

# ***Cannabis – suicide, schizophrenia and other ill-effects***

***A research paper on the consequences of acute and chronic  
cannabis use***



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**A review prepared for Drug Free Australia**  
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**Heather Ashton DM, FRCP** is Emeritus Professor of Clinical Psychopharmacology at the University of Newcastle upon Tyne, UK. Prof Ashton has conducted laboratory research on the effects of smoking THC on the brain and performance, and has carried out surveys on the extent of cannabis use in UK university students, including separate surveys on medical students, dentists and junior doctors. She has written extensively in professional journals on the adverse effects of cannabis use.

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**Herschel Mills Baker** – President of Australian Parents for Drug Free Youth. Mr Baker was author of 'Suicide/Schizophrenia - Consequences of Acute and Chronic Cannabis Use' (1988 and 1996). He was responsible for updating the 'Drug Awareness' booklet for Lions International District 201.Q5 Zone 2 of Queensland, Australia. He also developed a drug prevention resource for parents entitled 'Drug Free Kids: A Parent's Guide' and developed a series of 'Parent Drug Education Courses' successfully used by Queensland TAFE and many organisations in Wide Bay Queensland such as the Lions Clubs, Quota Club and churches.

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## FOREWORD

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This research paper gives a concise, clear, accurate and logical account of the main mental and physical risks of cannabis consumption, particularly for young users. The aim is to provide information and advice to politicians, decision-makers and researchers in order to ensure that the level of cannabis use in Australia is markedly reduced. The report provides practical recommendations towards this end and makes a valuable contribution to public knowledge and to the framing of government policies.

It is right that the emphasis is on young people since the age of first cannabis use is declining, and children and adolescents are the most vulnerable to the adverse effects. These include severe psychiatric disorders, cognitive impairment, and progression to other illegal drugs. It may be noted that the age of continuing cannabis use is also increasing and contributing to public risks, such as traffic and other accidents. These issues underline the importance of the addictive nature of cannabis, particularly in its increasingly more potent forms – unfortunately nurtured by burgeoning trafficking in hydroponically grown cannabis.

The widespread use of this pervasive and addictive drug demands urgent attention to the problem of quitting in people already cannabis dependent. None of the present methods, which rely mainly on psychological approaches, is highly effective. Further research, perhaps including the judicious use of cannabinoid antagonists combined with psychological therapies, needs to be explored, instigated and financed.

The report is written in a style easily accessible to the layman but is firmly based on hard scientific evidence, carefully selected from the vast amount of literature on cannabis that has accrued over the years. Policy makers would do well to heed its messages and recommendations.

**Heather Ashton DM, FRCP**

## EXECUTIVE SUMMARY

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Cannabis is the most commonly used illicit drug in Australia<sup>1</sup>, with one in three aged 14 years and older using the drug in their lifetime<sup>2</sup>. With the age of first use declining and the potency and popularity of the drug increasing there is clear incentive to ensure we understand the ramifications of its use on our health and communities.

This paper seeks to provide an introduction to the available literature on cannabis and the issues arising from cannabis use today, including: a description of the drug and its use; the increased potency of cannabis in the market; cannabis as a “gateway” to harder drug use; the issues of dependence and withdrawal; the significant cannabis harms on mental health, brain function and development, and physical conditions such as cancer; and, the problems encountered when trying to quit cannabis and the generally poor outcomes today.

The paper also provides recommendations on how we can effectively answer the questions surrounding cannabis use in Australia.

Throughout, we return to the issue of age of first use. Overwhelming evidence exists to support the fact that the age of first cannabis use is an important predictor of progression to heavier drug use and need for treatment (for example, see Pope et al, 2003; Anthony et al, 1994; Warner et al, 1995; Kandel et al, 1997). Clearly, there is a significant problem when boys aged 9 and 10 are discovered with cannabis in Brisbane schools<sup>3</sup>.

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<sup>1</sup> Department of Parliamentary Services Research Note, June 2007.

<sup>2</sup> 2004 National Drug Strategy Household Survey

<sup>3</sup> “Children caught with pot”, Sunday Mail, October 26, 2003

## SECTION ONE: CANNABIS USE

### A DESCRIPTION OF THE DRUG

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Cannabis is the term most frequently used to refer to the drug deriving from the plant *Cannabis sativa*, the most commonly used illicit drug in Australia.

Cannabis is generally found in three forms, all of which contain delta-9 tetrahydrocannabinol (“THC”) as the main psychoactive ingredient. The most common and least potent of these forms is marijuana, a mix of the plant’s dried leaves and flowers. Cannabis in the form of hashish, or dried cannabis resin, produces stronger effects through its higher concentration of THC. Hashish oil, a thick oily liquid, is the third and most powerful form of cannabis.

Of the active constituents of cannabis there have been over 60 cannabinoids identified; however, only a few, and primarily THC, have been studied intensely. The primary metabolite, 11-hydroxy-THC, is also psychoactive and even more potent and, as with all cannabinoids, acts on the endogenous receptors in the brain and body.

Cannabis is well absorbed through inhaling its smoke or its inclusion in cakes or cookies and is very slowly metabolised by the body as it becomes deeply absorbed and entrenched in the body’s fatty tissues, with the brain a primary target. The complete elimination of a single dose from a user’s system may take up to thirty days (Cabral, 1989) and its acute effects can last several hours. In the case of chronic and frequent use, cannabis concentrations accumulate and can cause a chronic intoxication and dependency.

Further, the endocannabinoid system moderates many of the body’s vital functions, including motor control, cognition and memory, cardiovascular and endocrine activity, appetite, mood and immune responses. The endocannabinoid system’s regulation of these functions is fundamental to the brain’s normal performance and as such is central to understanding the pervasive effects of cannabis. THC overwhelms this system with long-lasting and extensive effects on both cannabinoid receptor type 1 (CB<sub>1</sub>), in the brain, spinal cord and peripheral nerves; and cannabinoid receptor 2 (CB<sub>2</sub>), in the body’s immune tissues. Physically, this means a decrease in the release of neurotransmitters, decreased neural firing and transmission of nerve impulses. The body’s natural substances which interact with CB<sub>1</sub> and CB<sub>2</sub> receptors are called anandamides. These modulators are released locally in discrete brain areas, and in contrast to THC, are rapidly de-activated in minutes.

It has also been argued that 27% of the population carry a high risk genetic variant which produces a weaker Catechol-O-Methyl Transferase (COMT) enzyme which is responsible for the break down of dopamine in the brain. Henquet (2005) argues that the excessive amounts of dopamine released by cannabis use places those with the weaker COMT enzyme at 10 times greater risk of developing psychosis and, later in life, of developing schizophrenia (see Section 4: Cannabis Harms, Mental Health).

Over 1,500 toxic chemicals have been identified in the smoke of cannabis, including carbon monoxide, carcinogens and irritants. These all greatly affect the body's respiratory and cardiovascular systems in a similar manner to the known effects of smoking tobacco. Moir et al's 2007 study of marijuana smoke found ammonia at levels up to 20-fold greater than that found in tobacco, hydrogen cyanide at concentrations 3-5 times those in tobacco smoke, and confirmed the presence of known carcinogens and other chemicals implicated in respiratory diseases.

The Institute of Medicine of Washington DC<sup>4</sup> produced the table opposite, which shows a comprehensive comparison of the chemicals in cannabis and tobacco:

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<sup>4</sup> Sources cited by the Institute of Medicine, Marijuana and Health, Washington DC: Hoffmann, D et al, 1975; Hoffman, D et al, 1976; Brunnemann, KD et al, 1976; Brunnemann KD et al, 1977.



**Table 1 – Comparison of Chemicals – Cannabis and Tobacco**

<b>A. Cigarettes</b>			
	<b>Units</b>	<b>Marijuana</b>	<b>Tobacco</b>
		(85mm)	(85mm)
Average Weight	(mg)	1115	1110
Moisture	(%)	10.3	11.1
Pressure Drop	cm	14.7	7.2
Static Burning rate	mg/s	0.88	0.80
Puff Number		10.7	11.1
<b>B. Mainstream Smoke</b>			
<b>I. Gas Phase</b>	<b>Units</b>	<b>Marijuana</b>	<b>Tobacco</b>
Carbon Monoxide	%	3.99	4.58
	mg	17.6	20.2
Carbon Dioxide	%	8.27	9.38
	mg	57.3	65.0
Ammonia	mcg	228	199
HCN	mcg	532	498
Cyanogen (CN) <sub>2</sub>	mcg	19	20
Isoprene	mcg	83	310
Acetaldehyde	mcg	1200	980
Acetone	mcg	443	578
Acrolein	mcg	92	85
Acetonitrilebenzene	mcg	132	123
Benzene	mcg	76	67
Toluene	mcg	112	108
Vinyl chloride	ng	5.4	12.4
Dimethylnitrosamine	ng	75	84
Methylethylnitrosamine	ng	27	30
pH, third puff		6.56	6.14
fifth puff		6.57	6.15
seventh puff		6.58	6.14
ninth puff		6.56	6.10
tenth puff		6.58	6.02
<b>II. Particulate phase</b>			
	<b>Units</b>	<b>Marijuana</b>	<b>Tobacco</b>
TI particulate - dry	mg	22.7	39.0
Phenol	mcg	76.8	138.5
o-Cresol	mcg	17.9	24
m- and p-Cresol	mcg	54.4	65
Dimethylphenol	mcg	6.8	14.4
Catechol	mcg	188	328
Cannbidol	mcg	190	
D9 THC	mcg	820	
Cannabinol	mcg	400	
Nicotine	mcg		2850
N-Nitrosomicotine	ng		390
Naphthalene	mcg	3.0	1.2
1-Methylnaphthalene	mcg	6.1	3.65
2-Methylnaphthalene	mcg	3.6	1.4
Benz(a)anthracene	ng	75	43
Benzo(a)pyrene	ng	31	21.1

## INCREASED POTENCY

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Of particular concern in recent years is the cultivation of high potency cannabis, often referred to as “skunk” or “super skunk”<sup>5</sup>. This increase in potency, which in real terms refers to increased THC concentrations, is in addition to the existing hybrid varieties of cannabis which are continuing to gain popularity in Australia. High potency cannabis, or cannabis containing high THC concentrations, is currently cultivated in all states of Australia, largely through the use of hydroponics cultivation, and is also brought into Australia from countries such as Papua New Guinea, India, Lebanon, Morocco, Holland, Afghanistan, Thailand and Canada.

The effects of THC in the cannabis user, including those which are negative, are dose-related – the higher the dose of THC, the greater the effects – hence, the significance of increased cannabis potency (Raemaekers, 2006).

It is important to note that some publications dated as recently as 2006, be treated with caution on this matter, as the evidence base has now substantially changed. For example, the Australian National Council on Drugs (ANCD’s) position, outlines in the papers “*Cannabis: answers to your questions*” (2006) and “*Evidence-based answers to cannabis questions: a review of the literature*” (2006), is that in the past few decades Australia has only seen small increases in THC levels.

Of interest is the fact that, more than a decade ago, the Australian Bureau of Criminal Intelligence (1993) reported a THC content in cannabis plants of up to 30%, a substantial increase from the early 60’s when the typical cannabis joint contained as little as 0.5%. One example of our concerns regarding the increase of potency of cannabis in Australia is that of ‘Drug Kingpin’, Alexander Malcolm Lane, who used drug mules, paying up to \$30,000 a trip to travel to Amsterdam and bring back thousands of high-potency cannabis seeds.

The Courier-Mail August 17 2007. <http://www.news.com.au/story/0.23599.22257426-2.00.html>

In both the United States (US) and United Kingdom (UK) public offices have acknowledged THC potency increases. A joint report of the US’s Office of National Drug Control Policy (ONDCP) and the National Institute on Drug Abuse recently found that levels of THC in cannabis have reached the highest-ever levels since analysis of the drug began in the late 1970’s. They found the average to have increased from just below 4% in 1983 to a new high of 9.6% in 2008, a doubling of potency. John Walters, former Director of ONDCP, states “Baby boomer parents who still think marijuana is a harmless substance need to look at the facts. Marijuana potency has grown steeply over the past decade, with serious implications in particular for young people”.

The UK’s Home Office “Cannabis Potency Study 2008”, while finding a lesser increase over time (from 13.98% to 15.0%), nevertheless presents a startling average percentage of THC content at 15% potency. These figures, while not based on Australian data, cannot be ignored. It would be imprudent to assume the increases in potency seen in overseas cannabis markets are not mirrored within Australia.

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<sup>5</sup> See Appendix A and Appendix B for media reports

When it is considered that there is a well-demonstrated dose-response relationship between cannabis and its related drug-induced psychosis, where the greater the amount of cannabis consumed correlates to a higher degree of risk of psychosis, any three to fourfold increase in potency is absolutely critical in any assessment of cannabis harms.

When it is further considered that changed usage patterns, whereby young users smoke only the multiple potent heads of the cannabis plant and also use a more concentrated mode of drug delivery via the use of 'bongs', the ANCD paper's approach to potency is of concern. By emphasising their assessment of a narrow understanding of the thirty-fold claim, which makes three to fourfold increases pale into insignificance, the very significant conjuncture of these real and significant three to fourfold increases in cannabis potency, along with new usage patterns which deliver significantly higher doses of cannabinoids, is downplayed for the Australian reader at the very time that the scientific community has expressed alarm at this very same conjuncture and its relationship to psychosis. Concluding their discussion in ANCD Research Paper (2006, p.11), the authors cite US figures which do in fact show increases in potency which have more than tripled:

*"Between 1980 and 1997 THC content increased from 1.2 per cent to 4.2 per cent. Cannabis samples (excluding hash and hash oil) analysed between May and August 2003 had average THC levels of 6.37 per cent (see 1.2 for details on potency for different forms of cannabis). This finding suggests definite rises in cannabis THC content. However, over the last two decades, such increases are not consistent with claims of a thirty-fold increase. While Australia has not collected such comprehensive data, moderate changes as seen in the United States and New Zealand data are likely to be replicated in Australian trends given that, with isolated exceptions, the majority of THC levels in studies of cannabis seizures have remained under 5 per cent."*

## **GATEWAY DRUG**

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The term “gateway drug” is used to illustrate the tendency of cannabis to introduce the user to other illicit drugs, and arguments for and against the hypothesis have a long history.

There are multiple studies that have reached a conclusion in support of the gateway hypothesis (see Kandel, 1992 and 1996; Clayton, 1992; Bailey, 1992; Poikolainen et al, 2001). Specifically, the Centre on Addiction and Substance Abuse (CASA) at Columbia University found that children who use drugs, including cannabis, are up to 266 times more likely to use cocaine than those who do not use any of the gateway drugs identified (cannabis, tobacco and alcohol).

There are critics of the gateway theory who argue that a clear link between cannabis use and other illicit drugs does not reflect a causal sequence, relying upon the presence of confounding factors such as a user’s socio-economic status and family history (see Johnson, 1973; Hays et al, 1987).

In contrast, the US Office of National Drug Control Policy’s “2008 Marijuana Sourcebook” clearly states that recent research supports the gateway hypothesis, specifically that “its use creates greater risk of abuse or dependency on other drugs, such as heroin and cocaine”.

Further, a study on 311 sets of same-sex twins, in which only one twin smoked cannabis before age 17, found that early cannabis smokers were up to five times more likely than their twin to move on to harder drugs (Lynskey, 2003). Also, Hurd (2006) concluded that findings supported the gateway hypothesis when she conducted a study on rats. Hurd found that rats trained to self-administer heroin would administer greater doses if they had previously been exposed to THC. A further study of 75,000 adolescents and young adults found teenage cannabis smokers had a 50% higher risk of developing an alcohol-use disorder and specifically stated “Addictive drugs all act on a part of the brain that is described as the central reward circuitry. Once this system is exposed to one drug, the brain may become more sensitive to the effects of other drugs, as demonstrated by a number of rodent studies” (Gruzca, 2006).

In summary, as Kandel states (1992), very few try illicit drugs other than cannabis without prior use of cannabis.

(Behrendt 2009) results revealed several interesting findings. First and foremost, early onset users were shown to have an elevated risk of substance use disorder, even if the transition to substance use disorders was not immediate. The transition from first cannabis use to cannabis dependence occurred more rapidly than transitions to alcohol disorders and nicotine dependence. Substance use onset later in adolescence was associated with a more rapid progression to substance use disorders for alcohol and nicotine. Overall early onset of substance use was not shown to lead to a rapid progression of substance use disorders. But delaying first substance use is still important.

## DEPENDENCE

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There is general consensus that cannabis is addictive and the addiction carries with it all the adverse affects of dependence, including symptoms of withdrawal (see Ramstrom, 2003, in *A Survey of Scientific Studies*).

In fact, in 1992 the World Health Organisation (WHO) identified cannabinoid dependence syndrome and described that dependence as existing where three or more of the following diagnostic guidelines were experienced or exhibited during a year:

- a) a strong desire or sense of compulsion to take cannabinoid;
- b) difficulties in controlling cannabinoid-taking behaviour in terms of its onset, termination or levels of use;
- c) a physiological withdrawal state when cannabinoid use has ceased or been reduced, as evidenced by: the characteristic withdrawal syndrome for cannabinoid; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms;
- d) evidence of tolerance, such that increased doses of cannabinoid are required in order to achieve effects originally produced by lower doses;
- e) progressive neglect of alternative pleasures or interests because of cannabinoid use, increased amount of time necessary to obtain or take the substance or to recover from its effects;
- f) persisting with cannabinoid use despite clear evidence of overtly harmful consequences, such as depressive mood states consequent to periods of heavy substance abuse, or drug-related impairment of cognitive functioning; and
- g) efforts should be made to determine that the user was actually, or could be expected to be, aware of the nature and extent of the harm.

Haney et al (1999) demonstrated withdrawal symptoms from pure THC delivered under laboratory conditions in humans and those symptoms such as anxiety and insomnia lead to difficulty in stopping cannabis use.

Budney et al (2004) reviewed the validity of cannabis withdrawal syndrome and concluded that the evidence of laboratory and clinical studies indicates that withdrawal syndrome reliably follows discontinuation of chronic cannabis use and further noted that the severity of withdrawal symptoms appeared substantial.

Later, in 2006, Budney & Hughes found evidence of a withdrawal syndrome in cannabis use and noted “demand for treatment of cannabis dependence has grown dramatically (and) the majority of people who enter treatment have difficulty in achieving and maintaining abstinence from cannabis”. They found laboratory studies had established the reliability, validity and time course of a cannabis withdrawal syndrome and pointed to the discovery of an endogenous cannabinoid system, the identification of cannabinoid receptors and demonstrations of precipitated withdrawal with cannabinoid receptor antagonists as the neurological basis for cannabis withdrawal.

In a wide ranging appraisal of the literature, Gardner reviewed 224 scientific papers in 2003 and concluded “cannabinoids act on the brain reward processes and reward-related behaviours in strikingly similar fashion to other addictive drugs”.

Budney (2006) also asked if specific dependence criteria were necessary for different substances in a report for *Addiction* and found that “cannabis dependence is much more similar to, than different from, other types of substance dependence, even with regard to withdrawal”.

Vandrey (2008) more recently suggested withdrawal from cannabis use is similar to that experienced when quitting smoking tobacco, in a controlled comparison based on the self-reporting of twelve heavy users of both cannabis and tobacco. The participants’ abstinence was confirmed objectively, procedures were identical during each abstinence period and abstinence periods occurred in a random order. The strength of this study is in the same participants reporting on withdrawal symptoms for tobacco and marijuana, eliminating the likelihood that results reflect physiological differences between subjects.

Vandrey found that “since tobacco withdrawal symptoms are well documented and included in the DSM-IV<sup>6</sup> and the ICD-10<sup>7</sup>, we can infer from the results of this comparison that marijuana withdrawal is also clinically significant and should be included in these reference materials”.

Also, Cambridge University Press recently published “Cannabis Dependence: Its Nature, Consequences and Treatment” (2006), a report on over 2,500 adult daily cannabis users where 1, 111 adults met the DSM-IV criteria for dependence and reported significant associated problems, such as depression and lower levels of motivation and satisfaction with life.

Coffey et al (2003) related dependence to a user’s starting age and reported that weekly use of cannabis marks the threshold for an increased risk of later cannabis dependency, specifically amongst young users. Coffey et al reported “30% of teenagers smoking more than once a week became addicted by their early twenties, those between 14 and 17 were twenty times more likely”.

Interestingly, dependent cannabis users reported compulsive and out-of-control use more frequently than dependent alcohol users, withdrawal to a similar extent and tolerance considerably less often.

Chambers’ study (2003) supported the increased vulnerability of adolescent brains to addiction compared to adults. He suggested that drug addiction should be thought of as a development disorder in the brains of teenagers, in that the adolescents’ changing brain circuitry leaves them especially vulnerable to the effects of addictive drugs.

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<sup>6</sup> Diagnostic and Statistical Manual of Mental Disorders, 4<sup>th</sup> Edition

<sup>7</sup> International Classification of Diseases, 10<sup>th</sup> Edition

Finally, *Science Threads of Addiction, Substance Use and Health* (STASH January 2007) looked at the transition from drug use to dependence in over 8,000 participants. They found the probability of drug initiation and developing dependence both peaked at 18. Interestingly, male users were found to be approximately twice as likely to develop dependence in the first two to five years as female users.

## SECTION TWO: CANNABIS HARMS

### INTRODUCTION TO THE ADVERSE HEALTH CONSEQUENCES OF CANNABIS

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Sweden was the first country in the world to extensively research the evidence on the adverse health consequences of cannabis use and has since adopted a strategy of community wide information sharing regarding the health hazards posed by the drug. Renowned psychiatrist Jan Ramstrom has compiled extensive reviews for the Swedish National Board of Health Welfare (in 1998) and National Institute of Public Health (in 2003) on the health implications of cannabis use. A result of Ramstrom's reviews was the report "*Adverse Health Consequences of Cannabis Use*", which provides outlines of mental disorders, physical injury, psychological and psychosocial injury. More recently in the United Kingdom, Brett (2008) produced "*Cannabis – A General Survey of its Harmful Effects*" in a review of the ever-widening range of negative effects upon health caused by the substance, including childhood development, mental illness and cognitive functioning.

In this section we shall discuss only a limited portion of the available literature on adverse health consequences in three primary areas including mental health, brain function and physicality.

#### MENTAL HEALTH

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The harms of cannabis use on the user's mental health have been well documented and include specific research into the onset of schizophrenia (see Boydell, 2006; Solowij, 2007; Fergusson, 2005; Ferdinand, 2005, Veling 2008) and other mood disorders including depression, bi-polar disorder and amotivational syndrome (see Bovasso, 2001; Hayatbakhsh, 2007; Corcoran 2008). Research has also explored the links to suicide, especially in young people (Dervaux, 2003; Greenblatt, 1998; Beautrais, 1999).

Firstly, severe mental disturbances, such as momentary short-term psychosis or the long-term illness of schizophrenia, have been linked to cannabis use and especially so when cannabis use begins in adolescence. As a stimulant of the dopamine system, cannabis offers the user a pleasurable 'high'; however, this 'high' can become dangerous when dopamine levels become excessive. Murray (2005) discusses the impact of early cannabis use on the developing adolescent brain and specifically dopamine receptors, indicating early cannabis use may damage these receptors permanently, leaving a young cannabis user at a much higher risk of developing schizophrenia or experiencing psychosis.

A significant study in Sweden (Andreasson, 1987) examined, over fifteen years, the link between heavy cannabis use and schizophrenia in 50,087 members of the Swedish Army and conclusively found schizophrenia occurred more frequently in heavy consumers of cannabis.



The results were re-analysed and replicated in additional studies (Zammit, 2002; Fergusson, 2003) with the British Medical Journal (BMJ) reporting in 2002 heavy consumers of cannabis at age 18 were over 600% more likely to be diagnosed with schizophrenia over the next fifteen years than those who did not use cannabis. The BMJ report also clarified that it was cannabis use and not other drugs that was associated with schizophrenia.

Moore et al concluded in 2007, that “there is now sufficient evidence to warn young people that using cannabis could increase their risk of developing a psychotic illness later in life”. In fact, Moore et al found, in a review of 35 longitudinal studies that cannabis use increased the risk of developing a psychotic illness, such as schizophrenia, by 40%. This figure was doubled for frequent or heavy users. Reports by Hollis et al (2008); Henquet (2005) and Konings (2008) have found a significant positive association between cannabis use and mental health disturbance in young people who are genetically predisposed to mental health problems, such as schizophrenia.

Interestingly, Ramstrom (2003) demonstrated the association between adolescent cannabis use and adult psychosis persists even after controlling for many potential confounding variables, such as low IQ and education levels, unemployment, social integration, gender, age, ethnic group, tobacco smoking and previous psychotic symptoms. This finding was supported by recent studies of Finnish adolescents (Jouku et al, 2008) which showed an association between cannabis use and psychosis symptoms not caused by other drug use, family background or behavioural problems.

Further, researchers in Spain recently found a strong and independent link between cannabis use and the onset of psychosis at a young age, reporting that compared with nonusers, the age of psychotic onset was lowered by 7, 8.5 and 12 years among users, abusers and dependents respectively. These results are supported by multiple studies (Fergusson, 2005; Ferdinand, 2005; Solowij, 2007) and all highlight the notion of the younger the user, the worse the effects.

A second mental health issue frequently associated with cannabis use is depression and numerous studies support the connection.

For example, an Australian study of 3,239 young adults, from their birth to the age of 21, found a relationship between early initiation to and frequent use of cannabis and depression (Hayatbakhsh, 2007); a 16-year study of individuals not initially suffering from depression, but who then frequently used cannabis, were found to be four times more likely to develop depression at follow up (Bovasso, 2001); and, Fergusson (2002) studied 1,265 children over a 21-year period and concluded that cannabis use, particularly heavy or regular use, was associated with a later increase in depression and suicide. Recent articles in *The Age* newspaper (September 29, 2008) discuss Australian statistics showing that cannabis' toll on mental health, expressly causing depression, is more prevalent than that caused by the well known impact of amphetamines.

Thirdly, cannabis use can induce amotivational syndrome, a mental state characterised by apathy, an inability to carry out plans, deal with frustration or concentrate for any length of time (Cohen, 1982). While equivocal, amotivational syndrome strikes a chord in that it aptly describes the 'personality' of a chronic cannabis smoker and is supported by numerous studies (Newcomb & Bentler, 1988; Tunving, 1987; Cohen, 1982). Musty & Kaback (1995) maintain that amotivational syndrome exists and is a manifestation of depression.

Finally, multiple studies have linked cannabis use with suicide<sup>8</sup>. A study by Beautrais et al (1999) examined and found a relationship between cannabis abuse and suicide. Greenblatt (1998) found that young people, aged 12 to 17, who smoke cannabis weekly are three times more likely than non-users to have thoughts about committing suicide, and this ratio was confirmed by Lynskey et al (2004). Dervaux (2003) examined the link between cannabis abuse and the suicide attempts of schizophrenics, finding a close correlation.

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<sup>8</sup> See Appendix B for media articles on this issue

## **BRAIN FUNCTION**

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It is undeniable that cannabis affects the brain, and affects the brain's functioning adversely. Conclusive evidence shows that heavy marijuana use for five years or more may impair memory and slow cognitive function (Lambros, 2006; Ashtari, 2005; Robbe, 2006; Karila, 2005; Lundqvist, 2005; Fisk 2008; Solowij, 2008), with specific research completed on impaired driving ability (Kurathaler, 1999; Menetry, 2005; Drummer, 1994, 1998, with Gerostamoulos, 1999).

The short-term effects of cannabis use on brain function can include things such as problems with memory and learning, difficulty in thinking and problem solving, loss of coordination. Long-term effects include permanent memory impairment and overall slower cognitive function.

Importantly, Chambers (2003) and Pistis (2004) found the adolescent brain, while still under development, was particularly vulnerable to the ill effects of substance abuse, including cannabis. Researchers have concluded that repeated exposure to cannabis as an adolescent was related to abnormalities in the development of the specific fibres associated with higher aspects of language auditory functions (Ashtari, 2005). Giedd et al (1999) also discusses the development of the adolescent brain which does not reach physical maturity until the mid-twenties, and warned drug abuse could alter the normal course of brain growth. He later specifically looked at regions of the brain that control impulse and risky behaviours, reconfirming his previous findings that cannabis use on a developing adolescent brain can negatively affect overall and specific brain functions. In a study of brain abnormalities in schizophrenics as compared to the brain abnormalities presenting in adolescents frequently using cannabis, Kumra (2007) concluded the deficiencies were the same and in that part of the brain which develops during adolescence – emotional associations and other higher cognitive functions such as language, perception, creativity and problem solving.

Most recently, Medini et al (2008) confirmed the adverse brain impact of adolescent cannabis use in a study presented to the American Academy of Pediatrics. The research team found that the chronic use of cannabis during adolescence – a critical period of ongoing brain development – slowed psychomotor speed, led to poorer complex attention, verbal memory and also planning ability. Perhaps, most startlingly, these impacts continued after one month's abstinence from cannabis use.

Recent evidence on cannabis and cognitive functioning also comes from Greece (Messinis et al, 2006) where they found that those who smoked at least four joints per week for several years performed significantly worse than non-users in several areas, particularly verbal learning (the ability to remember previously learned words) and executive functioning (organising and coordinating simple tasks). Further, Ranganathan (2006) reviewed the literature on the acute effects of cannabis on memory, concluding that cannabinoids impair all stages of memory (including encoding, consolidation and retrieval).

Solowij et al (2002) examined the effects of the duration of cannabis use on specific areas of cognitive functioning among users seeking treatment for cannabis dependence. Their results also confirmed that long-term heavy cannabis users show impairments in memory and attention, and in fact that endure beyond the period of intoxication and with increasing years of regular cannabis use. Bolla (2002) found a dose-response relationship in that the more cannabis used, the worse they performed in cognitive testing, especially memory. It is very clear that regular cannabis use is associated with impaired functioning – both by objective measures and by the admission of users themselves (Pope Jr, 2004).

Alternate studies (Niveau & Dang, 2003; Howard & Menkes, 2007) also looked at the effects of cannabis use upon neural mechanisms controlling impulse and found a connection with acts of violence and aggression. Additionally, the latest evidence of brain abnormalities in long-term, chronic cannabis users further confirms that heavy daily use exerts harmful effects on brain tissue (Yucel, 2008) and in similar ways to those seen after long-term abuse of other major drugs (de Fonseca, 1997).

Specific research on the impacts of cannabis on driving ability has increased of late. Drummer (1994; 1998; with Gerostamoulos, 1999) has done significant research on the issue and found road fatalities related to cannabis intoxication have steadily increased over the last ten years. Consistent with Drummer's findings, past research examining the effects of THC on driving ability indicate it impairs both car control (Moskowitz, 1985) and the driver's awareness of the vehicle's position in traffic (Ramaekers et al, 2000). Hansteen et al (1976) also found THC intoxication is more likely to result in collisions with obstacles on a driving course than when not intoxicated. Studies by Papfotiou (2001, 2005) found that driver errors occurred more frequently when the driver was under the influence of both cannabis and alcohol. Since the two are frequently taken together it is concerning to note that a 2005 study (Laumon et al) found the risk of accident when cannabis was combined with alcohol was 16 times higher than when using either drug alone.

These findings indicate that cannabis impairs driving ability and given the prevalence of cannabis use (upward of 300,000 Australians smoke it daily; 750,000 smoking it weekly<sup>9</sup>) this poses a significant risk on our roads.

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<sup>9</sup> Australian Institute of Health and Welfare 2005. Statistics on drug use in Australia 2004. AIHW Cat. No. PHE 62. Canberra: AIHW (Drug Statistics Series No. 15). p 22

## PHYSICAL HARMS

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Cannabis smoke contains many of the same known carcinogens as tobacco smoke. In fact, studies have found the tar from cannabis contains 50% more of some of the carcinogens found in tobacco, notably benzopyrene, a potent carcinogen and key factor in the development of lung cancer (Hoffman et al, 1997; Tashkin et al, 1997; Novotny et al, 1976; Leuchtenberger et al, 1983), and so it should not be surprising to see cannabis use as a factor in a wide range of adverse physical conditions, including lung cancer, chronic obstructive pulmonary disease, increased risk of heart or stroke due to adverse impacts on the cardiovascular system, weakened immune system and birth defects. Cannabis cigarettes also have a higher combustion temperature than tobacco cigarettes.

There is research to support the connection between cannabis use and cancer of the digestive and respiratory tracts (Hall, 2002), lung cancer (Berthiller 2008), lung (Sridar, 1994) and breast (McKallip, 2005). Aldington (2007; et al, 2008) found that long term cannabis use specifically increased the risk of lung cancer in young adults, particularly in those who started smoking cannabis at a young age. Tashkin (2006) explains that cannabis smokers typically hold their breath four times longer than tobacco smokers, allowing more time for particles to be deposited in the lungs. In addition, cannabis is usually smoked without an adequate filter.

Researchers have interviewed lung cancer patients in seeking to identify the main risk for the disease, such as smoking habits, family history and occupation (Tetrault et al, 2007). The patients were questioned about cannabis consumption and results showed lung cancer risk rose by 5.7 times for patients who had smoked a joint a day for 10 years, or two joints a day for five years, and after adjusting for cigarette smoking.

A study in 2006 (Terris et al) reported that, of 52 men with transitional cell bladder cancer, 88.5% had a history of smoking cannabis and almost 31% were still using the drug. Terris et al found that cannabis metabolites have a half-life in urine about 5 times greater than tobacco metabolites, and warned smoking cannabis may be a more potent stimulant than tobacco smoking of malignant cell transformation, a hallmark of cancer.

In relation to chronic obstructive pulmonary disease (COPD), the period of cannabis use seems to play an important role, particularly in regard to lung emphysema and various other respiratory complications such as asthma, dyspnea, pharyngitis and chronic cough (Tetrault et al, 2007). Beshay (2007) researched emphysema in young adults and agreed the period of cannabis use was influential. A further study Tan (2007) on people aged 40 and over found that smokers were two and a half times as likely as non-smokers to develop COPD and that adding cannabis to tobacco increased the risk again by one-third.

With regard to the body's cardiovascular system, the harms of cannabis use are again significant. At first, the intoxication produced by cannabis causes an increase in heart rate of between 20% and 50% (Huber et al, 1988; Jones, 1984) as THC increases the production of chemicals which stimulate the heart.

The increase in heart rate caused by cannabis is additive with the increased heart rate caused by nicotine in tobacco. THC is also found to have analgesic properties, lessening chest pain which Jones (1982, 1984) argues may delay the seeking of treatment, decrease the supply of oxygen to the heart and place it under greater strain. Maykut (1984) also found a rise in blood pressure if the person is sitting or lying, but upon standing drops drastically, in some cases causing the person to faint.

It must be added that tolerance can develop quickly to the acute cardiovascular effects of cannabis, with people receiving daily doses by mouth developing tolerance within 7 to 10 days, in a possible explanation of why effects can sometimes be missed (Benowitz & Jones, 1975; Nowlen & Cohen, 1977; Jones, 1984).

Supporting research as to the cardiovascular harms of cannabis use are found in Herring et al (2001), who used sound waves to measure cerebral artery blood flow resistance and found that prolonged cannabis use in 18 to 30 year olds increased the resistance in arteries and restricted blood flow to the brain; in Geller et al (2004) who detail an incident in which three teenagers, aged 15 to 17, “binge smoked” cannabis and suffered strokes from which two later died; and, in Mittleman (2001) who interviewed 3,882 patients of heart attacks and found the risk of myocardial infarction rose almost 5 times in the hour following the smoking of a joint.

We still do not know the long term effects of exposure to cannabis smoke on the cardiovascular system over extended periods, but experience with the problems of tobacco smoke should urge caution. Jones (1984) suggests “after years of repeated exposure, there may be lasting, perhaps even permanent alterations of the cardiovascular system function. There are enough similarities between THC and nicotine’s cardiovascular effects to make the possibility plausible” and this is supported by a multitude of research (Mukamal et al, 2008; Lindsay, 2005; Fisher et al, 2005; Korantzopoulos, 2008).

There is also significant supporting research on the effects of cannabis use during pregnancy on newborns, with THC readily crossing the placenta (Bada, 2006; Cornelius, 1995; Bailey, 1987) – Bluhm (2006) discusses an increased risk of neuroblastoma; Robinson et al (1989) identified an eleven-fold increase in leukaemia; and, there are multiple abnormalities in physical appearance, size, weight and hormonal functions discussed by Fried, 1980 and 1984; Zimmerman, 1991; Zuckerman, 1989; Barnett, 1983; El Marroun 2008; Mendelson, 1985 and 1986).

A paper by Klonoff-Cohen et al (2006) studied the effects of cannabis use on the outcomes of IVF and GIFT fertility treatments and concluded cannabis use lowered the prospects of successful treatments. They found females produced fewer eggs and the child once successfully conceived had a significantly lower birth weight.

The risk of miscarriage of ectopic pregnancy of women smoking cannabis in the early stages of pregnancy was highlighted in recent research by Day (2006). THC was found to mimic anandamide and its control over embryo development, disrupting the process and creating cell abnormalities in mice. Day also concluded that, “Prenatal exposure to marijuana, in addition to other factors, is a significant predictor of marijuana use at age 14”.

This study's findings warrant further attention to puberty as a sensitive period in an individual's development. With regards to prevention, there is a need to understand more about the pathways between pubertal development, child behaviour and substance and cannabis use. (Hayatbakhsh, 2009).

A review by Huizink & Mulder (2006) came to the conclusion that pre-natal exposure to cannabis use is related to some common neuro-behavioural and cognitive outcomes, including symptoms of ADHD such as inattention and impulsivity, decreased general cognitive functioning and deficits in learning and memory tasks.

Barros and colleagues, writing in *The Journal of Paediatrics* in January 2007, found that marijuana-exposed infants born to adolescent mothers scored differently on measures of arousal, regulation and excitability compared to non-exposed infants, where they displayed subtle behaviour changes in the first few days of life, i.e. they cried more, startled more easily and were more jittery. The authors said this may also interfere with mother-child bonding.

Harkany et al. (2007) found that endocannabinoid signalling modulates central nervous system patterning, so that "pharmacological interference with endocannabinoid signals during foetal development leads to long-lasting modifications of synaptic structure and functioning. Marijuana abuse during pregnancy can impair social behaviours, cognition and motor functions in the offspring with the impact lasting into adulthood".

Another paper in May 2007 had similar findings. Endocannabinoids in the human body play a vital role in the development of a baby's brain in that they are responsible for controlling how the complex system of nerves develop in the embryonic brain. Dr Ann Rajnicek states "Smoking cannabis could interfere with the signals that are being used in the brain to wire it up correctly in the first place. As the brain develops further, there will be functional problems – potential brain damage" (Berghuis et al. 2007).

The reason for the late appearance of this damage is assumed to be that the functions involved are "executive" cognitive functions that are not taken into use until the child is four to six years old. Another long-term study shows similar associations between exposure during the foetal stage and relatively late (at age 6 and 10 respectively) behavioural disturbances (Ramstrom, 2003).

## SECTION THREE: QUITTING CANNABIS

It is not only important to have strategies to help people quit cannabis but prevention must be the aim of the policy makers. Student drug testing is intended as prevention and as a deterrent. It offers young people a tool to refuse drugs among their peers. Student drug testing, which include anonymity, privacy, non-coercion, also encourages families to seek help for their children in need. (McKinney 2005, DuPont 2002, Ticker 1997, Goldberg 2007).

While it is acknowledged that it is far easier and less expensive to adopt preventative measures than invest in treatment, for those who are addicted to cannabis, it is important to provide the means to be able to stop – just as we have seen implemented with other common drugs such as tobacco and alcohol. This section discusses symptoms, the need for treatment, effective treatment techniques and the high incidence of relapse.

Contributors to “*Cannabis Dependence, Its Nature, Consequences and Treatment*” state the symptoms of cannabis withdrawal are “irritability, anger, nervousness, sleep difficulty, change in appetite, physical discomfort” (2006) and Kouri (1999) found previous reports of an abstinence syndrome associated with chronic marijuana use were confirmed and also suggested aggressive behaviour as a component. There is also research to suggest staying clean for cannabis addicts is as hard as for heroin addicts (Roffman, Stephens, Marlatt; 2006).

Extensive research has found a connection between early cannabis use and the likelihood of need for treatment (Kandel & Yamaguchi, 1985; Robins & Przybeck, 1985; Adams & Gfroerer, 1988; Glants & Pickens, 1992; Anthony & Petronis, 1995).

There is a need for effective treatment of cannabis misuse. Psychological therapies have been developed based on principles of motivational interviewing, cognitive-behavioural therapy and relapse prevention. The evidence base for these therapies is explored in a review by Maddock & Babbs (2006), and studies targeting both adult users and young people are considered. They also discuss new pharmacological treatments.

Increased recognition that marijuana can cause addiction and significant negative consequences in a subset of users has prompted the development of marijuana-specific interventions and treatment materials paralleling those for other substance use disorders. These advances have increased users’ and caregivers’ perceptions that it is acceptable to seek and provide treatment for cannabis use and have contributed to an increase in the number of individuals requesting help (Budney, 2007). In light of the recognition that people smoke cannabis mainly for pleasure (euphoria/“high”) it is noted that none of the available treatments are highly effective.

The Substance Abuse and Mental Health Services Administration (SAMHSA) released a treatment manual titled “Brief Counselling for Marijuana Dependence – a Manual for Treating Adults” and outlined procedures for individuals who use cannabis as their primary drug. The manual suggested chronic cannabis users tended not to seek treatment in traditional drug treatment settings, but that when given the opportunity would respond positively.



Increasing evidence suggests that counselling for cannabis dependence is effective (Steinberg et al, 2002; SAMHSA, 2005).

Clients in treatment require a sense of hope and positive expectations are especially critical when facing a protracted period of withdrawal (Zweben & O'Connell, 1992). Programs designed to aid cessation should focus on the negative effects of marijuana and should offer alternative ways to relieve negative physical and psychological conditions such as stress (Weiner, 1999).

Professionals working with cannabis dependent people often see them relapse repeatedly. Relapse may involve the length of detoxification; ease of access to the substance; social pressures in schools, work, entertainment, social and family settings; persistent denial; or the high level of functioning many addicts have when they enter recovery. Marijuana addicts who have not previously shown extensive drinking histories often believe they can consume alcohol and this can lead to a cannabis relapse (Chacin, 1996). Budney et al (2002) found clinical trials evaluating treatment for cannabis dependence suggest that the withdrawal syndrome, like other substance dependence disorders, is responsive to intervention but the majority have difficulty achieving and maintaining abstinence.

In recent years, multiple sources have released suggested treatment programs, ranging from counselling treatments for adults (SAMHSA, 2005), intervention programs (Maddock & Babbs, 2006) and specific treatment programs developed for women (Chacin, 2006). The work of Roffman & Stephens (2006) and Budney et al (2007) also discuss treatment options and are recommended reading on the topic.

## SECTION FOUR: RECOMMENDATIONS

The evidence is clear that the younger the age of initiation to cannabis use, the greater the risk of harmful effects to the individual. The following recommendations aim to provide advice and strategies to politicians, decision-makers and researchers to ensure that the level of cannabis use in Australia is markedly reduced, within the next few years.

Drug Free Australia's research recommends:

1. That all Australian Governments urgently implement effective preventative drug education in all States and Territories, focusing on education, in both primary and secondary schools that includes the latest scientific research into the harmful effects of cannabis on the developing brain, together with information on issues related to the risk of suicide, drug-induced psychosis, schizophrenia and depression.
2. That the Federal Government urgently implements a national media campaign, similar to the "Bloody Idiot" alcohol campaign, in order to inform the community of the harmful effects of cannabis use on all community members. This would be an appropriate response to the concerns of the Australian community, as measured in the Pfizer/NDARC report of 2007, in which 77% of Australians expected the government to run a public health campaign alerting the public to the harms of cannabis.
3. That clear cannabis prevention policies be issued by the Commonwealth Department of Health and Ageing, to be implemented in all schools and further, that these be regularly updated and reinforced.
4. That Federal, State and Territory police are resourced to implement NOAH (Narcotics, Opiates, Amphetamines, Hashish 1992) blitzes every three months for a two year period. This should target users and potential users; it should deal with plantation and hydroponically grown cannabis, trafficking, financing, and/or selling drugs to children. Further, that the Proceeds of Crime funds be used to continue a NOAH cannabis campaign after the two-year period.
5. That all professionals working in drug and alcohol fields be required to strongly discourage any cannabis use by those whom they counsel or to whom they provide treatment for drug related problems.
6. That the Federal and all States and Territory Governments resource and conduct a long-term cannabis QUIT campaign, to be organised and implemented along lines similar to the successful "QUIT Tobacco" campaign. Further, that the Cancer Council of Australia be encouraged to promote the message that cannabis has carcinogenic properties that cause the same adverse health consequences as tobacco.

7. That greater penalties be introduced to prosecute suppliers and traffickers of drugs to children while young offenders be directed toward compulsory treatment rather than jail.
8. That clear messages about the harmful effects of cannabis on the young body should be issued by the Commonwealth Department of Health and Ageing with the cooperation of the State and Territory Governments be used in all schools and be constantly reinforced.
9. That recommendation Number 70 of the report to the *Ampe Akelyernemane Meke Mekarle* "Little Children are Sacred" Inquiry be fully implemented. This recommends that government develop and implement a multi-faceted approach to address the abuse of illicit substances in Aboriginal communities, in particular cannabis. This approach to include strategies for prevention, intervention and enforcement strategies which:
  - a) Recognise the geographic context of substance abuse, which occurs in both urban and remote locations, and its implications; and
  - b) Are population-based, youth-focused and integrate substance abuse, mental health and other health and welfare concerns into youth programs.
10. That drug testing in schools be encouraged, giving a clear message that drug use including cannabis, is not permitted. Many youngsters do not see cannabis as a drug or that it will harm them.
11. That roadside testing be implemented to identify drug-driving and related safety issues, in all States and Territories.

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## APPENDIX A: UNITED KINGDOM

An article by David Wilkes in the Daily Mail dated 5 September 2007 see link:

[http://www.dailymail.co.uk/pages/live/articles/news/news.html?in\\_article\\_id=480162&in\\_page\\_id=1770](http://www.dailymail.co.uk/pages/live/articles/news/news.html?in_article_id=480162&in_page_id=1770)

**"Mother blames cannabis for suicide of promising violinist daughter"**

Talented, bubbly and pretty, Laura Bower-McKnight had it all to live for. A gifted musician, the 22-year-old studied at the prestigious Royal Welsh College of Music and seemed destined for a career in the performing arts. But her life once so full of promise was prematurely ended when she killed herself after cannabis turned her into a shambling wreck and left her an depressed recluse terrified of going outdoors. She was found dead at her family's home last week after hanging herself from the end of her bed. Her heartbroken mother told how smoking a single joint of the potent "skunk" variety of the drug triggered a psychotic episode in her violinist daughter and set her on the road to her death.

Mrs. McKnight said: "People think nothing of cannabis nowadays. They just don't realise this drug can tip you over the edge. "A lot of people try it". With the government downgrading it, I think young people assume it is completely harmless." But it can destroy your mind."

Having returned to the family home in North Hykeham, troubled Laura, who had previously smoked normal cannabis with friends, tried a joint of skunk - and the experience proved devastating. Mrs. McKnight said: "It wasn't the real Laura, the always-on-the-go, lovely young woman, the musician, the passionate writer, the artist." It tipped her into psychosis. We lost our wonderful girl for a while. Her behaviour became completely erratic. She was doing very odd things. Mrs. McKnight said she and her husband Malcolm, Laura's stepfather, now only hoped their daughter's death would serve as a warning to others.

She said: "Laura would have wanted us to highlight these issues. We were so close. It's just a massive, irreplaceable loss from our lives. "There are a lot of young, vulnerable people. Expectations of them are so high. Drug use, depression and suicide among them is a growing problem." Mr. McKnight, 44, an engineer, added: "Different people have different limits with drugs. For some even the tiniest amount can be too much."

An article by Paul Britton in the Manchester Evening News on 17 April 2006 see link:

[http://www.manchestereveningnews.co.uk/news/s/210/210885\\_parents\\_blame\\_cannabis\\_for\\_sons\\_suicide.html](http://www.manchestereveningnews.co.uk/news/s/210/210885_parents_blame_cannabis_for_sons_suicide.html)

**"Parents blame cannabis for son's suicide"**

A grieving family blames cannabis for causing the mental illness that drove their son to suicide. Lee Michael Wellock, 24, was found hanging from a tree with a note in his pocket indicating that he intended to kill himself. Lee had smoked the drug since he left Elton High school in Bury to work at a computer company. His parents, Michael and Denise, of Newington Drive in Bury, said it "took over and controlled" their son's life and ultimately led to his death. Lee, who did not drink alcohol, smoke cigarettes or take any other drugs, developed mental health problems at the age of 18 and was diagnosed with schizophrenia at 22, an inquest in Bury was told.

An article by Richards Edwards in the Telegraph Newspaper on the 25 September 2007 see link:

<http://www.telegraph.co.uk/news/main.jhtml?xml=/news/2007/09/25/nsuicide125.xml>

**"Suicide girl jumped to death at hospital"**

The daughter of an aristocratic couple jumped to her death following an eight-year descent into mental illness triggered by cannabis, it has emerged. Genevieve Butler, 28, the daughter of Lord and Lady Dunboyne, the Anglo-Irish family, threw herself from a balcony at a London hospital after breaking free from a nurse who was taking her for a cigarette break.

Her parents told of how their "clever, bright and quick-witted" daughter had been lost to them eight years ago when she was diagnosed with drug-induced -paranoia after using cannabis. "*Potent marijuana blamed for remote youth suicides*" reported in 'The Australian' on Wednesday 21 November 2007 highly potent marijuana is being blamed for youth suicides and psychotic episodes in a remote central Australian community, which is struggling to cope with increasing levels of drug use over the past 12 months. Susie Low the head of the Internationally-recognised substance abuse program at Mt Theo outstation said "In two out of the last three (suicides), the young men were under the influence of alcohol and marijuana". Ms Low's anecdotal concerns support the findings of two reports on marijuana use in the Territory, the most recent of which said 60 per cent of people in some Arnhem Land communities were cannabis users.

## APPENDIX B: AUSTRALIA

Spencer Gear in a Letters to the Editor, Fraser Coast Chronicle Maryborough Queensland on the 15 March 2007 wrote. Sadly, I have conducted the funeral of a 27-year old who committed suicide. Her family told me that the doctor said that her psychosis was probably marijuana induced. Herschel Baker (FCC 31-3-07) is right in challenging Dr. Kees Nydam's incorrect statement that "finding a clear-cut association between marijuana and mental health was not easy." It is clear in the research literature.

**"Potent marijuana blamed for remote youth suicides"** reported in The Australian on Wednesday 21 November 2007 highly potent marijuana is being blamed for youth suicides and psychotic episodes in a remote central Australian community, which is struggling to cope with increasing levels of drug use over the past 12 months. Susie Low the head of the Internationally-recognised substance abuse program at Mt Theo outstation said "In two out of the last three (suicides), the young men were under the influence of alcohol and marijuana". Ms Low's anecdotal concerns support the findings of two reports on marijuana use in the Territory, the most recent of which said 60 per cent of people in some Arnhem land communities were cannabis users.

### **Cannabis may trigger psychosis: experts**

The Sydney Morning Herald March 7, 2005 - 1:24AM [www.SMH.com.au](http://www.smh.com.au/news/Health/Cannabis-may-trigger-psychosis-experts/2005/03/07/1110044267823.html)  
<http://www.smh.com.au/news/Health/Cannabis-may-trigger-psychosis-experts/2005/03/07/1110044267823.html>

Cannabis is not the harmless drug many people believe it to be, with new evidence showing today's genetically engineered crops are more potent and may trigger psychotic illnesses, Australian scientists say. One in five Australian teenagers smoke cannabis every week, some as young as 10, and 10 per cent of those become addicted. Psychologists, bioscientists and counsellors are seeing more young Australians developing psychoses, depression and anxiety disorders through cannabis use, the ABC's *Four Corners* program has been told. Professor Vaughan Carr, Scientific Director of the Neuroscience Institute, said he believed there were similarities between the effects of cannabis on the brain, and schizophrenia. "I think that the odds are better than 50-50 that cannabis use in sufficient quantities beginning early enough in life may produce some cases of schizophrenia in people who otherwise would not have developed it," he told *Four Corners*, which airs tonight. "But that's my gut feeling. Roughly one in five adolescents overall are cannabis users in reasonable quantities. "I would have to say that all of them are at risk, but the earlier the onset of cannabis use and the greater the frequency of use, the higher the risk."

Sydney psychologist Andrew Campbell said there was much debate about whether cannabis uncovered an existing psychosis, or caused it. "My view is that it is bringing on new cases of psychosis," he told the program. "I see a lot of people with long-standing psychosis and if I see one in 10 people in a day, seven of them will have used cannabis on a daily basis at the first time of onset of psychosis."

The experts also say new hydroponically grown crops have been engineered into a much more toxic drug than 30 years ago. Dr Campbell said the new variety grew only about a metre high with little leaf and a lot of heads. As a result, the main chemical, tetrahydrocannabinol, or THC, is much more concentrated. "So when you buy \$25 worth of cannabis these days you're mainly getting heads. You don't get the leaf which is much lower in concentration of cannabis," Dr Campbell told the program. The experts also say that because new research has shown the brain is not fully wired until a person is in their early to mid-20s, teenage users are most at risk of developing mental illness.

Melbourne's Early Psychosis Prevention and Intervention Centre (EPPIC) director, Pat McGorry, said at least 70 per cent of young people who attended the centre had used cannabis. "The proportion of patients using it that we see has gone up. I would say it's doubled since the early '80s when we started to look at this group of patients," Professor McGorry said.

### **Convicted of manslaughter after relying on cannabis psychosis re diminished responsibility.**

Daily Telegraph by Michele Tydd 3<sup>rd</sup> September 1991

In the Supreme Court at Wollongong on the 3<sup>rd</sup> September, 1991, a Bega man pleaded guilty to slashing his neighbour's throat and stabbing him in the stomach and anus, on the spur of the moment, in the victim's caravan at Burragate on 3<sup>rd</sup> September, 1991. He was a long term user of marijuana and a friend of the deceased.

He raised diminished responsibility and was found to be suffering from a marijuana-induced psychosis. He was freed by the Judge after being held in custody for some two years.

**“Skunk Sparks a stink”** by Christopher Taylor The Sunday Mail 9 April 1994.

Drug Counsellors are concerned that skunk weed is 10 to 15 times more potent than normal cannabis strains and that is a conservative estimate. Experts say the strain has an almost hallucinogenic effect. Where marijuana gives the user a sense of euphoria, skunk can leave the user in a state that could easily be mistaken for mental imbalance.

The user can become intensely paranoid even exhibiting extreme schizophrenic traits. Experts said the strain can create “users with retarded motivation and responses.

**“Video dream made me stab brother”** Daily Telegraph 9 November 1988.

A 19 year old who cut his brother’s throat while he was asleep. He had seen the film Platoon and he believed he was an American soldier and his brother a member of the Vietcong. He had used 4 cones of marijuana and was said to be hallucinating, a psychiatrist gave evidence that he was suffering from a cannabis induced toxic psychosis. He was convicted of murder. The trial Justice, Justice Yeldham remarked “So much for those who would legalise marijuana”.

**“Debbie’s alleged killer sobbed, say police”** The Sydney Morning Herald September 15, 1987  
[www.SMH.com.au](http://www.SMH.com.au).

A 21-year-old man who is a heavy user of cannabis and lived with his family and nine-year old sister at Maitland in NSW, he was directed by voices (auditory hallucinations) to kill a member of his family and hence sexually assaulted and bashed his sister to death in their flat they both occupied. His plea of diminished responsibility as a result of cannabis induced psychosis was accepted. He was sentenced to three years imprisonment with a parole period of two years.

**Innisfail Advocate of Saturday July 18, 1992.**

“In the Townsville Bulletin newspaper on Thursday was the shocking story of two teenagers facing committal proceedings for murder, who, after smoking 20 cones of marijuana, allegedly battered a man to death with a shifting spanner and a large lump of wood. Police asked the youth (about the marijuana): “How effective was it?” to which the youth answered: “Well, I can’t remember much after it happened”. The youth also allegedly told police: “I wish I’d never had that first cone of marijuana”.

This horrifying, yet pathetic, story involving marijuana usage is not an isolated case of marijuana smoking leading to a shocking allegedly criminal act.

## Need Help?

The following websites are recommended for additional information:



[www.drugfree.org.au](http://www.drugfree.org.au)



<http://ncpic.org.au>



<http://www.theantidrug.com>



<http://www.nida.nih.gov>

