & Millman, 1987).

Blood borne viruses

In the United States, injecting cocaine use has been associated with higher levels of needlesharing, increased sexual risk-taking, and a higher HIV seroprevalence than injecting heroin use (Bux *et al.*, 1995; Chaisson *et al.*, 1989; Schoenbaum *et al.*, 1989). Studies conducted in Europe (Torrens *et al.*, 1991) and Australia (Darke *et al.*, 1992) have also revealed an association between cocaine injecting and higher levels of HIV risk-taking behaviours.

Dependence

Due to its powerful reinforcing effects, cocaine is regarded as having a high dependence liability (Jacobs & Fehr, 1987). Cocaine dependence can develop after a relatively short history of use and can be associated with a range of use patterns (APA, 2000; Dackis & O'Brien, 2001). Problematic use may be chronic, such that cocaine is used on a daily, or almost daily, basis, or it may be episodic, where the pattern may be to binge on cocaine over a period of hours or days and then abstain for several days. An early indicator of cocaine dependence is when the user finds it increasingly difficult to resist using cocaine if it is available (APA, 2000). Smoking and injecting cocaine is associated with a greater dependence liability than intranasal use, with use often progressing to dependence in a matter of weeks or months (Chen & Kandel, 2002; Gossop *et al.*, 1994; Hatsukami & Fischman, 1996). The onset of dependence as a consequence of intranasal use is much more gradual, typically taking months or even years (APA, 2000).

While much is now known about the underlying mechanisms of cocaine dependence, there are still no pharmacological treatments that have been deemed effective in promoting abstinence from cocaine use (Lima *et al.*, 2002). Previous research has suggested that a combination of psychotherapy and pharmacotherapy may be more effective in the treatment of cocaine dependence than either approach on its own (Carroll, 1993).

Hospital admissions

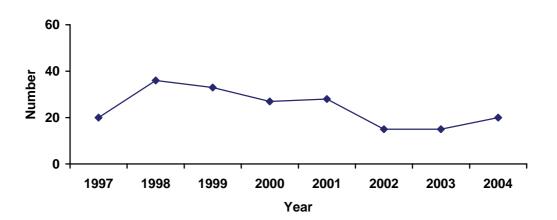
An indicator of cocaine-related harm is the number of hospital separations (i.e. episode of care for an admitted patient), where cocaine is the principal reason for the hospital stay. Data from the National Hospital Morbidity Database reveal that, between 1993 and 2005, the number of cocaine-related hospital separations fluctuated, with sharp increases in 1998/99, 2001/02 and 2004/05, the 2004/05 increase due primarily to an increase in cocaine dependence separations (Roxburgh & Degenhardt, 2006). The rise in cocaine-related separations in 2001/02 was consistent with the increase in cocaine use during the heroin shortage and increased user reports of problems associated with cocaine use (Roxburgh *et al.*, 2004; Stafford *et al.*, 2006b). The increase in hospital separations observed in 2004/05 is in accordance with IDU reports of an increase in daily use and a greater availability of cocaine during this period (Black *et al.*, 2006). Cocaine-related separations between 1993 and 2005 were lower than those related to other illicit drugs, i.e. opioids, amphetamines and cannabis, and did not exceed 300 per year. The majority of cocaine-related separations during this period occurred in NSW (Roxburgh & Degenhardt, 2006).

Mortality

Deaths due to cocaine toxicity typically occur among male, experienced drug users who are, on average, in their mid-30s. Cocaine-related fatalities often involve multiple drugs, most commonly heroin and alcohol (Darke *et al.*, 2007). Deaths due to cocaine toxicity are due primarily to cardiovascular complications, such as myocardial ischaemia and infarction and cardiac arrhythmias (Darke *et al.*, 2007; Karch, 2002). The risk of these events occurring is increased in the presence of underlying coronary artery disease and cardiac hypertrophy, conditions which have been associated with the chronic use of cocaine. Cerebrovascular accidents (strokes), cerebral haemorrhage in particular, are also a well-recognised cause of cocaine-related death (Darke *et al.*, 2007; Karch, 2002).

According to data from the Australian Bureau of Statistics, there were 20 accidental druginduced deaths in Australia in 2004 where cocaine was mentioned as either the primary cause of death or noted in "toxic quantities" where another drug was the primary cause of death (Figure 8.4; Degenhardt *et al.*, 2006).

Figure 8.4: Total mentions of cocaine in accidental drug-induced deaths in Australia: 1997-2004.



Current trends in cocaine use

Between 1985 and 1995, recent cocaine use remained stable at around 1% (Makkai & McAllister, 1998), increasing slightly to 1.4% in 1998 and 1.3% in 2001, and decreasing again to 1% in 2004. While the NDS household surveys indicate that the prevalence of cocaine use among the general population is relatively low and stable, changes in cocaine use have been particularly apparent among injecting drug users, heroin users in particular, in Sydney. The use of cocaine is most prevalent in NSW. Accordingly, the availability of cocaine is greater in NSW than in other Australian jurisdictions.

The IDRS detected an increase in the use of cocaine in the preceding six months among IDU in NSW from 34% in 1997 to 84% in 2001. Following the peak of use in 2001, there was a decline in use in NSW to 47% in 2004 and then an increase to 60% in 2005 and 67% in 2006. Much of the increase in cocaine use in NSW was reported among heroin users and the sharp increase in 2001 was attributed to the reduction of the availability of heroin in late 2000/early 2001 (Roxburgh *et al.*, 2004). Nationally, recent cocaine use by IDU decreased from 35% in 2001 to 16% in 2004, increased to 22% in 2005, and remained relatively stable in 2006 at 20%. In NSW, the frequency of cocaine use in the preceding six months decreased from 90 days in 2001 to 6 days in 2004. An increase in frequency was observed in 2005, with the number of days used doubling from 6 in 2004 to 12 in 2005. In 2006, frequency of cocaine use increased further to 20 days in the preceding six months. In other Australian jurisdictions, the frequency of recent cocaine use in 2006 remained low and sporadic, as it has been in previous years (Black *et al.*, 2006).

The availability of cocaine remained stable between 2002 and 2005 with the majority of cocaine using IDU in NSW reporting that cocaine was "easy" to "very easy" to obtain. Approximately two-thirds of NSW IDRS participants in 2005 regarded the purity of cocaine as medium to low and that purity had remained stable over the preceding six months. The price of a "cap" of cocaine in NSW remained stable at \$50.

The prevalence of recent cocaine use among regular ecstasy users across Australia increased between 2003 and 2005 (2003: 24%; 2004: 27%; 2005: 41%), and then decreased slightly in 2006 (37%). In NSW, a similar trend was observed, with recent use reported by 46% of REU in 2003, 46% in 2004, 55% in 2005, and 45% in 2006 (Dunn *et al.*, 2006).

Summary of cocaine use in Australia

Population based surveys in indicate that cocaine has been used by approximately 5% of the general population, with 1% having used in the preceding 12 months. Lifetime and recent cocaine use is more prevalent among males and among those aged 20-39 years. The overwhelming majority of cocaine users use the powdered form of the drug and, whilst there is a small minority who report the use of crack, there is little evidence that there is a significant crack market in Australia. Among the general population, cocaine is typically snorted. Cocaine injecting is more frequent among existing IDU. Cocaine injectors are more likely to have higher levels of unemployment, lower levels of education, and higher levels of dependence. Cocaine injecting is also associated with a greater number of health problems related to injecting, a greater risk of contracting blood-borne viruses due to increased injecting and sexual risk behaviours, and higher rates of psychopathology. Whilst cocaine use among the general population has remained low and stable, the recent increase in the prevalence and frequency of cocaine use among IDU in NSW indicates the need for continued monitoring, as is done annually by the IDRS, in order to detect any further increases in use.

References

Adhikari, P. & Summerill, A. (2000). *1998 National Drug Strategy Household Survey: Detailed findings*. AIHW cat. no. PHE 27. Canberra: AIHW (Drug Statistics Series No. 6).

Advisory Council on the Misuse of Drugs. (2000). *Reducing Drug Related Deaths*. A report by the Advisory Council on the Misuse of Drugs. London: The Stationery Office.

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. Text Revision). Washington, D.C.: American Psychiatric Association.

Australian Institute of Health and Welfare. (2002). 2001 National Drug Strategy Household Survey: detailed findings. AIHW cat. no. PHE 41. Canberra: AIHW (Drug Statistics Series No.11).

Australian Institute of Health and Welfare. (2005). 2004 National Drug Strategy Household Survey: Detailed Findings. AIHW cat. no. PHE 66. Canberra: AIHW (Drug Statistics Series No.16).

Benzaquen, B.S., Cohen, V., & Eisenberg, M.J. (2001). Effects of cocaine on the coronary arteries. *American Heart Journal*, 142, 402-410.

Black, E., O'Brien, S., Campbell, G., & Degenhardt, L. (2006). *An overview of the 2006 IDRS: the injecting drug user survey findings. IDRS Drug Trends Bulletin, December 2006.* Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Bux, D.A., Lamb, R.J., & Iguchi, M.Y. (1995). Cocaine use and HIV risk behaviour in methadone maintenance patients. *Drug and Alcohol Dependence*, *37*, 29-35.

Carroll, K.M. (1993). Psychotherapeutic treatment of cocaine abuse: models for its evaluation alone and in combination with pharmacotherapy. In F.M. Timms & C.G. Leukefeld (Eds.), *Cocaine Treatment: Research and Clinical Perspectives*. NIDA Research Monograph, Number 135. Rockville, Maryland: Department of Health and Human Services. National Institute on Drug Abuse.

Chaisson, R.E., Bacchetti, P., Osmond, D., Brodie, B., Sande, M.A., & Moss, A.R. (1989). Cocaine use and HIV infection in intravenous drug users in San Francisco. *The Journal of the American Medical Association*, 261, 561-565.

Chen, K. & Kandel, D. (2002). Relationship between extent of cocaine use and dependence among adolescents and adults in the United States. *Drug and Alcohol Dependence*, *68*, 65-85.

Cregler, L.L. (1994). Cocaine: a risk factor for cardiovascular disease in women. *Cardiovascular Risk Factors*, *4*, 39-44.

Cregler, L.L. & Mark, H. (1986). Special report. Medical complications of cocaine abuse. *The New England Journal of Medicine*, *315*, 1495-1500.

Dackis, C.A. & O'Brien, C.P. (2001). Cocaine dependence: a disease of the brain's reward centers. *Journal of Substance Abuse Treatment*, 21, 111-117.

Darke, S., Baker, A., Dixon, J., Wodak, A., & Heather, N. (1992). Drug use and HIV risk-taking behaviour among clients in methadone maintenance treatment. *Drug and Alcohol Dependence*, *29*, 263-268.

Darke, S., Degenhardt, L., & Mattick, R. (2007). *Mortality amongst illicit drug users*. *Epidemiology, causes and intervention*. Cambridge: Cambridge University Press.

Darke, S., Kaye, S., & Duflou, J. (2006) Comparative cardiac pathology among deaths due to cocaine toxicity, opioid toxicity and non-drug related causes. *Addiction*, *101*, 1771-1777.

Darke, S., Kaye, S. & Topp, L. (2002) Cocaine use in New South Wales, Australia, 1996-2000: 5 year monitoring of trends in price, purity, availability and use from the Illicit Drug Reporting System (IDRS). *Drug and Alcohol Dependence*, *67*, *81-88*.

Degenhardt, L., Roxburgh, A., Black, E., & Dunn, M. (2006). 2004 Cocaine and amphetamine related drug-induced deaths in Australia. Sydney: National Drug and Alcohol Research Centre.

Derlet, R.W. & Albertson, T.E. (1989). Emergency department presentation of cocaine intoxication. *Annals of Emergency Medicine*, 18, 182-186.

Dunn, M., Campbell, G., & Degenhardt, L. (2006). An overview of the 2006 EDRS: the regular ecstasy user survey findings. EDRS Drug Trends Bulletin, December 2006. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Egred, M. & Davis, G.K. (2005). Cocaine and the heart. *Postgraduate Medical Journal*, *81*, 568-571.

Ferri, C.P. & Gossop, M. (1999). Route of cocaine administration. Patterns of use and problems among a Brazilian sample. *Addictive Behaviours*, 24, 815-821.

Gossop, M., Griffiths, P., Powis, B., & Strang, J. (1994). Cocaine: patterns of use, route of administration, and severity of dependence. *British Journal of Psychiatry*, 164, 660-664.

Halikas, J.A., Crosby, R.D., Pearson, V.L., Nugent, S.M., & Carlson, G.A. (1994). Psychiatric comorbidity in treatment-seeking cocaine abusers. *American Journal on Addictions*, *3*, 25-35.

Hando, J., Flaherty, B. & Rutter, S. (1997) An Australian profile on the use of cocaine. *Addiction*, 92, 173–182.

Hatsukami, D.K. & Fischman, M.W. (1996). Crack cocaine and cocaine hydrochloride: are the differences myth or reality? *The Journal of the American Medical Association*, 276, 1580-1588.

Jacobs, M.R. & Fehr, K.O'B. (1987). *Drugs and Drug Abuse: A Reference Text* (2nd ed.). Toronto: Alcoholism and Drug Addiction Research Foundation.

Johanson, C. (1986). Cocaine. The New Epidemic. New York: Chelsea House Publishers.

Karch, S.B. (2002). Karch's Pathology of Drug Abuse (3rd edition). Boca Raton: CRC Press.

Kaye, S. & Darke, S. (2002) Determining a diagnostic cut-off on the Severity of Dependence Scale (SDS) for cocaine dependence. *Addiction*, *97*, 727-731.

Kaye, S. & Darke, S. (2004a) Non-fatal cocaine overdose among injecting and non-injecting cocaine users in Sydney, Australia. *Addiction*, *99*, 1315-1322.

Kaye, S. & Darke, S. (2004b) Injecting and non-injecting cocaine use in Australia: physical and psychological morbidity. *Drug and Alcohol Review*, 23, 391-398.

Klonoff, D.C., Andrews, B.C, & Obana, W.G. (1989). Stroke associated with cocaine use. *Archives of Neurology*, *46*, 989-993.

Kontos, M.C., Jesse, R.L., Tatum, J.L., & Ornato, J.P. (2003). Coronary angiographic findings in patients with cocaine-associated chest pain. *The Journal of Emergency Medicine*, *24*, 9-13.

Lange, R.A. & Hillis, L.D. (2001). Medical progress: cardiovascular complications of cocaine use. *The New England Journal of Medicine*, *345*, 351-358.

Lexau, B.J., Nelson, D., & Hatsukami, D.K. (1998). Comparing IV and non-IV cocaine users: characteristics of a sample of cocaine users seeking to participate in research. *The American Journal of Addictions*, 7, 262-271.

Lima, M.S., Soares, B.G.O., Reisser, A.A.P., & Farrell, M. (2002). Pharmacological treatment of cocaine dependence: a systematic review. *Addiction*, *97*, 931-949.

Majewska, M.D. (Ed). (1996). *Neurotoxicity and neuropathology associated with cocaine abuse*. NIDA Research Monograph 163, Rockville, US Department of Health and Human Services.

Makkai, T. & McAllister, I. (1998). *Patterns of drug use in Australia, 1985–95.* Canberra: Australian Government Publishing Service.

Nnadi, C.U., Mimiko, O.A., McCurtis, H.L., & Cadet, J.L. (2005). Neuropsychiatric effects of cocaine use disorders. *Journal of the National Medical Association*, 97, 1504-1515.

Pettiti, D.B., Sidney, S., Quesenberry, C. & Bernstein, A. (1998). Stroke and cocaine or amphetamine use. *Epidemiology*, *9*, 596-600.

Platt, J.J. (1997) *Cocaine Addiction : Theory, Research and Treatment*. Cambridge: Harvard University Press.

Rounsaville, B.J., Anton, S.F., Carroll, K., Budde, D., Prusoff, B.A., & Gawin, F. (1991). Psychiatric diagnoses of treatment-seeking cocaine abusers. *Archives of General Psychiatry*, 48, 43-51.

Roxburgh, A. & Degenhardt, L. (2006). *Drug-related hospital stays in Australia, 1993-2005*. Sydney: National Drug and Alcohol Research Centre.

Roxburgh, A., Degenhardt, L., & Breen, C. (2004). Changes in patterns of drug use among injecting drug users following changes in the availability of heroin in New South Wales, Australia. *Drug and Alcohol Review*, 23, 287-294.

Satel, S.L., Southwick, S.M., & Gawin, F.H. (1991). Clinical features of cocaine-induced paranoia. *American Journal of Psychiatry*, 148, 495-498.

Sbriglio, R. & Millman, R.B. (1987). Emergency treatment of acute cocaine reactions. In A.M. Washton & M.S. Gold (Eds.) *Cocaine. A Clinician's Handbook* (pp. 87-95). New York: The Guildford Press.

Schoenbaum, E.E., Hartel, D., Selwyn, P.A., Klein, R.S., Davenny, K., Rogers, M., Feiner, C., & Friedland, G. (1989). Risk factors for human immunodeficiency virus infection in intravenous drug users. *The New England Journal of Medicine*, *321*, 874-879.

Shearer, J., Johnston, J., Kaye, S., Dillon, P., Collins, L. (2005). Characteristics and dynamics of cocaine supply and demand in Sydney and Melbourne. *National Drug Law Enforcement Research Fund Monograph Series No. 14*.

Stafford, J., Degenhardt, L., Dunn, M., Fischer, J., George, J., Johnston, J., Matthews, A., Newman, J., Proudfoot, P., & Weekley, J. (2006). *Australian trends in ecstasy and related drug markets 2005: findings from the Party Drugs Initiative (PDI)*. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Stafford, J., Degenhardt, L., Black, E., Bruno, R., Buckingham, K., Fetherston, J., Jenkinson, R., Kinner, S., Newman, J. and Weekley, J. (2006) *Australian Drug Trends 2005: Findings from the Illicit Drug Reporting System (IDRS)*. NDARC Monograph No. 59. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Torrens, M., San, L., Peri, J.M., & Olle, J.M. (1991). Cocaine abuse among heroin addicts in Spain. *Drug and Alcohol Dependence*, 27, 29-34.

van Beek, I., Dwyer, R., & Malcolm, A. (2001). Cocaine injecting: the sharp end of drug-related harm! *Drug and Alcohol Review*, 20, 333-342.

Vasica, G. & Tennant, C.C. (2002). Cocaine use and cardiovascular complications. *Medical Journal of Australia*, 177, 260-262.

Warner, E.A. (1993). Cocaine abuse. Annals of Internal Medicine, 119, 226-235.

Washton, A.M. & Gold, M.S. (1987). Recent trends in cocaine abuse as seen from the "800-cocaine" hotline. In A.M. Washton & M.S. Gold (Eds.) *Cocaine. A Clinician's Handbook* (pp.10-22). New York: The Guildford Press.

Williamson, A., Darke, S., Ross, J., & Teesson, M. (2006). The association between cocaine use and short-term outcomes for the treatment of heroin dependence: Findings from the Australian Treatment Outcome Study (ATOS). *Drug and Alcohol Review 25*, 141-148.

Wilson, L.D. (1998). Rapid progression of coronary artery disease in the setting of chronic cocaine abuse. *The Journal of Emergency Medicine*, 16, 631-634.

Yeager, R.A., Hobson, R.W. 2nd., Padberg, F.T., Lynch, T.G., & Chakravarty, M. (1987). Vascular complications related to drug abuse. *Journal of Trauma-Injury Infection & Critical Care*, 27, 305-308.

Chapter 9: Heroin

Joanne Ross

Summary of key changes in heroin use over the past decade:

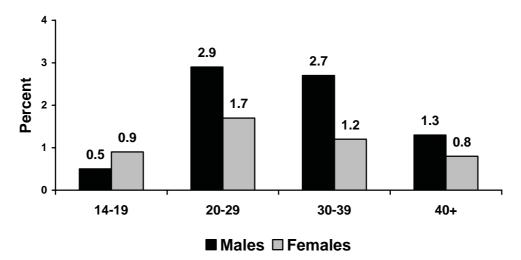
- Early in 2001 the availability of heroin was suddenly reduced, accompanied by an increase in the cost of heroin and objective declines in its purity.
- These changes were sustained throughout 2001, and monitoring systems suggest that while the availability of heroin has increased since 2001, it has not returned to pre 2001 levels.
- Prior to the 2001 heroin shortage, increases were seen in rates of heroin use, and a decline in age of initiation among successive cohorts of people born in Australia was noted. This was particularly evident in the late 1990's.
- Since the 2001 heroin shortage the number of regular heroin users in Australia has reduced dramatically, and opioid deaths have declined from 938 in 2000 to 357 in 2004.
- The greatest reduction in heroin use and opioid fatalities is evident among the younger age groups.
- It is estimated that the number of regular heroin users decreased from 10 per 1000 persons aged 15-54 in 1998, to 4.5 and 4.0 per 1000 in 2001 and 2002, respectively.
- According to the 2004 NDS household survey, recent heroin use was reported by 0.2% of the population aged 14 years and older. This is equivalent to the population prevalence reported back in 1993.

Epidemiology of heroin use

The 2004 NDS Household Survey found that 1.4% of Australians aged 14 years or older had used heroin in their lifetime. This is similar to the seven previous NDS household surveys (1985, 1987, 1991, 1993, 1995, 1998, 2001), which have consistently found the lifetime population prevalence of heroin use to be in the range of 1.0-2.2% (Makkai and McAllister, 1998; Australian Institute of Health and Welfare, 2003; 2005). Heroin had been used in the preceding 12 months by an estimated 0.2% of Australians aged 14 years or older, again falling within the range reported by earlier surveys (lowest: 0.2% in 1993 and 2001, highest: 0.8% in 1998).

Overall males appear more likely to have ever used heroin (1.8% v. 1.0% of females), but this does not appear to be the case among the 14-19 year age group (Figure 9.1). Lifetime heroin use was most common among the 20-29 year age group, with males being almost twice as likely as females in this age category to have ever used heroin (2.9 v. 1.7).

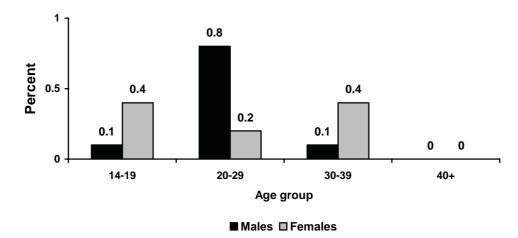
Figure 9.1: Prevalence of lifetime heroin use by age and gender.



Source: National Drug Strategy Household Survey, 2004

According to the 2004 NDS Household Survey, the prevalence of recent (past 12 month) heroin use was 0.2%. Estimates of recent heroin use among the population have ranged from a minimum of 0.2% in 1993, 2001 and 2004, to 0.8% in 1998 (AIHW 2004). Care should be taken in interpreting the prevalence estimates of current heroin use, due to the small numbers upon which the estimates are based. Nonetheless, an examination of age and gender patterns is presented below (Figure 9.2). The 20-29 year age group had the highest proportion and number of recent heroin users (0.5%, 12,900).While similar proportions of males and females had recently used heroin, in terms of numbers, there were more male (14,800) than female (11,100) recent heroin users. Recent heroin use by females and males remained stable from 2001 to 2004, but there were approximately 12,000 fewer recent heroin users in 2004 when compared with 2001 (AIHW, 2005).

Figure 9.2: Prevalence of 12-month heroin use by age and gender.



Source: National Drug Strategy Household Survey, 2004

In 2000, Hall and colleagues estimated the number of dependent heroin users in Australia, by applying three different methods of estimation (back-projection, capture-recapture, and multiplier) to data on national opioid overdose deaths in Australia, first time entrants to methadone maintenance treatment, and heroin-related arrests in New South Wales. The best estimates of the number of dependent heroin users in Australia in 1997-1998 from the three methods of estimation were between 67,000 and 92,000, with a median estimate of 74,000. This represents a population prevalence of 6.9 per 1000 adults aged 15-54 years.

In more recent years there have been significant changes in the heroin market in Australia. Early in 2001, there were anecdotal reports of a sharp reduction in the availability of heroin. These reports were confirmed by timely research using convenience sampling (Day, Topp *et al*, 2003; Weatherburn *et al*, 2001), and in monitoring systems that had documented the increasing heroin availability during the previous five years (Darke *et al*, 2002; Topp *et al*, 2002). As a result of the reduced heroin availability, Degenhardt and colleagues (2004) sought to estimate the number of current regular heroin users for the period 1997 to 2002, by applying a variety of estimation methods to different data sources. They estimated that there were 7.4 regular heroin users per 1000 persons aged 15-54 in 1997, 10 per 1000 in 1998 and 1999, 9.0 per 1000 in 2000, and that this reduced dramatically to 4.5 and 4.0 per 1000 in 2001 and 2002, respectively. These estimates suggest that the number of regular heroin users decreased following the reduction in the heroin supply, and that this decrease was maintained in 2002.

All estimates of the prevalence of heroin use should be treated with caution, due to the illicit nature of heroin use and the stigma attached to admission of its use. Estimates are, in all probability, conservative. Broadly speaking, however, the 2004 NDS household survey indicates that approximately 1.4% of the adult population have ever used heroin, and 0.2% have done so in the last 12 months.

Characteristics of heroin users

Australian studies have consistently reported a ratio of male to female heroin users of approximately 2:1 (ANAIDUS, 1991; Darke & Ross, 1997; Darke *et al*, 2002; Stafford *et al*, 2006). In 2005 the IDRS (Stafford *et al*, 2006) recruited a national sample of 943 injecting drug users (IDU), of whom 64% were male, with a relatively small range from Victoria (60%) to the Northern Territory (71%). While this was a study of IDU, heroin had been used by the majority (91%) of the sample. Studies that have specifically examined heroin users have reported similar results (Darke & Ross, 1997; Day *et al*, 2003).

Prior to the heroin shortage, there was evidence that the age of initiation to heroin use was falling. Increases were seen in rates of heroin use, and a decline in age of initiation among successive cohorts of people born in Australia was noted (Degenhardt *et al.*, 2000; Lynskey & Hall, 1998). Across Australian birth cohorts from the 1940s to 1970s, it has been estimated that there was a 4-year reduction (from 20.5 to 16.5 years) in the mean age of initiation to heroin use and injection (Lynskey and Hall, 1998). Earlier initiation to heroin use has been associated with greater polydrug use, accidental overdose and criminal behaviour, independent of the length of heroin use career (Lynskey & Hall, 1998). A study of heroin dependent persons found that users aged 18-24 years had progressed twice as fast to heavier heroin use and heroin treatment than older users (Mills *et al*, 2004). Rates of comorbid mental disorders were extremely prevalent among this younger group (Mills *et al*, 2004).

The 2004 NDS Household Survey (AIHW, 2005) indicates that the mean age at which Australians first used heroin was 21.2 years, with age of initiation having remained fairly stable between 1995 and 2004. There is evidence, however, that the heroin shortage resulted in a

reduction in the number of new heroin injectors in Australia. Data collected from regular IDU over the period 1996-2004 indicates that the proportion of people interviewed in the IDRS who were aged ≤ 24 years decreased from 46% in 1996 to 12% in 2004, with the most marked drop in 2001, the year in which there was an abrupt and marked reduction in heroin availability (Day *et al*, 2006). Of those who first reported injecting between 1993 and 2000, similar proportions reported heroin and amphetamine as the first drug injected. After 2000, methamphetamine was the drug most often reported as being the first injected.

The mean age of heroin users in cross-sectional samples has typically been in the early thirties, with ages ranging from the late teens to late fifties (Day *et al*, 2003; Ross *et al*, 2005; Stafford *et al*, 2006; Stafford *et al*, 2005). While no recent studies of heroin use have examined the age of heroin users according to gender, the IDRS indicates that female injecting drug users are approximately 3.8 years younger than there male counterparts (Breen *et al*, 2005). As mentioned above, the vast majority of the IDRS IDU samples report a lifetime history of heroin use.

The overwhelming majority of heroin users surveyed in Australia have been unemployed (Darke & Ross, 1997; Darke *et al*, 2002; Ross *et al*, 2005; Stafford *et al*, 2006). In a study of heroin users entering treatment (Ross *et al*, 2005), only 18% obtained their main source of income for the preceding month from a wage or salary. Heroin–related fatalities are also overwhelming unemployed at the time of death (Darke & Zador, 1996; Darke *et al*, 2000).

Patterns of heroin use

Among the general population a notable proportion (45.0%) of recent heroin and/or illicit methadone users reported using these drugs weekly or more frequently (AIHW, 2005). The highest rate of weekly or daily use was found among those aged 30-39 years (61.1%). Heroin rock had been used by 83.5% of recent heroin users, while heroin powder had been used by 67.8%. Heroin had been injected by 86.8% of recent users and smoked by 64.6% On a day when heroin had been used, the majority of recent heroin users had one or two injections, with an overall average of 1.9. Of recent heroin users only 10.3% had not used another drug in conjunction with heroin at some stage in the preceding 12 months. Marijuana/cannabis had been used in conjunction with heroin by 66.4% of recent heroin users, and alcohol by 55.8%.

Throughout the late 1990s Australia's illicit heroin markets experienced a rapid expansion, during which time heroin was readily available, the price steadily decreased, purity increased (Topp *et al*, 2003), and heroin was the drug most frequently injected by IDU nationally (MacDonald *et al*, 2001). As discussed earlier, a dramatic shift in the heroin market occurred early in 2001, when the availability of heroin was suddenly reduced, accompanied by an increase in the cost of heroin and objective declines in its purity (Topp *et al*, 2002). These changes were sustained throughout 2001 in all jurisdictions with a viable heroin market, and monitoring systems suggest that while the availability of heroin has increased since 2001, it has not returned to pre 2001 levels (Stafford *et al*, 2006).

The IDRS gives an indication of the patterns of heroin use among regular IDU. In 2005, 24% of the national IDRS IDU sample reported daily heroin use, but this varied according to jurisdiction. In 2000 the proportion of daily heroin users was similar (47-49%) across the three major heroin markets (NSW, VIC and the ACT). In the last four years, however, the proportions reporting daily use have been considerably reduced in jurisdictions other than NSW (Stafford *et al*, 2006). While the proportion reporting daily use in NSW was 42% in 2005 (Black *et al*, 2006), the median number of days on which heroin was used in the six months preceding interview decreased dramatically among NSW IDU over the preceding three years from 170

days (i.e. almost daily use in 2003) to 120 days in 2004 (approximately five days a week), and again in 2005 to 96 days (i.e. use on approximately every second day).

Given that the IDRS sample was selected because they injected drugs, it is not surprising that injection is the primary route of administration used for heroin, with only 7% having smoked heroin in the preceding 6 months. It is noteworthy, however, that 47% of IDU had smoked heroin at some stage, a phenomena that was first reported in Australia in the 1990s. Darke *et al* (2002) indicate that from 1996 to 2000 between 17 and 24% of the IDRS IDU sample in each year reported having smoked in the preceding 6 months. Smoking of heroin (sometimes referred to as 'chasing the dragon'), appeared to emerge first in the Indo-Chinese community in Sydney, and spread from there to younger Caucasian heroin users (Darke *et al*, 2004). Following the heroin shortage in 2001, however, the 6-month prevalence of heroin smoking reduced to 9% in 2001 and 2002, 6% and 7% in 2003 and 2004, respectively.

Polydrug use

Polydrug use is the norm among heroin users, and illicit drug users in general (Darke *et al*, 2003; McGregor *et al*, 1998; Stafford *et al*, 2006). In a specific study of polydrug use, heroin users had used an average of 8.5 different drug classes in their lives, and 5.2 in the preceding six months (Darke & Hall, 1995). Among a sample of 825 heroin users, 90% of whom were entering treatment for heroin dependence, a mean of 9.1 drug classes had ever been used and 5.0 had been used in the preceding month (Ross *et al*, 2005). Large proportions of Australian heroin users also meet criteria for dependence on other drugs, including alcohol, meth/amphetamines and benzodiazepines (Darke & Ross, 1997; Ross *et al*, 1996, 1997).

Benzodiazepine use among heroin users has been associated with benzodiazepine dependence, greater polydrug use, an increased risk of heroin overdose, injection-related risk-taking behaviour, and injection-related health problems (Darke et al, 2002; Gutierrez-Cedollada et al, 1994; Klee et al, 1990; Ross et al, 1997; Ross et al, 2000; Breen et al, 2004). Due to increasing concern about the negative health effects associated with the injection of temazepam capsules in particular, the 10mg formulation required an 'Authority prescription' (i.e. prior approval from the Health Insurance Commission) from May 1st 2002. Temazepam 10mg tablets remained unrestricted on the Pharmaceutical Benefits Scheme (PBS), and temazepam 20mg capsules remained available without authority as a non-PBS item (i.e. they could still be prescribed by any doctor and purchased without subsidy). Breen and colleagues examined the effect of the restricting the prescription of the 10mg temazepam capsules by interviewing IDU about their use of benzodiazepines before and after the policy change, and by examining PBS prescription data. While there was a decrease in temazepam 10mg capsule prescriptions after the policy change, IDU survey data suggested that IDUs continued to inject benzodiazepines, including temazepam capsules. The frequency of the injection of capsules appeared similar to that before the policy change. In response to continued concerns, gel capsules were subsequently removed from the market at the end of March 2004. The 2005 IDRS then reported a reduction in the number of IDU reporting the recent injection of benzodiazepines in all jurisdictions except the NT. NSW reported the greatest drop from 13% in 2004 to 2% in 2005. As a result of public health measures within the state, Victoria had experienced substantial decreases in benzodiazepine injecting since 2001, reducing from 40% in 2001, to 6% in 2005. The injection of benzodiazepines is still an issue of particular concern in the NT and Tasmania, where 21% and 23% respectively had recently injected benzodiazepines (Stafford et al, 2006).

Of further concern is the widespread injection of methadone syrup that has been documented in Australia (Darke *et al*, 1996; Darke *et al*, 2002; Humeniuk *et al*, 2003; Stafford *et al*, 2006). The injection of methadone syrup has been associated with abscesses and infections at injection sites, venous thrombosis, poorer general health, higher levels of psychiatric distress, and a history of

heroin overdose (Darke *et al*, 1996). According to the 2005 IDRS 26% of the national IDU sample had injected methadone in the preceding six months (Stafford *et al*, 2006).

Psychopathology associated with heroin use

Heroin users have high rates of psychiatric morbidity, with the most common diagnoses being mood disorders, anxiety disorders, and Anti-Social Personality Disorder (ASPD) (Darke & Ross, 1997; Darke *et al*, 2005; Mills *et al*, 2005; Swift *et al*, 1990; Teesson *et al*, 2005). In addition, high rates of post traumatic stress disorder (PTSD) and borderline personality disorder (BPD) have been reported among dependent heroin users (Trull *et al*, 2000; Villagomez *et al*, 1995).

Darke and Ross (1997) examined polydrug dependence and psychiatric comorbidity among heroin injectors, and revealed high levels of polydrug dependence and lifetime anxiety and mood disorders that did not vary by treatment status or gender. Overall, the more psychologically distressed participants were, the more different types of drugs they were dependent on. Two thirds of participants met the criteria for a lifetime, and 55% for a current diagnosis of anxiety and/or depressive disorder. The Australian Treatment Outcome Study (ATOS) reported high degrees of psychiatric comorbidity among entrants to treatment for heroin dependence (Ross *et al*, 2005). Almost half (49%) of the ATOS cohort had severe psychological distress, 28% had current major depression, 37% having attempted suicide in their lifetime, and 42% having a lifetime history of PTSD. Personality disorders were also prevalent, with 72% meeting criteria for ASPD, and 47% screening positive for BPD.

Studies have consistently shown that between a quarter and a third of heroin users meet the criteria for a lifetime diagnosis of major depression, a prevalence many times greater than that seen in the general population (Dinwiddie et al, 1992; Darke & Ross, 1997; 2002). Depression as a risk factor for suicide has particular relevance for heroin users. It has been repeatedly demonstrated that opiate users with a history of attempted suicide have a higher prevalence major depression and/or higher levels of depression as measured on instruments such as the BDI Murphy et al, 1983; Kosten & Rounsaville, 1988; Best et al, 2000; Darke & Ross, 2001). The higher levels of depression seen among those with a history of suicide appear to persist after an attempt has been made (Darke & Ross, 2001) The completed suicide rate among heroin users is estimated to at 14 times that of the general population (Harris & Barraclough, 1997). Attempted suicide also occurs at a rate well in excess of the general population, as does suicidal ideation (Darke & Ross, 2002). The major general population risk factors for suicide also apply to heroin users (gender, psychopathology, family dysfunction and isolation), but heroin users have extremely wide exposure to these factors. They also carry additional risks specifically associated with heroin and other drug use, with drugs as a method of suicide playing a larger role in suicide among heroin users than in the general population (Darke & Ross, 2002).

The prevalence of ASPD among methadone maintenance patients has been specifically examined by Darke and colleagues (1994; 1998). Darke *et al*, (1994) reported a prevalence of ASPD of 61%. In a comparative study of prison and community patients, the rates were 44% for community MM patients, and 65% among prison patients (Darke *et al*, 1998). It should not be assumed that because a heroin user meets the criteria for ASPD that they are "psychopathic". While rates of ASPD are high, reflecting high levels of crime, rates of psychopathology are low (Darke *et al*, 1998). A recent paper from the Australian Treatment Outcome Study (Darke *et al*, 2004) examined the relationship between ASPD, BPD and harm among heroin users. While initial analyses suggested an increased risk among ASPD patients for suicide and psychopathology, these relationships disappeared after BPD was taken into account, suggesting that, from a clinical perspective, screening for BPD among heroin users would be of far greater

utility than assessing ASPD. BPD is clinically related to a broader range of harms (Darke *et al*, 2004).

Despite international epidemiological evidence suggesting that people with heroin dependence may be at increased risk of PTSD (Clark *et al*, 2001; Hien *et al*, 2000; Milby *et al*, 1996; Villagomez *et al*, 1995), this comorbid disorder has only recently received research attention in Australia. According to a study based on the Australian National Survey of Mental Health and Well-Being, the prevalence of PTSD among individuals with an opioid disorder is 33.2% (Mills *et al*, 2006). Among the ATOS cohort trauma exposure (92%) and lifetime PTSD (41%) were also found to be highly prevalent (Mills *et al*, 2005). PTSD was found to impact negatively on the ATOS cohort's treatment outcomes over a 24 month follow-up period (Mills *et al*, in press). Despite improvements in substance use, PTSD was associated with continued physical and mental disability, and reduced occupational functioning throughout the 2 year follow-up.

Other harms associated with heroin use

Overdose

The major harm associated with heroin use in Australia is the risk of fatal and non-fatal overdose (Darke & Zador, 1996; Warner-Smith *et al*, 2001). In the 1990s there were substantial rises in the number of deaths related to heroin overdose (Hall *et al*, 1999). The overdose rate (per million adults aged 15 to 44 years) increased 56-fold from approximately 1.3 in 1964 to 71.5 in 1997 (Hall *et al*, 2000). Degenhardt and colleagues (2005) indicate that following the 2001 reduction in the heroin supply, NSW ambulance callouts to suspected heroin overdoses reduced from 302.7 per month to 111.6 per month, representing an overall decrease of 63%. Similarly, data on suspected drug-related deaths between January 1995 and June 2003 showed a 43% decrease in the number of drug-related deaths after the reduction in the supply of heroin (Degenhardt *et al*, 2005). Decreases in heroin overdoses were of a similar magnitude for men and women, but there were bigger decreases among younger age groups and no detectable change among those aged 45 years and older. As the authors suggest, this may indicate that older users remained in the heroin market, whereas younger users may have ceased or reduced their use.

According to the 2004 Australian Bureau of Statistics (ABS) data on opioid overdose deaths (Degenhardt, Roxburgh *et al*, 2006), there has been a stabilisation in the number of opioid-related deaths. In 2004 there were 357 deaths in which opioids were determined to be the underlying cause of death (i.e. the primary factor responsible for the person's death) among those aged 15-54 years (Degenhardt, Roxburgh *et al*, 2006). The rate of accidental deaths due to opioids was 31.3 per million persons aged 15 to 54 years. This rate is effectively unchanged compared to 2003 (31.5 per million). With regards to non-fatal overdose, the 2005 IDRS found that, of those IDU that reported heroin use in the six months preceding interview, 58% had ever overdosed, and 2% had done so in the last month (Stafford *et al*, 2006). The proportion of IDU reporting heroin overdose in the last year fell from 31% in 2000, to 13% in 2005.

The 'typical' fatal heroin overdose case has been described as a long-term opiate dependent male in his early 30s, who is not in treatment for heroin dependence, and has consumed other drugs in combination with heroin, primarily alcohol and/or benzodiazepines (Darke, Ross *et al*, 2000; Darke *et al*, 2007). Rather than novice users, it is older heroin users who constitute the bulk of heroin overdose deaths. A feasible explanation offered by Darke and colleagues (2007) is that the rigours of the heroin lifestyle may become more difficult to maintain after a decade or so of use. Consequently, older heroin users may cut down on their use, and increase their use of other drugs, such as alcohol and benzodiazepines, to compensate for their reduced heroin use (Darke *et al*, 2007). Darke and colleagues (2007) indicate that this would be consistent with the low blood morphine concentrations detected in many fatal overdose cases, which may reflect less frequent use, and correspondingly lower tolerance to opioids, among older heroin users.

Blood borne viruses

Blood borne viruses (primarily HIV and the hepatitises) represent a major harm associated with the injection of heroin and other drugs. Approximately, 80% of people with hepatitis C virus (HCV) have a history of injecting drug use, and 61% of those who use needle and syringe programmes are HCV positive (Day, Ross *et al*, 2003; National Centre in HIV Epidemiology and Clinical Research, 2006). In contrast, the national HIV prevalence among IDU remains low (less than 2%). High levels of the hepatitis B infection have been reported among cohorts of IDU (Kaldor *et al*, 1996; Crofts *et al*, 1997), and the uptake of the HBV vaccine among this group appears poor (Day *et al*, 2003). Despite the risks involved, the 2005 IDRS indicates that 11% of the national IDU sample reported having used a needle after someone else in the past month, and 17% reported that someone else had used a needle after them in that time (Stafford, 2006).

Dependence

Heroin dependence is one of the major health risks associated with heroin use. In terms of clinical importance and public health, it is long-term dependent users who represent the primary target group. Heroin dependence is distinguished from heroin use by a number of behavioural characteristics. The Fourth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association, 2000) defines opioid dependence as opioid use accompanied by signs and symptoms of compulsive prolonged self-administration. These signs include tolerance, withdrawal, using in larger amounts or for a longer time than intended, a persistent desire or unsuccessful efforts to control use, and continued use despite recognition of adverse effects. The Australian National Household Survey of Mental Health and Well-Being estimated the population prevalence of current (past year) opioid dependence in 1997 to be 0.2% (Andrews *et al*, 1999).

Large scale community surveys of mental disorders in the USA, such as the National Comorbidity Survey (Warner *et al*, 1995), indicate that approximately 25 percent of those who report ever having used heroin go on to develop dependence on the drug. The estimated one in four heroin users who become dependent on heroin typically report a one to two year period between their first use of heroin and their first period of sustained daily use. The development of heroin dependence is likely to require daily heroin use over several weeks or months (Hall *et al*, 1999).

While some have argued that heroin injecting is associated with more severe dependence than heroin smoking (Gossop *et al*, 1992), others have found no difference between heroin smokers and injectors (Swift *et al*, 1997; Stohler *et al*, 2000). A novel finding from the Australian Treatment Outcome Study was that nearly 1 in 10 of the heroin users entering treatment for dependence were heroin smokers. The majority of these had never injected heroin, yet had developed severe enough dependence to seek treatment (Darke, Hetherington, *et al*, 2004).

Hospital admissions

Hospital admissions for opioid use have reduced from 6833 in 2000-01 to 4076 in 2004-05 (McKetin, 2006), reflecting the reduction in heroin use since the 2001 shortage. Despite reduced heroin use, this drug still accounts for 34% of treatment episodes in Australia. In addition to drug treatment centre presentations (23,193 in 2004-05), there were over 36,000 clients registered in opioid pharmacotherapy (methadone or buprenorphine) in 2004 (McKetin, 2006). This reflects an ongoing increase in opioid pharmacotherapy enrolments over the past decade (Stafford *et al.*, 2006).

Current trends in heroin use

Subsequent to the heroin shortage of 2001, there was a notable reduction in the number of regular heroin users in Australia (Degenhardt *et al*, 2004). Opioid deaths also declined markedly from 938 in 2000 to 386 in 2001, to 364 in 2002 and 357 in 2003 and 2004 (Degenhardt, Roxburgh *et al*, 2006). As described above, hospital admissions for opioid use were also reduced. The 15-24 year age group appears to have benefited most from the heroin shortage, with a 26% reduction in new enrolments for opioid pharmacotherapy, a 49% reduction in heroin possession/use offences and a 65% decline in heroin–related deaths (Degenhardt, Day *et al*, 2005). While heroin availability, price, purity, prevalence of use and frequency of use have not returned to pre-shortage levels, the 2005 IDRS reported that 83% of those IDU who felt able to comment on heroin availability indicated that it was easy or very easy to obtain (Stafford *et al*, 2006).

Summary of heroin use in Australia

Approximately 1.4% of the population have tried heroin, and 0.2% in the preceding 12 months. A number of data sources indicate that heroin market has stabilised since the shortage in 2001, but that it has not returned to pre-shortage levels. The "typical" heroin user is an unemployed male in his twenties or thirties, who injects heroin and uses a variety of drugs in addition to heroin. While the age of onset of heroin use had declined prior to the heroin shortage, the effect of the shortage on age of initiation is yet to be examined. Importantly, the shortage has resulted in a much needed reduction in opioid related deaths.

References

American Psychiatric Association (2000) Diagnostic and statistical manual of mental disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association.

ANAIDUS (1991) Neither a borrower nor a lender be: First report of the Australian National AIDS and Injecting Drug Study. Sydney: ANAIDUS.

Andrews, G., Hall, W., Teesson, M. & Henderson, S. (1999) The Mental Health of Australians. National survey of mental health and well-being. Report 2. Canberra: Mental Health Branch, Commonwealth Department of Health and Family Services.

Australian Institute of Health and Welfare –AIHW (2003) Statistics on drug use in Australia 2002. AIHW cat. no. PHE 43. Canberra: AIHW (Drug statistics series no. 12).

Australian Institute of Health and Welfare –AIHW (2005) Statistics on drug use in Australia 2004. AIHW cat. no. PHE 62. Canberra: AIHW (Drug statistics series no. 15).

Breen, C., Degenhardt, L., Bruno, R.B., Roxburgh, A.D. & Jenkinson, R. (2004) The effects of restricting publicly subsidised temazepam capsules on benzodiazepine use among injecting drug users in Australia. *MJA*, *181*, 300-304.

Breen, C., Roxburgh, A. & Degenhardt, L. (2005) Gender differences among regular injecting drug users in Sydney, Australia 1996-2003. *Drug & Alcohol Review, 24, 353-358.*

Clark, H.W., Masson, C.L., Delucchi, K.L., Hall, S.M. & Sees, K.L.(2001) Violent traumatic events and drug abuse severity. *Journal of Substance Abuse Treatment*, 20, 121-127.

Darke, S., Degenhardt, L., & Mattick, R. (2007). *Mortality amongst illicit drug users*. *Epidemiology, causes and intervention*. Cambridge: Cambridge University Press.

Darke, S., Hall, W. & Swift, W. (1994) Prevalence, symptoms and correlates of anti-social personality disorder among methadone maintenance clients. *Drug & Alcohol Dependence, 34*, 253-257.

Darke, S., Hetherington, K., Ross, J., Lynskey, M. & Teesson, M. (2004) Non-injecting routes of administration among entrants to three treatment modalities for heroin dependence. *Drug & Alcohol Review, 23,* 177-183.

Darke, S., Kaye, S. & Finlay-Jones, R. (1998) Antisocial personality disorder, psychopathy and injecting drug use. *Drug & Alcohol Dependence*, *52*, 63-69.

Darke, S. & Ross, J. (1997) Polydrug dependence and psychiatric comorbidity among heroin injectors. *Drug & Alcohol Dependence*, 48, 135-141.

Darke, S., Ross, J., Teesson, M., Lynskey, M. (2004). Attempted suicide among entrants to three treatment modalities for heroin dependence in the Australian Treatment Outcome Study (ATOS): prevalence and risk factors. *Drug and Alcohol Dependence* 73, 1-10.

Darke S. Ross J. Zador D. Sunjic S. (2000) Heroin-related deaths in New South Wales, Australia, 1992-1996. *Drug & Alcohol Dependence*. 60(2):141-50.

Darke, S., Topp, L., Kaye, S. & Hall, W. (2002) Heroin use in New South Wales, Australia, 1996-2000: 5 year monitoring of trends in price, purity, availability and use from the Illicit Drug Reporting System (IDRS). *Addiction*, *97*, 179-186.

Darke, S., Topp, L. & Ross, J. (2002) The injection of methadone and benzodiazepines among Sydney injecting drug users 1996-2000: 5-year monitoring of trends from the Illicit Drug Reporting System. *Drug & Alcohol Review*, *21*, 27-32.

Darke, S., Williamson, A., Ross, J., Teesson, M. & Lynskey, M. (2004) Borderline personality disorder, antisocial personality disorder and risk-taking among heroin users: findings from the Australian Treatment Outcome Study (ATOS). *Drug & Alcohol Dependence*, *74*, 77-83.

Darke, S. & Zador, D. (1996) Fatal heroin "overdose": a review. Addiction, 91, 1765-1772.

Day, C., Degenhardt, L.& Hall, W. (2006) Changes in the initiation of heroin use after a reduction in heroin supply. *Drug & Alcohol Review*, 25, 307-313.

Day, C., Ross, J. & Dolan, K. (2003) Hepatitis C- related discrimination among heroin users in Sydney: drug user or hepatitis C discrimination? *Drug & Alcohol Review*, 22, 317-321.

Day, C., Topp, L., Rouen, D., Darke, S., Hall, W. & Dolan, K. (2003) Decreased heroin availability in Sydney in early 2001. *Addiction, 98,* 93-95.

Degenhardt, L., Conroy, E., Gilmour, S. & Hall, W.D. (2005) The effect of a reduction in heroin supply on fatal and non-fatal drug overdoses in New South Wales, Australia. *Medical Journal of Australia*, 182, 20-23.

Degenhardt, L., Lynskey, M. & Hall, W. (2000) Cohort trends in the age of initiation of drug use in Australia. *Australian & New Zealand Journal of Public Health*, 24, 421-426.

Degenhardt, L., Rendle, V., Hall, W., Gilmour, S. & Law, M. (2004) Estimating the number of current regular heroin users in NSW and Australia 1997-2002. NDARC Technical Report No.198. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Degenhardt, L., Roxburgh, A., Black, E. & Dunn, M. (2006) Accidental drug-induced deaths due to opioids in Australia, 2004. Sydney: National Drug and Alcohol Research Centre.

Dinwiddie, S.H., Reich, T. & Cloninger, C.R. (1992) Psychiatric comorbidity and suicidality among intravenous drug users. *Journal of Clinical Psychiatry*, 53, 364-369.

Gossop, M. Griffiths, P., Powis, B. & Strang, J. (1992) Severity of dependence and route of administration of heroin, cocaine and amphetamines. *British Journal of Addiction*, 87, 1527-1536.

Gutierrez-Cedollada, J., De La Torre, R., Ortuno, J *et al* (1994) Psychotropic drug consumption and other factors associated with heroin overdose. *Drug & Alcohol Dependence*, *35*, 169-174.

Hall, W., Degenhardt, L. & Lynskey, M. (1999) Opioid overdose mortality in Australia, 1964-1997: birth-cohort trends. *Medical Journal of Australia, 171,* 34-37.

Hall, W., Lynskey, M. & Degenhardt, L. (1999) Heroin use in Australia. Its impact on public health and public order. NDARC Monograph No. 42. Sydney: National Drug and Alcohol Research Centre.

Hall, W.D., Ross, J.E., Lynskey, M.T., Law, M.G. & Degenhardt, L.J. (2000). How many dependent heroin users are there in Australia? *Medical Journal of Australia*, 173, 528-531.

Hall, W.D., Ross, J.E., Lynskey, M.T., Law, M.G. & Degenhardt, L.J. (2000). How many dependent heroin users are there in Australia? NDARC Monograph No. 44. Sydney: National Drug and Alcohol research Centre, University of New South Wales.

Hien, D.A., Nunes, E., Levin, F.R. & Fraser, D. (2000). Post-traumatic stress disorder and short-term outcome in early methadone maintenance treatment. *Journal of Substance Abuse Treatment*, *19*, 31-37.

Humeniuk, R., Ali, R., McGregor, C. & Darke, S. (2003) Prevalence and correlates of intravenous methadone syrup administration in Adelaide, Australia. *Addiction*, *98*, 413-418.

Klee, H., Fluagier, J., Hayes, C., *et al.* AIDS-related risk-behaviour, polydrug use and temazepam. *British Journal of Addiction*, 85, 1125-1132.

Lynskey, M.T., Hall, W. (1998). Cohort trends in age of initiation to heroin use. *Drug & Alcohol Review*, *17*, 289-297.

Makkai, T. & McAllister, I. (1998) Patterns of drug use in Australia, 1985-95. Canberra: Australian Government Publishing Service.

McKetin, R. (2006) Patterns and Trends in Drug Use in Australia. In: 2006 Asian Multi-city Epidemiology Work Group (Meeting Proceedings), November 7-10, 2006, Taipei. In press.

Milby, J.B., Sims, M.K., Khuder, S., Schumacher, J.E. & Huggins, N. (1996) Psychiatric comorbidity: prevalence in methadone maintenance treatment. *American Journal of Drug & Alcohol Abuse*, *22*, 95-107.

Mills KL, Teesson M, Darke S, Ross J. Reliability of self-reported trauma exposure among people with heroin dependence: A longitudinal investigation. *Journal of Traumatic Stress*. In press

Mills, K., Teesson, M., Lynskey, M., Ross, J., Darke, S. (2005). Post Traumatic Stress Disorder (PTSD) among people with heroin dependence in the Australian Treatment Outcome Study (ATOS): Prevalence and correlates. *Drug and Alcohol Dependence* 77, 243-249.

Mills, K., Teesson, M., Ross, J. & Peters, L. (2006) Trauma, PTSD, and substance use disorders: findings from the Australian National Survey of Mental Health and Well-Being. *American Journal of Psychiatry*, *163*, 652-658.

National Centre in HIV Epidemiology and Clinical Research (2006) Australian NSP survey national data report 2001-2005. National Centre in HIV Epidemiology and Clinical Research. Sydney: University of New South Wales.

Ross, J. & Darke, S. (2000) The nature of benzodiazepine dependence among heroin users in Sydney, Australia. *Addiction*, *95*, 1785-93.

Ross, J., Darke, S. & Hall, W. (1997) Transitions between routes of benzodiazepine administration among heroin users in Sydney. *Addiction*, *92*, 697-705.

Ross, J., Teesson, M., Darke, S., Lynskey, M., Ali, R., Ritter, A. & Cooke, R. (2005) The characteristics of heroin users entering treatment: findings from the Australian Treatment Outcome Study (ATOS). *Drug & Alcohol Review*, *24*, 411-418.

Stafford, J., Degenhardt, L., Black, E., Bruno, R., Buckingham, K., Fetherston, J., Jenkinson, R., Kinner, S., Newman, J. & Weekley, J. (2006) Australian drug trends 2005. Findings from the illicit drug reporting system (IDRS). NDARC monograph No.59. Sydney: National Drug and Alcohol Research Centre, UNSW.

Stohler, R., Dursteler-Mac Farland, K.M., Gramespacher, C. Petitjean, S. Battegay, R. & Ladewig, D. (2000). A comparison of heroin chasers with heroin injectors in Switzerland. *European Addiction Research*, *6*, 154-159.

Swift, W., Maher, L., Sunjic, S. Doan, V. (1997) Transitions between routes of administration among Caucasian and Indochinese heroin users in South-West Sydney. NDARC Technical Report No. 42. Sydney: National Drug and Alcohol Research Centre

Teesson, M., Havard, A., Fairbairn, S., Ross, J., Lynskey, M. & Darke, S. (2005) Depression among entrants for heroin dependence in the Australian Treatment Outcome Study (ATOS): prevalence, correlates and treatment seeking. *Drug and Alcohol Dependence*, *78*, 309-315.

Topp, L., Kaye, S., Bruno, R., Longo, M., Williams, P., O'Reilly, B., Fry, C., Rose, G., Williams, P. & Darke, S. (2002) Australian drug trends 2001: findings from the illicit drug reporting system (IDRS). NDARC monograpph No. 48. Sydney: National Drug and Alcohol Research Centre, UNSW.

Trull, T., Sher, K., Minks-Brown, C., Durbin, J. & Burr, R. (2000) Borderline personality disorder and substance use disorders: a review and integration. *Clin Psychol Rev, 20,* 235-53.

Villagomez, R., Meyer, T., Lin, M. & Brown, L. (1995) Post-traumatic stress disorder among inner city methadone maintenance patients. *Journal of Substance Abuse Treatment*, *12*, 253-7.

Warner, L.A., Kessler, R.C., Hughes, M., Anthony, J.C. & Nelson, C.B. (1995) Prevalence and correlates of drug use and dependence in the United States. Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *52*, 219-29.

Warner-Smith, M., Darke, S., Lynskey, M. & Hall, W. (2001) Heroin overdose: causes and consequences. *Addiction*, *96*, 1113-1115.

Weatherburn, D., Jones, C., Freeman, K. & Makkai, T. (2001) The Australian heroin drought and its implications for drug policy. Crime and Justice Bulletin No. 59. Sydney: NSW Bureau of Crime Statistics and Research.

Appendix A: Prevalence of illicit drug use by age and gender, 2004 NDS survey

	Lifetime use	12-month use
Persons	33.6	11.3
14-19	25.5	17.9
20-29	54.5	26.0
30-39	54.5	15.9
40-49	41.6	8.7
50-59	22.1	3.2
60+	4.4	0.3
Males	37.4	14.4
14-19	24.9	18.4
20-29	57.4	32.4
30-39	59.1	21.4
40-49	47.0	11.9
50-59	27.6	4.3
60+	5.7	0.4
Females	29.9	8.3
14-19	26.2	17.4
20-29	51.6	19.5
30-39	50.0	10.6
40-49	36.2	5.7
50-59	16.5	2.1
60+	3.3	0.2

 Table 1: Prevalence of lifetime and 12-month cannabis use by age and gender

Table 2: Prevalence of lifetime and 12-month meth/amphetamine use by age and gender

	Lifetime use	12-month use
Persons	9.1	3.2
14-19	6.6	4.4
20-29	21.1	10.7
30-39	16.0	4.1
40-49	3.6	0.4
Males	11.0	4.0
14-19	6.6	4.0
20-29	24.3	12.4
30-39	19.8	5.7
40-49	4.6	0.7
Females	7.3	2.5
14-19	6.5	4.9
20-29	17.9	9.0
30-39	12.3	2.5
40-49	2.6	0.2

	Lifetime use	12-month use
Persons	7.5	3.4
14-19	6.2	4.3
20-29	22.0	12.0
30-39	12.5	4.0
40+	1.5	0.3
Males	9.1	4.4
14-19	5.7	3.9
20-29	25.8	15.1
30-39	15.2	5.8
40+	2.3	0.6
Females	6.0	2.4
14-19	6.8	4.7
20-29	18.2	8.8
30-39	9.9	2.3
40+	0.8	0.1

Table 3: Prevalence of lifetime and 12-month ecstasy use by age and gender

Table 4: Prevalence of lifetime and 12-month hallucinogen use by age and gender

	Lifetime use	12-month use
Persons	7.5	0.7
14-19	3.0	1.5
20-29	14.2	2.3
30-39	12.8	0.7
40-49	4.4	0.1
Males	9.4	1.1
14-19	3.0	1.6
20-29	17.5	3.5
30-39	16.4	1.2
40-49	5.6	0.1
Females	5.6	0.4
14-19	3.1	1.3
20-29	10.9	1.0
30-39	9.3	0.2
40-49	3.3	0

	Lifetime use	12-month use
Persons	4.7	1.0
14-19	1.9	1.0
20-29	8.9	3.0
30-39	8.8	1.8
40+	2.6	0.2
Males	5.8	1.3
14-19	1.3	0.6
20-29	10.6	3.7
30-39	10.6	2.4
40+	3.5	0.2
Females	3.7	0.8
14-19	2.6	1.4
20-29	7.1	2.3
30-39	7.1	1.1
40+	1.7	0.1

Table 5: Prevalence of lifetime and 12-month cocaine use by age and gender

	Lifetime use	12-month use
Persons	1.4	0.2
14-19	0.7	0.2
20-29	2.3	0.5
30-39	2.0	0.2
40+	1.0	-
Males	1.8	0.2
14-19	0.5	0.1
20-29	2.9	0.8
30-39	2.7	0.1
40+	1.3	-
Females	1.0	0.1
14-19	0.9	0.4
20-29	1.7	0.2
30-39	1.2	0.4
40+	0.8	-

Table 6: Prevalence of lifetime and 12-month heroin use by age and gender